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Principles of Manual Sports Medicine

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PRINCIPLES OF MANUAL SPORTS MEDICINE

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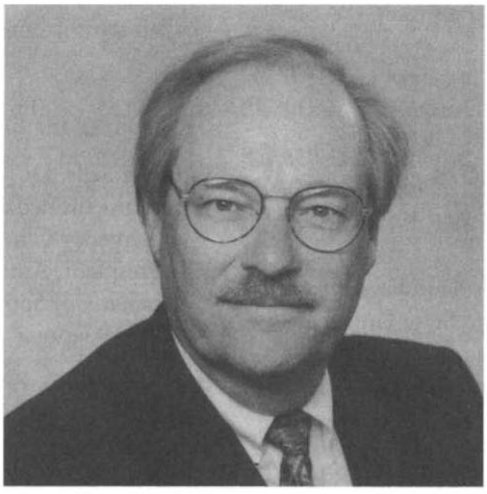

Principles of Manual Sports Medicine is dedicated to the life of one extraordinary physician and human being. Allen W. Jacobs, DO, PhD (1942–2001) impacted many osteopathic physicians throughout his career, most notably as Dean of Michigan State University College of Osteopathic Medicine from 1995–2001.

He was a man who pursued life's passions to their fullest; family, friends, medicine, and sports, especially the Chicago Cubs. He was a leader, teacher, healer, father, husband, and mentor, all while still keeping a childlike wonder for the athletes and teams he cared for. To this end, he impacted and trained many team physicians in his life, unselfishly opening opportunities for others, teaching at all times, and quietly assuming an iconic status within the community. His gift was not measured best by curriculum vitae or money, but by a greeting and handshake. He made the world a better place, one person at a time. We hope this book continues the strong legacy that Al left behind.

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PREFACE

This book is the first attempt to define manual sports medicine, and I hope it will not be the last. For the hundreds of sports doctors, therapists, and athletic trainers who use various forms of manual medicine techniques on their athletes, this book is dedicated to putting a name and a face to this unique brand of medicine that we practice.

These concepts and skills have been passed down to the next generation primarily by experience, such as courses, hands-on teaching, and rotations. Many sports doctors had their approaches and techniques passed around like campfire stories or tales of ancient lore. Proof of their effect is in physical therapy and athletic training, where many PTs and ATCs sit side-by-side with DOs and even MDs taking manual medicine courses to apply techniques to athletes.

Manual sports medicine is mainly based upon the holistic principles of osteopathic medicine, the kinetic chain principles of biomechanics, the rehabilitation principles of physical therapy, and standard approaches to sports and orthopedic injury. By applying these in a comprehensive approach to acute and chronic sports, recovery can be quicker, return to play can be sooner, and injury prevention can be better. These principles in the book are addressed in several ways.

First, we discuss commonly used manual medicine techniques, including physical therapy modalities and massage. For more in-depth discussion of these and other techniques not included, review Greenman's *Principles of Manual Medicine*, DiGiovanna and Schiowitz's *An Osteopathic Approach to Diagnosis and Treatment*, and the *Foundations for Osteopathic Medicine* text sponsored by the AOA.

The second section discusses exercise in various forms; the core principles behind its use in manual medicine, prescriptions, and stretching. Two exercise programs commonly used on athletes are core stabilization and return to throwing. These are discussed and depicted in detail.

After the basics of manual sports medicine are laid out, we really get into the meat of the book. We discuss how these techniques and approaches are applied in each region of the body. The bedrock of any good sports medicine clinician is solid knowledge of anatomy and physical exam skills, so each region covers anatomy, physical exam, and common conditions affecting this region, from head to toe, including gait analysis. Each region discussion on common injuries discusses how to use manual medicine techniques to improve recovery and return-to-play.

The last two sections discuss the use of manual medicine techniques in 14 different sports and 6 different athlete populations. Many of the unique perspectives and pearls for treating athletes are found in these chapters, such as understanding specific aspects of an unfamiliar sport, treating injuries in different populations (geriatric vs. pediatric), or adopting a different approach to treating specific athletes.

My hope is that this book inspires more research on manual sports medicine, looking at specific techniques and recovery time with athletes. Over the coming years, manual sports medicine will become more refined with a more distinct roadmap for using manual medicine techniques; this is simply the first step.

Whether it is a refresher for manual techniques, a manual for musculoskeletal exams, a reference for stretches, or a new approach to sports injury treatments, this book will have multiple uses and applications to your practice. This represents the heart and soul of many wonderful physicians, athletic trainers, and therapists, who believe in the power of touch and the body's amazing capacity for healing.

Enjoy the journey. S.J.K.

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S.J.K.

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1

INTRODUCTION

MICHAEL HENEHAN

Many excellent textbooks have been written on the topics of sports medicine and manual medicine. So what is different about this one? The idea behind this book is to integrate the principles of manual medicine with sports medicine, highlighting the natural overlap between them.

A fundamental concept in sports medicine is the use of active rehabilitation techniques that result in the safe and timely return of the injured athlete to participation in his or her sport. Coaches as well as athletes expect sports medicine physicians to use all available clinical tools to effectively rehabilitate the injured athlete. Manual medicine techniques are useful tools that can help in this process and are widely accepted by the athletic community. Consequently, we as sports medicine physicians need to be familiar with the concepts and practice of manual medicine. Even if one chooses not to actively use these therapies, many coaches and athletes have the expectation that a sports medicine physician will be familiar enough with these techniques to refer an athlete for manual medicine therapy when it is appropriate and may be beneficial. This text is designed to serve as a resource to sports medicine clinicians experienced in the practice of manual medicine as well as an introductory text to those interested in learning more about manual medicine techniques and their application in sports medicine.

WHAT IS MANUAL MEDICINE?

Manual medicine is not a single technique or type of therapy; rather, it encompasses a spectrum of therapeutic elements. Many of the techniques traditionally used by physical therapists and athletic trainers fall into the realm of manual medicine. Massage and stretching, for example, are types of manual medicine. When a physician refers a patient to physical therapy, there is a good chance that some type of manual medicine will be part of his or her treatment.

The basic concepts of manual medicine probably represent one of the oldest forms of medical therapy. Early references are found in the writings of Hippocrates (ca. 460–377 B.C.) and in the early Chinese medical literature. More recent developments in manual medicine techniques started after Andrew Taylor Still founded osteopathic medicine in 1874 and started the first college of osteopathy in 1892 (1). Chiropractic practitioners have also contributed to the development of manual medicine since Daniel David Palmer founded the first chiropractic school in 1895. Manual medicine concepts have continued to develop throughout the twentieth century on an international level. Some of the more prominent figures include Cyriax and Mennell (Great Britain), Lewit (Czech Republic), Dvorak (Switzerland), and Greenman (United States).

Manual medicine terminology is not completely standardized and can be confusing. There are, however, categories of manual therapy techniques and general terms that are consistently used by most practitioners. A modification of the categories used by Greenman (2) and Schneider and Dvorak (3), including the techniques most commonly used in sports medicine and, in particular, those techniques described in this text, is outlined below.

1. **Soft tissue techniques.** These techniques enhance muscle relaxation, flexibility, and circulation of body fluids. The focus is primarily on restoring physiologic movements

to altered joint mechanics. Techniques covered in this text include massage, stretching, strain–counterstrain, myofascial release, "muscle energy," "unwinding," and indirect functional techniques.

2. **Mobilization techniques** [also known as mobilization without impulse (the term *impulse* refers to a quick force of moderate intensity that is applied across a joint) or "articulation" techniques]. In these techniques, the joint is gently carried repeatedly and passively through the normal range of motion. The purpose is to increase the range of motion in a joint where the normal motion has become restricted. Examples used in this text include facilitated positional release and joint play.
3. **Manipulation techniques** [also called high-velocity, low-amplitude (HVLA) technique or manipulation with impulse]. These have been developed to restore the symmetry of the movements associated with the vertebral or extremity joints.

A basic concept in manual therapy is that the techniques are on a continuum based on the amount of force needed to perform them. The three categories described differ mainly in the focus of the maneuver (soft tissue versus joint) and the amount and type of force placed on the joint (impulse versus no impulse). Soft tissue techniques require the least force, and manipulation techniques require the most force. The amount of force used in a technique relates not only to the risk of injury to the patient but also to the skill and experience needed by the clinician to perform it effectively and safely. With greater force, there is a greater risk of complications, which requires a highly trained clinician to safely perform the technique. This continuum is illustrated in Figure 1.1.

In general, acute problems respond best to the least forceful techniques, and subacute and chronic problems respond to a combination of techniques. Specific technique(s) chosen depend on the nature of the problem and the training and experience of the clinician.

HOW DOES IT WORK?

A good model for conceptualizing how manual medicine works is outlined by Schneider and Dvorak (3). They describe two barriers (physiologic and anatomic) to normal range of motion in a joint and a third barrier (pathologic) that can develop with injury. The physiologic barrier is a normal restriction to range of motion resulting from joint capsule and ligament restraints, as well as muscle activity. Beyond this, the anatomic barrier presents an additional, small range of motion through which the joint can move before injury occurs. In a musculoskeletal injury, the physiologic barrier can change due to factors such as muscle spasm or voluntary muscle contractions secondary to pain. The result is a restricted range of motion at a segmental level that is called the pathologic barrier.

Many factors can cause restriction in joint range of motion. Acute causes include muscle spasm, joint effusion, soft tissue swelling, and synovial fold entrapment. Chronic causes include fibrosis, ligament shortening, muscle contracture, and degenerative changes. When mobilization or manipulation techniques are applied to a joint, the joint is carried through the pathologic barrier. It is moved as close to the preinjury physiologic barrier as the injury will comfortably (and safely) allow, but always short of the anatomic barrier. Stretching soft tissue structures and reducing swelling can also increase joint range of motion without the "impulse" used in HVLA techniques.

FIGURE 1.1. Force continuum for execution of various manual medicine techniques.

The physiologic barrier is thereby "reset" closer to the preinjury location. Ideally, there is relief of pain and muscle spasm with improvement in the range of motion and function of the joint.

What exactly is the therapeutic effect with manual medicine techniques? This is a difficult question because there is not a definitive, conclusive answer. In fact, there are probably multiple effects occurring at different anatomic and physiologic levels. Evans (4) has proposed the following effects of joint mobilization: (a) release of entrapped synovial folds or plica, (b) relaxation of hypertonic muscle by stretching, (c) disruption of articular or periarticular adhesions, (d) unbuckling of motion segments that have undergone disproportionate displacements, and (e) neurophysiologic effects. He concluded that separate mechanical and neurophysiologic effects occur in response to joint mobilization.

Manual medicine techniques are proposed to normalize mechanoreceptor function. Haldeman (5) has reviewed the proposed mechanical and neurophysiologic theories including alteration of mechanoreceptors in the spinal apophyseal joints as a source of pain and altered joint function. He also discusses hormonal factors such as endorphin release that may play a role in pain modulation with the use of manual medicine techniques. The therapeutic benefit of manual medicine therapies appears to occur at multiple levels including the macrolevel of joints, ligaments, and muscles as well as at the neurophysiologic and hormonal level. In some patients, a placebo effect probably also plays a role. It is not surprising that such a complex system is difficult to study.

DOES MANUAL MEDICINE WORK?

Numerous clinical trials have investigated manual medicine techniques in the management of chronic back and neck pain. Several authors who have written comprehensive reviews of the medical literature have concluded that the body of evidence suggests a demonstrable therapeutic benefit for manual medicine, particularly in patients with acute or subacute back pain (6–8). The long-term benefits still remain unclear, and further research needs to be done. Studies of manual medicine techniques are challenging to do because both the interventions and outcomes are difficult to quantify objectively. Because many techniques fall under the umbrella of "manual medicine," it is difficult to standardize interventions. Even with these difficulties, the body of evidence does suggest a therapeutic benefit, particularly in acute musculoskeletal problems.

RISKS

As with any medical therapy, manual medical therapy has associated risks. In their review of the literature, Assendelft (9) and Stevinson (10) have concluded that the risk of severe injury with mobilization therapy is relatively low. The risk of serious injury ranges from approximately 1 in 400,000 to 1 in one million manipulations. The most common serious adverse events include vertebrobasilar accidents, disc herniations, and cauda equina syndrome. The risk of serious injury appears ro be highest with manipulations of the cervical spine. As with any medical therapy, the key to success is an accurate diagnosis and application of the appropriate therapy. Greenman (2) has identified the following conditions as contraindications to high-velocity techniques: severe osteoporosis, vertebral tumors, fractures or unstable joints, severe degenerative joint disease, and an uncooperative patient. Very experienced clinicians may find these to be relative contraindications for carefully performed manual medicine techniques. Gentle soft tissue procedures and modalities can be done carefully and are of benefit in most situations. Part of the art of applying manual medicine techniques is understanding which techniques to use in a given situation.

USING THIS TEXT

Manual medicine skills can be a great asset to the sports medicine physician. This text is designed to provide information at both introductory and reference levels. Section I describes

manual medicine techniques. Section II describes the general principles of exercise as applied to the athlete. Section III provides a review of anatomy and physical examination skills. Sections IV and V describe the application of manual medicine principles in specific sports and specific populations.

It is the hope of all of us working on this text that sports medicine physicians will find it to be a useful resource that they turn to frequently in their care of athletes.

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SECTION

I

TECHNIQUES

2

FUNCTIONAL TECHNIQUE

RICHARD G. SCHUSTER

OVERVIEW

Functional techniques are among the oldest but least understood techniques in the osteopathic armamentarium. There is no single functional technique, but rather they are represented by a diverse group of techniques related by their dependence on the functional, rather than the structural, component of the structure–function relationship. Because they are difficult to teach, they have often been forgotten or neglected. It is only recently that some of these procedures have been standardized and are again being more widely used. Much of the credit for this revival goes to three individuals—William Johnston's *functional methods* (1), Stanley Schiowitz's *facilitated positional release* (FPR) (2), and Richard VanBuskirk's *Still technique* (3). Although one may argue that these methods are different, they all share fundamental characteristics.

HISTORY

The New England Academy of Applied Osteopathy (NEAAO) first introduced the term *functional technique* to the osteopathic profession in the 1950s (4). They defined a group of techniques characterized by establishing an "ease-response" relationship with the restricted segment, maintenance of the "ease-response" by the clinician as the segment was carried through its range of motion, then returning the segment to neutral, at which point the athlete's body could maintain the "ease-response" on its own. Using these criteria for defining functional technique, it becomes readily apparent that the methods described by the NEAAO were rediscovered techniques rather than truly original ones.

This resurgence is best demonstrated by the recent work of Richard VanBuskirk, who rediscovered many of the techniques thought to have been practiced by Andrew Taylor Still, the founder of osteopathy (5). In recognition of this fact, VanBuskirk named it the *Still technique*. He also recognized the similarity of the Still technique to the functional methods taught by Johnston, FPR as taught by Schiowitz, and a group of unnamed techniques (6) taught by Edward Stiles, who learned them from George Laughlin, a direct descendant of Still. All of these techniques have in common the basic tenets described by the NEAAO in 1955.

STRUCTURE–FUNCTION RELATIONSHIP

Functional technique relies on the evaluation of a segmental tissue response to specific motion demands. Rather than assessing the position of a given segment with respect to somatic dysfunction, specific motions are introduced to test how the segment moves (i.e., its function). Particularly, an attempt is made to determine in which direction the segment has the greatest amount of motion—the "ease-response." This is in contradistinction to direct techniques, such as the high-velocity, low-amplitude (HVLA) thrust, which depend on determination of the motion restriction or barrier.

Functional evaluation is not interested in an isolated plane or direction of motion, but rather in their summation. The motion of a vertebral segment (the examples presented later in the chapter assume that we are discussing vertebral motion unless otherwise stated) is described as rotation around the three ordinal

axes, x, y, and z: spinal flexion-extension is rotation around the x-axis, axial rotation is rotation around the y-axis, and sidebending is rotation around the z-axis.

EVALUATION AND DIAGNOSIS

Functional evaluation begins with identification of a vertebral segment that has a limited range of motion. The clinician can use any method of screening to identify somatic dysfunction. Once the dysfunctional segment has been identified, its motion characteristics must be identified. In the presence of somatic dysfunction, the greatest ease of motion is no longer the midline neutral position, but rather somewhere between the restrictive barrier and the anatomic-physiologic end point. It is the duty of the clinician to find the position of greatest ease, because it is from this position that treatment will begin.

Each direction of motion is tested to determine where the point of maximum ease is to be found. This is accomplished by placing one hand over the dysfunctional segment (the receiving hand), while the other hand (the motive hand) introduces gross motion down to the affected segment. The receiving hand evaluates the sense of ease or bind at the affected segment. Increasing ease is associated with decreased sense of tissue texture and tension. Each motion is tested sequentially in combination: first flexion-extension, then rotation and side bending. An attempt is made to find the point of maximum ease around all three axes of motion simultaneously. Fine-tuning this sense of maximum ease is accomplished by testing the very subtle translatory motions of movement along each axis. Bowles stated, "It is not position, nor is it motion, which is a change of position, which you feel. It is a physiological tissue response to demand for performance. The bind you feel may be a lesioned response or a normal response to the specific motion demand, but it is always a physiological response and its source lies in organized body processes." (4) The key point to this evaluation is that although the motion demands involve gross motions, the response is specific and focused to a single segment: the evaluation of a specified segment undergoing motion.

INDICATIONS

Functional treatment is very easy on both the athlete and clinician; it takes only seconds to accomplish and is atraumatic. Although the previous description was confined to vertebral segments, the basic principles are applicable to any joint, most ligaments, and even many tendons. It is equally effective for both acute and chronic somatic dysfunction.

Functional techniques are indicated for the treatment of any somatic dysfunction in an athlete. Its speed and tolerability make it especially effective in an athletic population. It can be accomplished easily on a sideline or in a training room. Because it starts from a position of ease, even an *acutely injured* joint tolerates treatment very well. If it is not effective on a first attempt, it can quickly be attempted again with little guarding on the part of the athlete. If a non–functional technique is deemed necessary, the basic motion characteristics of the joint have already been established, and the next technique can be readily begun.

In the case of chronic conditions, the primary advantage of functional technique is that it addresses the abnormal neuromuscular mechanisms that maintain the somatic dysfunction. This is in contrast to many direct techniques that attempt to alter the position of a joint, but may not affect the neuromuscular mechanism.

Another use of functional technique that is particularly applicable to the athletic population is its ability to assess the effects of other interventions. After a functional evaluation has been conducted, an intervention such as bracing, taping, heel lift, or injection can be accomplished, and the functional evaluation repeated. This gives an objective assessment of the intervention that may be prognostically valuable. The evaluation itself is a unique aspect of functional technique, lending important advantages such as this.

As in any joint evaluation, significant muscle spasm at the end range of active or passive motion testing may be indicative of a more serious injury. Should the athlete develop significant muscle spasm or guarding as the clinician approaches the position of ease, assume serious underlying pathology and abandon

manipulative corrections until such pathology is ruled out.

TREATMENT PRINCIPLES

Treatment is simply a matter of maintaining this "ease-response" relationship while returning the segment through its range of motion and restoring it to normal. Once the point of maximum ease is found, a vector force is generated from the motive hand toward the affected segment and the sensing hand. This vector force is then used to carry the tissue back toward the initial restriction, all the while maintaining the point of balanced ease, hence the term "ease-response." The sense of "ease" is maintained as the segment "responds" to the motion. As the vector force moves the segment beyond the initial restriction, there is usually, though not always, a palpable "release" of tissue tension, signifying the end of the treatment. The vector force may be released at this point, and the segment returned to normal. At this point, the segment must be retested to ensure that normal motion has returned.

TREATMENT

The following examples demonstrate how functional technique is used to treat specific somatic dysfunction. These examples do not represent the only way treatment may be accomplished. They have been chosen to demonsttate some of the many positions that can be used. It is up to the clinician to adapt each treatment to the individual athlete based on the circumstances existing at the time.

Examples

Thoracic Spine: Type 2 Somatic Dysfunction, Extended [extended, rotated, and side-bent left (ERS L)] (Fig. 2.1A and B)

1. The athlete is seated on the table, while the clinician stands behind the athlete.
2. The clinician's sensing hand is placed over the transverse process of the dysfunctional segment (left). Any part of the hand can be used, although the thumb, index, or ring finger is most typical.
3. The motive hand reaches around the front of the athlete's shoulders (Fig. 2.1A).
4. Extension, rotation, and side bending are induced until a decrease in tissue texture and tension is noted. Maximum relaxation—"fine-tuning"—is achieved by translation along the three ordinal axes.
5. A compressive vector force (typically about 5 lb of pressure) is generated by the motive hand, toward the sensing hand.
6. The athlete is then flexed, rotated, and side bent in the opposite direction, using the vector force as an operating lever, until release of the segment is noted. An attempt is made to sense and maintain the "ease-response" relationship throughout this motion (Fig. 2.1B).

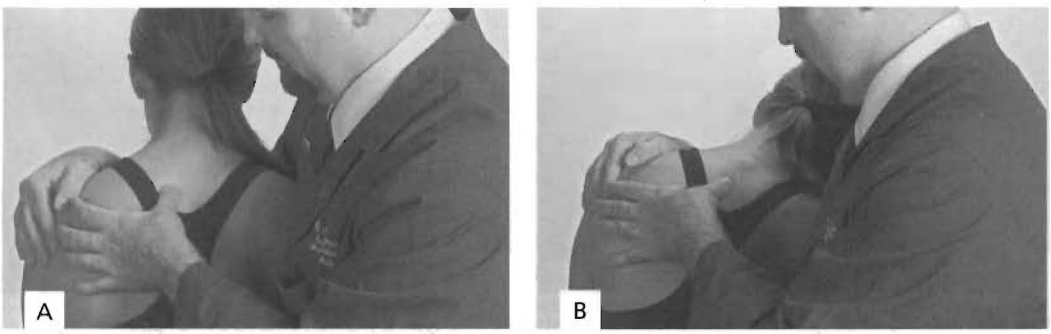

FIGURE 2.1. A, Initial position for functional treatment of a type 2 flexed somatic dysfunction of the thoracic spine [flexed, rotated, and side-bent left (FRS L)]. **B,** Final position for functional treatment of FRS thoracic lesion.

7. The athlete is returned to neutral position, and the segment is rechecked to be sure that correction has been accomplished.

In the case of an extended somatic dysfunction, the motions are exactly the same except that extension is used as the initial direction. The same technique can be used in the same manner to treat the lumbar spine in the seated position. Type 1 somatic dysfunction would be treated in the same manner except that neither flexion nor extension would be introduced, and side bending and rotation would be in opposite directions.

Lumbar Spine: Type 2 Somatic Dysfunction, Flexed [flexed, rotated, and side-bent (FRS R)] (Fig. 2.2A–D)

1. The athlete is supine on the table, while the clinician stands on the side of the somatic dysfunction.
2. The sensing hand is placed beneath the athlete, on the transverse process of the dysfunctional segment.
3. The motive hand flexes the knee until motion is felt at the involved segment by the sensing hand (Fig. 2.2A).
4. The knee is now moved medially, adducting the hip, until decreased tissue texture and tension is noted (Fig. 2.2B).
5. A compressive vector force (typically about 5 lb of pressure) is generated by the motive hand, toward the sensing hand.
6. Using the force vector as a lever, the knee is carried laterally, adducting the hip (Fig. 2.2C).
7. The knee is then carried inferiorly, straightening the leg. Release is typically felt sometime during this final motion (Fig. 2.2D).
8. The segment is rechecked to ensure that correction has been accomplished.

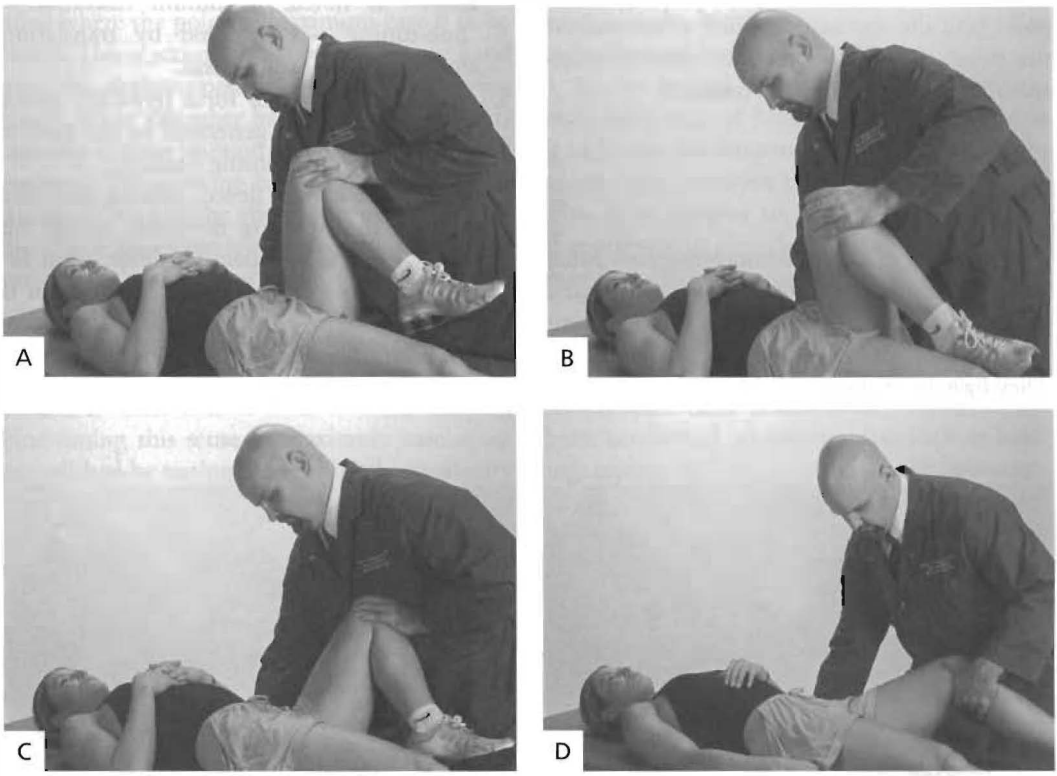

FIGURE 2.2. A, Initial position for functional treatment of a type 2 flexed somatic dysfunction of the lumbar spine [flexed, rotated, and side-bent right (FRS R)]. **B,** Intermediate position for lumbar functional treatment; the leg is flexed and adducted. **C,** Leg is abducted slowly. **D,** Leg is now extended.

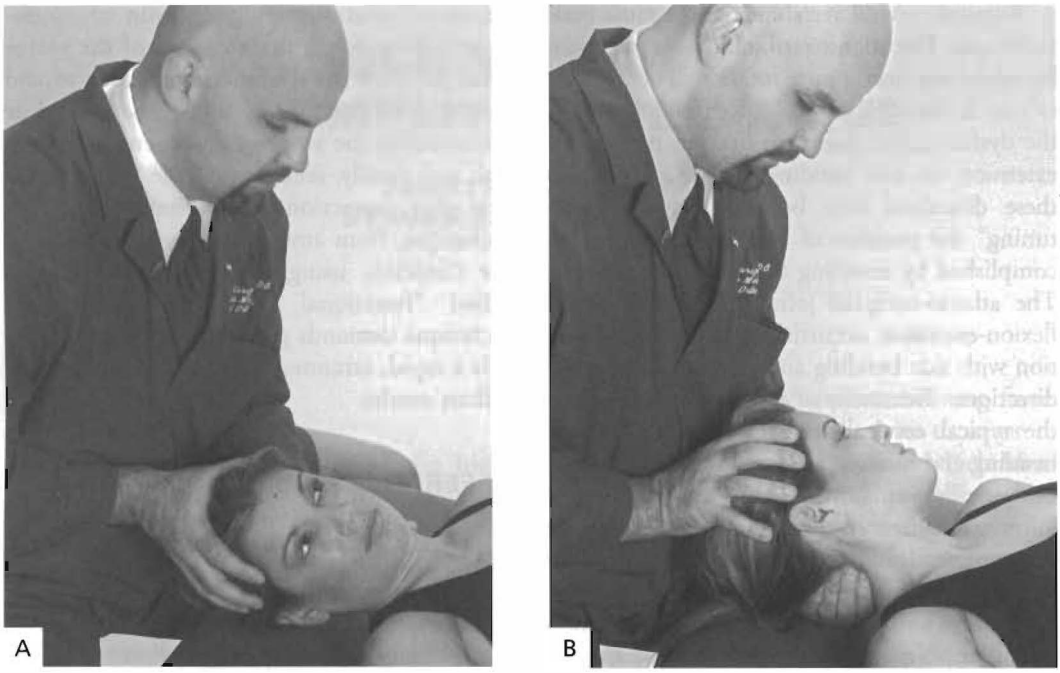

FIGURE 2.3. A, Initial position for functional treatment of a flexed cervical spine lesion [flexed, rotated, and side-bent right (FRS R)]. **B,** Final position for cervical treatment.

In the case of an extended lumbar segment, extension is achieved by moving the knee laterally, abducting the hip. A compressive vector force is then generated toward the sensing hand, and the knee is carried medially, reversing the treatment sequence. Although a neutral lumbar segment can be treated in the supine position, treatment is much easier accomplished in a seated position.

Cervical Spine, Typical Vertebrae: Somatic Dysfunction [flexed, rotated, and side-bent right (FRS R)] (Fig. 2.3A and B)

1. The athlete is supine on the table, while the clinician is seated at the head of the table.
2. The sensing hand is placed beneath the neck with a finger on the articular mass of the dysfunctional segment.
3. The neck is supported primarily by the remainder of the sensing hand.
4. The motive hand is placed on the top of the head, flexing the athlete's head until motion is sensed to the dysfunctional segment.
5. Side bending and rotation are introduced until decreased tissue texture and tension are noted.
6. A compressive vector force (typically about 5 lb of pressure) is generated by the motive hand, toward the sensing hand.
7. Using the vector force as a lever, the head is carried into extension, side bending, and rotation to the opposite side. An attempt is made to maintain the sense of ease throughout the maneuver.
8. The athlete's head is returned to neutral position and the segment is rechecked to ensure that the correction has occurred.

Recall that for the typical cervical vertebrae, C2-T1, all motions involve type 2 mechanics, as determined by the plane of the facet joints.

Treatment of an extended somatic dysfunction is essentially the same except that the initial and final positions are reversed. The same technique can be used with the athlete in a seated position and the clinician standing in front of the athlete.

Atypical cervical vertebrae use the same basic technique. The atlantoaxial joint is characterized by exhibiting nearly pure rotation. The position of ease is therefore achieved by rotation toward the dysfunctional side with little or no flexion-extension or side bending (subtle motions in these directions may be necessary for "fine-tuning" the position of ease). Treatment is accomplished by reversing the rotational element. The atlanto-occipital joint is characterized by flexion-extension occurring as a combined motion with side bending and rotation in opposite directions. Treatment of this joint is similar to the typical cervical vertebrae except that side bending and rotation are opposite. From a practical standpoint, however, the atlanto-occipital joint is much more difficult to correct because the motion is much more complex and the "ease-response" is far more difficult to maintain.

These are some basic examples of how functional technique can be used to treat specific spinal segmental dysfunction. Keep in mind that there is no single, regimented way to do functional technique.

CONCLUSION

Functional technique is distinguished by its underlying principles: identifying the "ease-response" relationship; generation of appropriate vector force; maintenance of the vector force to carry the dysfunctional tissue to and through the previous restrictive barrier while maintaining the sense of ease; return to neutral; and finally, reevaluating the tissue to ensure that correction was accomplished. Any technique, from any posture by the athlete or the clinician, using these principles can be called "functional technique." Functional technique demands precise palpatory skill, yet it is a rapid, atraumatic technique yielding excellent results.

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3

HIGH-VELOCITY, LOW-AMPLITUDE TECHNIQUE

JONATHAN D. TAIT
STEVEN J. KARAGEANES

OVERVIEW

The thrust technique [also called the high-velocity, low-amplitude (HVLA) technique, or mobilization with impulse] is one of the oldest and most widely used forms of manual medicine. Although there are different types of thrust techniques, including the low-velocity, high-amplitude technique, this chapter focuses on HVLA techniques. HVLA had been the main technique taught in colleges of osteopathic medicine until recent years, where curricula now contain a variety of direct and indirect techniques as well. Several physiotherapists and other practitioners such as Maitland, McKenzie, and Mennell heavily influenced many of the HVLA techniques used and taught by early osteopathic physicians.

Many theories exist regarding the prevalence of the technique in osteopathic colleges. A. T. Still was known to use thrust sparingly in favor of myofascial release and indirect techniques (1). The faculty and students who assisted in teaching may have been largely responsible for the popularity of thrust techniques over other techniques known at the time. Teachers were able to very accurately describe the joint restriction and provide techniques for correcting the dysfunction. These techniques were much more easily learned and practiced than the myofascial and indirect techniques preferred by Still, which required much more skill in assessing the motion patterns in the tissues in order to precisely treat the dysfunction (1).

Thrust is a direct technique, meaning that the joint exhibiting somatic dysfunction is moved through its restrictive barrier of physiologic motion by an extrinsically applied thrusting force. This extrinsic force allows the joint to return to its appropriate physiologic motion, which Mennell refers to as "joint play" (2). He defines joint play as the movement allowed within a synovial joint, which is independent and cannot be introduced by voluntary muscle contraction (2). The movement is small (less than 1/8 inch) with the range dependent on the contour of the joint surfaces. He also postulates that this motion is necessary for normal, pain-free movement of the synovial articulation and, if restricted or absent, voluntary movement will, in turn, become restricted and frequently painful (2, 3). In this sense, the goal of treatment is to use a small thrust—not to force the joint "back into place," but to restore motion loss at the dysfunctional segment by gapping the joint. Joint gapping can be accomplished with or at right angles to the plane of the joint, or with distraction of the joint (3). Key points to remember regarding joint play are that it is a small, precise movement that is elicited passively, it is independent of voluntary muscle action, and it is found in all synovial joints. Effective treatment results in an immediate increase in range of motion, a decrease in muscle hypertonicity, and often a decrease in pain associated with the dysfunctional area.

CONCEPTS AND PRINCIPLES

Somatic Dysfunction

To successfully use the HVLA technique, one must accurately assess the athlete to diagnose an articulation or vertebral segment that is not exhibiting normal range of motion (4). This

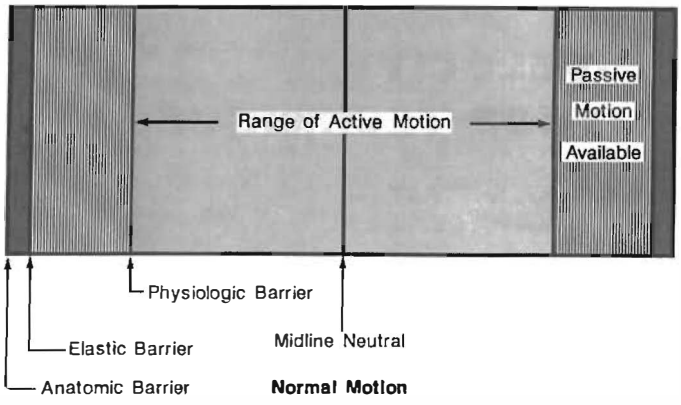

FIGURE 3.1. Active range of motion with physiologic and anatomic barriers. (From Greenman PE. *Principles of manual medicine*, 2nd ed. Baltimore: Williams & Wilkins, 1996, with permission.)

requires a focused, detailed vertebral structural examination after obtaining an accurate history of the athlete's symptoms. Muscles usually contribute to the joint restriction, and will have characteristic palpatory tissue texture changes, feeling boggy, stringy, or ropy in nature. These findings, as well as local tenderness with palpation or movement, and asymmetry of bony structures, also help the clinician localize the segmental problem (5). The somatic dysfunction usually demonstrates some or all of these characteristic tissue changes, and will also exhibit a change in the quantity and quality of joint motion.

Barrier Concepts

In joint mechanics, a "barrier" generally refers to a motion restriction. It is most commonly produced by either abnormal muscle contraction or capsular-ligamentous shortening, causing a "limit" to the normal range of motion. Within the total range of motion of each joint there exists a range of active motion limited by a physiologic barrier (Fig. 3.1). This physiologic barrier is created by the accumulation of soft tissue tension as the joint moves through its normal range of motion. There also exists a range of passive motion that is limited in each direction by an elastic barrier (Fig. 3.2). The elastic barrier is a physiologic limit of the joint, or the point at which all "slack" or tension has been passively taken out of the joint and surrounding muscle and ligamentous structures. The normal end-feel is due to the resilient nature and tension of these structures. The anatomic barrier of the joint represents the limit to motion beyond which tissue damage occurs. There is a very small potential

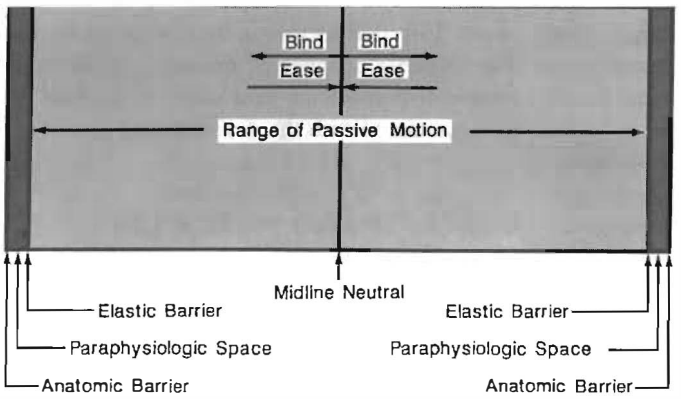

FIGURE 3.2. Passive range of motion with restrictive barriers. (From Greenman PE. *Principles of manual medicine*, 2nd ed. Baltimore: Williams & Wilkins, 1996, with permission.)

space existing between the anatomic and elastic barriers, known as the paraphysiologic space, and it is within this area that the thrust takes place, often generating a "popping" sound (3). The popping sound is not required, however, and may not always signify a successful treatment.

One can appreciate the differences found within joint motion when a joint exhibits somatic dysfunction. Joint motion will also have a characteristic feel of bind, or a barrier to movement. This barrier, in the direction of motion loss, is known as the "restrictive barrier." The restrictive barrier and the normal physiologic barrier on the opposite side of the joint limit the amount of active motion remaining within a joint.

Loss of motion is described with these principles in mind, as the asymmetry diagnosed on examination confirms the new position of the dysfunctional segment. The examiner must accurately identify normal and abnormal motion, as well as normal and restrictive barriers to these motions in order to diagnose the somatic dysfunction. Effective treatment will correct the segment, returning its full range of motion. It must be remembered that treatment is not just a change in the static position, but involves the dynamics of motion involving the following general principles (1):

1. The anatomic barrier is the end point for permitted passive motion; any motion beyond the anatomic barrier damages anatomic structures.
2. Normal active motion occurs between physiologic barriers.
3. The restrictive or pathologic barrier is the end point of permitted motion with motion loss in somatic dysfunction.
4. The normal midline, or neutral point, in the range of motion of a joint is frequently shifted to a new position in the presence of somatic dysfunction.

The dysfunctional segment will exhibit asymmetrical quantity of motion as well as a change in the feel, or quality, of the motion. This aspect is much more difficult for the novice practitioner, but it is a key component in the assessment of motion and positioning for treatment.

Restrictive Barriers

Restrictive barriers cause a limitation in the normal range of motion, and will have palpatory characteristics different from normal anatomic, physiologic, and elastic barriers. Skin, fascia, muscles (long and short), ligaments, and joint capsules and surfaces can all act as barriers (3). Pain can also be a cause of joint restriction. The barriers may involve a single segment or joint (short restrictor), or they may cross more than one joint or series of spinal segments (long restrictor). It is necessary to evaluate the total range of movements, the quality of movement during the range, and the end-feel of the movement to accurately diagnose the restrictive barrier.

Engaging the Barrier

The use of any direct technique requires the practitioner to engage the restrictive barrier. The term "barrier" can be misleading if it is thought of as a rigid obstacle that one is attempting to push through. Rather, as the joint reaches the barrier, restraints in the form of tight muscles and fascia serve to inhibit further motion. Engaging the barrier involves pulling against restraints rather than pushing against some anatomic structure (1). The practitioner monitors the increasing restriction caused by the tissues, positioning the joint at the limit of its motion, effectively engaging this end point, or barrier.

The restrictive barrier must also be conceptualized in three dimensions, rather than a single plane of motion. Most joints demonstrate motion in flexion-extension, rotation, side bending, and translation, which must then be tested separately to properly diagnose the restriction. (Some joints do not have all of these motions.) For an HVLA treatment to be effective, each plane of the barrier must be engaged simultaneously, creating a solid end-feel prior to the thrust. This "locking out" in all planes is necessary to limit induced motion to the specific plane desired for the thrust, giving an added

sense of control. The experienced clinician can make subtle alterations to engage the barrier in all planes quickly and simultaneously, while the novice may prefer to engage each plane separately to localize the restriction.

Localization of Force at the Restrictive Barrier

After an accurate diagnosis and positioning to engage the barrier in all planes, forces must be accumulated and directed at the restriction prior to performing the thrust technique. Once the barrier is engaged and forces are localized to the restrictive barrier, the final thrust is given from this maintained position. A controlled thrust is applied to the involved articulation in the direction perceived as limited, and a small motion in the desired direction (either flexion, extension, rotation, or side bending) occurs as the articulation transverses the barrier, effectively gapping the joint (5).

A common mistake is to "back off" prior to the thrust. This means that the localization is lost when the practitioner attempts to generate more force for the thrust than is required if it had been properly localized. This effectively disengages the barrier, causing poor thrust localization, resulting in a less specific treatment. Specificity of treatment is measured by how accurately the force is directed at the point of restriction. The more specific is the localization of forces, the less force (or amplitude) that is needed, resulting in a decreased risk of treatment side effects. Another common mistake made by novices is to compensate for poor localization by increasing the thrust amplitude. This may still correct the dysfunction, by essentially a "shotgun" approach at many segmental levels, but it also increases the likelihood of adverse side effects from the treatment.

A principle of localization that is very useful is the introduction of converging convexities in two different planes, with the goal of treatment being to place the segment under treatment at their apex (3). In order to accomplish this, one can first use flexion and extension movements to place the dysfunctional segment at the apex of the convexity created in either direction. Next, a side-bending convexity is created. And finally a rotational convexity is created. Often, once the flexion or extension and side-bending convexities are created, the appropriate rotational convexity is simultaneously created, which simply requires fine adjustments.

While localizing the dysfunctional segment, the hand serves a twofold purpose. The clinician will sense the aforementioned subtleties of localization by noticing changes beneath the hand. Once properly localized, the hand can also be used as a fulcrum to block the motion of a lower segment, allowing the thrust to move the dysfunctional upper segment. Although this applies more to the thoracic spine, various hand positions can be used to help facilitate localization in other areas of the spine and extremities.

HOW IT WORKS

Abnormal tone or spasm in the paraspinal muscles is usually responsible for holding the joint in a position of restriction. It has been postulated that this restricted position puts inappropriate force or load on the joint capsule altering the input from the afferent nerve innervating type I and type II mechanoreceptors (3). This alters the positional sense of the articulation. In response, this alteration in neural control is thought to affect the muscles surrounding the segment, causing increased tone and further restricting the movement of the joint. The segment becomes what is known as a "facilitated segment," with increased motor activity, and less afferent stimulation required to trigger a muscle contraction. The result, referred to by one author as the "pain-spasm-pain" cycle, must be disrupted in order for the joint to return to a normal range of motion (6).

The exact physiologic mechanism underlying the effects of spinal manipulation is not clearly known, but several theories exist. One recently proposed theory is that spinal manipulation may initiate afferent discharges from cutaneous receptors, muscle spindles, mechanoreceptors, and free nerve endings in the zygapophyseal joint

capsule and spinal ligaments (7). The afferent discharges may then synapse on inhibitory interneurons to inhibit alpha motor neuron pools of the paraspinal musculature (7). Another recent study by Dishman and Bulbalian (8) has shown that thrust manipulation may lead to short-term attenuation of alpha motor neuron activity, and their findings have also suggested that the cutaneous receptors, muscle spindles, and Golgi tendon organs rather than the velocity-dependent joint mechanoreceptors contributed more to the afferent discharge to the inhibitory pool. It may then be reasonable to suggest that thrust manipulation may cause a reflex inhibition effect to disrupt the "pain-spasm-pain" cycle causing a change in neural input to the muscle restrictors, resulting in a "temporary electrical silence of the segmental-related muscles with a refractory period before normal electrical activity returns" (3).

INDICATIONS

HVLA techniques are useful in restoring loss of motion, decreasing pain, and improving biomechanical function at spinal or extremity articulations. Thrust is also effective in reducing somoatovisceral reflexes associated with a somatic dysfunction (4). Treatment tends to be more successful for subacute and chronic dysfunctions, but it can also be useful in some acute settings. HVLA treatment also seems to be more successful in dysfunctions that appear to be caused by short restrictors of single segments, rather than long restrictors spanning several segments. The main limitation to treatment of acute injuries is the associated muscle hypertonicity and spasms that can accompany the joint restriction. It is often useful to precede the thrust treatment for an acute injury with another form of direct treatment, or with an indirect treatment, such as soft tissue, myofascial release, counterstrain, functional energy, or muscle energy. These preparatory treatments can help alleviate muscle hypertonicity, are more easily tolerated by an athlete in acute pain, and facilitate the effectiveness of the thrust treatment.

CONTRAINDICATIONS

The ability to accurately diagnose somatic dysfunction and specifically treat only the dysfunctional area will reduce the potential contraindications for thrust techniques. All thrust techniques have the potential to cause iatrogenic side effects if the treatment is not well localized to the specific segment to be treated. Even when an effective, specific treatment is performed, the technique itself inherently carries more contraindications than other manual medicine techniques due to the forces involved.

There are differing opinions among authors when they discuss contraindications to the thrust technique. One author suggests thinking in terms of the risk-benefit relationship for each individual athlete. If the risk of harming the athlete exceeds the potential therapeutic benefit, the technique should be avoided (1). The risk of side effects is also decreased in the hands of a skilled physician. That being said, most authors agree that the conditions listed in Table 3.1 are the few absolute contraindications to the use of

TABLE 3.1. ABSOLUTE CONTRAINDICATIONS TO THE USE OF THE HVLA TECHNIQUE

Hypermobility and/or instability of any joint
Ligamentous instability
Objective radicular signs
Cauda equina syndrome
Spondyloarthropathies
Presence of inflammatory joint disease (e.g., rheumatoid arthritis)
Active infection
Aseptic necrosis
Osteoporosis
Myelopathy
Traumatic contracture
Advanced degenerative joint disease
Metastatic bone cancer
Vertigo
Vertebrobasilar disease
Anatomic or pathologic fractures

TABLE 3.2. RELATIVE CONTRAINDICATIONS TO THE USE OF THE HVLA TECHNIQUE

Vertebral artery ischemia
Atherosclerotic plaques
Cervical spondylosis
Scheuermann's disease
Anticoagulation, whiplash
Chronic herniated discs
Pregnancy
Agenesis of the odontoid process
Down's syndrome

the HVLA technique. Relative contraindications are listed in Table 3.2 (1, 3–5).

ADVANTAGES IN ATHLETICS

In the hands of skilled and experienced clinicians, the HVLA technique is highly effective in treating athletic dysfunctions. It can be effective during competition when dysfunction develops but time is limited, as in halftime of a football game. Treatments are often effective in relieving discomfort quickly, and the kinesthetic awareness of the athletes gives the clinician immediate treatment feedback on the problem's persistence. This can be effective in returning athletes to competition in situations where they could not compete or would be limited otherwise. This benefit can sometimes strengthen the physician-athlete relationship.

This relationship from thrust manipulation can be a double-edged sword. Many athletes will feel the immediate relief and seek instant gratification in the future from various aches, even if they do not affect performance. Athletes may encourage clinicians to treat them with HVLA manipulation just to hear the "pop," especially since a pop signifies in the athlete's mind a successful treatment, regardless of whether the dysfunction is actually better. Manipulation then becomes a crutch or a comfort rather than a treatment. This abuse should be avoided in high-velocity direct techniques. Also, overusing HVLA techniques can hypermobilize and loosen segments of the axial spine, leading to instability and chronic dysfunctions. The clinician then begins to chase lesions that develop because of the hypermobility created from the excessive treatments.

The other issue with the HVLA technique is the number of absolute and relative contraindications, as listed in Tables 3.1 and 3.2. No other technique has more of them, and the use of HVLA should be deferred if there is a question about whether the athlete has a contraindication. For instance, a mature athlete with radicular symptoms down the arm may have a chronic herniated disc, which is a relative contraindication. Instead of taking the risk of using HVLA on the affected segment, other techniques like counterstrain and the functional technique would likely be safer and less likely to produce unwanted sequelae.

USE OF EXERCISE

Athletes who require HVLA manipulation need exercises to help stabilize the treated area and thereby prevent future dysfunctions. After all, when a spinal segment is out of alignment, the clinician puts it back in alignment; but how does that segment stay in alignment? This is achieved by the musculature that supports the vertebrae, and the firing patterns and strength of those muscles play a role in stabilizing vertebral segments. Biomechanical issues should also be addressed, such as other areas of dysfunction, short leg syndromes, flaws in throwing or running technique, and scarring. When stabilization is assured, the muscles then need to be trained to support the dysfunctional segments.

For example, a pitcher who throws 200 innings a year in the major leagues may have chronic thoracic dysfunction, particularly group lesions. Treating his dysfunction with HVLA manipulation is effective in resolving the restriction, but if the pitcher fails to improve the endurance and firing patterns of the scapular retractors and stabilizers, these problems will likely return, possibly leading to other problems along the kinetic chain.

The muscles supporting dysfunctional vertebral segments need to be treated after the

HVLA manipulation resolves a dysfunction because of their adaptation to the previous dysfunction. For instance, after treating a cervical dysfunction with HVLA, rhe head will often lie on the table tilted to one side involuntarily. Muscle length and tension adapt to the dysfunction, and when the dysfunction is treated with HVLA, the muscle still retains its length until rhe muscle itself is treated or the athlete sits up and normalizes cervical positioning. This is why muscle energy, a direct rechnique that works on the muscle spindle that supplies the memory to muscle, is an effective treatment before HVLA. Indirect techniques such as myofascial release and counterstrain also work well.

TREATMENT

The previously mentioned concepts and principles were defined for the application of thrust technique to somatic dysfunction of rhe spine. However, with some modifications, these principles apply to all synovial joints, including those of the extremities. In addition to the concepts discussed earlier, Mennell defines ten rules for effective thrust technique (2):

1. The athlete must be relaxed
2. The clinician must be relaxed. Therapeutic grasp must be painless, firm, and protecrive.
3. One joint is mobilized at a time.
4. One movement in a joint is restored at a time.
5. In performance of a movement, one aspect of a joint is moved upon the other, which is stabilized.
6. The extent of movement is nor greater than that assessed in the same joinr on the opposite, unaffected side.
7. No forceful or abnormal movement must ever be used.
8. The manipulative movement is a quick thrust, with velocity, to result in approximately 1/8-inch gapping at the joint.
9. Therapeutic movement occurs when all of the "slack" in the joint has been taken up.
10. No therapeutic maneuver is done in the presence of joint or bone inflammation or disease (heat, redness, swelling).

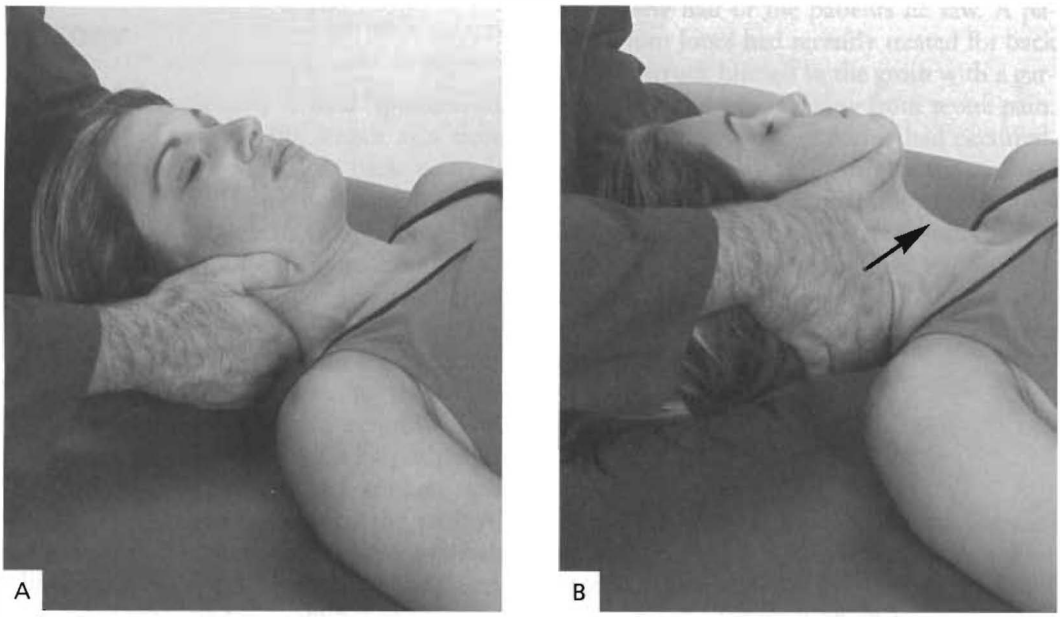

FIGURE 3.3. A, Setup for high-velocity, low-amplitude (HVLA) manipulation of the cervical spine at C6-7 [extended, rotated, and side-bent right (ERS R)]. **B,** Thrust position for HVLA at C6-7.

Done properly, the thrust technique has an immediate effect, and signs of improvement should be apparent within 1 week of initial treatment. Possible side effects experienced by some athletes include a transient increase in discomfort or mild autonomic effects. If there is lack of improvement after 2 to 4 weeks, the diagnosis should be reevaluated by the physician (5).

Example

Cervical Spine, C6-7 Lesion [extended, rotated, and side-bent left (ERS L)] (Fig. 3.3A and B)

1. The athlete is supine with the clinician at the head of the table.
2. The second finger of the operator's thrusting hand (right) is placed against the C6 vertebra while the opposite hand holds the athlete's head around the occiput (Fig. 3.3A).
3. The cervical spine is side-bent right and rotated left (wrong-way rotation) at the lesion, then slight flexion is introduced at the lesion to engage the restrictive barrier (Fig. 3.3B).
4. The clinician introduces a sudden impulse by the thrusting hand toward the contralateral hip.
5. Retest is performed.

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4

COUNTERSTRAIN TECHNIQUE

RANCE McCLAIN

DEFINITION

Counterstrain (CS) is defined as "a system of diagnosis and treatment that considers the dysfunction to be a continuing, inappropriate strain reflex, which is inhibited by applying a position of mild strain in the direction exactly opposite to that of the reflex; this is accomplished by specific directed positioning about the point of tenderness to achieve the desired therapeutic response" (1). Other terms commonly used today to describe the technique include Jones technique, strain–counterstrain, and spontaneous release by positioning. The term "positional release by therapy" is frequently used by physical therapists (2).

HISTORY

Counterstrain, originally termed "spontaneous release by positioning" (3), began as a treatment for a patient with low back pain. In 1955, Lawrence H. Jones was working as an osteopathic practitioner in Oregon. One day, Jones was treating an otherwise healthy 30-year-old man for recalcitrant low back pain. Jones had been unsuccessful in all his prior attempts at treatment, as had two chiropractors. The pain was severe enough to allow only short bouts of sleep at night, before it woke the patient. In an attempt to find a position in which the patient could obtain enough relief to allow sleep, Jones fine-tuned the patient's position until the pain was almost completely relieved. When this position had been achieved, Jones left the examination room to attend to another patient. On returning, Jones allowed the patient to arise from the table. To their mutual surprise, the man was nearly pain free (4).

Jones worked on refining this accidentally found treatment technique for many years after his initial discovery, and his failure rate continued to decline. It was during this refinement period that Jones discovered other important aspects to treatment with the counterstrain method. Jones noted that the need for a passive and slow return from the treatment position was required to minimize the possibility of recurrence of the dysfunction.

The final important piece of the counterstrain puzzle was unlocked in much the same accidental fashion as the initial piece. Jones had been able to find the tender point in only approximately half of the patients he saw. A patient whom Jones had recently treated for back pain had struck himself in the groin with a gardening hoe and was suffering from severe pain. After concluding that no hernia had occurred, Jones began a second treatment for the patient's back pain. During idle time waiting for the requisite treatment period to elapse, Jones began to once again probe the inguinal region. The pain was almost completely gone. This discovery led to a 3-year search of the anterior aspect of the body for tender points (5).

HISTORICAL PERSPECTIVE

The idea of relieving dysfunction and enhancing body function by position is not a new concept. For over 5,000 years, many forms of yoga have included positioning as a method to place areas of the body under stress while other

parts are placed in a position of relaxation (6). One of the eightfold paths of yoga is known as the Asana, or postures and poses. It is during these Asana that the yogin attempts to give the body stability and strength. To achieve this effect, the yogin should be able to hold the body in a particular position for long periods of time without effort (7).

Jones's counterstrain points also compare favorably with certain points in acupuncture. Local tender points in an area of dysfunction are considered spontaneous acupuncture points. These were termed *Ah Shi points* in Chinese medicine. These points were used in the treatment of painful conditions dating back to the Tang dynasty. Needling treated these points in the same manner as points along the acupuncture meridians of the body (8).

PHYSIOLOGIC THEORY

Although osteopathic physicians have used counterstrain for nearly 50 years, research to establish an exact physiologic basis for the counterstrain, as with all manual techniques, has been limited. It is by extrapolation that osteopathic physicians and researchers are able to understand how tender points occur and how counterstrain may work. Combining research results from the muscular system with results from the neurologic and circulatory systems, one can begin to understand an interrelationship that forms the basis of counterstrain.

To understand the probable mechanism of tender point generation, one can use an agonist/antagonist model of muscle action. Both muscles maintain a baseline firing rate when at rest in a neutral position. Activity can then induce lengthening in muscle A and contraction in muscle B. This increases the proprioceptive activity in muscle A, while a decrease occurs in muscle B's activity. When these muscles are called on to return from this position of moderate strain, if the motion occurs too forcefully or rapidly, muscle B is stretched against this increased firing rate. This can induce a reactive hypertonicity in muscle B with sustained increased firing, and a tender point develops (9). This theory of proprioceptor activity in somatic dysfunction was first delineated by Irvin Korr in his article "Proprioceptors and Somatic Dysfunction" (10).

The underlying mechanism in treatment using counterstrain proposed by Jones was strengthened by Korr's research. If the affected muscle could be placed in a position of maximum comfort, this would allow the muscle to shorten enough so it would no longer report the strain. As stated previously, this position would need to be held for 90 seconds to adequately affect the changes in the neural system processes as well as the myofascial tissues and the microcirculation. The initial reduction in discomfort may be explained by an instant change in the neural component, whereas the myofascial and circulatory changes would occur slowly over the remainder of the 90-second treatment.

HOW IT WORKS

The goal of osteopathic manipulative treatment is to maximize the function of the body by achieving optimum structure—a basic premise in place since the origin of osteopathy. The interdependence of structure and function is at the heart of osteopathic treatment. Without proper structure there cannot be proper function, and when proper function is disturbed there will be structural changes that follow. It is the differences in the musculoskeletal system, the circulatory system, the neurologic system, and all other body systems that dictate the type of treatment best suited to address any particular dysfunction.

Counterstrain tender points are found in muscles, ligaments, and tendons. These tender points can manifest themselves in many different ways, including pain, limitation of motion secondary to muscle hypertonicity, reduction in local circulation, decreased muscular strength, reduced joint mobility, and increased fascial tension (11). A successful counterstrain treatment would reverse any of these manifestations from which the athlete suffered.

TECHNIQUE

There are five basic steps in the use of counterstrain technique. The success of the treatment technique is intimately associated with addressing each step precisely and completely before proceeding to the next step. It is this precision that makes counterstrain a technique that is easy to understand, but more difficult to master. The basic steps for treating with counterstrain are as follows:

1. Identification of a tender point.
2. Positioning of the athlete to a point of maximum comfort.
3. Maintenance of the position for 90 seconds.
4. Slow passive return to a neutral position.
5. Reevaluation of the tender point.

The first step in locating tender points is to gain a general knowledge of tender points and their location. Tender points usually lie within the belly of the muscle or at the tendinous attachment. These areas are at least four times more tender than surrounding tissues. There may or may not be overt palpable evidence of tissue changes.

Many practitioners are confused by the similarities and differences between tender points, Chapman's reflexes, and trigger points. There may be some overlap, but there are also distinct differences in these conditions. Chapman's reflex points (neurolymphatic points) are not specifically related to joint dysfunction. They can be related to underlying visceral disturbances, such as appendicitis. Trigger points will often refer pain, sensitivity, or other symptoms to a target atea when pressure is applied (8).

Following identification of the tender point, the athlete must be placed in a position of maximum comfort. It is not necessary to completely alleviate the pain at the tender point, only to reduce it to about 30% of its maximal discomfort. This is a major benefit of treatment with counterstrain, as it allows the practitioner to individualize the position to each athlete. The general positioning is similar between athletes with a tender point located in the same place, but the final position necessitates individualization. The reduction to 30% is achieved by advising the athlete to consider the initial palpatory pressure to be the maximum discomfort the point can have. The athlete is to notify the clinician when the positioning has achieved a 70% reduction in that initial discomfort.

The position is then maintained for 90 seconds. During this step, the athlete must remain relaxed, and not be allowed to contract the affected tissue. It should also be noted that the clinician must also achieve a position of comfort during this time, as a position of discomfort would not allow the clinician to maintain the athlete's position for the requisite 90-second treatment duration.

Following the 90-second maintenance of the treatment position, the athlete is returned to a neutral position. The athlete must remain relaxed as the clinician slowly moves the athlete back to a neutral position. If the clinician notices any activity by the athlete, he or she should pause the return to neutral and instrucr the athlete once again to relax and not aid in the technique.

The final aspect of treatment is to recheck the tender point on return to a neutral position. A complete resolution of pain at the tender point is the desired outcome of the treatment. However, a result of the treatment could leave the athlete with no more than 30% of the original discomfort to be considered a success. If the treatment fails to achieve the desired outcome, it could be due to improper positioning, shortened duration of rreatment, or incorrect return to a neutral position. Failure of the treatment warrants a second attempt at treatment of the tender point with counterstrain. A second failure of the technique would be an indication to reassess the diagnosis or consider a different treatment technique.

INDICATIONS AND CONTRAINDICATIONS

Because counterstrain is an extremely gentle technique, most athletes tolerate the technique very well. As with any treatment, there are indications and contraindications to be addressed.

A complete history and physical examination can identify these situations and should be carried out on every athlete in a sports medicine practice. Details elicited in the history can be vitally important to the correct identification of the dysfunctional area.

Most athletes will recall an inciting injury incident that preceded their complaint. It is important to discuss any atypical position that may have occurred during the incident, because this may shed some light on positioning to the point of maximum comfort. It is this position of comfort that correlates with the position of original insult. Examples of situations in which tender points should come to mind are falls, sprains and strains, improper lifting, and sudden, unexpected movements. Because many athletes present in the immediate postinjury setting, still in the acute phase, counterstrain is a great technique choice. Many other techniques can exacerbate the athlete's pain, but counterstrain eases the athlete's pain by its inherent principles. Finally, keep in mind the potential for using counterstrain in an athlete with repetitive strain injuries.

A careful history and physical examination can also identify situations in which the physician may want to exercise care in using counterstrain, or treat with a different technique. The most notable contraindication would be the athlete's inability to cooperate with the examination. If an athlete is unable to communicate to the physician, the tender point cannot be elicited. Likewise, if an athlete cannot understand the initial rating of the tender point and the reduction in pain required to successfully treat, then the technique is also set up to fail.

Physical conditions that may or may not limit the ability of the physician to treat with counterstrain would include open wounds, sutures, healing fractures, hematoma, hypersensitivity of the skin, systemic or local infection, and aneurysm (9). It is difficult to assess and monitor a tender point if that point underlies a skin wound or sutures. A hematoma or hypersensitive area of skin may make it difficult for the athlete to perceive a change in the level of discomfort necessary to the technique. An athlete may not be able to be correctly positioned if a fracture is present, especially if the fracture is stabilized with a hard cast.

One final condition to note is that during the treatment of a posterior cervical tender point, a position of maximal comfort may also be a position of compromised vertebral artery function. When combining the motions of cervical extension with side bending and rotation to the ipsilateral side, the athlete should be continuously monitored for arterial compromise. The athlete should keep his or her eyes open, so that the clinician can easily identify nystagmus. The clinician can also converse with the athlete, asking about any dizziness. These measures should aid in quickly identifying a potentially harmful situation.

APPLICATION IN SPORTS MEDICINE

As mentioned previously, counterstrain is an optimal technique to use in the acute injury setting. It is best used when an exact mechanism of injury can be identified, so that the clinician can understand the undue stress placed on the athlete during the incident. The athlete's higher level of physical conditioning allows for a speedier return to function than a nonathlete. Early in the treatment process, a combination of counterstrain with articulatory techniques is effective. As the recovery progresses, the treatments can become more aggressive. Injuries that may respond well to counterstrain include ligamentous sprains, muscle strains, tender points resulting from extreme exertion during a highly competitive event, and musculoskeletal dysfunctions resulting from changes or increases in training.

Examples

Elevated First Rib (left) (Fig. 4.1)

1. The athlete is sitting while the clinician is standing behind.
2. The clinician holds the athlete's head with one hand on top while the opposite thumb palpates the first-rib tender point in the supraclavicular fossa.

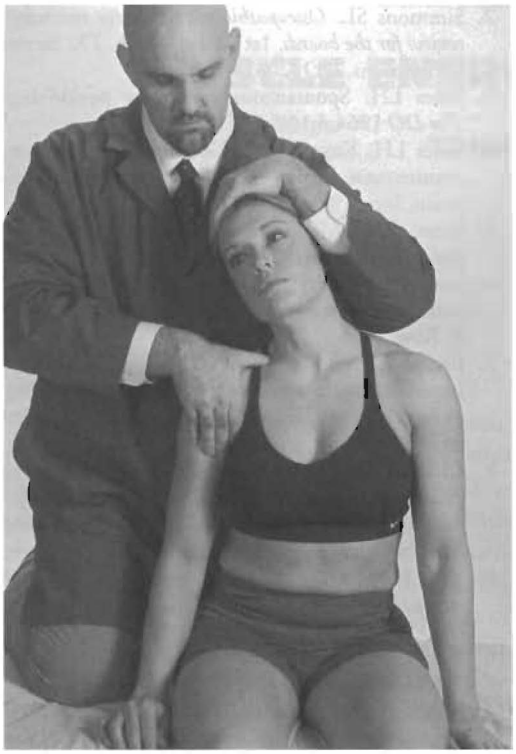

FIGURE 4.1. Setup for counterstrain technique on an elevated left first rib.

3. The clinician maneuvers the athlete's head into extension and minimal rotation toward the rib, feeling for the position of ease on the first rib.
4. The clinician holds the athlete's head and thumb in position for 90 seconds, then reassesses.

Posterior Lumbar Vertebral Dysfunction (right)

This counterstrain technique is three-in-one, treating L3, L4, and L5 dysfunctions.

1. The athlete is prone with the clinician standing opposite the dysfunctional side.
2. The clinician holds the athlete's right leg just proximal to the knee with the right hand.
3. The clinician uses his or her own knee to support the athlete's right distal leg and allow more variance and control of the limb.
4. The clinician can introduce more lumbar rotation and less extension by lifting the athlete's leg 6 in. proximal to the knee while the clinician's knee supports distal to the knee. This decision is based on how the dysfunction feels to the clinician.

First Position (Fig. 4.2A)

1. The clinician's left hand palpates the L5 tender point, located superomedial to the right posterior superior iliac spine, while the athlete's right leg is brought into extension to where a position of ease is felt.
2. This position is held up to 90 seconds.

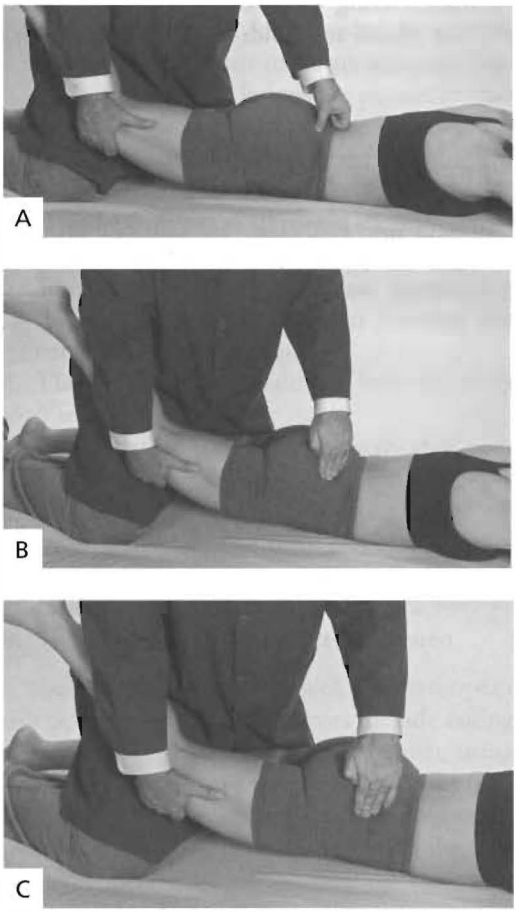

FIGURE 4.2. Setup for counterstrain technique for posterior lumbar dysfunctions. **A,** L5 tender point located just superomedial to the right posterior superior iliac spine. **B,** L3 tender point between the PSIS and the posterior aspect of the greater trochanter. **C,** L4 tender point about 1 cm behind the tensor fascia lata halfway up between the trochanter and the iliac crest.

Second Position (Fig. 4.2B)

1. The clinician moves the left hand to put pressure on the L3 tender point, between the posterior superior iliac spine and the posterior aspect of the greater trochanter.

Third Position (Fig. 4.2C)

1. The clinician moves the left hand to the L4 tender point, 1 cm behind the tensor fascia lata halfway up between the trochanter and the iliac crest. This spot should line up with the L5 and L3 tender points.
2. The clinician puts pressure on the point, maneuvering the athlete's leg for the position of ease and holds for up to 90 seconds.

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5

MUSCLE ENERGY TECHNIQUES

TODD J. MAY

OVERVIEW

The first description of the muscle energy technique is attributed to Fred Mitchell Sr., who first published his work in 1958. This publication was a summary of his work from the 1940s and 1950s. He described "one method of correction" using the effort of an extrinsic guiding operator as the activating force plus the use of respiration and muscular cooperation. However, a similar technique was developed independently by Kabat et al., called proprioceptive neuromuscular facilitation (PNF). PNF, although similar to muscle energy, was not widely known at the time Mitchell published his work.

The first techniques were directed at the pelvis, specifically targeting the sacroiliac joints, which at that time were considered immovable. As the muscle energy technique developed and grew, more of the spine was incorporated, then the extremities. In 1970, Mitchell began to teach his principles in a 5-day tutorial. In 1981, the existing five osteopathic medical schools began teaching these tutorials. This technique has now become part of the standard osteopathic manipulation therapy (OMT) curriculum in all osteopathic medical schools (1). Its popularity has crossed over into physical therapy and some athletic training programs, where many of these clinicians become trained in this technique to apply in their clinical settings.

PRINCIPLES

Muscle energy is classified as a direct technique in which the restrictive barrier is actively engaged to contract a muscle in a precisely controlled fashion against a distinct counterforce (2). It can be used to stretch out tight muscles and fascia, or mobilize a restricted joint. When treating segmental dysfunctions using muscle energy, eight essential steps must be followed (3):

1. Due to the precise force needed to use this technique, one must make an accurate diagnosis. Osteopathic lesions are named for the direction of ease or motion; the direction opposite the one of ease is rhe restrictive barrier.
2. Once the restrictive barrier is located, it must be engaged in all planes (flexion/extension, side bending, and rotation).
3. The force between the athlete contracting the muscle and the clinician resisting the contraction must be equal.
4. The contraction should be held for 3 to 5 seconds.
5. The athlete must relax completely after each muscle effort.
6. The athlete is repositioned into the new restrictive barrier in all three planes.
7. Steps 2 through 6 are repeated three to five times.
8. The restrictive barrier must be retested.

If these steps are not followed, the rreatment will be ineffective. Typical errors include failing to monitor the segment during treatment, using too much force or too short a duration of contraction by the athlete, not allowing for complete relaxation before or not engaging the new barrier, and/or not retesting after the treatment is completed (3).

PHYSIOLOGY

Engaging a restrictive barrier and then using an isometric contraction causes inhibition of the

agonist muscle through the Golgi tendon organ (GTO) (1). Muscle energy technique uses the GTO's reciprocal inhibition of the agonist and, to a lesser extent, antagonist muscle. This brief relaxation allows for a further engagement of the restrictive barrier. In 1967, Houk and Henneman demonstrated the GTO's sensitivity to minute changes in tension of the tendon and its complementary functioning with the muscle spindle fiber to execute a smooth, coordinated motor performance. This reciprocal relationship modulates muscular tension (3).

The treatment goal of muscle energy, as it is with many other manipulative techniques, is to restore the normal physiologic motion to the joint or area. By using muscle energy, the clinician can continue treating the restrictive barrier until the normal physiologic barrier is regained. By its actions on the GTO and muscle spindle, muscle energy can reduce the tone of hypertonic muscles and reestablish normal muscle resting length. This is especially important in regions that have chronic pain that cause muscular restrictions, hypertonicity, and spasm.

APPLICATION IN ATHLETICS

The defined uses of muscle energy techniques, as explained in *Foundations for Osteopathic Medicine*, are as follows (1):

1. Mobilize joints in which movement is restricted.
2. Stretch tight muscles and fascia.
3. Improve local circulation.
4. Balance neuromuscular relationships to alter muscle tone.

This last point is particularly relevant to athletics. Elite athletes tend to have high kinesthetic intelligence or awareness, their neurology is typically more finely tuned than the general population, with much of this due to muscle biofeedback. However, in segmental dysfunction the muscles that stabilize the involved segments are not necessarily treated at the same time, and this can lead to recurrent dysfunction and instability, especially if the athlete is not given exercises to restore proper function. Muscle energy gives the clinician the ability to restore alignment and mobility, and treat the supportive muscles as well.

Furthermore, muscle spasms seem to present earlier than the segmental dysfunction pattern, so treating the spasm may prevent, or minimize, the complete dysfunctional pattern from developing. The muscle energy technique can be helpful in mobilizing soft tissues in preparation for a high-velocity, low-amplitude (HVLA) thrust, ultimately making the HVLA treatment more effective.

Muscle energy is an effective technique in athletes because the athlete controls the action, not the clinician. Athletes know their limits and levels of discomfort better than the clinician, so by incorporating the athlete in the treatment, it empowers him or her to take responsibility for the dysfunction or injury, and that can be motivational. In addition, because there is no direct thrust through a physiologic or restrictive barrier, the muscle energy technique introduces minimal force, lowering the risk of discomfort after treatment. If it becomes too painful, the athlete can easily halt the treatment.

Muscle energy is a more practical and comfortable technique to use in acute situations. There is no impulse introduced into a painful area, the clinician can stop if the pain is too great, and it can be applied to almost any joint. For that reason, muscle energy is helpful in treating acute spasm and dysfunction, which can help athletes return to play faster.

INDICATIONS

Because the muscle energy technique uses athlete-directed force, it is effective in acute injuries. During the acute phase of an injury, muscles tend to contract in order to protect the body from further injury, such as in low back injuries. However, because muscle energy is a direct technique and acute injuries have a pain-spasm cycle that can be easily aggravated, manipulations should be stopped if pain or spasm increases. This requires close monitoring of the athlete's comfort during the contractions. The technique also is helpful in chronic myofascial

restrictions, muscular imbalances, and respiratory restrictions (3).

CONTRAINDICATIONS

Contraindications to the muscle energy technique are mainly relative and include fracture or avulsion injuries, muscle cramps, severe osteoporosis, metastatic bone or muscle disease, and open wounds. Generalized muscle soreness may be aggravated with this treatment. Because of the interaction between the athlete and clinician, an athlete who is unable to cooperate either from a lack of understanding or unresponsiveness makes the procedure nearly impossible to perform. If either of these two situations exists, it is advisable to use another technique (3).

ADVANTAGES IN ATHLETES

Muscle energy works in a similar fashion to the HVLA technique in that both techniques treat the restrictive barrier; in muscle energy, however, there is no mobilizing thrust. Because the athlete controls the action, it is a more comfortable technique in acute situations. If it becomes too painful, the athlete can halt the treatment. The athlete cannot do the same for HVLA. As mentioned previously, because muscle spasms present earlier than the segmental dysfunction pattern, treating the spasm may prevent or minimize the complete pattern from developing. This technique can also be helpful in mobilizing soft tissues in preparation for a HVLA thrust, ultimately making the HVLA treatment more effective.

TECHNIQUE

The particular positioning of the clinician and athlete depends on the area to be treated and the particular restriction. If the segment is sidebent left and rotated to the left, it means that the restriction in motion is to the right.

Muscle energy technique works the same for sttetching a tightened muscle as for treating segmental dysfunction. If lengthening a shortened muscle is the main goal, the shortened muscle is stretched to its limit and then isometrically contracted against resistance for 3 to 5 seconds. The muscle is stretched and the process is repeated three to five times. For treatment of a restrictive barrier, the restrictive barrier is engaged and then isometrically contracted against resistance for 3 to 5 seconds. The new restrictive barrier is engaged, and the process is repeated three to five times (3). During treatment, athletes tend to use maximal force. If the athlete is positioned correctly, however, very little force needs to be generated. Having the athlete match the clinician's force is the best way to control the amount of force.

Examples

Atlanto-occipital (C0-C1) Dysfunction (restriction of motion to the right) (Fig. 5.1)

1. The athlete lies supine on the table with the clinician at the head of the table.
2. The clinician holds the athlete's head in both hands and rotates the head to the right, engaging the resistance (Fig. 5.1).

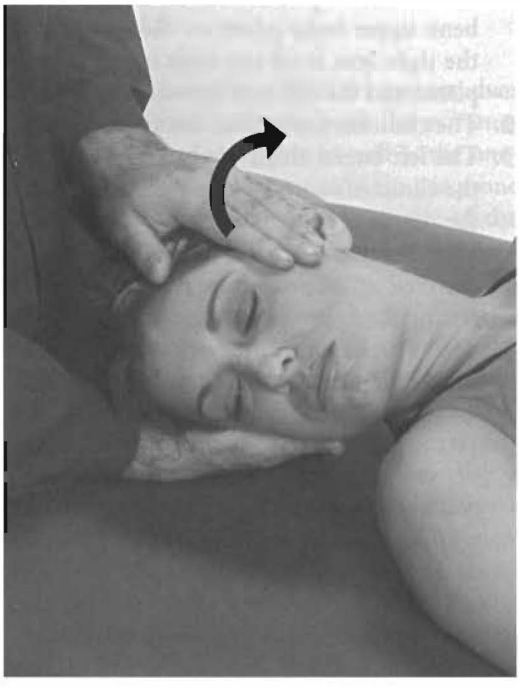

FIGURE 5.1. Cervical spine atlanto-occipital muscle energy technique (head turned to the right).

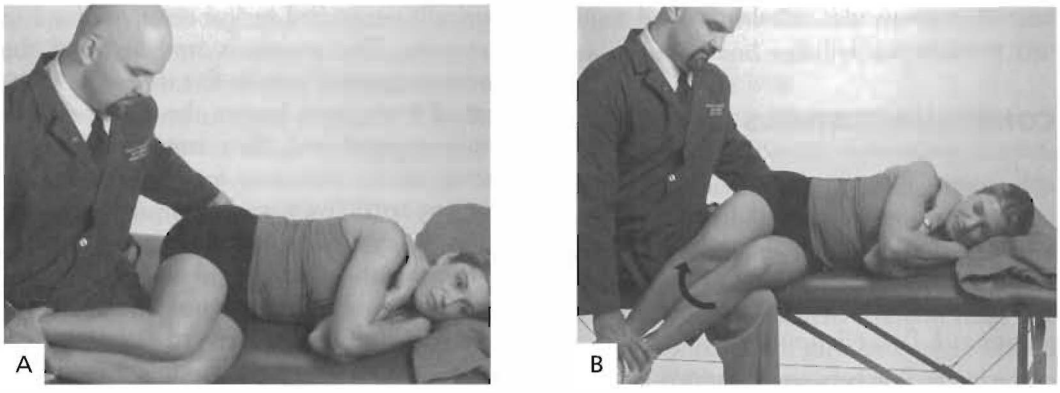

FIGURE 5.2. Setup for muscle energy treatment of sacral torsion, with posterior infero lateral angle on the left (setup with legs parallel to ground) **(A)** and with legs down lying on left side **(B)**.

3. The athlete attempts to rotate the head to the left for 3 to 5 seconds.
4. Relax, reposition, repeat, and reassess (1).

Sacral Torsion (posterior infero lateral angle on the left) (Fig. 5.2)

1. The athlete is positioned left lateral recumbent, upper body prone on the table so that the right arm is off the table in the anterior plane, and the left arm is posterior.
2. The clinician sits behind the athlete.
3. The left lateral thigh of the athlete rests on the clinician's anterior thigh just proximal to the patella (Figure 5.2A).
4. The clinician flexes the hips until motion is detected at the second sacral segment while flexing the knees at 90 degrees.
5. The clinician grasps the athlete's ankles and rotates toward the floor until the barrier is engaged, keeping the angle at the hip and knee the same (Fig. 5.2B).
6. The athlete directs the ankles upward toward the ceiling against the clinician's resistance for 3 to 5 seconds.
7. Relax, reposition, repeat, and reassess (1).

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6

MYOFASCIAL RELEASE

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WAYNE ENGLISH

OVERVIEW

Myofascial release refers to a class of manual techniques that are used to relieve the abnormal constriction of tense fascia. Myofascial treatments are widely used by physical therapists, massage therapists, and osteopathic and allopathic clinicians. Myofascial release is especially useful in sports medicine, as it helps athletes to relieve pain, resolve structutal dysfunction, restore function and mobility, release emotional trauma, and promote healing.

Myofascial release is of significant benefit in rreating a wide range of conditions that exist in sports medicine. Fascia acts much like a flexible skeleton, moving in response to complex activities. The fascial system and musculoskeletal system are anatomically connected in a complex and intricate framework that has many implications in the overall well-being of the body.

The benefits of myofascial therapy are not limited to muscle and fascia, but extend also to bones, joints, ligaments, skin, blood, cartilage, tendons, and other collagenous tissue as well as soft tissues of the body. Because fascia exists in virtually every part of the body, myofascial treatment can be tailored to any limb or body part. Myofascial techniques can be extremely useful in treating any type of athlete, from novice to elite and in any sport, from ballet to bodybuilding.

Many variations of myofascial techniques continue to be developed; in addition to myofascial release, the technique is also referred to as myofascial unwinding, myofascial manipulation, active release technique, myofascial massage, and soft tissue manipulation. All of these techniques view a healthy fascial system as integral to good health. The art of myofascial technique lies in understanding its basic concept, and learning to apply it anywhere it is useful in the body, trunk, or extremity.

Because the fascial system is very comprehensive, myofascial release is a very broad topic with many indications beyond the scope of this chapter. Some osteopathic physicians consider the realm of myofascial release to be very extensive, including counterstrain, direct fascial release, cranial osteopathy, facilitated positional release, and visceral manipulation in its repertoire. The goal of this chapter is to address the basic general concepts of myofascial release and its application in sports medicine.

RATIONALE

The athlete's body is subject to greater than normal biomechanical stress with the extremes of movement and repetition as well as the one-sided nature of many sports, and thus is prone to asymmetrical restrictions. The nature of the particular sport and the athlete's dynamic posture within the sport dictate the most common injuries and causes of pain. Asymmetry causes the body to adopt compensatory patterns. The reason for compensatory patterns may be handedness, eye dominance, or foot preference. Postural imbalances such as leg-length discrepancies and eye-level imbalance may also play a role. The tendency to move in one direction results in the body's attempt to maintain a center of gravity and a balance of all forces to attain equilibrium.

Problems in the myofascial system are recognized by loss of function, such as decreasing ability of the muscles to work properly, as well as nerve numbness, tingling, and pain. In addition,

myofascial problems may result in decreased range of motion, weakness, stiffness, soreness, and even in slower reaction time.

Myofascial pain is relatively quick and easy to assess, and myofascial techniques are usually quick and easy to perform. As long as the athlete is stationary, the treatment setting can be anywhere from the field or office, in the sitting or standing position. The benefits of myofascial release may be both immediare and long term. The technique has relatively few contraindications, and it does not aggravate hypermobility. Because myofascial release is noninvasive and safe with virtually no side effects, it comes with a record of very good results. Performance of any sport or task—ranging from basketball, golfing, bodybuilding, and running to administrative work—can be improved significantly with myofascial release.

CONCEPTS AND PRINCIPLES

Fascial Pain

Fascia is continuous from head to toe, surrounding every muscle, tendon, nerve, blood vessel, bone, and organ. It is interconnected in various sheaths or planes, holding structures together, giving them their characteristic shapes and support. The ability of fascia to absorb and redistribute forces makes it a compensatory organ.

Fascia is comprised of three layers, superficial, deep, and subserous. *Superficial fascia* is a continuous layer of connective tissue over the entire body between the skin and the deep fascia. Holding muscles and other structures in place are the connective sheets and bands of *deep fascia.* Lining the body cavities and viscera between deep fascia and the subserous membrane is a *subserous fascia,* addressed more commonly in visceral techniques. Its elastic and flexible properties afford fascia its dynamic capabilities and fluid movements. In addition, movement and warmth increase the elasticity of fascia as it stretches and moves freely to accommodate the biomechanical stresses of the body.

The repetitive stress and mechanical loading of tissues by athletic training cause increased strain on the fascial system. This increase in muscle tension commonly disrupts flow in the delivery of nutrients and removal of waste products in the tissues. The accumulation of waste products acts as an injurious stimulus, inducing further tissue irritation, pain, and inflammation. Up to this point, the process can be thought of as *acute.* Acute processes generally manifest as severe, sharp tenderness, edema, erythema, and bogginess in the soft tissues, with increased moisture and hypertonicity.

The continuation of this course results in a fibrous tissue reaction leading to a *chronic* process. Chronic changes are less likely to be reversible, making the soft tissue damage more permanent. Furthermore, the body experiences muscle shortening, decreasing stretch, and restricting movements. Manifestations of chronic processes are usually dull, achy, burning, ropy, cool, dry skin with fibrotic changes and slight tension.

Both acute and chronic tissue changes often present with compensation in other parts of the body. The pervasiveness and interconnectedness of fascia throughout the body create a scenario in which restriction in one part of the body will not only affect local, adjacent structures but may also affect areas distal to the site of injury. It then becomes necessary to address dysfunction in both the local area of injury as well as in distant areas.

Myofascial pain is sometimes associated with *fibrositis,* defined by fibrous tissue inflammation and connective tissue hyperplasia. Frequently, a hyperirritable region within a taut band of tissue with referred pain is known as a *myofascial trigger point.* Trigger points have a predictable distribution and are commonly found within areas of myofascial pain. The concept and principles of myofascial trigger points can be further studied in works by Janet Travell.

Because of the fascia's integration with the neuromuscular system, many symptoms are mediated by the sympathetic nervous system. Myofascial release elicits neuroreflexive changes in rhe musculoskeletal system, from the skin to deep spinal joints. During myofascial release, the afferent stimulation caused by a stretch movement results in the sequential relaxation

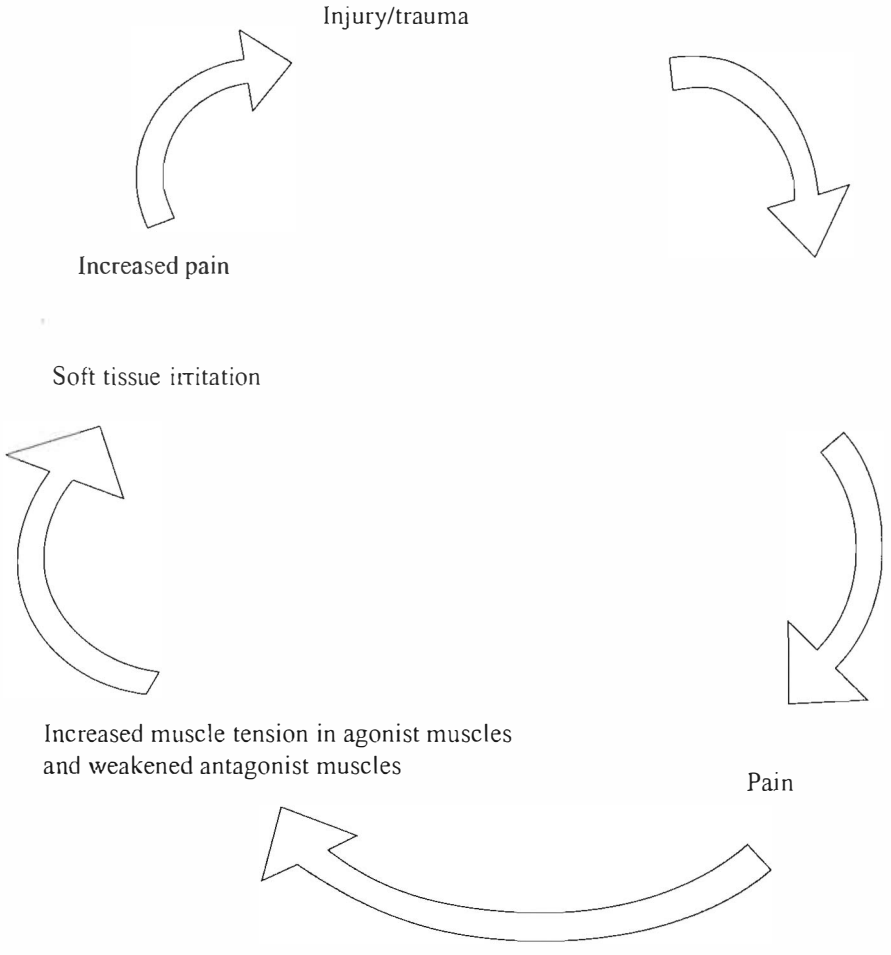

FIGURE 6.1. Positive feedback cycle of myofascial pain. The initial injury or trauma causes soft tissue irritation. The body interprets the irritation as pain and reacts by increasing muscle tension in the agonist muscle and weakening the antagonist muscle, which increases pain and further soft tissue injury and irritation.

of tight tissues by efferent inhibition, providing instant relief on many occasions.

Positive Feedback Cycle of Myofascial Pain

The soft tissue irritation caused by injury or dysfunction is read by the body as pain, and it reacts by increasing the tension in the agonist muscle and weakening the antagonist muscle. An increase in muscle tension is interpreted as increasing pain leading to increased muscle injury, thus positively feeding back into the cycle as soft tissue irritation (Fig. 6.1). Some examples of the multiple causes of myofascial pain are included in Table 6.1. Myofascial therapies are useful in reversing this damage.

Tight–Loose Concept (Direct-Indirect Barriers) in Myofascial Release

Tightness creates asymmetry, and looseness permits asymmetry. Three main barriers are encountered in osteopathic manipulation: the physiologic, anatomic, and restrictive (pathologic) barriers. A

TABLE 6.1. CAUSES OF MYOFASCIAL PAIN

Causes of Myofascial Pain	Examples
Poor posture	Chronic muscular strain, scoliosis, resting and exercise posture, leg-length discrepancies, anatomic variants, skating
Malpositioning/maladjustments	Bicycle seat, sleep position, weight training
Poor biomechanics	Improper techniques, shifting of center of gravity, jogging, running, snowboarding, skiing
Repetitive stresses, overuse injuries	Golf, tennis, soccer, swimming, baseball
Immobility	Sedentary lifestyle, prolonged rest
Steady contractions and position	Weight training, yoga, Pilates exercises, wrestling, prolonged sitting
Prolonged constriction of soft tissues	Ski and snowboard boots, rollerblades, ill-fitting sports bra or sportswear, tight shirts, collars, belts, shoes
Nutritional/vitamin deficiencies	Vitamin B_{12}, folate, thiamine deficiency, calf cramps, paresthesias
Infections/other processes	Abscesses, local inflammations, allergies, edema, effusions
Psychological factors	Depression, sleep disturbance, anxiety, tension
Endocrine/metabolic	Hypothyroidism, hypoglycemia
Other	Lifestyle, accidents, trauma, drug use, tobacco, alcohol, physical fitness

restrictive barrier causes asymmetry and inhibits movement in one direction. This is a basic concept of myofascial release, and looking for three-dimensional tightness and looseness is essential. Areas of myofascial injury can vary in size, pattern, and depth (superficial to deep), and they can be tight or loose in relation to one another.

The term "end-feel" describes the sensation one feels when a mobilized joint moves into a barrier, or the end of its range of motion. Asymmetrically perceived end-feels are commonly referred to as direct and indirect barriers. Direct barriers suggest tethering and tightness, whereas indirect barriers suggest tissue laxity and looseness. Movements are easier in some directions, less so in others. End-feels that are hard and terminate abruptly occur with direct barriers, whereas indirect barriers have soft and easy-to-navigate end-feels. Restrictions involving one side of the body frequently affect the opposite side.

Tight muscles are not always sources of pain and altered neuromusculoskeletal functioning. On the other hand, loosened sites are often chronic, painful, and susceptible to injury at relatively low thresholds of stress. Tight muscles can be self-limiting or they can steady an unstable region by increasing fibrotic tissue formation.

Physiologic Principles

DiGiovanna and Schiowitz (1) have described several principles underlying myofascial techniques. Their effects cross over to several types of tissues and systems, which is part of the reason why myofascial techniques are effective. The principles are as follows:

- *Increased circulation* to the area of restriction delivers oxygenated blood and nutrients to the tissues and removes harmful metabolic waste products.
- *Increased venous and lymphatic drainage* decreases local swelling and edema caused by tissue inflammation.
- The *elasticity and flexibility of connective tissues* elongate connective tissues secondary to mechanical loading.
- Increased temperature causes an increase in elasticity and stretch of the muscle.
- The *stretch reflex* stimulates tone in hypotonic muscles in areas of looseness. Muscle stretching excites the muscle spindle causing reflex contraction of the muscle.
- The stretch reflex is sustained by the *muscle spindle reflex*. The Golgi tendon organ can cause prompt relaxation of the muscle (via

inhibition) when tension on the tendon becomes extreme. Relaxation of contracted muscles occurs decreasing the oxygen demand of the muscle, decreasing pain, and allowing normalized range of motion across a joint.

- *Reciprocal inhibition:* The stretch reflex acrivates one muscle (e.g., quadriceps), while simultaneously inhibiting its antagonist muscle (the hamstrings).
- *Crossed extensor reflex:* The stretch reflex stimulates one muscle (e.g., right quadriceps), while simultaneously activating the contralateral antagonist muscle (the left hamstrings), creating an "X" pattern.

MECHANICS OF MYOFASCIAL PATHOLOGY

The effects of multidirectional forces on both local and distant joints and soft tissues manifest in injury patterns. Mechanical loading, increasing strain, and repetitive stress on a soft tissue over time under a constant load are certain to cause deformation. All tissues are mechanically responsive, exhibiting stress–strain responses that affect the body neurologically and anatomically.

Two-handed palpation is generally required to interactively assess and modify dysfunctional patterns and the athlete's ability to adapt. Searching out tight and loose end-feels allows assessment and simultaneous treatment of dysfunctional soft tissue patterns and joint-related movements. Using compression, traction, and twisting maneuvers to mechanically load areas of restriction can help release barriers. Athlete-assisted release-enhancing maneuvers may be further integrated to complement the treatment process.

The fascia release phenomenon is also known as *melting* or *quivering* of rhe segment. As treatment is directed to an area of tissue injury using layer-by-layer palpation, the practitioner's hands move further into the restrictive barrier, which begins to soften, allowing the muscle to relax into the fascia. By permitting the fascia to guide the practitioner, the tissue will ease up progressively from superficial to deep. The fascia responds by becoming more pliable and increases in elasticity. In addition, the heat imparted by the hands further increases the stretch and melting of the segment. Appropriate application of stress on the tissue results in both muscle and fascia tissue relaxation, as the tightness "melts" and gives way under the application of load.

INDICATIONS

Myofascial release techniques are typically gentle and can be performed on a wide range of athletes including hospitalized athletes and elderly athletes who cannot tolerate more aggressive therapy. Additionally, they can aid in sleep disturbances, depression, and other psychologically caused disturbances. The techniques can be performed in multiple positions and on athletes who cannot tolerate much movement. The principles of myofascial therapy have been applied in treating many injuries, such as those of the rotator cuff, tendinitis, shin splints, golfer's elbow, tennis elbow, bursitis, muscle strains, frozen shoulder, adhesive capsulitis, knee and quadriceps problems, iliotibial band injuries, muscle weakness, strength imbalances, poor flexibility, poor posture, nerve entrapment syndrome, and chronic joint pain and muscle stiffness.

These techniques can be used alone or in conjunction with other techniques such as the high-velocity, low-amplitude (HVLA) thrust, muscle energy, counterstrain, facilitated positional release, and visceral techniques. Modalities such as acupressure, acupuncture, transcutaneous electrical nerve stimulation (TENS), ultrasound, and spray and stretch are widely used in managing injuries and pain in athletes and work well with myofascial release techniques. Spray and stretch is commonly used in the treatment of a trigger point in concert with treating myofascial pain. A combination of the last-mentioned modalities is especially useful when HVLA and muscle energy are contraindicated. Contraindications to myofascial release are bacterial infections, sepsis, fractures, cancer (risk of metastasis), and visceral trauma. It is best to use good judgment in individual cases, and precautions should always be taken.

Myofascial release is particularly effective in three areas:

1. *Soft tissue restriction or spasm.* Myofascial release has also been referred to as soft tissue mobilization. Soft tissue mobilization should be differentiated from joint mobilization. Whereas joint mobilization is guided by specific strategies to restore normal arthrokinetics, myofascial release adapts to the many different planes and directions of fascia. Myofascial release relaxes and reduces hypertonic spasms through its physiologic principles. Releasing myofascial restrictions over a large region can appreciably influence joint mobility.
2. *Restricted motion.* The stretching used in myofascial techniques is very effective in increasing the range of motion in any given area of the body. Increasing the range of motion helps to prevent future injuries and promotes lymphatic flow and circulation to tissues. As the myofascia increases the elasticity of the muscle unit, joint mobilization should subsequently be incorporated. Additional strengthening exercises are recommended to reeducate the neuromuscular system, encouraging new, more efficient movement postures.
3. *Pain.* Many subjects try to work around the pain before seeking treatment. Compensatory biomechanical shifting begins to take place. This readjustment in postural mechanics causes tissue damage and initiates the positive feedback cycle of myofascial pain in other parts of the body. Myofascial release is advantageous in both acute and chronic pain. Generally, acute cases tend to resolve in just a few treatments. The longer a condition has been present, the longer it will take to resolve. Occasionally, dramatic results will occur immediately after treatment.

ADVANTAGES IN ATHLETICS

Myofascial techniques are extremely useful in athletes because they remove restrictions in the fascia that cause limited mobility, postural distortion, poor cellular nutrition, pain, and a variety of other dysfunctions. Myofascial treatments can free fascial tissues systematically and restore structural integrity and proper alignment. Thus, myofascial release can be applied to any sport, including tennis, golfing, swimming, weight training, hockey, football, baseball, soccer, martial arts, running, gymnastics, discus throwing, skating, skiing, snowboarding, and rehabilitation.

Contractile muscle tissues are extremely susceptible to the wear and tear of the daily activities of athletes, and myofascial pain can be a part of everyday training for serious competitive and recreational athletes. Blunt or repetitive trauma causes tissue reaction and acute pain, which triggers the soft tissue–pain cycle. By intervening in this cycle quickly after known trauma with such techniques, recovery time can be shortened and standard physical therapy modalities can be used less often.

Additionally, chronic myofascial tissue fibrosis occurs often in athletes who have multiple acute processes that eventually develop into chronic changes. Treating acute processes before they become chronic is the ideal goal, and teams with knowledgeable athletic trainers treating athletes daily can assist in that process. The accumulation of stress on the body over years of athletics, however, produces chronic changes, which can limit performance. Myofascial techniques can at least help to minimize the fibrotic changes that occur and keep connective tissue loose and unrestricted. The aging athlete in particular can benefit significantly from myofascial release.

The body has an extraordinary capability to respond to myofascial release. Myofascial treatment aids in improving quality of life and athletic performance. Because the hyperirritability that develops can often be minimized, it is important to treat myofascial pain to prevent acute processes from becoming chronic.

TECHNIQUE

The goal of treatment is to identify areas of greatest restriction, local and general patterns,

TABLE 6.2. RULES OF MYOFASCIAL RELEASE

1. The athlete should be relaxed and in a position of ease.
2. The clinician should be relaxed and in a position of ease.
3. The clinician should use body weight more than upper arm strength during the techniques.
4. The force must be of low intensity, slowly applied, maintained for 3 to 4 seconds, and released slowly.
5. The force must not create pain.
6. Always push or pull muscle away from bone, not toward bone.
7. Push or pull muscle in a direction perpendicular to the long axis of the muscle fibers.
8. Avoid skin friction and irritation from rubbing.
9. Use leverage whenever possible.
10. The clinician's fingertips, and thenar and hypothenar eminences are the tools to apply pressure.
11. Apply a counterforce to maintain the athlete's position when applying a transverse force (push or pull) across a muscle body.
12. Muscle stretch can be achieved with lengthwise traction applied at the muscle's origin or insertion.
13. Compression can be used with multiple muscle layers to reach deeper tissue.

From Murphy T. Principles of manipulative techniques: myofascial techniques. In: DiGiovanna EL, Schiowitz S, eds. *An osteopathic approach to diagnoses and treatment*, 1st ed. Philadelphia: JB Lippincott, 1991:83–84.

and then to release tightness and reestablish functional symmetry without aggravating hypermobility. The aim is to alter the patterns that caused injury in the first place and prevent recurrence of problems. Using proper palpatory skills and responding interactively to shifting changes, the practitioner can efficiently treat the athlete in a reasonably short time. Furthermore, myofascial techniques promote homeostasis and optimize body performance.

The clinician must be aware of the rules of myofascial technique (Table 6.2). There are many ways to engage restrictive barriers; however, the two main approaches in myofascial treatments are indirect and direct techniques in any combination of counterclockwise, clockwise, traction, compression, and twisting movements bilaterally. The direction of tissue movements are important considerations: Cephalad (north) direction refers superiorly toward the head, and caudad (south) refers inferiorly toward the legs. Some myofascial techniques require the hands to be moved in opposite directions to relieve the tension. Others require both hands to move in the same direction. The force can be described as originating from the base of the palm and going to the fingertips. Some practitioners use the base of the palm to gently push or pull, and others use the fingertips to slowly push through the restrictive barrier.

Direct and Indirect Techniques

The direct approach addresses the agonist muscle(s) in question causing the problem while the indirect approach addresses the antagonist muscle(s). The *direct approach* moves body tissues and/or joints closer to the restrictive barrier. This is accomplished by engaging the dysfunctional tissues head-on. Direct myofascial release maneuvers strain (deform) areas of tightness. Releases are triggered by holding firmly against soft tissue resistance, toward ditect myofascial barriers. By making tightness tighter, releases occur quickly, often in multiple directions at the same time. For example, if the thoracolumbar fascia moved more freely caudad (inferiorly toward the legs) than cephalad (superiorly toward the head), the practitioner would hold the tissue cephalad (toward the barrier), allowing the tissues to stretch (Fig. 6.2A).

For each area of restriction, there is a three-dimensionally related area of looseness. The *indirect approach* follows the restrictions to the point of ease. In an indirect treatment, the practitioner moves tissues and/or joints away from the restrictive barrier. Generally, the looseness is located in the opposite direction from the tightness. Practitioners may find it easier to follow gently behind releases as they occur in sequence. Using the same example as shown in Fig. 6.2A,

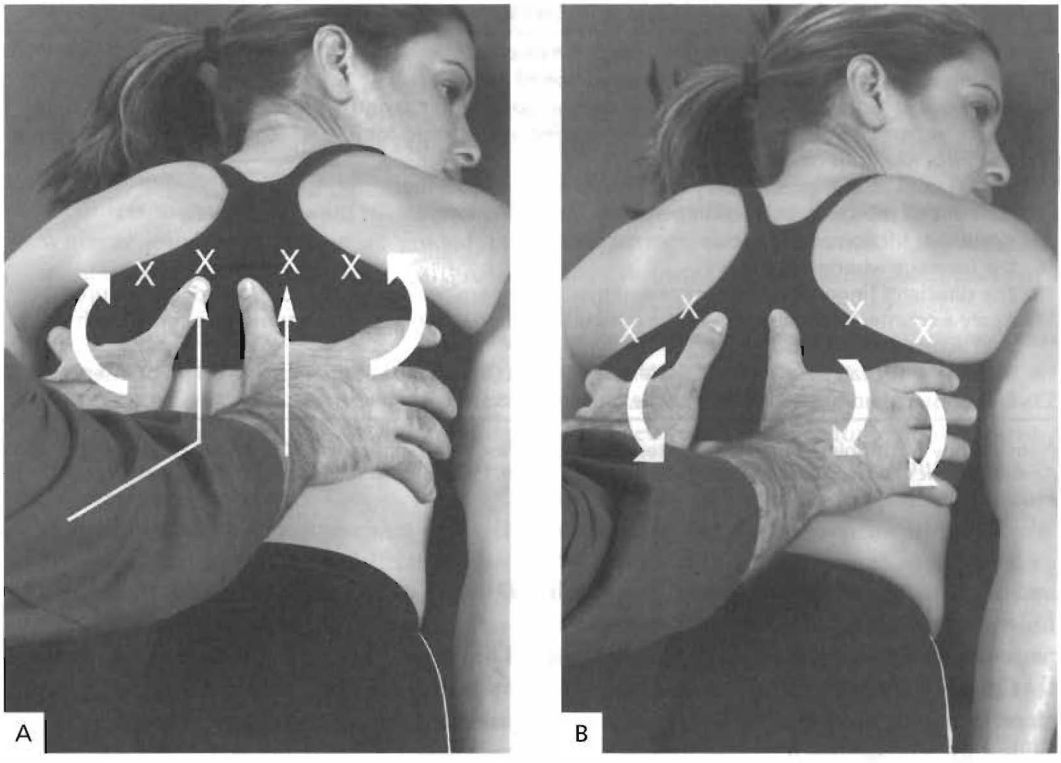

FIGURE 6.2. Myofascial release. **A,** Direct technique. The clinician's hands move cephalad into the restrictive barrier. **B,** Indirect technique. The force is directed away from the barrier and toward the area of looseness.

the tissue would be held caudad (away from the barrier) (Fig. 6.2B).

As the myofascial releases occur, the tissues often feel as though they are "quivering" or "melting" in multiple directions simultaneously. The art of the technique lies in being able to palpate both single and multiple releases following the changing patterns and arriving at new barriers until a point of freedom is attained.

Myofascial release treatment may be active or passive. In an active treatment, the athlete actively assists in the treatment, usually in the form of isometric or isotonic contraction. In a passive treatment, the athlete relaxes and allows the practitioner to move the body tissues.

Procedure

Restrictions in deep fascia may be located by visual analysis of the subject's posture, observing where fascia may be shortened or lengthened. Restrictions may also be identified through layer-by-layer palpation of tissues, finding where tissues seem to "stick together" or resist lengthening. Practitioners must be aware of subtle areas of resistance and areas of freedom in the movement of fascial tissues using tactile senses.

Starting from the skin superficially and working deeper, varieties of traction, twist, stretch, shear, and compression are applied three-dimensionally to detect changes in underlying structures. Inherent tissue and joint motions are monitored for shifting tightness (direct barriers) and looseness (indirect barriers). The stretching of the tissues and the heat imparted by the practitioner's hands help produce a softer consistency, allowing elongation of soft tissues.

Depending on whether the practitioner chooses direct or inditect technique, the

directions of both greatest and least resistance can be palpated. In response to gentle pressure over a period of time, the subsequent "quivering" or "melting" occurs as the fascia softens and releases in multiple planes and directions. Using the fascia as a guide, the practitioner follows the motion of the tissue, barrier upon barrier until freedom is felt. There may be intermittent points where the fascia will ease, and another barrier will be met. Using the same force, the new barrier may again melt and ease up, leading to another point of restriction. Generally, the force should move parallel to the muscle. If the athlete is lying prone and the paraspinal muscles, for example, are being addressed, then the force should be horizontal and parallel to the floor. On completion of treatment, the practitioner's hands should leave the tissues as gently as on entering.

Release-enhancing maneuvers such as the following are helpful:

- The respiratory phases of inhalation and exhalation may be sustained.
- Positioning the head right, left, or center is often helpful.
- Craniosacral manipulation.
- Soft tissue mobilization.
- Recruiting the use of muscles in isometric limb and neck movements against the table or chair creates postisometric muscle relaxation at various sites.

Additional useful modalities include

Transcutaneous electrical nerve stimulation (TENS)

Percutaneous electrical nerve stimulation (PENS)

Ultrasound

Trigger point therapy

Acupuncture

Acupressure

Spray and stretch

Examples

Because fascia surrounds and compartmentalizes all structures throughout the body, there are several hundred different types of applicable myofascial release techniques. This chapter outlines only a few typical techniques for common sports-related injuries.

Myofascial techniques can be modified and customized using the many different forms of stretching. Any part of the body can be treated using a combination of twisting, compression, and stretching in counterclockwise and/or clockwise directions. Release-enhancing maneuvers such as deep breathing are often beneficial during the treatment process.

Cervical Myofascial Release (Fig. 6.3)

What it is: Cervical release in the seated position.

What it does: Increases general cervical–upper thoracic range of motion, releases soft tissues in the cervical area.

Rationale: Many sports can cause myofascial pain in cervical muscle groups such as the trapezius, splenius cervicis, levator scapulae, and semispinalis capitis.

1. The athlete is seated in a relaxed position with the clinician standing behind the athlete (Fig. 6.3A).
2. With both hands, the clinician assesses cervicothoracic mechanisms for tightness, looseness, and asymmetry.
3. Place the hands around lateral and posterior cervicothoracic attachments.
4. Bilateral, anterior-inferior circumferential twist and stress is induced across and around the cervicothoracic junction against both direct and indirect barriers.
5. Releases generally move anteriorly and laterally. Follow tissue movements until symmetry occurs across the cervicothoracic junction.
6. Treatment is completed when symmetry has been established in relation to active and passive cervicothoracic, upper limb, respiratory, and costal cage mechanisms.

Thoracic Myofascial Release (Fig. 6.4)

What it is: Thoracic release in supine position.

What it does: Balances scapulothoracic, thoracic spine, and costodiaphragmatic relationships.

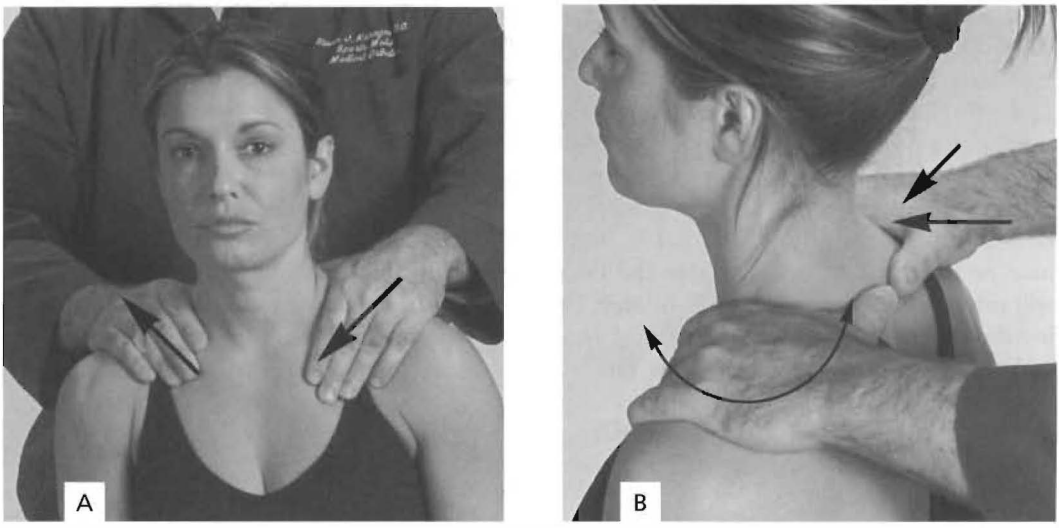

FIGURE 6.3. Cervical myofascial release with the clinician behind the athlete **(A)** and lateral view **(B).**

Rationale: Restoring symmetry to soft tissues in the thoracic area can optimize athletic performance.

1. The athlete is supine with the clinician seated at the head of the table.
2. Reaching between the athlete and the table, the clinician places the hands firmly against inferior costothoracic attachments on both sides of the thoracic spine, maintaining whole-hand contact across the erector spinae and around the costal cages.

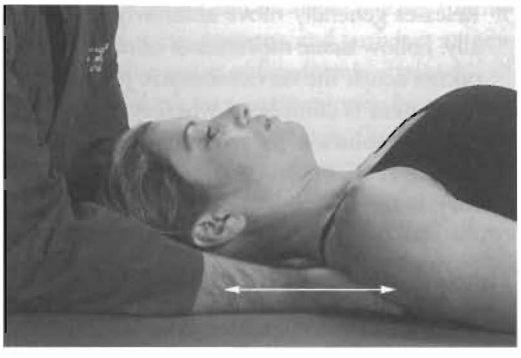

FIGURE 6.4. Thoracic myofascial release, lateral view, with hands under the supine athlete moving caudad to cephalad.

3. Palpate positional and tight–loose asymmetry patterns.
4. Gently but firmly lift the costothoracic attachments anteriorly and laterally, then apply twist and traction in the caudad and cephalad directions as deemed necessary. (Fig. 6.4).
5. Treatment is completed when costothoracic movements are as functionally symmetrical as can be expected.

Thoracolumbar Myofascial Release (Fig. 6.5)

What it is: Thoracolumbar release in prone position.

What it does: Balances the thoracolumbar junction in relation to both lumbopelvic and costothoracic mechanics.

Rationale: The high occurrence of and longer recovery from low back pain in sports injuries can be decreased using myofascial release.

1. The athlete is prone with the head turned to the more comfortable side—right, left, or center—while the clinician stands beside the athlete's hip facing cephalad.
2. The clinician places his or her hands at the thoracolumbar junction.
3. Place hands widely open with the thumbs pointed cephalad along both sides of the

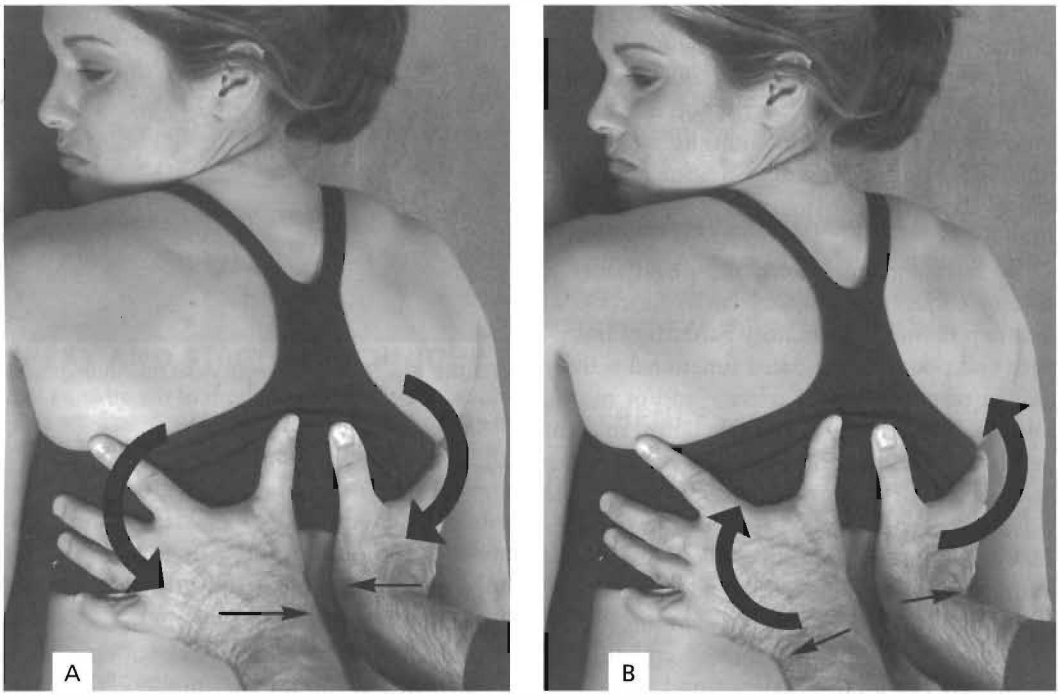

FIGURE 6.5. Thoracolumbar myofascial release. Posterior view, with the hands rotating opposite each other **(A). B,** Hands rotating toward each other.

spinous processes, while the remainder of each hand spreads over the posterior-inferior costodiaphragmatic areas (Fig. 6.5A).

4. Identify superficial and deep tightness and looseness patterns.
5. Separate the thumbs across the midline as the right hand creates clockwise and the left hand creates counterclockwise traction (Fig. 6.5A). The hands should not slide on the skin.
6. As traction and twists are maintained, tissues begin to relax and subsequently release.
7. Hands can be rotated toward each other to reverse the direction of ttaction (Fig. 6.5B).
8. Treatment is completed when segmental movements are functionally symmetrical.

MYOFASCIAL UNWINDING

Myofascial unwinding is another form of myofascial release. It is a very important and most useful hands-on technique for postinjury and postsurgical rehabilitation. It can also be used in injury prevention when, through the pre-participation physical (neuromusculoskeletal) examination, joint restrictions, "myofascial tightening," and somatic dysfunction are detected. With unwinding techniques, the practitioner can bring about spontaneous bending and twisting maneuvers to release fascial and articular restrictions in both upper and lower extremities. These techniques can be carried out by both single and multiple clinicians to obtain maximum benefit to release the tissues involved.

Myofascial unwinding techniques can be used in conjunction with other preliminary or follow-up techniques, such as muscle energy, myofascial "pillar" release, prolonged pressure on trigger point (acupressure), ligamentous strain, A. T. Still corrective, and Jones counterstrain techniques. These are all good techniques and can be used to precede low-velocity, high-amplitude and HVLA techniques.

The goal of these techniques is to restore normal resting muscle length of agonist and antagonist groups. This should result in correcting the myofascial tissue restriction and restoring normal joint range of motion. Myofascial unwinding ultimately reduces "tissue bind" and brings about normal "joint play," as defined by Mennell.

By releasing restrictions, the practitioner is able to correct musculoskeletal dysfunction as well as prevent compensatory patterns of imbalance and poorly coordinated functional activity that in turn can lead to overuse and/or misuse injuries. If injuries do occur, safe return to competition with restoration of peak performance can be brought about more quickly. The athlete will also have more rapid return of proper balance and coordination with restoration of maximum flexibility, agility, speed, and endurance.

At the time of injury, it is important for the clinician to approach the injured athlete with sensitivity. The athlete may not understand the injury and be fearful and concerned that even a physical examination will cause further pain and injury. By approaching him or her in a gentle, permission-seeking manner, the clinician can help the athlete avoid unnecessary guarding and/or overprotection of the injured part that can cause an altered pattern of behavior that would further restrict the activity in the involved area, causing a potential disuse or misuse phenomenon and thus slowing recovery and return to safe play.

Using a gentle, permission-seeking manner, the clinician more quickly gains the confidence and cooperation of the injured athlete. This cooperation gives the opportunity for a more accurate diagnosis and the beginning of active-assistive mobility that will prevent unnecessary congestion, swelling, and restriction of the injured part.

Example

1. Myofascial unwinding is most commonly performed with the clinician at the foot of the table.
2. With hands beneath the athlete's heels, the clinician picks up both legs with knees fully extended, creating 10 to 20 degrees of hip flexion.
3. The clinician proceeds to seek out tight–loose elements of hip rotator–lumbopelvic tissues both unilaterally and bilaterally. This is accomplished by carrying the extended limb into varying degrees of rotation, abduction, and adduction while applying a direct force (traction) or indirect force (compression).
4. The clinician takes the extremity to the barrier (direct or indirect) and holds it against the barrier while focusing on associated tight–loose relationships. Releases should occur as twist, traction, compression, and shear forces are used interactively against direct and indirect barriers.
5. From the foot of the table, long-lever unwinding maneuvers release the whole lower limb, including the foot and ankle (Fig. 6.6).

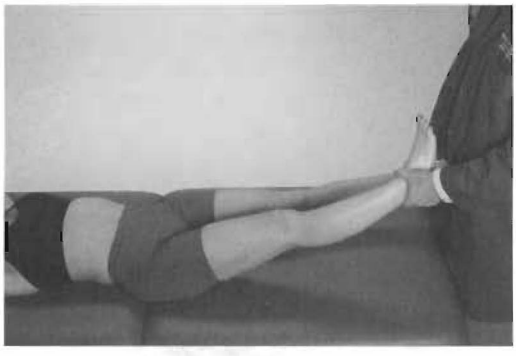

FIGURE 6.6. Setup for myofascial unwinding technique. The clinician holds both of the athlete's legs in extension. Using the legs as long levers, the clinician can move them around into and out of restrictive barriers.

Post-treatment

Athletes should be made aware of post-treatment discomfort similar to postexercise soreness that may be present after the initial treatment. In addition, it is essential that the athlete develop a simple, time-efficient exercise program for long-term success. The exercise program should include stretching areas of tightness without aggravating pain or instability. Looseness, or areas of inhibited muscle activities,

should have emphasis on strengthening, stabilization, and toning exercises. Both agonist and antagonist muscles should be addressed in maintaining and restoring function.

Proper exercise, nutrition, relaxation, and psychotherapy may also be incorporated for a holistic approach. Ultimately, the long-term success of treatments depends on the athlete's compliance with recommendations for aftercare.

SPRAY AND STRETCH TECHNIQUE

This technique is effective in single-muscle myofascial pain syndromes. It is frequently used after trigger point injections to inactivate all trigger points in a specific muscle. It is also helpful after trigger point injections and before passive range of motion and manual medicine procedures. Spray and stretch is effective on the sidelines to treat acute muscle spasm. The technique uses vapocoolant sprays to increase flexibility of the muscles. Commonly used sprays include Fluori-Methane and ethyl chloride, although Fluori-Methane is preferred because it is less volatile (5).

The purpose of the treatment is to inactivate the trigger points in a muscle. This is done by stretching the muscle out to its full range of motion without exciting reflex spasm. The athlete must be relaxed during the technique. Cutaneous stimulation by the vapocoolant spray blocks reflex spasm and pain, which allows passive stretching of the muscle (5).

To perform the spray and stretch technique effectively, the clinician must have understanding of the origin and insertion of muscles. The vapocoolant is sprayed approximately 18 inches from the surface of the skin and at a 30-degree angle in a pattern directed along the muscle fibers of the muscle group being addressed. The muscle is passively stretched as parallel sweeps of the spray are applied sequentially along the entire length of the muscle at a rate of approximately 4 inches per second. The procedure can be repeated approximately two to six times depending on the muscle group. Cooling occurs as the spray evaporates from the skin, but it is important to avoid prolonged skin exposure to the spray, because it will turn the skin white (5).

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7

JOINT PLAY

MATTHEW S. REEVES

OVERVIEW

John Mennell introduced the theory of joint play as a therapeutic manual medicine technique through his book *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques* in 1964. Since its introduction, this valuable and time-tested principle has not been further expanded on, nor has it received any research in today's age of evidence-based medicine. Nevertheless, joint play is readily used and essential to the pain-free function of synovial joints, both in principle and as a technique of manual medicine.

Joint play is defined by Mennell as small movements within a synovial joint that are independent of voluntary muscle contraction (1). These movements measure not more than 1/8 inch in any plane and follow the contour of the opposing joint surfaces. By nature, joint play is an involuntary movement that is inherent to the musculoskeletal system and cannot be introduced by voluntary muscles. It provides roll, glide, distraction, and spin combinations for joint motion and occurs in the shape of the joint surfaces (3). Capsular laxity allows for this motion, which is essential for the proper functioning of normal, painless active and passive range of movements (1–3). All movements of living anatomy are based on the summation of the movements of joint play and the movements of voluntary muscles. Although small, these precise involuntary movements are important, as their integrity greatly affects the performance of the gross voluntary movements of synovial joints.

JOINT DYSFUNCTION

To better understand the concept of joint play, joint dysfunction needs to be defined. According to Mennell, joint dysfunction is the loss of joint play movement that cannot be recovered by the action of voluntary muscles (1). This pathologic condition is readily reversible in its early stages and is common in life but cannot be demonstrated after death, which presents a difficulty in researching this concept. Yet, if joint dysfunction is left uncorrected, restricted voluntary movement and joint pain develop. Therefore, recognizing joint dysfunction as a pain-producing pathologic condition makes the restoration of normal joint play by manipulation a logical and reasonable treatment.

Joint dysfunction is further defined by Mennell as more than a *diagnosis*; it is also an invaluable *sign* of some serious pathologic process or joint disease (1). The distinction between these two entities, the clinical *diagnosis* and the physical examination *sign*, can be made only by careful evaluation including history, clinical examination, radiographic study, and often laboratory investigation.

PRINCIPLES AND CONCEPTS

In the traditional teaching of musculoskeletal medicine, emphasis is usually placed on the deficiencies of the muscles in the evaluation of functional loss, thereby focusing rehabilitation on muscular retraining and development. Joint disease, however, is often the cause of secondary muscle changes, particularly atrophy and spasm.

Even in light of this knowledge, treatment is often aimed at the joint only when gross clinical and/or radiographic changes are demonstrated, which then often neglects the muscles. Neither approach is complete. With this in mind, Mennell reminds clinicians of four basic truisms in practice (1):

1. When a joint is not free to move, the muscles that move the joint cannot be free to move it.
2. Muscles cannot be restored to normal if the joints that they move are not free to move.
3. Normal muscle function is dependent on normal joint movement.
4. Impaired muscle function perpetuates and may cause deterioration in abnormal joints.

Mennell states there is a vicious circle of effects that develops in any musculoskeletal problem, but the prime fault usually lies in the synovial joint (1). Mennell derived the manual medicine principle and technique of joint play from his work in determining that the prime fault of joint dysfunction is in the synovial joints. If the prime fault can be corrected, the secondary abnormalities are usually corrected also.

DIAGNOSIS

Diagnosis of joint dysfunction is best achieved by clinical means. Static radiographs do not demonstrate such subtle pathology, and although stress radiographs may be used to demonstrate the end range of joint play movement, the cost of films, repeated exposure, and time to complete the detailed examination for such small movements make this an unreasonable option. Because joint dysfunction does not cause any known biochemical alterations, laboratory methods are of no assistance. Although this sounds contrary to previous statements regarding the distinction between joint dysfunction as *diagnosis* or *sign*, the important difference is that laboratory and radiographic evaluations rule out underlying joint pathology when joint dysfunction is a clinical *sign* rather than the *diagnosis*. As is true for other techniques, the prerequisite for successful treatment is accurate diagnosis.

A much more readily available means of diagnosing joint dysfunction is a careful physical examination. There may be individual variations in the degree of joint play at any specific synovial joint, but there is no variation of technique in eliciting each movement at each joint. In the examination of a joint, Mennell clearly defines ten general rules that must be followed when using this manipulative technique (1):

1. The athlete must be relaxed with the joint being examined protected from unguarded, painful movement. Unguarded movements will result in spasm of the supporting muscles, thus preventing movements necessary for joint play.
2. The clinician must be relaxed and use a grasp that is firm and protective, but not restrictive.
3. One joint must be examined at a time, breaking up complex joints into their single components. For example, the wrist is examined by evaluating joint play at its individual articulations between the radial, ulnar, and carpal bones.
4. One movement at each joint is examined at a time.
5. In the performance of a movement, there is a mobilizing force and a stabilizing force exerted on opposing facets of the joint.
6. Comparison of joint play observed in the examined joint to the same joint on the opposite, unaffected limb is used to determine the extent of joint play.
7. No forceful or abnormal movement must ever be used, with the extent of movement being not greater than that assessed in the same joint on the unaffected limb.
8. The manipulative movement is a sharp thrust with velocity to cause gapping or sliding at the treated joint.
9. Joint play movements occur when all the "slack" is taken up in the joint.
10. No examining movements should ever be performed in the presence of obvious clinical signs of primary joint inflammation or disease.

TABLE 7.1. THE FOUR "NEVERS" OF JOINT PLAY

1. Normal ligaments are *never* tender to palpation.
2. One can *never* palpate a normal joint capsule.
3. One can *never* palpate fluid in a normal joint.
4. *Never* manipulate a swollen, warm, or inflamed joint.

Additionally, the clinician needs to be mindful of the four "nevers" when evaluating any joint (Table 7.1).

All normal movements of joint play are performed painlessly. Pain elicited during the performance of joint play movements on examination suggests joint dysfunction. An integral component of the examination is to compare joint play movements to the contralateral joint to more accurately assess the degree of impairment. Asymmetry in joint play can indicate a dysfunctional joint, particularly if the pain emanates from that joint.

When joint dysfunction is a *sign* of more serious joint disease, the dysfunction will remain after the primary disease has resolved. It can then be a *diagnosis*, or a primary cause of residual pain, often seen after resolution of disease, which can then be effectively treated with manipulation. A continuum exists between joint dysfunction as a *sign* and a *diagnosis*, however. Only clinical experience provides the practitioner with the knowledge to determine when the *sign* of disease becomes the *diagnosis*, or the primary cause of residual symptoms following the resolution of the disease (1).

One historical clue that strongly suggests the change from *sign* to *diagnosis* has occurred is that the nature of the symptoms has changed. Rest will now improve the symptoms when joint dysfunction is a *diagnosis*, whereas during the stage of inflammation when dysfunction was a *sign*, rest made the joint stiffer. In addition, manipulation of the actively inflamed joint, when joint dysfunction is a *sign*, will exacerbate the symptoms, whereas gentle but firm manipulation of dysfunction when it is the primary cause of symptoms will not increase pain.

Further clues indicating joint dysfunction as a primary cause of joint pain are sudden onset of pain, onset following unguarded joint movement, lack of marked swelling or warmth, pain limited to one joint, palliation by rest, lack of stiffness, and pain aggravated by activity. The nature of pain with joint dysfunction tends to be sharp and occurs when the joint is in use. Often this occurrence is intermittent, but the same movement always causes the same pain and is almost always relieved by rest. When joint pain follows the resolution or dormancy of a primary joint disease, a change in the nature of pain will occur. For example, during the active phase of a primary joint disease, the athlete may find that after rest, the joint is painful and stiff. This is not seen with joint dysfunction.

Understanding etiologic factors predisposing to joint dysfunction helps in identifying these historical clues. Primary joint dysfunction commonly results from the imposing load of an unguarded movement at a joint that at the time is actively going through a normal functional movement. This is an *intrinsic* trauma to the joint. It may also follow an *extrinsic* traumatic episode involving a joint that is only mild to moderate in nature such as a sprain or strain. According to Mennell, joint dysfunction is perhaps the most common cause of residual symptoms after severe bone and joint injury and after almost every joint disease when the primary pathologic condition has healed or is dormant (1).

Joint dysfunction also occurs after joints have been immobilized in the treatment of such injuries as fractures or severe soft tissue injuries, even when the joint itself was not involved in the injury. It also results as a residual symptom following any inflammatory joint disease, such as gout or rheumatoid arthritis.

INDICATIONS AND TREATMENT TECHNIQUE

Mennell emphasized that manipulative maneuvers in treatment are designed solely to restore normal anatomic and physiologic joint

TABLE 7.2. RULES OF JOINT PLAY

1. The athlete must be relaxed.
2. The clinician must be relaxed. Therapeutic grasp must be firm and painless, yet protective.
3. One joint is mobilized at a time.
4. One movement in a joint is restored at one time.
5. In movement, one end of the joint is stabilized while the other is mobilized.
6. The extent of movement on one joint is not greater than that of the opposite side.
7. No forceful or abnormal movement must ever be used.
8. Manipulative movement is a sharp thrust that gaps the joint approximately 1/8 inch.
9. Therapeutic movement occurs when all the slack is taken up.
10. No manual treatment is ever performed in the presence of joint or bone inflammation or disease.

motion (1). Treatment with joint play manipulation is more focused on the mechanical joint play that is present only in life and absent in death. Joint dysfunction is the only pathologic condition that will respond to the manipulative treatment of joint play. Therefore, the normal range of joint play movements must be learned as well as the ranges of voluntary motion before manipulative therapy should be used. Furthermore, the proper distinction between joint dysfunction as a primary *diagnosis* versus a *sign* of underlying pathology is essential to the proper use of the manual medicine technique of joint play.

Although the movements involved with examination and treatment are, in most cases, identical, it is the purpose with which they are used that is distinct. The proficient practitioner of the joint play technique is able to distinguish between these concepts and use them appropriately and thereby not apply the manipulative movement when it is not indicated. No therapeutic maneuver should be done in the presence of joint or bone inflammation, significant hypermobility, or disease indicated by heat, redness, and swelling (1–3).

Once a restriction in joint play is noted, a manipulative movement may be exerted at this barrier. The barrier reptesents the joint dysfunction. Therapeutic movement is introduced after the laxity, or "slack" in the joint has been taken up (2). Keep in mind that the manipulative movement is a sharp high-velocity, low-amplitude thrust in the plane of joint play tested to cause gapping at the joint being treated (usually no more than 1/8 inch) (2).

The rules for the use of joint play as a treatment are outlined in Table 7.2. The assessment of the amount of play in a joint should follow these rules as well. Joint play techniques are listed in Section III of this text because they are helpful in evaluating function and stability. Treatment comes from the diagnostic setup, so that one can diagnose a problem and correct it with a joint play maneuver in a matter of seconds.

APPLICATION IN ATHLETICS

The usefulness of the joint play technique and how it relates to athletics is fairly clear when one understands the etiology of joint dysfunction. As stated previously, the three primary causes of joint dysfunction are (1) prolonged immobilization of a joint, (2) intrinsic injury of an unguarded motion at a particular joint, and (3) the result of inflammatory conditions. These three conditions occur frequently in athletics. In fact, athletes commonly complain of unexplained soreness that often resolves when the stiffness does. When joint pain is persistent after the primary condition has been treated, one can unwittingly make an inaccurate diagnosis of tendinitis or capsular strain, when the loss of joint play is the residual cause of joint pain.

Joint play is also effective in treating the extremities. Most direct manual medicine techniques apply to the spine, but joint play is focused on one joint and its intrinsic motion. Joint play can more accurately assess each

individual joint, which is handy when treating the hand or foot with its myriad number of small joints. Athletes injure the extremities more often than the spine, and return to play is a pressing issue, particularly in the more elite athletes. Joint play can identify and treat the dysfunctional barriers without worrying about causing hypermobility. Various techniques are described in Section III of this text.

Athletes respond well to joint play techniques because they can be applied to specific and subtle dysfunctions that only athletes can notice with their heightened proprioception and awareness. Elite athletes function at high efficiency, and the kinetic chain principles apply through every joint in order to execute a skill. Any breakdown along a specific chain can have an impact on the whole action. For instance, an athlete recovering from an ankle sprain may have the requisite strength and flexibility when assessed after rehabilitation; however, his or her function may still be limited. Another example is seen in athletes with foot fractures who have been immobilized for weeks, then try to compete again. The early discomfort and dysfunction are often chalked up ro being "part of the process" or "it just takes time." However, restrictions in such areas as the cuboid, tarsal navicular, subtalar joint, and the metatarsals can impact function significantly, yet be easily missed on examination. Other problematic areas of note are the fibular head, elbow, and wrist.

CONCLUSION

The injured athlete requires careful attention from the clinician to evaluate and treat specific joint dysfunction. These dysfunctions can be small, but they can disrupt athletic skill and performance and lead to disability. Treating the athlete with joint play techniques can improve range of motion and overall function, particularly in the extremities. Specific examination and treatment techniques are discussed in Section III of this text, because joint play gives one the advantage of being *diagnostic and therapeutic* at the same time.

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8

PHYSICAL MEDICINE MODALITIES

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OVERVIEW

In the practice of sports medicine, the majority of injuries are musculoskeletal in nature. Acure traumas such as sprains, strains, and contusions are commonplace. Physical medicine modalities are an established method of treatment for these injuries. Manual medicine techniques are commonly used in conjunction with these modalities to treat sports injuries. Manual Combined, the two modalities aid in the athlete's recovery from injury and speed the return to play. This chapter discusses those modalities most useful in sports medicine.

THERAPEUTIC HEAT

Superficial Heat

Superficial heat affects the skin and subcutaneous soft tissues and stimulates a reflex relaxation in deeper muscles. Treatment with superficial heat elevates the pain threshold and thereby causes a decrease in the pain response. It may also cause a sedative effect and aid in muscle relaxation. Heat-induced changes in the viscoelastic characteristics of connective tissue aid in stretching activities. Superficial heat increases metabolism and oxygen consumption of the tissues. The metabolic rate increases two to three times that of normal for every 10°C increase in skin temperature. This heat modality also causes vasodilatation, which may aggravate swelling, edema, and superficial or subcutaneous bleeding. The therapeutic range of heating is between 40°C and 45°C. Anything above this range becomes painful. The length of treatment time is important. After approximately 8 minutes, superficial heating has extended only 0.5 cm into tissues. After approximately 15 to 30 minutes, the heating has extended 1 to 2 cm deep into muscles. After the application of heat, the area may be passively and actively stretched to improve flexibility and range of motion.

Technique

Moist heat packs (hydrocolator packs) stored at 70°C to 80°C may be used. The packs are placed on the athlete and covered with several layers of towels. They are maintained for 15 to 30 minutes prior to the athlete's therapeutic exercise. Paraffin baths are made with either commercially available paraffin bath wax or by melting 7 parts paraffin with 1 part mineral oil and heating to between 52°C and 54°C. They are used for hands, feet, and elbows. The body part is dipped seven times into the paraffin bath wax, allowing each layer of wax to cool before the next dipping. The area is next wrapped with plastic and towels, treated for 20 minutes, and then unwrapped. After treatment, the paraffin bath wax is peeled off and deposited back into the vat. The area then undergoes passive and active range of motion. Fluidotherapy is a technique in which hot air between 38.8°C and 47.8°C is forced through a container of fine cellulose particles. The area being treated, such as a hand or foot, is placed in these particles for 20 to 30 minutes and then removed and treated with passive and active range of motion.

Contraindications

Superficial heat modalities should not be used in athletes with cardiac insufficiency due to

susceptibility to heat stress, in malignant tumors due to potential stimulation of tumor growth and metastasis, in areas of bleeding or hemorrhage due to potential exacerbation of the bleeding, in acute inflammation due to exacerbation of the inflammation, and in peripheral vascular disease due to increased metabolic demands with decreased blood flow causing potential anoxia and tissue damage (1–5).

Deep Heat

Like superficial heating modalities, deep heating modalities aid in improving flexibility of muscles and joint range of motion. Deep heating modalities work below the skin and superficial layers of tissue to a depth of approximately 3 to 5 cm.

Ultrasound

Ultrasound is used to treat multiple soft tissue injuries such as tendinitis, bursitis, myositis, joint contractures, and scar tissue. Ultrasound uses a frequency between 0.8 MHz and 3 MHz to deliver sound waves through the tissues. The sound waves are emitted by a piezoelectric crystal that creates vibration and produces mechanical waveforms, which are transmitted from the ultrasound unit's sound head (transducer) through a coupling medium such as gel into the tissues. The sound waves travel through the soft tissues and are reflected off the bones in an irregular pattern, which is absorbed in the connective tissues as heat. As with superficial heat, ultrasound increases local metabolism, blood flow, and nerve conduction velocity. Heating of the collagen fibers in the connective tissues elevates the pain threshold and allows for greater tissue distensibility. Scar tissue may be denatured and become more amenable to stretching using this modality. Ultrasound increases tissue temperature up to 43.5°C.

Technique

The ultrasound unit uses a continuous sound wave setting to deliver therapeutic heating. The transducer is coupled to the skin using a gel or water interface, and it is moved over the treated area for 5 to 10 minutes in a back-and-forth or circular fashion. A water medium may be used for the hands or the feet, and where the body part is immersed in the water, the sound head is placed 0.5 to 1 inch away from the skin. After ultrasound heating, the area is treated with passive and active range of motion to improve flexibility.

Phonophoresis

Acute localized inflammatory processes such as tendinitis, tenosynovitis, and epicondylitis can be treated with a corticosteroid solution (e.g., 1% hydrocortisone cream or a local anesthetic such as 1% lidocaine) coupled with ultrasound. This technique is known as *phonophoresis.* The ultrasound transducer allows deeper penetration of these medications than by topical application alone. After phonophoresis, the area is further treated with passive and active range of motion, manipulative medicine procedures, and if indicated, deep friction massage to break up scar tissue.

Contraindications

Ultrasound with or without phonophoresis is contraindicated in peripheral vascular disease because it increases metabolic activity, in acute bleeding because it stimulates greater blood flow, and over fluid-containing organs such as the eye, the heart, or a pregnant uterus (including ultrasound over the low back in a pregnant female). Ultrasound should not be used over the testis due to potential orchitis, over malignant tissue due to potential tumor growth or metastasis, over the epiphyses in children due to abnormal growth stimulation, and over nerve roots with acute radiculopathy due to exacerbation of nerve root inflammation. Ultrasound should also not be used over pacemakers; however, it may be used over metal implants such as a joint replacement. Ultrasound may be used over anesthetized skin, but care should be taken to maintain the treatment within time parameters and monitor for side effects such as swelling or erythema.

Short Wave Diathermy

Short wave diathermy is used to treat acute and chronic pain complaints such as myositis, tendinitis, bursitis, and joint contractures. It uses radio waves at 27.12 MHz to heat the subcutaneous tissues. The unit uses a capacitor or inductor to transmit radio waves, which are absorbed in and heat the tissues. The temperature in subcutaneous fat is raised approximately 15°C while muscle is raised 4°C to 6°C by either continuous or pulsed waveforms from the unit. After short wave diathermy, the area is treated with passive and active range of motion or osteopathic manipulative medicine (OMM) to improve flexibility and range of motion.

Contraindications

Short wave diathermy is not used over fluid-containing organs such as a pregnant uterus, the eyes, or the head due to increased metabolic activity; it is not used over pacemakers due to possible interaction with the setting of the pacemaker. It is not used over areas of acute inflammation due to exacerbation of the inflammation, or over areas of acute infection due to exacerbation of the infection. It is contraindicated in joint replacements due to heating of the metal. It is contraindicated in cancer due to possible tumor growth and metastasis. It is not used over the epiphyses in children due to possible abnormal bone growth. It is contraindicated in athletes with cardiac problems due to potential increase in cardiac demand.

Microwave Diathermy

Microwave diathermy uses electromagnetic radiation at frequencies of 9.15 MHz and 24.50 MHz to heat subcutaneous tissues. Its effects are similar to short wave diathermy including raising subcutaneous fat temperature 10°C to 12°C and subcutaneous muscle temperature 3°C to 4°C. It has a lower depth of penetrance than short wave diathermy and therefore should not be used on deeper structures, such as hips or deep low-back musculature. It is primarily used around superficial muscles and joints. The area is treated for 15 to 30 minutes and is followed by passive and active range of motion and manual medicine procedures.

Contraindications are the same as with short wave diathermy (1–5).

THERAPEUTIC COLD

Therapeutic cold or cryotherapy is a technique used to cool the skin and subcutaneous tissues in an effort to obtain a therapeutic physiologic effect. Athletes feel a change in sensations during the treatment including cold, burning, warmth, aching, numbness, and tingling. The therapeutic effect is achieved by decreased blood flow, decreased metabolism, and decreased pain fiber conduction to the area treated. Tissue flexibility and spasticity may also be decreased. The area is generally treated for 15 to 30 minutes. Vasoconstriction usually occurs within 15 minutes. However, vasodilatation may occur after 15 minutes if the tissues are cooled to −10°C.

Cold Packs

Cold packs are either bags of ice chips in water or silicone gel packs that are refrigerated to −5°C. They are moldable to the shape of the body part being treated. Packs are placed on the skin for 10 to 15 minutes for superficial cooling, and for 15 to 20 minutes for deeper cooling. If the athlete is too sensitive, then a thin towel may be placed between the pack and the skin. If cooling is maintained for too long, the possibility of frostbite exists. Their thermal properties give ice packs a greater cooling effect than commercially available silicone gel packs. Therefore, if anesthesia is desired, a slurry of ice in water is preferential to a gel pack. A variation of this technique is to soak a terry cloth towel in an ice and water slurry, wring out any excess water, and then wrap the body part with the chilled towel. A disadvantage is that the towel needs to be changed every few minutes as it warms up. This technique is best used if no other cryotherapy equipment is available.

Ice massage is a common technique used in acute sports injury and on the sidelines. Ice is rubbed directly on the skin of the injured area for approximately 5 to 10 minutes to develop anesthestic and anti-inflammatory effects. An ice rub may be followed by deep friction massage or prolonged stretch to improve muscle spasm, trigger points, and tendinitis. An "ice lollipop" may be made before a sporting event by placing a tongue depressor and water in a Styrofoam cup and freezing it. The lollipop is kept frozen in a cooler until needed on the sidelines.

Ice baths are commonly used to cool extremities. A tank is filled with ice and water with the temperature maintained between 13°C and 18°C. The body part is immersed in the water and maintained for approximately 20 minutes to obtain optimal cooling. This treatment is followed by passive and active range of motion. A variation of this technique is the cold compression sleeve unit, which is commercially available. To use this unit, the clinician places the extremity in a sleeve that has an inner core where cold water is pumped and circulates. The sleeve cools and compresses the body part. This aids in the treatment of edema caused by acute sprains or contusions.

Contraindications

Cold modalities are contraindicated in athletes with impaired sensation due to anesthesia and possible tissue damage, in impaired circulation due to the cold effect of vasoconstriction and decreasing circulation, in nerve trauma where the nerve may be regenerating due to decreased nerve conduction, in cardiac conditions, and in Raynaud's disease and other diseases with cold sensitivity, such as cold urticaria. Open wounds and incision lines may be treated on an acute basis with cold, but after approximately 48 hours cold should be withheld due to the risk of vasoconstriction decreasing the healing (1–5).

ELECTRICAL STIMULATION

Electrical stimulation is a physical medicine modality that uses electrical current to decrease pain and improve muscle tone. In sports medicine, the most common forms are transcutaneous electrical nerve stimulation (TENS) and electrical muscle stimulation (EMS).

Transcutaneous Electrical Nerve Stimulation

Transcutaneous electrical nerve stimulation (TENS) is a technique for pain control. It is based on the gate theory of pain whereby large-diameter afferent fibers are stimulated with a frequency of 1 to 120 Hz and a pulse width of 50 to 300 microseconds and 10 to 50 mA. These electrical impulses close a gate in the nerve fibers that blocks pain stimulation to the higher central centers and thus blocks pain perception. The stimulation is delivered by a small, battery-powered stimulator through electrodes to electrode pads placed over the skin around the area of pain complaints. TENS is used for acute pain complaints, such as acute trauma or acute recurrent pain as seen in arthritic changes.

This modality is contraindicated in athletes with demand-type cardiac pacemakers. TENS should not be used around the neck due to stimulation of the muscles and/or carotid sinus. It should not be used over irritated skin due to the adhesive pads aggravating the irritation. An athlete can wear this device for several hours at a time, but its effectiveness decreases if it is worn continuously for more than a few days.

Electrical Muscle Stimulation

Electrical muscle stimulation (EMS), or neuromuscular electrical stimulation, is used to decrease muscle spasm, increase muscle mass, and strengthen muscle, especially after prolonged immobilization. EMS is also used to facilitate muscle education as well as increase range of motion. It is similar to a TENS unit in appearance. However, an EMS unit uses 1000 Hz for low voltage and 4000 to 4100 Hz for interferential current. Both techniques stimulate the peripheral motor nerves causing peripheral muscle stimulation, or they directly stimulate peripheral muscles. The electrodes are placed over the area of muscle spasm or muscle weakness.

The treatment session is generally for 20 to 30 minutes (1,5–7). EMS is contraindicated over demand-type pacemakers, over the carotid sinus, and in pregnancy and cardiac conditions.

Iontophoresis is the use of a corticosteroid or anesthetic solution coupled with electric stimulation. This is similar to phonophoresis, and its uses are similar.

HYDROTHERAPY

Hydrotherapy is a term used for several devices that are used to treat injuries and aid in the rehabilitation of the athlete. Several factors contribute to the effectiveness of hydrotherapy. The warm water creates a relaxing environment, which helps to reflexively decrease muscle spasm and decrease discomfort. The buoyancy of the water helps to support the trunk and limbs, and decrease gravitational forces allowing easier active range of motion in gravity-eliminated postures. The density of the water also creates resistance for low-level, active-resistance exercise training. Hydrotherapy is used for joint mobilization, in treatment of muscle spasm, in progressive resistive exercise training, in burn treatment, and in the treatment of athletes with arthritis.

WHIRLPOOL

A whirlpool is a tank of heated water that circulates through the tank with a mechanical pump. A whirlpool treatment consists of immersing an extremity or the body up to the chest in the circulating water. Upper limb treatment temperature is generally between 37.8°C and 40.6°C. Lower limb treatment temperature is generally between 37.8°C and 38.9°C, although temperatures up to 46°C may be tolerated. The athlete is treated for 5 to 20 minutes. It is believed that the circulating water causes mechanical stimulation to the skin, which causes a reflex anesthetic effect and a decrease in muscle spasm. The athlete may undergo passive and active range of motion exercises in the whirlpool or after completing the whirlpool treatment.

Hubbard Tank

Hubbard tanks are used for immersing the entire body up to the neck. The temperature is generally between 36.7°C and 37.2°C, or between 37.8°C and 38.3°C for more vigorous heating. The athlete is treated for 10 to 20 minutes, and may undergo passive and active range of motion during or after the Hubbard tank treatment. Due to the extensive immersion in the Hubbard tank, there is a contraindication to treating athletes with cardiac conditions and circulation problems.

Pool Therapy

Pool therapy can offer a higher level of conditioning exercise than exercise performed in either whirlpools or Hubbard tanks. The athlete is placed in a therapeutic pool with water up to the chest and he or she either stands on the bottom of the pool or is suspended in the deep end of the pool. Training is given in active range of motion and conditioning exercises. Various paddles and inflatable exercise devices add a greater resistance effect to the athlete's exercises. Pool therapy allows an athlete to move from range of motion exercises to progressive resistive exercise in his or her rehabilitation.

Contrast Baths

In this variation of the whirlpool technique, the athlete is placed alternately in warm water between 38°C and 44°C for 10 minutes and in cold water at 10°C to 15°C for 1 minute, then alternated between the warm water for 4 minutes and the cold water for 1 minute, for a total treatment time of 20 minutes. This technique benefits muscle strains, joint sprains, and edema. Contraindications include peripheral vascular disease and the contraindications previously noted in the sections on therapeutic heat and therapeutic cold (1–3,5).

BIOFEEDBACK

Sports medicine biofeedback may be used as an aid to training the athlete in the relaxation of chronic tight muscles. The technique is to

use sensing electrodes over the muscles in question. The electrodes are attached to the electromyographic biofeedback machine, which then gives an auditory or visual readout of the athlete's muscle activity. As the athlete relaxes, the activity is seen to diminish (1,8).

TRACTION

In the athlete, traction is primarily used in the axial skeleton to stretch the paraspinal soft tissues and separate the vertebrae. This typically decreases pain and muscle spasm while increasing flexibility and range of motion. Traction has been traditionally used with bulging or herniated discs in the axial spine. It may also improve blood supply by decompressing the intervertebral joint and reducing vascular restriction. Thoracic vertebral joints are stabilized by the thoracic cage and develop disc herniations less often than the cervical or lumbar spine, so traction is used most often with lumbar and cervical disc injuries.

Cervical Traction

Cervical traction is delivered manually or mechanically. Manual cervical traction is often used at the start of manipulative medicine procedures in an effort to improve muscle spasm, joint contractures, and somatic dysfunction. The athlete is placed supine with the neck flexed at approximately 15 to 20 degrees, and a longitudinal force is then placed through the neck with the clinician's hands either under the occiput or under the occiput and over the forehead. The traction is maintained for 1 to 2 minutes, allowing the muscles to relax. Mechanical cervical traction is performed either in sitting or supine position with the head flexed at approximately 15 to 20 degrees, and a longitudinal force is directed through a harness around the head to the weight or weight machine. Static cervical traction is delivered for approximately 15 minutes. Intermittent traction is delivered with a cycle of the traction being on for 10 to 60 seconds and off for 5 to 20 seconds. It is recommended that the traction force be started low at approximately 3 to 5 lb so that the athlete may become accustomed to the traction device. This is then increased progressively to approximately 15 to 18 lb. Cervical traction is contraindicated in cases of cervical instability such as spinal fracture, ligament or muscle rupture, metastatic disease, osteoporosis, rheumatoid arthritis, and spinal infections.

Lumbar Traction

Lumbar traction is indicated in cases of lumbar muscle spasm, joint contractures, and somatic dysfunction. It may be performed alone or prior to manipulative medicine procedures. As with cervical traction, lumbar traction decreases muscle spasm and increases soft tissue distensibility and joint mobility.

Manual lumbar traction is difficult to perform due to the size of the athlete's trunk versus the arm strength of the physician. However, two-person manual traction may be performed with the athlete lying supine with the hips and knees flexed. One person then distracts the pelvis by holding the superior aspect of the iliac crests and pulling caudally. The other person holds the athlete under the axillae and pulls cephalad.

Lumbar traction is performed with a mechanical device with greater efficiency. A number of mechanical units are available. The units use forces of approximately one half the athlete's weight to cause distraction of the lumbar region. Generally, these forces are between 60 and 150 lb. Most of these units are designed so that the athlete lies supine with the hips and knees flexed at 45 to 90 degrees, and traction is delivered in a caudal fashion. A variation of mechanical traction is gravity lumbar reduction traction in which the athlete is placed on a movable table. The athlete is secured with a strap across the trunk, and then the table is rotated so that the athlete is supported in a vertical position. This gives traction caudally using the weight of the lower half of the athlete's body as the traction force. Gravity inversion traction is another variation in which the athlete is supported at the ankles, and the table is then rotated so that the athlete is inverted. The traction force then

becomes the upper half of the athlete's body with gravity pulling downward, rostrally through the athlete's trunk. Contraindications to lumbar traction are the same as for cervical traction (1,4–6).

LIGHT THERAPY

Light therapy is a physical medicine modality that is often used in sports medicine. Ultraviolet radiation at wavelengths of 2000 to 4000 Å may be used to increase vascularization at wound margins, and at 2537 Å for bactericidal effects. This therapy may aid in wound healing following trauma. The dose is a one or two minimum erythema dosage concentration, or enough to cause first-degree erythema around the skin. The athlete is treated two to three times per week. Low-powered cold lasers such as helium-neon gas lasers (wavelength of 632.8 nm) or gallium-arsenide semiconductors (wavelength of 904 nm) are reported to be beneficial in pain complaints such as muscle strains, joint sprains, and headaches. The lasers are directed over acupuncture points or painful areas. Acute injuries are treated with a dose of 0.05 to 0.5 J/cm^2, while chronic conditions are treated at 0.5 to 3.0 J/cm^2. Athletes are treated three to six times. Although this information has been published and the equipment is available, at the present time more study is needed before this is a practical routine approach for these conditions (5,7).

CONCLUSION

Physical medicine modalities are designed to reverse damaging effects on soft tissues, decrease pain, resolve swelling, and improve mobility. These therapeutic benefits will allow the athlete to return to his or her sport more rapidly. The modalities alone or in combination with other appropriate interventions such as nonsteroidal anti-inflammatory drugs, modified exercise protocols, and manipulative medicine techniques will accomplish the goals of recovery efficiently.

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9

MASSAGE AND SOFT TISSUE MANIPULATION

RICHARD RADNOVICH

OVERVIEW

Massage as a therapeutic modality has been written about and used for hundreds of years (1). In the athletic community, massage has been popular for decades (2). Recently, awareness of its use in athletes has increased, and a newer form, "sports massage," has been coined. Sports massage maintains a well-established place in the care of athletes in other countries, particularly those of Eastern Europe. Most professional sports teams in the United States have massage therapists on staff. The underlying component strokes of sports massage are derived from traditional massage therapy strokes; it is the application and timing of these techniques that vary. Dynamic stretching through passive range of motion is frequently used in sports massage as a modality, although it is not generally found in other traditional massage techniques.

Sports massage has been promoted as beneficial to athletes for many reasons, such as pain relief, relief of muscle tension, improved athletic performance, improved flexibility, and reduced delayed-onset muscle soreness (3,4). Proposed mechanisms for its salutary effects include improved circulation, enhanced elimination of wastes, decreased motor neuron excitability, and lengthening of both tendons and muscle tissue. Rigorous trials have not supported these claims, however (5,6).

VALIDITY

The appeal and use of massage in the athletic community are well documented (3,6). Demonstrating the physiologic basis for these perceived benefits has proved more daunting a task. There are no large studies on athletic populations to demonstrate a competitive edge from massage or any study showing reduced injuries or better performance in a real-world setting. Such studies would, of course, be extremely difficult to design and execute. Physicians must rely on small sample sizes and studies that look at surrogate end points (e.g., decreased lactate level, increased blood flow, or reduced fatigue of an isolated muscle group as indicators of better performance). Few of these studies, however, are done on athletic populations. Evidence exists supporting the use of massage for depression and anxiety (7,8).

In addition, there is no scientific evidence claiming that sports massage is physiologically different from or superior to traditional massage. The term *sports massage* is more of a philosophical construct rather than a discipline based on science. Many of its benefits are plausible without supportive data, and few side effects or negative outcomes have been reported.

Despite these shortcomings, the physician who treats athletes should have a familiarity with massage techniques so that he or she can communicate with athletes and therapists. In addition, it is probable that the physician who can adequately perform some soft tissue techniques will have a hands-on connection with athletes that enhances his or her image, although this too remains unstudied. Because of its safety, massage deserves a continued place in the care of the athlete while the medical community awaits better methods to study its benefits (7).

PRINCIPLES

A fundamental premise of sports massage is that the treatment should be tailored to the athlete's stage of training (5). In sports massage philosophy, techniques used with a runner training for a competition, for example, will be different from the massage used for a runner recovering from a race, which again will differ from the massage techniques used in rehabilitation from injury. The various phases of massage treatment have been referred to as "pre-event," "post-event," "recuperative," and "rehabilitation." Others have described these phases as "training," "preparatory," "intermediary," and "warm-down" (5). Although we discuss the application of techniques for specific phases, none of this is validated through studies and no one really knows if, for example, effleurage is superior to petrissage before a race.

The components of massage therapy (i.e., the individual strokes) have been used for centuries. Over the years they have been "repackaged" in many different ways, sometimes with slight additions, modifications, or different emphasis. Massage has been called "Rolfing," "Hellerwork," myotherapy, muscle therapy, or sports massage, to name a few. None of the techniques has any rigorous scientific basis to convincingly show either the physiologic basis for the technique or an objective standard for success.

Several of the basic massage strokes are typically performed with some form of lubricant, usually oil or lotion, to prevent friction of the practitioner's skin on that of the athlete. There are many commercially available products but usually any oil will suffice. The strokes demonstrated in this chapter are shown without the use of lubricant, as performed by most physicians.

TYPES

The components of massage are classically described as the following five types (5,9):

1. Effleurage
2. Petrissage
3. Friction
4. Tapotement
5. Vibration

Effleurage

Long, slow stroking motions performed with the palmar surface of the hands characterize effleurage. It is usually executed distal to proximal in the extremities and generally parallels the tissues. Pressure is applied lightly at first and can get gradually deeper. This stroke is usually used at the beginning of the treatment, to distribute lubricant and establish contact with the athlete, and at the conclusion of treatment (10). Among the purported benefits for this stroke, when applied with light pressure, are reduction in muscle tone, generalized relaxation, and relief of muscle spasm (9). With deeper pressure, the supposed benefits include accelerated blood and lymph flow. Rapid strokes are supposed to be stimulating, and increase muscle tone. A deep, focused variation of this stroke (deep tissue, Rolfing, Hellerwork) includes applying abundant pressure along the fascial planes, within and between muscle groups, without using any lubricant. Pressure is applied with much smaller surface areas, for example, the fingertips, knuckles, and elbows, allowing greater force to be applied and helping to release adhesions that limit range of motion (3). This process can be painful due in part to the skin friction created between athlete and clinician.

The use of effleurage in sports massage includes different variations depending on the situation of the athlete. Rapid superficial strokes are usually applied before an event (pre-event) to ready the muscles for activity. Use of lubricants is discouraged because of potential impairment of sweat gland efficiency. After competition (post-event), deeper, slower strokes are applied without lubricant (6). During the recuperative phase in between days of competition or practice, both deep and superficial pressures are used (6). For extended rehabilitation, deep, focused strokes at sites of restricted range of motion can be applied (Fig. 9.1).

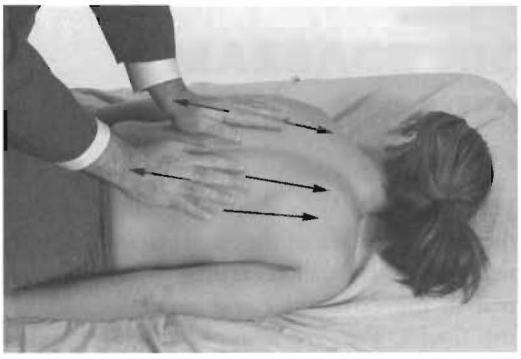

FIGURE 9.1. Effleurage on the athlete's back, using long, firm strokes.

Petrissage

Petrissage is characterized by squeezing of soft tissues for therapeutic benefit. It is performed by squeezing, pinching, or kneading the muscles and fleshy regions of the body (3,9). Tissues can be picked up in one hand and transferred to the other. This process is repeated the length of the region being worked from distal to proximal (Fig. 9.2A). Another petrissage technique includes pushing the skin and underlying tissues together using the heel of the hand or the dorsum of a loosely closed fist (Fig. 9.2B).

Compression is a form of petrissage frequently used in sports massage (3). The practitioner applies the heel of the hand or a fist to rhythmically compress tissues, in almost a pumping fashion. Pressure is applied perpendicular to the tissue. The technique can be performed with the muscle in a neutral or contracted position (Fig. 9.2C).

Another petrissage technique involves applying a compressive force to a relaxed, shortened muscle, then lengthening the muscle underneath the compression with only minimal movement of the force. An example is applying compression to the hamstrings with the knee in 90 degrees of flexion. The knee is slowly brought to neutral with resistance (Fig. 9.2D).

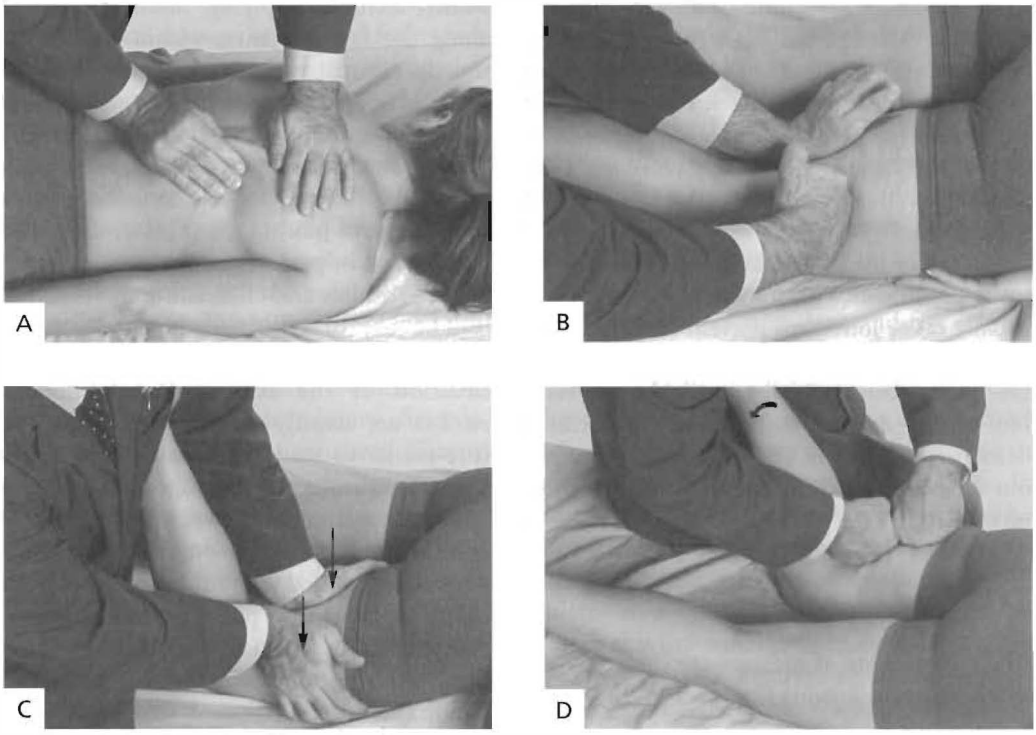

FIGURE 9.2. Petrissage techniques. **A,** Kneeding and picking up the soft tissue. **B,** Compression of the hamstrings with the fists or open hand in a rhythmic pumping motion. **C,** Pushing and pulling the hamstring soft tissue. **D,** Compression on the hamstrings with the knee flexed and introducing passive range of motion.

The use of petrissage in pre-event treatment includes rapid superficial strokes that gradually get deeper and slower. The purpose is to increase blood flow and prepare muscles and connective tissue for competition (11). The postevent technique is very similar to pre-event strokes and assists in "pumping" excess metabolic wastes from muscles (6,9). The use of petrissage in recuperation and rehabilitation is similar to the techniques previously mentioned.

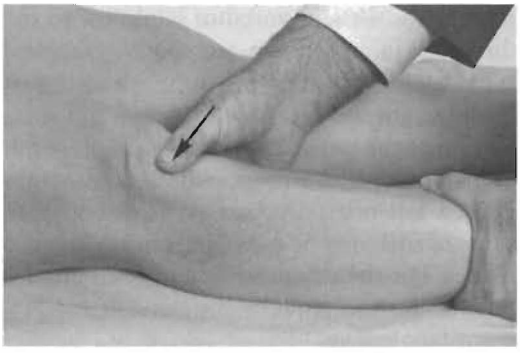

FIGURE 9.3. Cross-friction massage to the patellar tendon and supraspinous ligament.

Friction

Cross-friction massage involves circular or linear movements perpendicular to the alignment, or "grain," of the muscle or connective tissue fibers. It is also referred to as cross-fiber friction, transverse friction, and the Cyriax technique. The stroke is usually applied with the heel of the hand, tips of the fingers, or knuckles. Small instruments with soft tips can also be used. Generally, pressure is initially light then grows stronger during the treatment. It can become quite deep, and athletes usually have some discomfort, especially if the stroke is applied to damaged or inflamed tissues. When applied in a focal area, this stroke is supposed to create a localized area of hyperemia and increased metabolism that can accelerate or amplify the healing process (9,12).

Others believe that friction eliminates adhesions between tissue planes. Noted British orthopedic surgeon James Cyriax believed that certain connective tissue injuries would not heal adequately without applying this technique (13). He described using friction for 5 to 15 minutes on one spot (e.g., the anterior talofibular ligament or patellar tendon). Cross-friction is used in sports massage principally in the rehabilitative phase, particularly for ligament injuries and tendinosis. However, a more generalized form of friction can be incorporated into other phases of sports massage.

Friction massage is usually performed without a lubricant. The force is applied perpendicular to the muscle or connective tissue, in a back-and-forth motion (distal to proximal) usually with more pressure applied in the distal motion. The tissues being treated should be under some physiologic tension (i.e., the tissues should not be in a shortened position). Massaging is continued until erythema develops (Fig. 9.3).

Tapotement

Sometimes called "cupping" or "hacking," tapotement describes a rapid, rhythmic, percussive striking of the body with the hands (9). The palms of the clinician's hands may be used in a slightly concave position (cupping) (Fig. 9.4). Most physicians will know this form of tapotement as the technique used by respiratory therapists to aid in the elimination of lung secretions in athletes with pulmonary disease. Another type of tapotement uses the ulnar aspects of the hand, either open or closed, in a hacking or pounding stroke. Tapotement is generally used on large, fleshy areas such as the back, buttocks, or posterior aspect of the lower

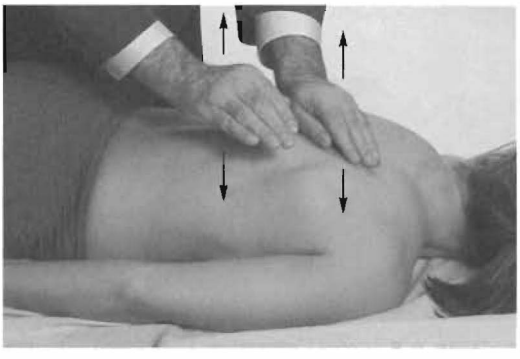

FIGURE 9.4. Tapotement, cupping and hacking on the back, is done forcefully and rhythmically.

extremities. Less importance is placed on the direction in which the technique is applied (i.e., distal to proximal) than in other strokes. It is thought to cause vasodilatation and stimulation of the muscles and is used in pre-event preparation for this purpose (6,9). This stroke is used less often after an event due to concerns of irritating or damaging already stressed tissues. For the recuperative and rehabilitative phases, the technique is the same as for pre-event tapotement.

Vibration

These motions range from superficial, high-frequency oscillations with small amplitudes (similar to friction) to literally shaking an entire extremity. This stroke is usually applied in a precise manner with the fingertips or, more generally, with the entire volar surface of the hand.

The fine oscillatory motions are presumed to stimulate nerve function and are frequently performed over major nerve pathways, parallel to the direction of the nerve (9). Larger oscillations are believed to stimulate muscle functioning and can be performed in a more general fashion either parallel or perpendicular to fibers (Fig. 9.5) (9).

Shaking the entire muscle or muscle group is performed by grasping the muscle or muscle group being treated and moving the tissues in short, back-and-forth motions. Additionally, the wrist or ankle can be grasped and shaken. These shaking motions can be performed in a transverse plane (i.e., front-to-back oscillations),

FIGURE 9.5. Coarse vibration over biceps tendon and muscle.

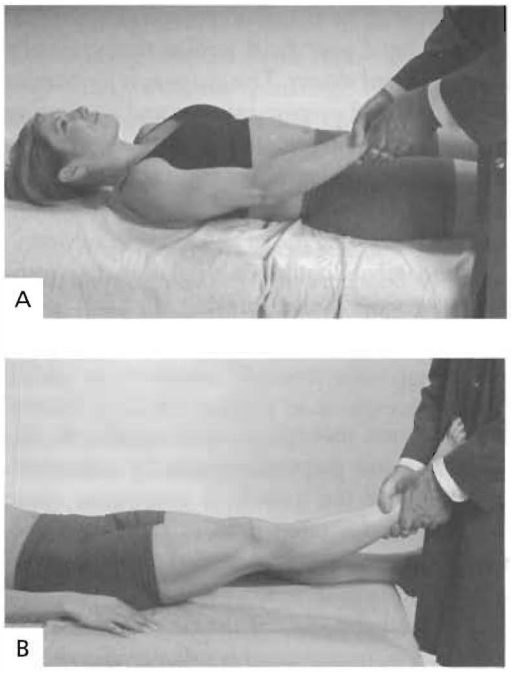

FIGURE 9.6. Shaking of upper extremity in the transverse plane **(A)** and of lower extremity in coronal and axial planes **(B)**.

coronal plane (side-to-side) or axially (rotational movements) (Fig. 9.6A and B).

Before an event, the clinician uses small oscillations over muscle bellies or nerve pathways to stimulate their functioning. Rapid shaking of large muscle groups or extremities is also used to stimulate muscle functioning and also to keep larger muscle groups relaxed and ready to perform (6,7). After an event, shaking large muscle groups and extremities is thought to prevent cramping and keep the muscles and extremities loose. During the recuperative and rehabilitative phases of training, vibration is used following bouts of exercise similar to postevent use.

Stretching

Although not a part of the five basic strokes, stretching remains an important component of care of the athlete and is often incorporated into sports massage treatments (14). Simple passive range of motion stretching involves taking the

muscle group to its testrictive barrier (the end of its passive range of motion) and holding the position for a length of time, usually 10 to 30 seconds. Chapter 12 discusses stretching in more detail.

Other Soft Tissue Techniques

Other manual techniques can be applied to soft tissue therapeutically to help prevent athletic injuries. Proprioceptive neuromuscular facilitation (PNF) is an effective technique that is similar to muscle enetgy or postisometric relaxation (PIR). Both of these techniques are popular forms of stretching with athletes. The crux of the treatment is to move a body part to the end of its passive range of motion (the restrictive barrier) and then have the athlete gently push against the isometric resistance of the clinician. The athlete's force is applied toward the "freedom of motion" or the neutral position. That is, the muscle being stretched is contracted against equal force. In a variation of this technique, the athlete applies force in the direction of the stretch; that is, the antagonist of the muscle being stretched is contracted isometrically against equal force. In both of techniques, the practitioner "takes up the slack" in the muscle by moving to the new barrier (or end of passive range of motion). This is typically repeated three times or until no further improvement is achieved with consecutive stretches.

In muscle energy, it is taught that the relaxation after an isometric contraction allows greater elasticity of the tissues. In PNF patterns, the contraction of a muscle leads to reciprocal relaxation of its antagonistic muscle and both claim a "resetting" of the Golgi tendon apparatus. Published data over the past decade, however, do not support this claim. Nonetheless, the effectiveness and popularity of these techniques remain steadfast, as our fundamental understanding of muscle physiology changes.

MUSCLE SPASM

Muscle spasms in the athlete merit special attention. Spasm is usually a result of anothet noxious stimulus or injury that the muscles are protecting. Spasm restricts motion at the affected joint and therefore is a physiologic response to injury by which the body attempts to limit range of motion so that no further injury can occut. Direct injury or stimulus to the spastic muscle may lead to primary spasm as well. Generally, muscle spasms are secondary to another cause.

Standard treatment for muscle spasm consists of ice, passive stretching, and fluid and electrolyte replacement. Two other methods should be mentioned. First, using PNF, PIR, or muscle energy techniques, followed by passive stretching, frequently curtails spasms. The antagonist of the spastic muscle is contracted against isometric force for 5 to 15 seconds, followed by stretching the spastic muscle to its new end point. This pattern is repeated until the spasm resolves or until no further imptovement is achieved.

A second method that is used less often but is still quite effective is sharply slapping the skin overlying the affected muscle with an open palm. The purpose of the slap is to create a sharp, stinging sensation on the surface of the skin. This is followed by passive stretch of the spastic muscle. The working theory is that the sharp slap temporarily interrupts nociceptive signals or acts as a counterirritant, allowing for momentary relaxation of the muscle (Fig. 9.7).

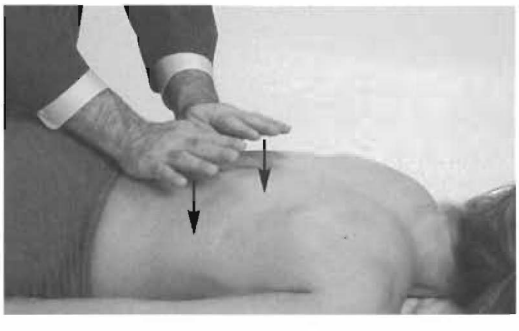

FIGURE 9.7. Open-palm slap to the lumbar erector spinae musculature.

CONCLUSION

Sports massage is widely accepted and used as an adjunct for treatment of injury and pre-event preparation. Several different forms exist, which allows for greater specificity in serving the needs of an athlete. Because massage is unlikely to cause harm, and has been used by athletes for decades, it deserves a place in the manipulating clinician's armamentarium. Sports massage needs more research to elucidate its therapeutic advantages and mechanisms of action in order for it to gain widespread acceptance in mainstream medicine.

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SECTION

II

EXERCISE APPLICATIONS

10

EXERCISE PRINCIPLES

SHAWN KERGER

OVERVIEW

Osteopathic philosophy and practice is truly at its pinnacle when dealing with exercise principles in the athlete. The mechanism at work (or play) as envisioned by Andrew Taylor Still in 1874 is in full stride and can be appreciated for its inherent wisdom and grace. Today, we study and expound on the principles established decades and even centuries ago by thinkers who observed phenomena on a larger scale to explain that which they could not see, but whose intuition told them must be present and thus created new facets and fields of medicine.

The three principles proposed by A. T. Still, which the faculty of the Kirksville College of Osteopathy and Surgery restated in 1953, are as follows (1):

1. The body is a unit.
2. Structure and function are reciprocally interrelated.
3. The body is self-healing.

Such concepts are uniquely suited to exercise and sport. They have tremendous implication in the diagnostic and therapeutic aspects of sports medicine.

More modern applications of these principles are easily seen both in the laboratory as well as on the playing field. Recent trends in exercise have largely focused on the principle of body unification: Pilates, dynamic lumbar stabilization (or core stabilization) training, yoga, and Tai Chi are all variations on this theme. Kinetic chain and sequencing studies in biomechanics laboratories across the country seek to quantify and qualify athletic skill. The human body's structure–function reciprocity and self-healing nature have been proved by the laws pertaining to its response to external and internal stimuli by structural change for some time now. These principles have advanced beyond the interactions of muscle and bone and currently incorporate neuron complexes, individual cellular structure, and proteomics into their milieu.

In an effort to facilitate freedom of thought and adaptation to clinical scenarios, one should focus on the study of normal functioning and only then on abnormal situations. By not classifying disease states as separate entities but rather as a misguided or an improper expression of a healthy physical state, the practitioner can more easily be of a frame of mind to find the health contained within the athlete.

BONE/JOINT/LIGAMENTOUS STRUCTURES

The skeleton tells a history of the body's formation: forces of tension and compression mold the shape and density of bone from its mesodermic origin. Tubercles are the result of tensile forces, such as the quadriceps has on the tibial tuberosity and the sartorius on the anterior superior iliac spine. Grooves are created by the growth of bone around a compressive force, such as the bicipital groove, or when tension around a future groove creates opposing ridges, such as in the groove for the radial nerve, which is bordered by the lateral and medial heads of the triceps. Subsequent adaptation to force follows Wolff's law: Bone is resorbed where force is not present and concentrated in areas of increased force (5).

By virtue of its inherent structure combining type I collagen and the minerals contained within bone matrix, compact bone has tremendous resistance to both compressive and tensile forces. Bone's resistance to tensile strength along its grain is superior to that of white oak, and its compressive resistance is more than 150% that of granite. Only steel has superior resistance to both compressive and tensile force (2). Much of bone's strength can be lent to its inherent adaptation of the principles of *tensegrity*—an architectural system in which structures stabilize themselves by balancing the counteracting forces of compression and tension (3). The chemical structure of hydroxyapatite (the cornerstone of bone formation) is a primary structure in tensegrity—the icosahedron. This principle has vast implications of which we are just recently becoming cognizant, and we will examine some of these later in the chapter.

Tendons and ligaments contain dense, collagenous tissue arranged in a linear fashion and thereby resist motion specifically in one particular plane in order to best serve their function. These fibers do not run independently of each other, but rather intertwine to better spread the force more diffusely across the entire structure, decreasing the chance of failure (4). Ligaments and tendons also serve a feedback function to help with proprioception, that is, the sensation of joint position that is critical to force generation and control.

Joints and their capsules similarly affect proprioception and the accordant neuromuscular adaptations. Although two of the three major types of joints are considered immovable by most anatomists (including the cranial sutures), further studies are demonstrating motion at these joints, albeit extremely small motions (5). With our focus on the athlete, this review will concentrate on the synovial joints, as these provide the greatest degree of motion.

Synovial joints generally have reciprocating surfaces, such as the cup-and-ball arrangement seen in the hip, and the bony structure strongly dictates the function of the joint (although the reverse situation might exist quite frequently during development, and certainly exists during adult life, as is demonstrated in osteoarthritic knees). The shallower the articular arrangement, the more reliant the joint is on soft tissue stabilization to control the expanded range of motion (ROM) available. An articular capsule filled with synovial fluid sandwiched between articular cartilages, usually hyaline, surrounds the joint.

Proprioceptive and pain output from the joint comes from the capsule, the surrounding ligaments, and the periosteum—not from the articular cartilage. It is this capsule and the joint's surrounding ligaments that respond with pain to a shear force, or more commonly, distension. This is why arthritic pain worsens with incoming storms or cold weather—the hydrostatic pressure inside the joint changes at a slower rate than the barometric pressure outside the joint (Fig. 10.1A and B).

The maintenance of intra-articular fluids is critical for several reasons: nourishment of the joint surfaces, cellular transport, lubrication, and cushioning. The synovial fluid carries with it not just the cells for maintenance of the joint, but also metabolic nutrients and waste products. Particulate matter, however, must be phagocytized. The very structure of synovial fluid (mostly hyaluronic acid) allows for cushioning and lubrication of the joint, and as one ages, this fluid decreases in volume and viscosity. This degeneration leads to a greater likelihood of chondral injury with a concomitant decrease in healing response. Hyaluronic acid supplements, both oral and injectable, have shown to help with osteoarthritic pain, but they do not seem to restore the articular cartilage.

Arthrokinetic responses are neuromuscular responses to joint position and output, and are a delicate and interwoven web of data processed by the central nervous system (CNS). These responses are separate from stretch reflexes, although some of the same nervous pathways are used. The four nerve types responsible for the afferent information from the joint are the following (6):

1. Globular (static and dynamic mechanoreceptors)

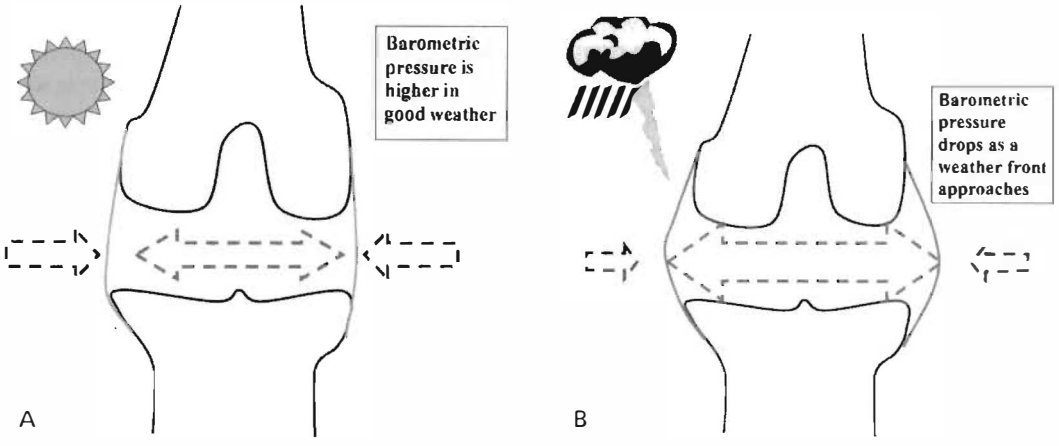

FIGURE 10.1. Effect of barometric pressure on a synovial joint. **A,** When the pressure is high (or after a period of acclimatization), the barometric pressure prevents distension of the capsule. **B,** Lower barometric pressures fail to counter internal joint pressure, allowing the capsule to distend.

2. Conical (dynamic mechanoreceptors)
3. Fusiform (mechanoreceptor)
4. Plexus (nociceptor)

These impulses are conducted to the spinal cord and interface with the data from the muscles, tendons, and ligaments serving not just the same joint but also the surrounding joints. This integration allows for a snapshot of the body's position for the CNS, on which it can act appropriately. When a capsule or ligament becomes stretched beyond its programmed length, or when motion occurs too quickly, two responses occur:

1. Inhibitory signals are sent to the agonist muscle responsible for loading the joint in the plane in question.
2. Stimulatory signals are sent to the antagonist musculature.

Thus, if the anterior hip capsule should be stretched more than its programming allows, the gluteal muscles will receive an inhibitory signal to reduce the forces acting on the capsule, while the hip flexot muscles will be stimulated. This is the case in both healthy and dysfunctional states, with the difference being *when* this process is activated.

Flexibility is the ability to move through a physiologic ROM. There are two types of stretches that can increase flexibility: elastic (temporary) and plastic (more permanent, but can still regress unless ROM is maintained). Joint ROM is primarily reduced due to connective tissue (muscle, ligament, tendon, or capsule). Muscles adapt their length more readily than their encasing fascia and attaching tendons, *unless* dysfunctional inputs to the muscle prevent adaptation. Therefore, most efforts to improve flexibility focus on plastic deformation of the connective tissues, and well-established techniques exist in the repertoire. Consideration of dysfunctional input to muscles will increase the success rate of practitioners dealing with recalcitrant or recurrent ROM problems in athletes.

MUSCLE

Similar to bone, muscle adapts its physical structure to the demands being placed on it. These structural changes occur at a macroscopic level with hypertrophy and increased tonicity of an exercised muscle, and also, to a limited degree, microscopically with regard to fiber type. The latter change reflects the *type* of demand repetitively placed on a muscle. It has been well documented that hypertrophy occurs in animal models to a *much* greater degree with

chronic loading of fibers than with the intermittent loading consistent with human exercise (7). Although initially classified by Engel (8) in 1962 as being one of two types of fibers, muscle fibers currently can be classified into one of seven fiber types, using pH sensitivity of myofibrillar adenosine triphosphatase histochemistry techniques (9). All muscles contain various percentages of fiber types, even within contralateral limbs of the same organism.

These fiber types respond to functional demands, hormonal signals, and neural input, but most of the muscle's fiber type composition is due to genetic control (9). In completely artificial circumstances—60 days of continuous (24 hours/day) low-frequency stimulation of a rabbit tibialis anterior muscle (10), for example—muscles *can* undergo complete fiber type changes. Most current research suggests that fiber type change in response to more reasonable types of exercise is not as significant as was anticipated years ago. For an unknown reason, both strength and endurance types of exercise result in the change of fast-twitch type IIB fibers to type IIA fibers (9). Therefore, if change in fiber type does not account for performance improvement with a certain form of exercise—what does? Hypertrophy of fiber types appropriate to the exercise, neurologic adaptations, skill development, metabolic changes, and other pathways are probably the answer. Muscle responds to and, to a small degree, can change its fiber composition, but generally adapts its structure within the existing fiber type and relies on other mechanisms of the body to assist with physical demands.

When considering muscular firing patterns in normal activities and in sports and their associated kinetic chains, origin-and-insertion thinking becomes obsolete. If a picture is worth a thousand words, a three-dimensional anatomic appreciation is certainly worth a million. Muscle action has classically been defined as concentric, eccentric, and isometric, with the defining characteristic of each being shortening, lengthening, or maintaining the same length of a muscle while under load, respectively. Recently, the concept of "econcentric" contractibility has been proposed as the way a muscle (or even portions of a muscle) may function in any of the three classic phases of action at one joint or multiple joints simultaneously (11). Such a term is used to more accurately define function in a variety of tasks, especially with regard to the kinetic chain. The kinetic chain is described as the sequencing of individual body segments and joints to accomplish a task (12). It generally functions from a base of support proximally and then proceeds distally, but this depends entirely on the task at hand: A bench press would follow the aforementioned path, but a push-up reverses the mechanics even though the muscles engaged are very similar, if not identical.

A helpful way of conceptualizing muscular action and function (and subsequently, dysfunction) is to categorize muscles by their *method* of functioning, as described early on by Rood's concept of stabilizer and mobilizer movements (13) and subsequently expanded on by Janda (14) and Sahrmann (15). This concept extends beyond simple contracture and relaxation, and more toward the characteristics exhibited during muscular activity. *Tonic* muscles are those maintaining a low level of tone nearly all the time, functioning primarily as postural muscles and using more fibers of an oxidative nature to avoid fatigue. Examples of tonic muscles are iliopsoas, rectus femoris, quadratus lumborum, scalenes, gastrocnemius, pectoralis major, and the biceps. *Phasic* muscles exhibit quicker, shorter bursts of activity with phases of rest in between and more often use the glycolytic pathway fibers. Examples of these muscles are vastus medialis and lateralis, gluteal muscle complex, midthoracic erector spinae, tibialis anterior and peroneal muscles, rhomboids, lower trapezius, and triceps.

Further defining function, one can consider the role played in *dynamic stabilization* created by the muscle's firing. There are two classes of muscle at work during dynamic stabilization: stabilizers and mobilizers. *Stabilizers* function to maintain joint congruence and control the range of motion at a joint, usually in a decelerative or eccentric fashion. They also tend to cross only one joint, or when crossing more than one joint, have multiple attachments along their length. *Mobilizers* produce or initiate motion at a joint, acting almost always in a concentric direction

and usually in the sagittal plane. They usually are larger muscles that cross several joints, and attach at points of significant leverage.

Using a more anatomic approach, Bergmark introduced the concept of local versus global musculature (16). This concept divides the muscles based on their anatomic locales and, in keeping with the structure–function interrelationship, their intended function. *Local* musculature functions similar to a tonic muscle, with low-load, continuous activity focusing on stabilizing joint congruity and controlling motion. *Global* muscles can function as both stabilizers and initiators of motion, like the mobilizers.

Comerford and Mottram have recently combined all the above-mentioned theories into a "unified theory" of muscle function, which acts symbiotically to fill in the gaps left by each individual system without diluting their stronger points (17). Their system uses a combination of location and function and categorizes muscles into one of three groups:

1. *Local stabilizers* maintain joint congruity and stiffness, contracting continuously (relatively independent of the joint's direction of movement), and providing proprioceptive data. These function typically as tonic muscles.
2. *Global stabilizers* generate force (usually eccentrically) to conrrol range of motion, especially rotation in the axial plane. This activity is direction dependent, and they therefore function as phasic muscles.
3. *Global mobilizers* generate motion concentrically, especially in the sagittal plane, and can also absorb shock load. This activity is direction dependent, and they therefore function as phasic muscles.

Examining the shoulder is an excellent starting point for classifying muscular function in such a conceptual method. Consider an abduction of the humerus and try to break down the muscles involved into rhese categories. In a simplistic model, the local stabilizers are the infraspinatus, the subscapularis, and the teres minor, acting to maintain joint congruency. The supraspinatus is the initiator of motion and subsequently the deltoid, so these muscles are the global mobilizers. The global stabilizers function to control, eccentrically, the motion of abduction and are therefore the levator scapula, rhomboid, and middle and lower trapezius (all three exerting influence through eccentrically contracting against the upward/medial rotation of the scapula) and to a lesser degree, the infraspinatus, the subscapularis, and the tetes minor.

Such synchronicity in a task is obviously difficult to achieve consciously, yet the neuromuscular system's adaptability allows for redundancy without repetition. It has been well documented that the more skill an athlete has with a certain task, the more he or she can reproduce a desirable technique despite variations in the circumstances (18,19). For instance, the ability of a professional golfer to reproduce a good, clean swing in various circumstances of footing, the lie of the ball, and wind conditions requires tremendous skill. This skill comes from neuromuscular adaptability ro achieve less variation in the outcome of the technique, usually from having trained pathways to use from experiencing similar situations in the past. This training cannot be replicated by redundant, collateral pathways using the same cascade of events, but rather through adaptation of early segments of the chain to permit a similar end point.

Power and speed are best created by spiral motion using the combination of a global mobilizer contraction in rhe sagittal plane with the axial direction from a global stabilizer. This performance increase in power and speed has also been noted in studies of various aspects of multiple sports (20,21) and is the prime focus of effective, powerful motion in the martial arts, such as aikido, Tai Chi, and Kenpo. Most athletes are familiar with the spiral motion generating increased speed and force when examining a terherball in motion, or a major-league pitcher throwing a ball.

NEUROMUSCULAR STRUCTURES

Performance of a task is more than a simple muscular contraction. As anyone who has tried to screw in a light bulb on their tiptoes can attest, balance, coordination, and skill are major

factors in determining success. Although the science of neuroanatomy is well established, the study of neurophysiology continues to break new ground, particularly with our understanding of the proprioceptive system as it relates to the gamma-efferent fiber and Golgi tendon complex feedback loop on alpha motor neuron activity.

Training results in activity-specific improvements in rate of force development, maximal force development, rate of activity, increased reflex response, improved coordination response, and/or task-specific coordination. Suggested mechanisms for such neural adaptations are the following (22):

- Increased activation of agonists
- Selective recruitment of motor units within agonists
- Selective activation of agonists within a muscle group
- Co-contraction of antagonists

Such physiologic adaptations are followed by structural and biochemical changes within motor nerves themselves (23). Although such changes have been noted in the peripheral and lower CNS, there is evidence for higher CNS involvement as well (24):

1. Force production can far exceed cross-sectional area changes.
2. Voluntary strength increases may occur after training without increases in twitch or tetanic tension when evoked with electrical stimulation.
3. Strength gains can be documented in the contralateral limb as well as the ipsilateral limb in single-limb training.
4. Increases in voluntary strength are specific to the training technique.

Training does not always result in an increase in excitatory activity, for some responses should be reduced or slowed to allow for a better outcome of the task at hand. *Summation* is the CNS's way of totaling feedback and intended motion before determining whether an action potential should be sent, while *recruitment* is the mechanism through which the body determines the number of muscle motor units to use for a desired action. If only a small amount of force is required for a fine motor skill, only the lowest-order, slow-twitch fibers will be activated, and so on, up to 50% to 75% of the various types of fibers available for a full-strength contraction.

After practicing a series of contractions for a variable number of times, the neuromuscular system will create an *engram*, that is, a memorized series of motor patterns. An example of an engram would be tying your shoes, or changing lanes when driving a car. Initially, an individual requires great focus on the task at hand, but subsequently, he or she may be able to have a conversation with someone concurrently. Such engrams are composed of muscle activation patterns (MAPs) acquired through trial-and-error with previously learned patterns and feedback. These engrams are critical for skill development, as they free up the conscious mind from the task at hand, allowing the athlete to focus on other tasks simultaneously. Sequencing kinetic chains toward a desired activity is what creates an engram. The development and "burning in" of successful engrams and kinetic chains frequently occur during the "plateaus" that athletes frequently report. Following such times, athletes will notice a dramatic improvement in technique and unrealized automaticity development, such as when a child can successfully ride a bike without training wheels for the first time ("Suddenly, I just *got* it!"). It is paramount that during these plateaus, the athlete is encouraged to concentrate on technique and form.

Such conscious and subconscious feedforward states are currently an area of intense interest and study. Training programs are available that use visual footage of athletes performing tasks with a desirable technique, so that athletes in training can pre-program the muscle activation patterns. Many athletes use visualization techniques, seeing themselves perform the desired activities during practice sessions or prior to competition. When this exercise is accomplished without *intentionally* trying to learn the skill in question, athletes frequently refer to this state as being "in the zone," and often evidence a superior level of athletic skill during these times. Martial artists, especially Zen swords-

men, will attempt to reach *mushin* or *mujin* (the state of no-mindedness) so that their reflexes and actions will be natural and unfettered by fear or other negative content. Athletes and their health care practitioners can use a variety of techniques to motivate and energize, calm and reduce tensions either before or during an event, or improve performance. Evidence also exists for improved muscle function with verbal encouragement (25).

METABOLIC PROCESSES

The energy conversion systems of the body are intricate and fairly well known. Tiered processes exist to create and utilize energy for immediate need (adenosine triphosphate–creatine phosphate system), as well as for short exercise sports (glycolytic metabolism) and prolonged exercise (oxidative metabolism). Although the substrate changes with the duration of exercise, the body does not particularly adapt its storage or utilization mechanisms over time, depending more on type and timing of nutritive replenishment as well as duration of rest. One of the few exceptions has already been discussed with modulation of fiber types depending on the type of exercise, where conversion to more oxidative fibers can result in free fatty acid (FFA) utilization at earlier stages of endurance exercise (26). Debate still exists over whether this utilization is primarily due to increased oxidation of plasma FFAs or intramuscular triglycerides (27). Although important, full review of the vitamin, mineral, and other nutrient considerations is beyond the scope of this chapter.

One has to be both awed and daunted when working within the scope and interwoven nature of the mind–body relationship. Beyond attitude and perceived effort, the allostatic load of an athlete can have tremendous implications for performance and health. *Allostasis* refers to the altered state of body functioning under a threat or strain (28), such as athletic activity. These include, but are not limited to, chemotactic, hormonal, and metabolic alterations to meet an immediate need. These changes begin with the central command effects as described earlier, as well as mobilization of energy reserves and reduction of nonessential system activity. Such modifications are meant to be temporary; following the removal of the stressor, the feedback mechanisms should engage and return the body to its normal homeostatic state. With prolonged stressors (including physical, psychological, or spiritual), or faulty feedback mechanisms, the intended beneficial response becomes maladaptive. Current trends in exercise suggest a shift toward these body–mind concepts—yoga; Pilates; aikido, Tai Chi, and other martial arts; triathlons; and cross-training. The mental and spiritual aspects of these current trends should not be ignored, as the athlete is without question unification of flesh and spirit.

THE QUANTUM ATHLETE

The culmination of all the systems we have described into a perfectly functioning athlete provides the concept for the "quantum athlete." Such an avatar would encompass, to the fullest, the qualities of endurance, strength, flexibility, proprioception, spatial awareness (i.e., "court sense" or "field vision"), and mental and spiritual harmony—the ideal of a fully unified, self-healing being, adapted structurally for the desired function. In such circumstances, athletic activity can almost seem prescient in nature (29).

The goal for the sports medicine practitioner should be to restore homeostatic mechanisms from an allostatic state and remove restrictions to healing, thereby allowing anatomic modification to better suit the needs of the athlete. Highest-level activity requires structurally and functionally healthy tissues, superior coordination of the neuromuscular network, and a clear and focused mind. It is not uncommon for a practitioner to focus only on the first, less on the second, and seldom the third. To realize maximal functional potential, the athlete must incorporate the above-mentioned principles with visual imaging—to see themselves successfully carrying out nearly impossible feats or high levels of performance. Cutting-edge research has led to new drills and programs to

enhance tissue healing and neuromuscular control, and progress should continue as we learn more about a high-performance body.

To expand our knowledge of the mind, however, we should to look to the historical practices of meditation, philosophy, religion, and even the martial arts for techniques of honing awareness. Their recent popularity in Western culture speaks to the innate connection an athlete can achieve with his or her body. Meditation and prayer have helped to stabilize the cardiovascular system, reduce perceived stress, improve mild hypertension and hormonal modulation of the sympathetic–adrenal medulla system, and advance intelligence and academic tests with considerable, albeit statistically insignificant, changes in psychomotor ability and cardiopulmonary functions (30–33).

Training and conditioning should be tailored to the athlete's needs and desires. Use of the concept of the quantum athlete can assist not only in alleviating injury and restoring function but also in promoting a level of achievement and skill the athlete may not have been aware was present within.

EFFECTS OF DISUSE AND MISUSE ON INJURIES AND RECOVERY

Having laid the groundwork for the healthy physical and mental state, we can now progress toward an examination of the misguided or improperly expressed states that precede disease and injuries.

Lack of tissue use (due to injury, improper pain management, altered or improper technique, joint or soft tissue restrictions, etc.) will reverse the processes we just reviewed. Bones lose density, joints stiffen, ligaments shorten, muscles atrophy, and neuromuscular control changes negatively. Metabolic processes will revert to a lower energy condition, with a drop in basal metabolic rate and exercise tolerance. Change or maladaptation of technique usually occurs much more rapidly than the body can accommodate structurally, and a race to see if the maladaptive state can overcome homeostasis begins. This deconditioning process is not independent, but rather is linked and greater than the sum of its individual parts. With improper performance by these links, the chance of injury rises rapidly and can occur not only at the site of the initial problem, but also in a tissue or organ system attempting to compensate for the original dysfunction or malfunction. It is therefore important to establish not only the correct diagnosis of the injury or problem in question, but also to seek out *why* the problem occurred in the first place—why did the body fail at *this* moment? Directing care in this fashion takes the practitioner from the state of treating just the disease toward treating both the disease and the host, moving away from the consequent illness (Fig. 10.2).

With regard to bone, stress reactions and fractures can develop due to resorption of bone from unloading, or due to increased loading on a portion of bone that has not previously been loaded in such a manner. The density of minerals in bone is lower and, without appropriate loading forces, is laid in a more diffuse pattern that supports forces in all directions equally, yet with less overall resistance than if a "grainy" pattern were present. Undergoing such processes, the bones cannot serve their tensegrity role in resisting compressive and tensile forces appropriately, and the entire structure is weakened.

The inserting tendons and ligaments lose their tensile properties as well, having either incorrect or even no particular fiber direction to follow. Proprioceptive inputs become less reliable and actually can become harmful as MAPs and their kinetic chains are altered, leading to abnormal loading of bone and the supporting soft tissues. Ligaments receive their blood supply only with motion. Therefore, immobilization retards healing, as confirmed by research in ankle sprain management and postsurgical rehabilitation of anterior cruciate ligament repairs. Synovial fluid stagnates, reducing the supply of nutrients and reparative cells to the articular surfaces and prolonging the time needed to restore damaged tissues. Joint capsules stiffen and shorten, promoting restricted ranges of motion and, by means of the arthrokinetic responses described earlier,

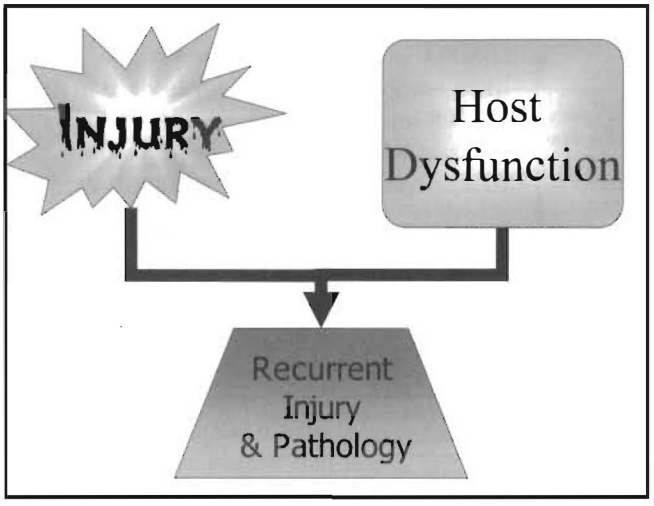

FIGURE 10.2. Combination required for recurrent/recalcitrant injury and pathology.

change muscle length and activation patterns (remember the inhibition of the gluteal muscles in response to a restricted anterior hip capsule mentioned earlier). Improper timing and strength of responses can reduce the loads absorbed by the soft tissues, and the articular surfaces will bear even more of the burden.

The muscular response to disuse is to change fiber types to less-energy-utilizing forms (type IIA fibers change to type IIB fibers) (13). The result is muscular atrophy and dysfunctions depending on the muscle classification. Tonic muscles actually increase their resting tone and become less pliable, which results in the corresponding (yet inappropriately timed) crossed extensor and reciprocal inhibitory responses of agonist–antagonist pairings, leading to an increased likelihood of strain or rupture of the particular muscle suffering disuse. The phasic musculature becomes weak and less responsive, resulting in compensation by other muscles to achieve the intended motion. Both responses carry a negative impact for the kinetic chain of any action, resulting in the "catch-up" phenomenon (16). Such compensation for dysfunction in the earlier components of the chain leads to a motion that is not as productive and can lead to injury in the later components, as the tissues cannot handle the load nor fire appropriately. Classic examples of this breakdown can be seen in overhead sports such as tennis or baseball, where recurrent elbow pain is the complaint that stems from inadequate lumbar or shoulder mechanics, or in the case of a pitcher's subscapularis tendon injury as the result of decreased internal rotation at the hip of the contralateral (stride) leg.

Having an imbalance in the global musculature allows microtrauma to accumulate insidiously. With repetitions of these dysfunctional MAPs, dysfunctional kinetic chains and engrams develop that cement the dysfunctional, but usually asymptomatic, paths even more. Pain and/or pathology usually begins in the local stability system, which cannot maintain proper function, thus perpetuating the loop (Fig. 10.3). Frequently, it is only after these dysfunctional pathways have been laid that athletes will notice a change in their performance and will typically blame the injury as the onset of their problem—not recognizing that the true issue was present earlier and the body had compensated around it. Close monitoring of technique will reveal that the typical, desirable spiral patterns of technique have begun to elongate and become more ovoid. The apex of this arc typically appears where the injured local stabilizer is unable to maintain an eccentric load during the desired motion. Investigating more proximally or earlier in the chain will usually

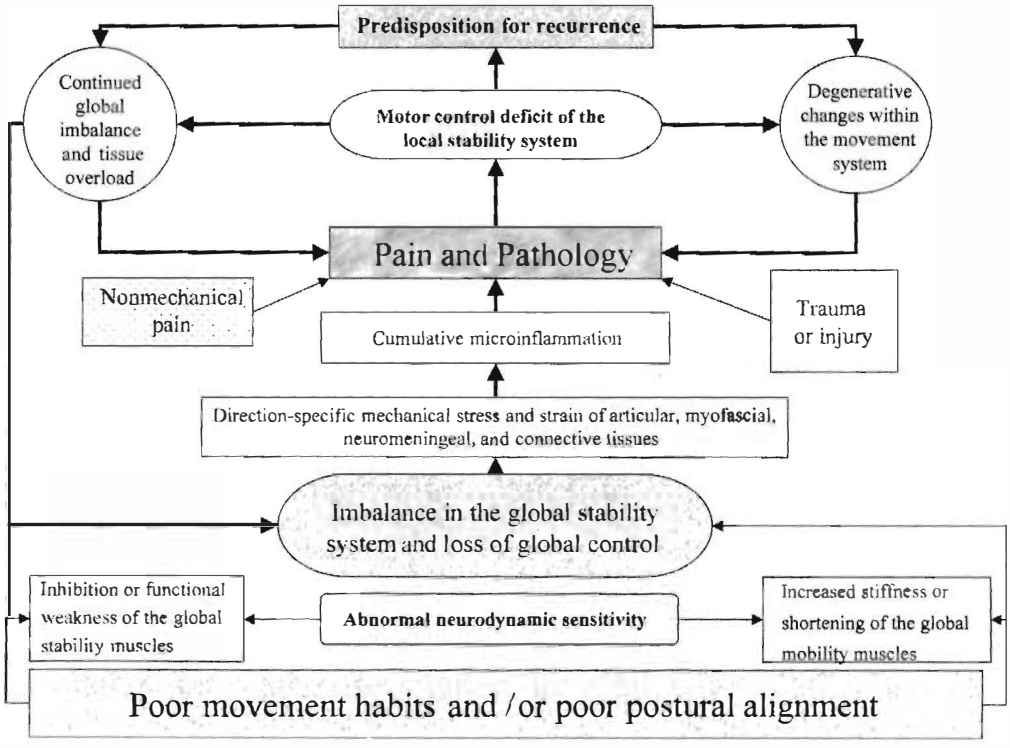

FIGURE 10.3. Feedback loop from poor posture or movement habits.

unveil the global mobilizer or stabilizer at the root of the dysfunction.

Until this happens, athletes may suffer recurrent or recalcitrant injury and the concomitant mental difficulties in handling it. They may be well aware of their inappropriate technique and experience frustration or anger when they are not able to release the hypertonic motion or activate the inhibited muscle. The sequelae of increased perception of difficulty and effort due to their frustration are increased resting heart rate and reduced resolution of fatigue via activated central command tone. This psychological injury can result in movement toward an allostatic milieu, which can physically retard healing, deplete energy reserves, reduce motivation to rehabilitate, and interfere with establishing new neuromuscular pathways. Many athletes experience a "tight gut" when faced with fearsome, frustrating, or difficult situations, which can manifest physically with a reduction in diaphragmatic breathing and the accompanying inefficient compensations discussed earlier. When things have progressed this far, the road back to recovery is long. However, with proper guidance, visual imaging, behavioral modification, and motivation, these athletes *can* find their way back.

CONCLUSION

In this chapter, we have reviewed the various tissues and systems within the body most involved in athletic performance, as well as in injury and dysfunction. Although research increasingly accumulates to explain the mechanisms by which these principles and practices help return athletes to a state of functionality quickly, clinical experience and application are the fruits of this labor. Applying these holistic principles means more than manipulating joints and stretching muscles. It is the *search*—seeing,

listening, feeling, thinking—more than the *manner* of treatment that returns optimal health to the athlete. By searching for the truth within the problem, listening to his or her body, and releasing its inherent ability to adapt and heal, the athlete will find the way back to optimal performance.

> There are people who, instead of listening to what is being said to them, are already listening to what they are going to say themselves.
>
> –ALBERT GUINON (1863–1923)

Thanks to all who helped review and critique this chapter for their insight, support, knowledge, and brutal honesty. This chapter is dedicated to Mike, who teaches me still from times long past, and to Amy, who opened my eyes.

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11

EXERCISE PRESCRIPTION

STEVEN J. KETEYIAN

OVERVIEW

Exercise is now touted as a treatment for many types of disease; in fact, lack of exercise and physical activity leads to disease and increases morbidity in many conditions. As society becomes more automated and modern conveniences make exercise less incidental to daily life, the depth of this problem will continue to grow. The body depends on motion and exercise to maximize healthy function. For example, lymphatics in the extremities depend on muscle contraction to pump lymph throughout the body. Joint motion aids in distributing nutrition to its cartilage and synovium, while an immobile joint quickly stiffens and loses range of motion.

Exercise is also important in helping athletes deal with chronic somatic dysfunction. In manual medicine, a common question a patient asks after treatment is "How do I prevent this from happening again?" Whether it is cervical dysfunction, sacral torsion, or other malalignments, exercise is the answer. Flexibility and muscle strengthening are the two most important aspects of dysfunction that go untreated. Many individuals just get manipulations repeatedly, thinking that the dysfunctional segment will eventually be forced into staying in place by the clinician's manipulations. Without exercises to specifically stabilize the region, the dysfunction will likely recur. After all, when a region is put back into alignment, it is the muscle group that keeps it aligned. Muscles have memory, and if the muscle believes that a dysfunctional position is correct, then the muscle will always fire dysfunctionally, unless it is retrained through exercise. Therefore, manual medicine should always incorporate an exercise prescription, either through a trainer or rehabilitation center, or with a home program the athlete maintains for himself or herself.

The task of prescribing exercise in high-performance athletes, in sedentary healthy people, and in patients with clinically manifest disease is more similar than one might think. Despite any obvious differences in performance level between the three groups or despite concerns one may have about exercise-related complications, there are two basic tenets that apply to everyone when establishing an exercise training program. These are *specificity of training* (e.g., mode of training) and *progressive overload* (i.e., intensity, duration, and frequency of training).

Before discussing each of these two tenets in detail, it is necessary to first identify and briefly discuss the major components or types of physical fitness. They include aerobic power or endurance, peak anaerobic power, anaerobic endurance or capacity, muscular strength, muscular endurance, body composition, and flexibility.

COMPONENTS OF PHYSICAL FITNESS

Table 11.1 summarizes various common methods for measuring the primary types of fitness in progression from the least costly and least complex to those of greater cost and complexity. In almost every case, in moving from lower cost and lesser complexity to higher cost and greater complexity, validity and measurement accuracy improve. For the physician routinely involved with sports medicine, having access to many of the more valid methods listed in

TABLE 11.1. METHODS OF ASSESSING COMMON FITNESS PARAMETERS

Complexity of Measure	Cost	Aerobic Power	Anaerobic		Muscular		Body Composition	Flexibility
			Power	Capacity	Power	Endurance		
Simple	Low	Submaximal test to predict Vo_2 max	—	—	Maximal (kg) using hand grip dynamometer	Repetitions to muscle fatigue	Skinfolds Bioelectrical impedance	Sit and reach
Moderate	Moderate	Maximal test to predict Vo_2	Margaria-Kalmen power test	—	One repetition maximum (isotonic)	—	Skinfolds Bioelectrical impedance	Gravity-dependent flexometer
Complex	High	Direct measure of gas exchange for Vo_2 max and pace at ventilatory threshold	Wingate test for peak power (hp)	Wingate test for anaerobic capacity or total work (joules) and fatigue index (%)	Isokinetic analysis of force/velocity curves	Isokinetic analysis of fatigue index at specific speeds	Hydrostatic weighing for body density Dual x-ray absorptiometry	Radiography Biomechanical analysis

Table developed courtesy of Steven J. Karageanes, DO, Henry Ford Health System.

Table 11.1 is important. However, before ordering any of these tests it is always prudent to consider whether the need to more accurately quantify fitness level or training effect offsets any increased cosr.

Aerobic Power

An approximation of peak aerobic power or oxygen consumption (V_{O_2}) can be easily accomplished using a submaximal or maximal bike or treadmill test. Previously established prediction equations are typically used to estimate peak V_{O_2}, and such data are useful when either categorizing one's fitness level or roughly assessing change in exercise capacity due to training. However, providing an estimate of peak V_{O_2} may not be helpful for highly competitive athletes interested in quantifying peak aerobic capacity or heart rate or pace at ventilatory threshold. Generally, submaximal and maximal tests tend to overestimate measured peak V_{O_2} by as much as 15% to 25% (1).

One accurate method for measuring peak V_{O_2} involves using open circuit, indirect spirometry. Because testing mode or type of exercise ergometer can influence results among highly trained athletes, it is appropriate to test the athlete using the mode that best simulates her or his activity—the principle of specificity of testing (2). Therefore, competitive cyclists should be tested on a cycle ergometer, swimmers in a swim flume, and runners on a treadmill. Among healthy untrained and less competitive individuals, treadmill testing is satisfactory and usually results in a higher peak V_{O_2}, by about 5% to 15%, when compared to testing performed using other ergometers.

Typically, peak V_{O_2} is reported in $mL \cdot kg^{-1} \cdot min^{-1}$ and, assuming normal pulmonary function and the absence of clinically meaningful anemia or a skeletal muscle disorder, it reflects the ability of the body to transport and utilize oxygen. A simple rearrangement of the Fick equation (Adolph Fick, 1870) provides a nice illustration of this concept:

$$V_{O_2} = Q \times a\text{-}vO_2 \text{ diff}$$

where V_{O_2} is oxygen consumption in $L \cdot min^{-1}$, Q is cardiac output in $L \cdot min^{-1}$, and $a\text{-}vO_2$ difference is arterial–mixed venous O_2 difference in $mL \cdot L^{-1}$.

In this equation, exercise cardiac output represents the transport of blood to the metabolically more active skeletal muscles and $a\text{-}vO_2$ difference represents the extraction and utilization of oxygen within the muscle. Typical values for peak V_{O_2} are shown in Figure 11.1.

Interestingly, at-rest cardiac output is the same in both healthy untrained and endurance-trained athletes, about $5 L \cdot min^{-1}$. However, at-peak exercise cardiac output may reach 22 to 25 $L \cdot min^{-1}$ in the nonathlete, and exceed $35 L \cdot min^{-1}$ in the athlete. In general, this difference in the ability to maximally transport oxygen to the active tissues is due to a greater stroke volume during exercise in the athlete versus the nonathlete (~170 $mL \cdot beat^{-1}$ versus ~120 $mL \cdot beat^{-1}$, respectively). In contrast, peak heart rate is influenced little by exercise training, and peak $a\text{-}vO_2$ difference is only slightly greater in the athlete (15.5 mL of $O_2 \cdot 100$ mL of $blood^{-1}$) versus the nonathlete (~13.8 mL of $O_2 \cdot 100$ mL of $blood^{-1}$).

Anaerobic Power and Capacity

Whereas the ability to generate adenosine triphosphate (ATP) over a long period of time in the skeletal muscle relates to chemical reactions within the aerobic metabolic pathways (i.e., Krebs cycle, oxidative phosphorylation, and beta-oxidation), a person's ability to generate ATP during sudden, short-duration and all-out tasks is related to the amount and rate of ATP produced via anaerobic pathways [i.e., phosphocreatine (PC) system and anaerobic glycolysis]. For example, the highly accomplished male 800-m college runner will complete his event in less than 1 minute and 50 seconds, requiring a high level of anaerobic fitness in the muscles of his legs to do so.

Tests that reflect or quantify one's ability to produce ATP through the ATP-PC system and anaerobic glycolysis have changed greatly over the past 40 years. In the 1960s, the common method for assessing anaerobic power involved

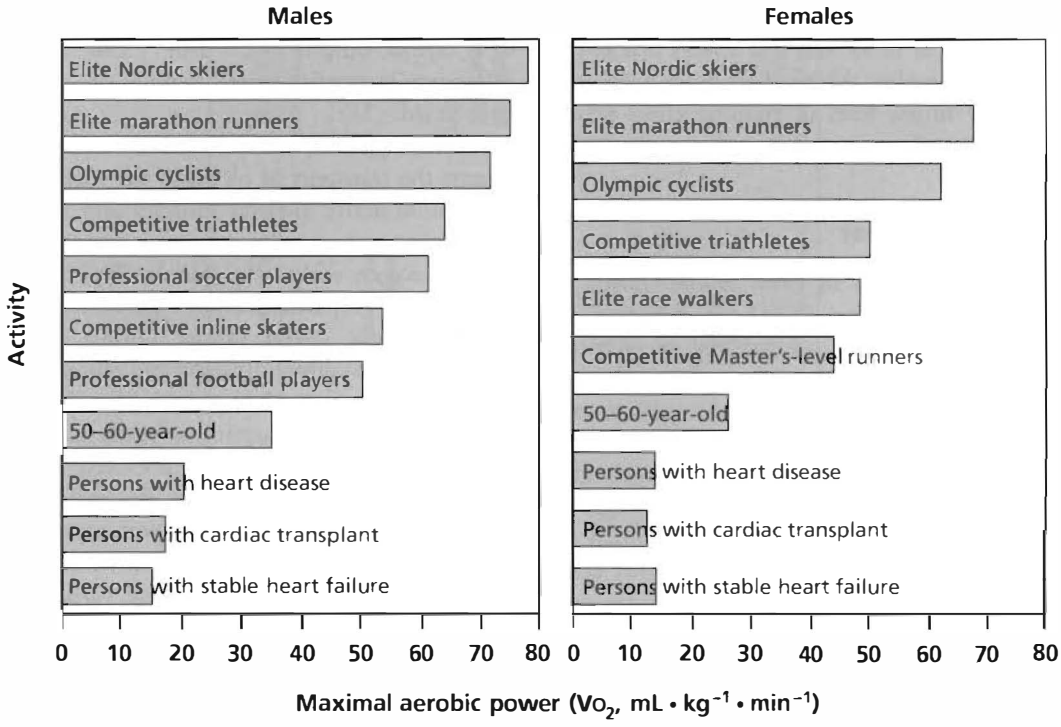

FIGURE 11.1. Typical values for peak Vo_2.

the Margaria-Kalmen power test (1,3). In this test, a subject runs up a flight of stairs as rapidly as possible, taking three steps at a time. Using electronic switch timer plates placed on the third and ninth steps and knowing the rise of each step, power is then calculated using the mass of the subject moved over the vertical distance between step 3 and step 9 and the elapsed period of time to do so.

The aforementioned method has given way over the years to the Wingate anaerobic test (4), which involves pedaling a cycle ergometer for 30 seconds using maximal effort against a fixed braking force that is set at two to four times a previously determined maximum. Peak power is determined in watts or horsepower, usually attained in the first 5 seconds of the test. Mean power represents the average power over the full 30-second test.

To appreciate the magnitude of the power generated in elite anaerobic athletes, consider the Olympic 1-km track cycling event that is conducted outdoors using an aerodynamically enhanced bicycle on a track called a velodrome. This event is often called the "killermeter" because it is an all-out sprint that typically takes 62 to 65 seconds to complete. Approximate Wingate test results for these athletes may approach 1385 W, 940 W, and 285 kJ for peak power, mean power, and total work, respectively. This is enough power to illuminate 100 light bulbs (each 60 W) for almost 5 seconds. Typical values for other athletes and nonathletes are shown in Figure 11.2.

Higher peak and mean power values among various athletes during testing are associated with the ability to achieve higher rates of ATP production through anaerobic pathways and higher concentration of muscle and blood lactate. Also, as one might guess, both of these variables are significantly correlated to the percentage of type II (fast-twitch) muscle fibers (5). However, although the Wingate test does identify people with increased capabilities for

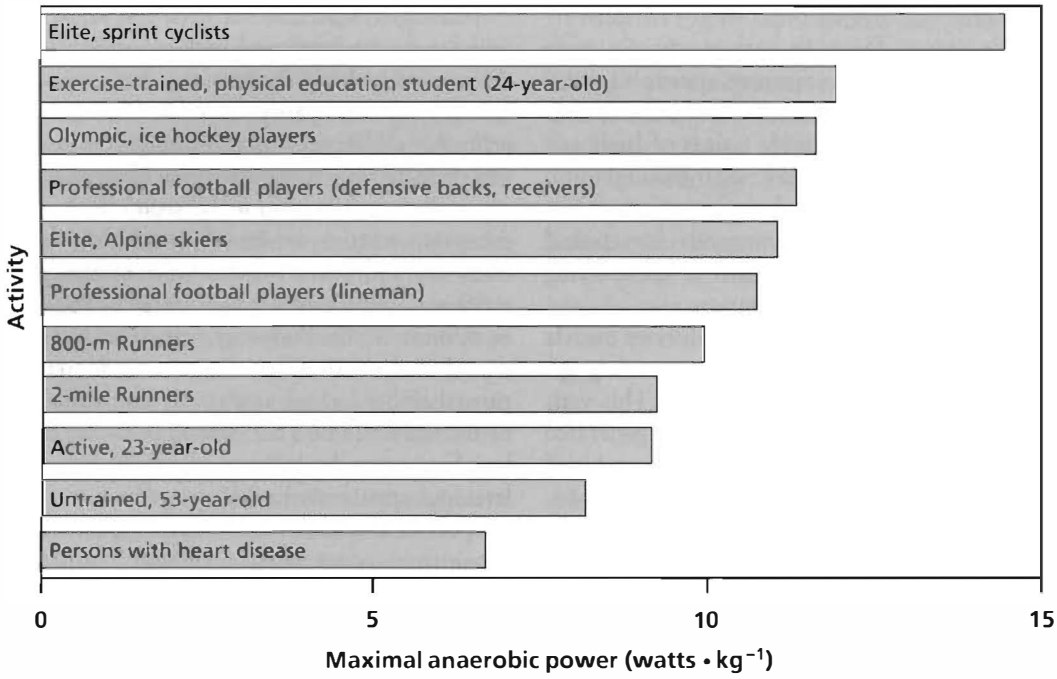

FIGURE 11.2. Typical values for power generated by different types of male athletes.

anaerobic performance, it does not always relate well to athletic performance. This is because other factors such as strategy, prior racing experience, and intrinsic motivation often differentiate success.

Muscle Strength and Endurance

Measures of muscle strength and endurance have long been performed, and established norms exist for boys and girls, men and women, and athletes and nonathletes. Generally, the more simple and inexpensive tests of muscle strength (e.g., hand dynamometer for grip strength, cable tensiometer for quadriceps strength) and muscle endurance [e.g., number of sit-ups or push-ups in 1 minute or until fatigue (3); flexed arm hang (girls) or pull-ups (boys)] do not necessarily correlate well with on-ice or on-field performance.

Another common method for assessing muscle function, more so for muscle strength than muscle endurance, is the one repetition maximum (1 RM). Using either free weights or fixed bar resistance machines, isotonic or concentric muscle strength is then measured as the maximum amount of weight that can be lifted during one repetition, thus 1 RM. Because multiple joints and muscles are involved in muscle strength testing when using free weights, this testing may be better for sport-specific movements or athletic performance. Conversely, fixed bar and isokinetic resistance machines better isolate specific muscles or muscle groups and as a result, often serve for evaluating treatment or rehabilitation outcomes.

Recent work by Robergs and Keteyian summarized grip strength and age-specific chest press norms in men and women using grip dynamometer and bench press, respectively (3). Similar information, expressed as percentiles, is available for upper body strength (bench press) and leg strength (leg press) (6). When 1 RM is not advised or available for whatever reason, equations exist to predict 1 RM for leg press and chest press using submaximal effort (3).

Over the past 30 years, the use of isokinetic or other accommodating resistance devices have

flourished because of their ability to quantify muscle power, force, or torque across a wide range of fixed joint movement speeds—from 0 to 300 degrees per second. With this methodology, the tester can identify points of high and low force output that may occur throughout a limb's measured range of motion. Such information is generally advantageous for clinical evaluation and research aimed at monitoring training progress or rehabilitation.

Additionally, with isokinetic devices muscle endurance can be assessed using a testing approach that provides a *fatigue index*. This variable represents the loss of maximally generated force at a given joint movement speed and over a given period of time (e.g., 20 to 30 seconds). Generally, the smaller the difference in mean force when comparing the first five repetitions in a test to the last five repetitions, the greater the muscle endurance. Restoring to less than 10% any differences in fatigue index (loss of power) that exists when comparing a previously injured limb to a healthy limb is often used as a guide when providing return-to-play recommendations among competitive athletes.

Body Composition

Although often more true for athletes than nonathletes, determining the percentage of body mass that is fat versus fat-free tissue (lean body mass) is sometimes included when evaluating physical fitness. This is partly due to the strong relationship between muscle mass or cross-sectional size and muscle performance (i.e., strength and endurance). Likewise, a lower percentage of body fat favors athletes involved in activities that demand balance, agility, and moving their body mass through space (e.g., jumping).

Among competitive runners, less fat is also associated with improved performance. In fact, differences in body fat between elite male and female runners partly account for gender-atributed differences in running performance. On the other hand, performance in swimming, especially women swimmers, seems to be the exception relative to the relationship between body fat and performance. Current thoughts are that a slightly greater percentage of body fat in swimmers aids buoyancy, which leads to reduced drag and metabolic cost and an improved ability to keep one's body on the surface of the water. Table 11.2 depicts common findings for percent body fat among endurance athletes and apparently healthy people.

The measurement of body composition is prone to a variety of technical and instrument errors. Therefore, it is important that both tester experience and laboratory reliability be ascertained before accepting results. As mentioned in Table 11.1, one inexpensive and simple method for body fat determination is the use of skinfold measures. In highly trained hands this approach estimates body density, which is then used to compute body fat using existing equations, to within ±3% to 4% (6). If correctly performed, this approach is usually sufficient when setting performance goals and categorizing athletic participation (e.g., weight classes in wrestling). One advantage of skinfold measures is that numerous prediction equations have been developed to estimate body density for a variety of groups including the general population, athletes and nonathletes, men and women, and children and older adults. Another obvious advantage is its lower cost and ease of testing. Subjects can be tested in just about any setting.

TABLE 11.2. COMMON VALUES FOR PERCENT BODY FAT IN ENDURANCE ATHLETES AND APPARENTLY HEALTHY PEOPLE

	Endurance Athlete	Health Maintenance				
		Excellent	Good	Average	Fair	Poor
Men	5–9	10–14	14–18	18–22	22–25	>25
Women	10–16	17–20	20–23	24–30	30–34	>34

Unlike skinfold measurement, and despite its simplicity and popularity for use at health fairs and in fitness centers, bioelectrical impedance analysis is not recommended for the routine measurement of body fat. Technically, this methodology is based on the passing of an undetectable current from electrodes placed on a hand and foot to electrodes placed on an ankle and wrist. Unlike body fat, fat-free tissue contains most of the body's water and electrolytes and is therefore a good electrical conductor. Thus, the amount of current flow through tissue reflects the amount of fat and fat-free tissue.

It should be noted that body fat determined from bioelectrical impedance (or conductivity) analysis is often underestimated in nonathletic people and overestimated in lean athletic people. One reason for this is that correct pretest instructions aimed at controlling confounding variables are often either not srated or followed. Specifically, the influence of prior alcohol, diuretics, and caffeine consumption; prior exercise; avoiding fluids for 4 hours; voiding before testing; and phase of menstrual cycle on total body water should be addressed. Given the above concerns and the often improper fitting of one population-specific prediction equation to people of different race, gender, age, and ethnicity, body composition determinations using bioelectrical impedance should be viewed with caution. It is important to point out, however, that with proper pretest instruction and the use of the correct population-specific equation, bioelectrical impedance is reasonably accurate.

Although newer and generally accurate commercially available systems are now available to measure body volume using plethysmography, the gold standard two-component method for determining body density remains hydrostatic or underwater weighing. Like skinfold testing, this approach is also subject to tester error. However, if performed correctly it can be used to determine body density and, therefore, body fat to within 1% to 2.5%. Measuring body density using hydrostatic weighing, when done in conjunction with the newer formula meant to convert body density to body fat in a variety of ages, gender, and ethnicity, is quite valid (7).

Another highly sophisticated method for determining body composition, one that represents a three-component model (i.e., bone mineral content, body fat, and lean body mass), is dual energy x-ray absorptiometry, or DEXA. This methodology is accurate, is safe, requires little pretest subject preparation, and provides little subject discomfort. Its only drawback, due to its price, is availability, which is becoming less of an issue as more and more units are purchased by clinics and hospitals.

Flexibility

Important for performance in both athletic activities (e.g., gymnastics, wrestling) and routine activities of daily living, joint flexibility is influenced by variables such as distensibility of joint capsule, adequacy of warm-up, muscle viscosity, and any pain or discomfort due to acute or chronic injury. Just as no single exercise can measure total body muscular strength or endurance, no single exercise can evaluate total body flexibility. Typical instruments for measuring joint flexibility include tape measures, the Leighton flexometer, and goniometers. In the fitness center setting, static flexibility of the hips and lower back (i.e., trunk flexion) is routinely measured using the sit and reach test (i.e., trunk flexion), both with and without the use of a sit and reach box.

PRESCRIBING EXERCISE

As mentioned at the beginning of this chapter, improving an individual's capacity to perform a certain task involves working specific muscles or organ systems at a progressively increased resistance. The two key training principles to accomplish this are *specificity of training* and *progressive overload* or resistance. These principles apply to both anaerobic and aerobic activities.

Specificity of Training

To develop the predominant energy or organ system(s) needed to perform a sport, one must first identify its associated performance time.

For example, near--world-caliber rime for the men's 5000-m ice speed skate is approximately 6 minutes and 33 seconds. Similarly, a near-record rime for the men's 3000-m run is about 7 minutes and 10 seconds. Although these two forms of locomotion are different, skating versus running, they both have similar performance times. As a result, the energy contributions from the muscle's energy systems are similar as well. Specifically, for both events approximately 35% of training time should be spent developing the anaerobic glycolysis system, with the balance spent in training the aerobic metabolic pathways.

Contrast the previous example to shorter duration events such as the 100-m dash; the 25-m swim; or the shuffle, jump, and block maneuver in volleyball—all of which predominantly rely on the ATP-PC and glycolysis systems for energy production. Obviously, less training time should be spent on aerobic training in these athletes and more on high-intensity, short-duration drills. Finally, the 3200-m distance runner should spend 4 or 5 days each week training the cardiorespiratory system and aerobic metabolic pathways, and no more than 1 or 2 days developing anaerobic glycolysis.

To summarize, the energy sources for a given activity are time- and intensity-dependent. Whether a person is chopping wood, shoveling dirt, swimming, ot lifting weights, the primary source of energy is dependent on the performance time maintained at a given intensity.

Having said this, it is important to also mention that although training is sport specific, the ability to *repeatedly* perform anaerobic activities relies on an efficient aerobic system. This means that even the striker in soccer, center forward in ice hockey, or running back in football, all of whom should spend a great deal of their training time developing the ATP-PC and anaerobic glycolysis systems of the legs, must also spend some time training the aerobic system. The reason for this is that it is the aerobic metabolic pathways that are responsible for restoring muscle PC stores in between bouts of repetitive exercise. The more efficient the aerobic systems, the quicker the recovery one has between anaerobic bouts.

Additionally, the more proficient an athlete becomes (or desires to become) within a certain sport, the greater the need to focus training time on sport-specific tasks and drills. Specifically, elite runners should run, volleyball players should play volleyball, and tennis players should play tennis. Although there is some benefit gained by having volleyball players do sprints or tennis players do repetitive jumps because both sports rely on the ATP-PC sysrem and anaerobic glycolysis, this is not an optimal training model for the elite competitive athlete. Among these individuals, conditioning exercises and skill development drills should mimic the athlete's sport. Training in this manner allows the athlete to not only develop the metabolic pathway(s) for the particular sport, but the necessary neuromuscular patterns as well.

Progressive Overload

Although the concept of overload training also applies to resistance training, we begin our discussion by first focusing on whole-body anaerobic and aerobic training programs. To accomplish this, it is necessary to introduce intensity, duration, and frequency of training. Ultimately, these concepts are used to provide a sufficient "overload" stimulus of the energy pathways, in hopes that corresponding adaptations lead to improved athletic performance.

Of the three factors mentioned, *intensity* is clearly the most important. The three methods that are typically used to guide intensity are blood lactate level, heart rate, and training velocity or training pace.

Blood lactate is an important biomarker indicating whether the athlete is predominantly stressing the muscle aerobic systems, anaerobic glycolysis, or both. Measuring this blood chemistry during exercise requires specialized equipment, but fingerstick methodology now makes it relatively easy—in that blood lactate levels are often measured poolside among swimmers. As shown in Figure 11.3, blood lactate increases with increases in exercise pace or power output, from around 1 mmol $\cdot$ L^{-1} to values that may exceed 12 mmol $\cdot$ L^{-1} during exhaustive work. Note, however, that at or around 4 mmol $\cdot$ L^{-1} there is

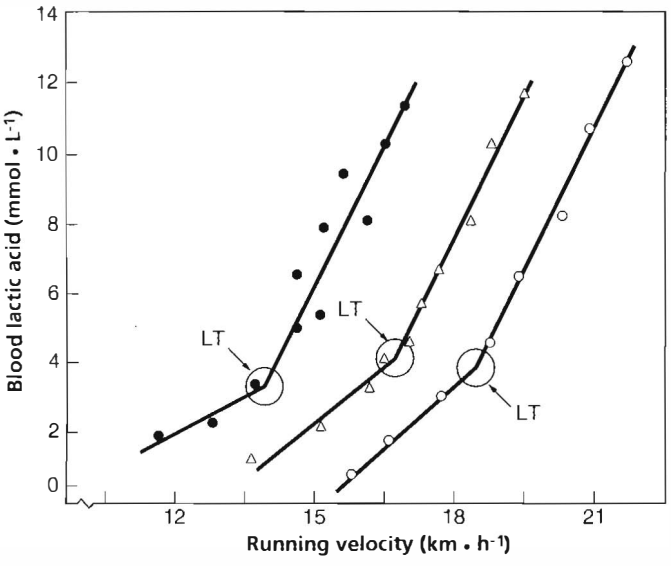

FIGURE 11.3. Blood lactic acid with regard to movement velocity. Regardless of whether the athlete is running, cycling, or Nordic skiing, lactate threshold (LT) occurs when movement velocity results in a blood lactic acid concentration exceeds ~4 mmol · L^{-1}. As shown, LT can vary a bit when comparing one athlete to other athletes performing the same event. In this example, athlete 1 (*black circles*) is relatively untrained, athlete 2 (*white triangles*) is moderately trained, and athlete 3 (*white circles*) is highly trained. Note that the onset of LT is delayed based on training state.

a definitive break in the curve that is referred to as lactate threshold (LT). It is at this point that lactate production, predominantly from anaerobic glycolysis in the more quickly recruited type II muscle fibers, exceeds the ability of the liver and kidney to clear lactate from the blood (1).

For example, members of the U.S. Olympic men's sculling team rely heavily on anaerobic glycolysis to perform the various events and as a result, spend a great deal of practice time doing sprint work at a velocity that elicits a heart rate that is 5% or more above heart rate at LT. Conversely, among aerobic athletes it is common to train up to 3 to 4 beats below LT, with only occasional periods of time spent at or just above LT. However, as mentioned previously, the lactate-based approach to guiding training intensity is not practical or available for most coaches and athletes. Therefore, monitoring heart rate or training pace is more common.

Because the relationship between heart rate and exercise intensity (i.e., power output or peak V_{O_2}) is generally quite linear (Fig. 11.4) in healthy athletes, it is appropriate to train them at the heart rate that will elicit the necessary overload stimulus on the muscles or organ systems that the sport requires. The two common formulas for determining a heart rate–based training intensity are the straight percent of peak heart rate method and the heart rate reserve method.

Table 11.3 depicts training heart rates for younger and older untrained individuals using both heart rate–based formulas. Typically, for the health and general fitness needs of apparently healthy nonathletes, training intensities set at 60% to 85% of peak heart rate or 50% to 80% of heart rate reserve are sufficient to produce the metabolic and organ system overload stimulus needed to improve aerobic performance. For athletes involved in competitive aerobic or anaerobic sports, heart rate during exercise may be maintained at 90% of peak heart rate or 85% of heart rate reserve.

Duration and Frequency

Duration can refer to either the length of a single training bout (number of minutes or hours) or how long the athlete has been training overall (months vs. years). Generally, the more frequent the program (5 days per week vs. 2 days per week) and the longer the overall program (months vs. weeks), the greater will be the increase in fitness or performance.

The recommended frequency for most endurance athletes is, at minimum, 5 days per week. However, many train seven or more

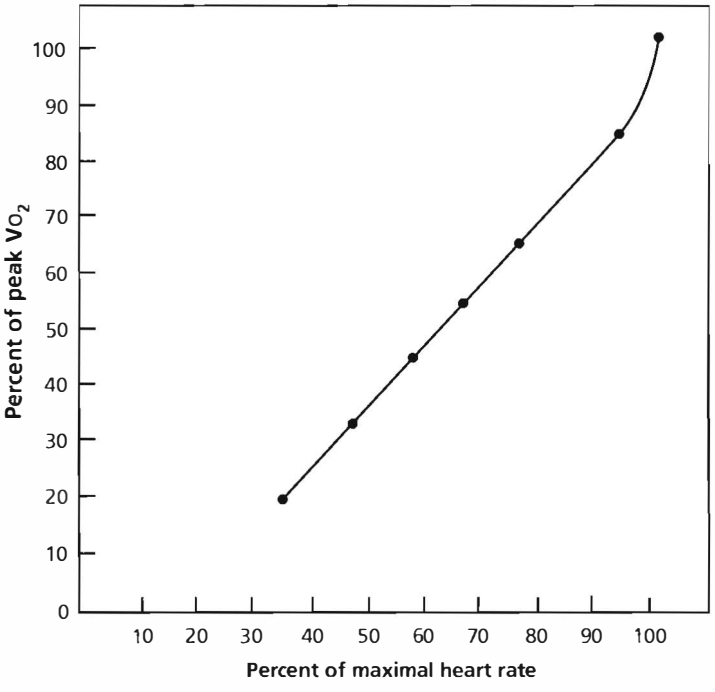

FIGURE 11.4. Relationship between heart rate and peak Vo_2, expressed as a percentage of maximum for each.

times in a week. Obviously, the latter may require more than one workout in a day during the week, which is common in swimmers and track athletes. Interestingly, despite this practice there is no conclusive evidence that multiple workouts each day actually lead to improved performance (8).

What we do know is that, assuming training intensity is unchanged, duration and frequency of training can, to a certain extent, be traded off. If, for some reason, training one week is decreased from 6 to only 4 days in a week, then total duration per training bout for that week should be increased to ensure that the overload

TABLE 11.3. EXAMPLES OF HEART RATE–BASED TRAINING INTENSITIES IN YOUNGER AND OLDER UNTRAINED INDIVIDUALS

	Estimated or Measured Peak Heart Rate (beats • min^{-1})	Resting Heart Rate (beats • min^{-1})	Exercise Training Heart Rate Range (beats • min^{-1})	
			Lower Limit	Upper Limit
70-year-old				
HRR	150	70	110	134
% peak HR	150	NA	90	128
20-year-old				
HRR	198	60	129	170
% peak HR	198	NA	119	168

Heart rate reserve (HRR) is computed as: (peak HR – resting HR) × 0.50 + resting HR = lower limit and (peak HR - resting HR) × 0.8 + resting HR = upper limit.
% peak HR computed as: peak HR × 0.60 = lower limit and peak HR × 0.85 = upper limit.
HR, heart rate; NA, not applicable.

TABLE 11.4. GENERAL TRAINING GUIDELINES FOR AEROBIC AND ANAEROBIC SPORTS

Training Factor	Aerobic Sport	Anaerobic Sport
Intensity	80–90% of peak HR 75–85% of HRR Just below (or at) HR at lactate threshold	HR 5–15% above HR at lactate threshold HR
Frequency	4–6 days/wk	3 to 4 days/wk
Sessions/day	1 (maybe 2)	1 (maybe 2)
Duration	≥8 wk	≥10 to 12 wk
Duration/session		
ATP/PC system	—	Repeated work bouts of 25 seconds or less
Anaerobic glycolysis	—	Repeated work bouts of 3–4 min or less
Aerobic	20–25-min fast work bouts with 5 min of slow work in between; or slow, continuous bout for 30–60 min or more	

HR, heart rate; HRR, heart rate reserve.

stimulus that is applied is similar to that of a 6 day per week workout regimen.

Table 11.4 shows a typical training program for both aerobic and anaerobic athletes. Notice that the frequency of anaerobic training occurs fewer times each week. This is because of the marked strain or overload that high-intensity training provides on the metabolic systems. Adequate time must be given for recovery and adaptation. Compare the training models shown in Table 11.4 to the general health guidelines shown in Table 11.5. Clearly, an athlete's exercise endeavors far exceed what is needed to acquire general health and fitness.

TABLE 11.5. GENERAL TRAINING GUIDELINES FOR IMPROVING HEALTH AND AEROBIC FITNESS

Training Factor	Guideline[a]
Intensity	60–85% of peak HR 50–80% of HRR
Frequency	4–6 days/wk
Sessions/day	One
Duration	≥8 wk
Duration/session	≥30 min/session

[a]Target goal is 150 kcal · day^{-1} (1000 · kcal · wk^{-1}).
HR, heart rate; HRR, heart rate reserve.

Resistance Training

All of the earlier discussion concerning specificity of training and progressive overload has been focused on activities that involve the entire body during an anaerobic or aerobic activity. Common among athletes, however, is the use of weight training or resistance training to improve athletic performance (1,3). Clearly, it is well documented that resistance training–induced increases in muscle strength due to improved neuromuscular coordination and increased cross-sectional muscle size lead to improvements in athletic performance. The difference here is that resistance training does so by focusing on a single muscle or muscle group while performing a regional or joint-specific activity. As a result, the concepts of intensity, duration, and frequency of activity take on slightly different meanings.

There are several important points to consider when discussing or recommending resistance training to athletes (1,3).

1. Specificity of weight training in athletes means that the training exercises or lifts the clinician recommends must be relevant to the energy source and movement pattern of the event.
2. Maximal strength gains are best achieved by an overload program that incorporates higher

intensities (more weight), more sets, and fewer repetitions. Interestingly, maximal gains in endurance are also derived from this same type of program. Therefore, because most competitive athletes require optimal gains in both muscle strength and endurance to perform optimally, a high-weight, low-repetition concentric or isotonic program might be recommended, for example, four sets of six to eight repetitions at ≥85% of 1 RM. Obviously, any recommendations made for resistance training must consider individual safety and population-specific issues. It is often prudent to use a lower weight, fewer set, and higher repetition training model for older people and those with clinically manifest disease, for example, two sets of 12 repetitions at 50% to 80% of 1 RM.

3. No scientific reports presently define the optimal number of sets and repetitions for resistance training in children and adolescents. Having said this, it is worth noting that the American College of Sports Medicine, the American Physical Therapy Association, the National Strength and Conditioning Association, and the American Orthopedic Society for Sports Medicine all support *supervised and moderate intensity* resistance training in children and early adolescents as a means to improve sports performance.

TYPES OF TRAINING PROGRAMS

Interval Training

As the name implies, interval training requires the completion of a series of repeated exercise bouts, alternated with periods of relief. The *work interval* is that period in which exercise intensity or pace is high, such as completing a 100-m swim in no more than 3 seconds above an athlete's best time from a moving start.

The *relief interval* is the time in between work intervals and there are two types. The first is called *rest-relief*, which means it contains very light work. The second is a *work-relief* interval, and it may include mild or moderate work.

If the specific metabolic system the athlete is trying to enhance is the ATP-PC system, then a rest-relief interval is used to train and allow the aerobic metabolic pathways to more quickly replenish muscle ATP-PC stores during recovery. If, however, the athlete is striving to improve anaerobic glycolysis in order to improve performance during either a longer duration anaerobic event or an aerobic event, then a work-relief interval is used. This approach is effective as it partially blocks the complete restoration of the ATP-PC system during the relief phase. As a consequence, anaerobic glycolysis is forced to provide more energy during the subsequent work bout.

A final point about interval training. It is important to determine the correct ratio between work and relief. Generally, the shorter the duration of the sport, the greater the number of workout sets, the greater the number of repetitions, and the greater the duration or time of the relief interval. For example, the 25-m swimmer might do three sets of eight repetitions with a work to relief (rest-relief) ratio of 1:3. Compare this to the 800-m runner who might do two sets of three repetitions, with a work to relief (work-relief) ratio of 1:1.

Fartlek. A Swedish word meaning "speed play," fartlek training is felt to be the precursor to interval training. It usually involves alternating slow exercise pace with a faster pace. Often used by distance swimmers, runners, cross-country skiers, and cyclists, fartlek training should be employed no more than once a week. In this method, the duration of the faster and slower paced periods are not prespecified, and all exercise is performed continuously. Most often used among aerobic athletes to improve aerobic metabolic pathways, there is some improvement in anaerobic glycolysis as well. The net effect is an improvement in race pace.

Tempos. In this method of endurance training, exercise intensity is set at heart rate at LT, with the plan to continue to exercise for 30 to 60 minutes. Occasionally, the intensity can be increased if duration is shortened to just 10 to 15 minutes or so. Sometimes called aerobic intervals, the main objective of tempos is to improve tolerance to racing at LT. Among athletes

who have plateaued relative to increasing peak V_{O_2}, tempos can improve race performance with little increase in peak V_{O_2} (1).

OTHER IMPORTANT TRAINING CONSIDERATIONS

Cross-training

Although some athletes use cross-training as a method to transfer training effects from one similar sport to another (e.g., in-line skating use in the summer among ice speed skaters), this is not always correct for elite level athletes in whom the principle of training specificity dictates they train within their sport. Among these athletes, any time spent in another sport may limit even the small amount of improvement in neuromuscular function or metabolic performance that is specific to their sport, and make the difference between winning and losing.

Among sedentary, lesser skilled, or less competitive athletes, cross-training provides an excellent diversion during the off-season, as well as resulting in transfer of athletic improvements from one sport to another. Ruby et al. (9) showed that running, cycling, or both activities combined resulted in similar improvements in peak V_{O_2} among sedentary women.

Having said this, the use of cross-training to facilitate recovery from injury or to lessen the occurrence of overtraining is useful among all athletes. Another practical example of cross-training is the use of swimming or cycling for the runner who strives to prevent major loss of cardiorespiratory function while he or she recovers from an ankle sprain.

Overtraining

Overtraining occurs when high-volume or high-intensity training (or both) is combined with inadequate recovery between workouts. An imbalance between training and recovery results.

Although both the volume of training required to produce an overtrained state and the consequences of overtraining can vary from one athlete to another, every attempt should be made to avoid overtraining because once it occurs, it may take weeks if not months for the athlete to recover. Milder forms of overtraining may result in fatigue, loss of appetite, periods of anger or frustration, menstrual irregularities, and depressed immune function. More advanced cases can also include insomnia and depression (10).

Athletic consequences of overtraining can include loss of muscle strength, loss of coordination, and decreases in peak V_{O_2} and peak power output. Biologic signs of overtraining are an increased resting heart rate or blood pressure relative to baseline, an increased heart rate or oxygen consumption during standardized submaximal work, muscle soreness lasting more than 24 to 48 hours, or a decrease in body mass.

Although the causes of overtraining are not well defined, it appears that both intrinsic and extrinsic factors can contribute. Associated intrinsic factors that have been identified include gender, age, and personality type (i.e., type A personality). Extrinsic factors are poor nutritional and sleep habits, intensity of training, total training volume, time of year, alcohol and drug use, frequent travel, and training history.

Common phrases often made by athletes who are overtrained are "feeling washed out," "feeling sluggish" and "just can't seem to get going" (11). Obviously, every attempt should be made to avoid overtraining, which means a coordinated effort between athletes, coaches, trainers, and parents. However, as long as any one of these groups remains obsessed with athletic success (especially the athlete), overtraining will remain a problem (12,13).

Taper

Across a variety of sports at the high school, college, and professional levels, the concept of taper is often employed. Most often used among swimmers and runners, tapering involves an approximate 80% to 90% reduction in training volume some 5 to 21 days before an event, with or without a reduction in training intensity. Such an approach has been shown to improve performance by as much as 3%.

EXERCISE PRESCRIPTION AND TRAINING IN PATIENTS WITH CLINICALLY MANIFEST DISEASE

Not all athletes are free of disease and some people with clinically manifest disease desire to train and participate in competitive sports. For these individuals, the aforementioned training principles and considerations generally apply. However, due to the pathophysiology of each disease, certain modifications or unique adjustments in the usual training guidelines may be needed. These considerations are listed in the following sections for several common disease states.

Heart Disease

Coronary Artery Disease

Following hospitalization for a myocardial infarction or coronary revascularization, it is important that stable patients initiate a regular exercise program. This can be accomplished by participating in a formal cardiac rehabilitation or secondary prevention program or training on one's own. The effectiveness of cardiac rehabilitation or exercise training on improving exercise tolerance, lessening symptoms, and improving mood is well established (14,15). Generally, patients can expect an approximate 15% to 35% increase in peak V_{O_2} or exercise capacity as a result of exercise training.

In addition, subsequent all-cause and cardiac mortality is reduced by approximately 25% over 3 years among patients participating in cardiac rehabilitation (14,15). Interestingly, patients who do not join a comprehensive risk-reduction cardiac rehabilitation program do not receive the same magnitude of benefit, inasmuch as an exercise-training-only programs (on their own or in a group) result in an approximate 15% decrease in all-cause or cardiac mortality.

As mentioned earlier, training principles for patients with coronary artery disease on standard therapy (aspirin, beta-adrenergic blockade) are really no different from patients free of disease. This is especially true for type, duration, and frequency of both aerobic and resistance training (see Table 11.5). Obviously, however, these patients need to train at an intensity that is free of myocardial ischemia and any angina or angina-like symptoms. Completing a graded exercise stress test prior to initiating a moderate-intensity exercise program is advised to help assess safety and guide exercise intensity (16).

In patients free of symptoms and ischemia, prescribing exercise intensity so that it is progressively increased up from 60% to 80% of heart rate reserve is satisfactory, regardless of beta-adrenergic blockade therapy. However, in patients with stable symptoms and/or evidence of ischemia during exercise, intensity should not exceed 10 beats below the heart rate where symptoms or electrocardiographic evidence of ischemia appears (17).

Heart Failure

A hallmark complaint of patients with heart failure is exercise intolerance and until the mid-1990s, exercise training was routinely withheld because of concern that the strain of exercise would worsen cardiovascular function. However, over two dozen randomized, small sample–sized trials conducted over the past 15 years have shown that when used in conjunction with optimal medical therapy, exercise training appears to safely improve exercise capacity between 15% and 30% (18). Quality of life is improved as well.

The mechanism(s) responsible for the increase in exercise capacity include improved central transport (increased peak heart rate, stroke volume, and cardiac output) to the metabolically more active skeletal muscles, improved regional blood flow due to improved endothelial-dependent vasodilatation, and partial normalization of the heart failure–related metabolic abnormalities known to occur in the skeletal muscles, such as mitochondrial density, percentage of type I fibers, and capillary density. To date, no large-scale, multisite trials exist evaluating the efficacy of exercise training on clinical outcomes in these patients.

Similar to patients with coronary artery disease, prescribing exercise in patients with heart failure is associated with few differences when

compared to recommendations for healthy people. For patients with ischemic cardiomyopathy, training intensity should not exceed 10 beats below any ischemia that may be evident. Because of the exertion-related fatigue that sometimes occurs at relatively low levels of work, the initiation of a training program may require an intermittent model, such as three 10-minute bouts with a rest in between each bout. As patients adapt and exercise tolerance improves, duration should be progressively collapsed to just one 30- to 40-minute bout.

Finally, skeletal muscle strength is reduced (19) in these patients, yet very preliminary evidence suggests that moderate-strength training helps partially restore exercise tolerance as well (20). Concerning the latter, three sets of ten repetitions using the knee extension machine improved both isokinetic leg torque and peak VO_2.

Peripheral Artery Disease

Although the benefit of regular exercise in patients with intermittent claudication due to peripheral artery disease is well established (21,22), many patients do not start or stay with this adjunctive therapeutic approach because of exercise-related symptoms.

Generally, a weight-bearing exercise training program improves total exercise time and time to pain onset by as much as 150%. Proposed mechanisms responsible for the improved exercise capacity include improved endothelial function and improved function within the aerobic metabolic pathways (21).

However, the methods used to achieve these improvements are not usually free of patient discomfort, for they require the patient to walk until she or he can no longer tolerate the pain. Following a brief rest to let the leg discomfort partially resolve, the patient should resume walking and repeat this pattern until a total of 30 to 40 minutes of exercise is completed.

Because exercise intensity is limited due to claudication symptoms, heart rate often does not increase to well within the 50% to 80% range that is usually prescribed. Therefore, two training bouts per week using a non–weight-bearing activity (e.g., stationary cycling) are recommended to elicit the heart rate response needed to derive the other physiologic benefits associated with exercise training.

Hypertension

Several excellent reviews have been published addressing the effects of exercise training on both normotensive and hypertensive patients. Generally, a mild to moderate exercise-training-induced decrease in blood pressure is observed in both groups, with the latter demonstrating the biggest decrease of 6 to 10 mm Hg for both systolic and diastolic pressures (23). There is no definitive evidence that resistance training provides a blood pressure-lowering effect. Instead, intense weightlifting remains a relative contraindication in patients with hypertension. Conversely, mild to moderate resistance training may be helpful in people interested in improving daily function and muscle strength and endurance.

Two key issues worth pointing out relative to prescribing aerobic exercise in these patients are the importance of regular exercise and the use of moderate- versus high-intensity training. It appears that exercise needs to be repeated within 24 to 48 hours to achieve the decrease in pressures indicated earlier. Also, training at 60% to 70% of heart rate reserve seems to provide the same, if not a greater, blood pressure-lowering effect when compared to training at higher intensities (23).

Hyperlipidemia

Contrary to popular misconceptions, regular resistance or aerobic training has literally no effect on lowering either total cholesterol or low-density lipoprotein cholesterol (24). Any change that is attributed to exercise is likely due to a change in body weight or eating habits. Having said this, triglycerides are decreased approximately 10% to 20% as part of a general aerobic training regimen, as shown in Table 11.5. Research addressing the effects of resistance training on blood cholesterol values has produced conflicting results, and at this time no comment can be made relative to recommending this type

of training as a means to help normalize blood lipid profiles.

With respect to high-density lipoprotein (HDL) cholesterol, it is true that regular exercise training may increase this negative risk factor for coronary artery disease. Several trials show that increasing total exercise up to 1000 to 1200 kcal · week^{-1}, for 4 or more months, is associated with an 8% to 10% increase in HDL cholesterol. This increase in duration may occur independent of any changes in exercise intensity.

Chronic Obstructive Lung Disease

Patients with emphysema, chronic bronchitis, and other forms of chronic lung disease do benefit from a program of regular physical training (25–27). Evidence from several randomized trials indicates that submaximal exercise tolerance, respiratory muscle efficiency, and mood are improved, as well as reductions in dyspnea with exertion and general and respiratory muscle fatigue. No definitive evidence exists relative to improvements in peak V_{O_2} or static or dynamic measures of pulmonary function.

Given the high frequency of inactivity in this patient population (28), it may be prudent to enroll eligible patients into a comprehensive pulmonary rehabilitation program (25). Doing so appears to provide additional benefits beyond exercise training alone, such as improvements in a patient's knowledge about the disease process, smoking cessation, breathing mechanics, administration of metered-dose inhalers, and self-management skills.

A contemporary example of the importance that thoracic surgeons and pulmonologists place on pulmonary rehabilitation is the recently completed National Emphysema Treatment Trial (NETT). In this study, subjects underwent 6 to 10 weeks of pulmonary rehabilitation before randomization to either medical treatment alone or medical therapy plus lung volume reduction surgery. After randomization, patients then participated in a maintenance pulmonary rehabilitation program.

Any discussion about exercise in patients with pulmonary disease would not be complete without mentioning the methods of prescribing exercise and the benefits of exercise in patients with either asthma or exercise-induced bronchoconstriction. With respect to exercise and asthma, of the many stimuli that can precipitate asthma-related symptoms, it is known that exercise is one of the more common. In fact, asthma symptoms induced from exercise may provide the first clue that an individual has the disorder.

Preventing asthma symptoms and exercise-induced bronchoconstriction requires an effective medical regimen and avoiding or controlling those environmental factors that can trigger symptoms. With respect to exercise, the latter may include instructing patients to train in warmer, more humid environments whenever possible. Also, because total ventilation (rate times tidal volume) may precipitate symptoms, it is sometimes necessary to restrict exercise pace as a means to reduce rate and depth of breathing. For example, this might involve asking a patient to continue a walking versus initiating a running or jogging program. Otherwise, once patients are symptom-free they can generally be exercise trained in a manner that is consistent with improving general health and athletic performance.

Obesity

The mechanisms by which exercise contributes to weight management are many and include an increase in energy expenditure, enhanced mobilization of fat, a small increase in postexercise metabolic rate, a minimization of loss of nonfat tissue during weight loss, and retardation of the decline in basal metabolic rate that occurs with calorie restriction (3).

Despite the benefits of exercise and popular belief, exercise alone is not an effective method for weight loss, but it does play an important role. For example, with no change in eating habits a program of modest walking five times per week for 30 minutes per bout should result in an approximate decrease in body mass of 10 to 14 pounds in 1 year. With respect to prescribing exercise, consider initially limiting exercise intensity to 60% of heart rate reserve, so that total duration can first be progressively

increased to 60 minutes per bout (29). This approach is designed to not only expend more calories but also minimize risk for activity-related injury.

With respect to mode of activity, low-impact exercise is preferred, such as swimming and stationary biking. Also, resistance training aimed at improving muscle strength and endurance should be included after the individual has shown the ability to tolerate a regular exercise training regimen.

Diabetes

Patients with type 1 or type 2 diabetes can benefit from participation in a regular exercise training program. In fact, the prescription of exercise in these two patient groups is quite similar. A general aerobic activity program involving walking ot biking should be performed for 20 to 60 minutes per bout, at a pace equivalent to 50% to 85% of heart rate reserve. Measuring heart rate to guide exercise intensity is not mandatory, given that patients can also train using ratings of perceived exertion (using the 6 to 20 scale, train at 11–13 on that scale).

Patients with type 1 diabetes and insulin-dependent type 2 diabetes should engage in aerobic training almost daily. Non–insulin-dependent type 2 diabetes patients should engage in aerobic training a minimum of four, preferably five, times per week. In both cases, consistency of training is important.

Resistance training is recommended for these patients as well; mild and moderate intensity training works well for nonathletes with type 1 and type 2 diabetes, respectively. Athletes with diabetes that is well controlled and without disease-related complications can do resistance training at higher intensities, as well as participate in anaerobic-type sports and activities.

The unique issues associated with training these patients often involve parameters other than frequency, intensity, duration, or type of exetcise. For example, insulin should be injected in a nonexercising site for those requiring insulin; check blood glucose levels regularly; practice good foot care; withhold exercising until the person consults a physician if blood glucose is greater than 250 mg · dL^{-1}; adjust insulin dose and/or consume 15 to 30 g of carbohydrates before (and during) exercise if needed; avoid exercising late in the evening to decrease risk of nocturnal hypoglycemia and maintain adequate hydration (30). These are a few of the factors that require individualizing of the medical therapy and exercise prescription for patients with diabetes.

CONCLUSION

The task of prescribing exercise in apparently healthy people, athletes, and patients with clinically manifest disease is actually quite similar. After an adequate assessment of physical fitness, the individual is prescribed the correct amount of *overload* stimulus that is *specific* to the sport or activity of interest.

The benefits and adaptations that occur in each of these different groups is similar as well. Proper training will result in the safe achievement of the fitness and performance goals set out by each individual.

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12

STRETCHING

ANGELA VITALE

INDICATIONS AND CONTRAINDICATIONS

Tight or shortened muscles are vulnerable to injury, especially when there is demand for repetitive use of a particular muscle as required in sports-related events. Tendiniris, the inflammation of a tendon, is usually the result of overuse. Just as muscles may benefit from lengthening with a safe stretching program, tendons too may benefit from stretching that is needed to combat initial trauma and prevent further inflammatory processes.

Stretching is a term used to describe manual or passive lengthening of soft tissue such as muscle or tendon that has been pathologically shortened. Stretching may be indicated for muscle tightness, adhesions from immobilization, and scar tissue adhesions from mechanical or chemical irritation (1). Stretching is as important to an athlete as strength training. Stretching of a muscle can assist in strengthening, help restore the normal pattern of movement and reduce the pain of musculoskeletal injuries, diminish a muscle spasm, or control muscle length in hypertonicity from a central nervous system (CNS) lesion.

Certain athletic activities, such as dance and gymnastics, require ranges of motion greater than those needed by the average person. Certain sports demand overstretching in order to participate. A ballet dancer that lacks good enough "turnout" may use a physiotherapist to improve this motion, usually through active training, warm-ups, and manual stretching techniques (1).

Although the benefits of stretching generally outweigh the risks, it is still important to remember that there is the potential for injury from "overstretch." Stretching could be harmful if a joint is unstable or hypermobile during activity. If the structures supporting the joint, that is, the muscles, ligaments, or capsule, have been damaged or compromised, then both therapist and trainer should demonstrate caution i.e. in the treatment of a shoulder dislocation. Stretching is often contraindicated or at least limited for a period of time, for example, during treatment of a Bankart repair of the shoulder or an Achilles tendon rupture.

Signs of injury from overstretch may include edema and inflammation of the involved area. Microtearing of the muscle fibers is possible with overstretch trauma and could result in hemorrhage and decreased ability to produce tension, especially the first few days following injury. In the anterior ribialis muscle of a rabbit, scar tissue developed when a stretch injury was induced in the elastic region of the load-deformation relation, while the resulting damage occurred near the myotendinous junction of this same muscle.

Prolonged joint pain or muscle pain lasting greater than 24 hours may be an indication of overstretching. Stretching is contraindicated at a fracture site that is immobilized and healing. Stretching should be provided with caution for individuals with osteoporosis. And individuals that have a chronic history of anticoagulant drug use or steroid use are also at potential risk of injury, if the stretching is too aggressive.

MUSCLE SPINDLE INFLUENCE

Muscle lengthening is necessary for the physiologic movement we observe and is controlled

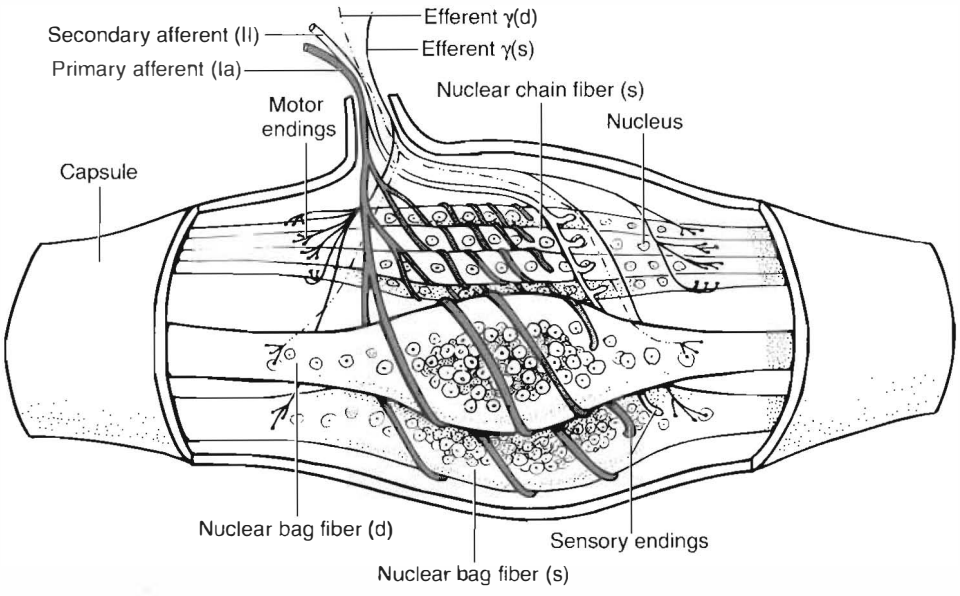

FIGURE 12.1. Anatomy of a muscle spindle, showing the chain fibers and nuclear bag. (From Shumway-Cook A, Woollacott M. *Motor control: theory and practical application*. Baltimore: Williams & Wilkins, 1995:53.)

by input into the CNS. The muscle spindle, a neuroreceptor within the muscle, stretches when a muscle is overstretched and provides information to the CNS that controls the length of the muscle (2) (Fig. 12.1). There are several muscle spindle receptors in a muscle and the spindle density varies from muscle group to muscle group. Muscle spindles are described as having intrafusal fibers enclosed in connective tissue capsules, which exist within the muscle bulk. The intrafusal fibers are arranged in parallel to the extrafusal fibers. The center of the muscle fibers is nonstriated and has a concentration of nuclei. The nuclei can exist within a central bunch (nuclear bag fibers) or as a central chain (nuclear chain fibers) (2). The ends of the intrafusal fibers are striated and can contract (3). Both ends of the spindle are attached to connective tissue of the muscle and therefore indirectly anchored to the muscle tendons (3).

The Ia and II afferent fibers of the muscle spindle both increase their firing rate as the length of the muscle increases. They decrease their firing rate as the length of the muscle decreases. The afferent fibers keep their firing rate constant if the length of the muscle remains constant. In addition to informing the CNS of change in muscle length, the Ia fiber also signals of the velocity with which the muscle lengthening is occurring (2). When a muscle is stretched too much, the muscle spindle is stretched as well. This leads to a contraction of the muscle and the spindle, and less overall stretch. This is the body's reflexive protection. The Ia afferent neuron of the spindle fibers sends a message that travels via the dorsal horn of the spinal cord. Sensory integration occurs between the neurons of the dorsal horn and the neurons of the anterior horn of the spinal cord. The gamma motor neuron of the anterior horn stimulates motor neuron output in the muscle. This causes an increase in muscle contraction that causes a decrease in muscle stretching (2).

Muscle spindles prevent muscle cells from stretching too much during an unexpected movement. The spindle's influence makes the muscle contract during these unexpected movements. This phenomena occurs automatically and protects the muscle from overstretching. During slow intentional stretching, however, the

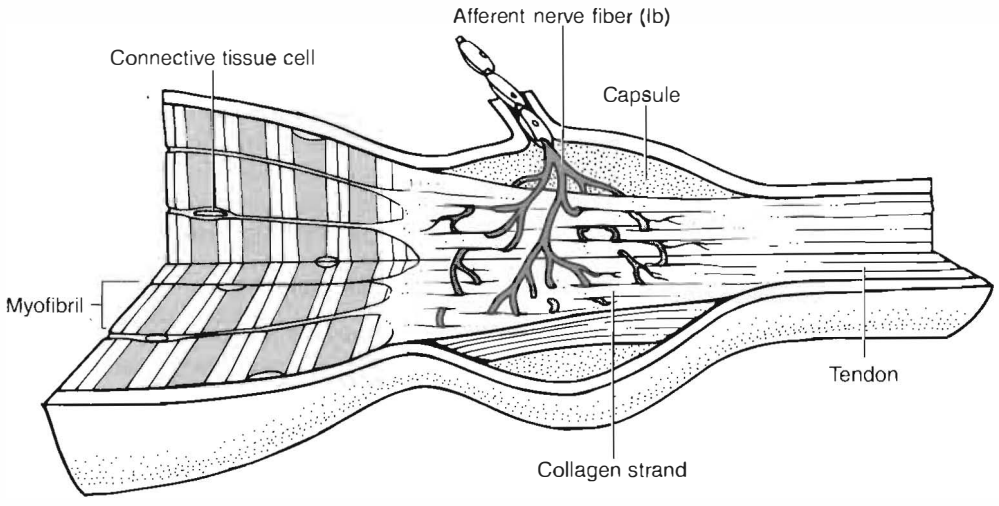

FIGURE 12.2. Anatomy of the spindle-shaped Golgi tendon organ (GTO) located at the musculotendinous junction and connected to 15 to 20 muscle fibers. (From Shumway-Cook A, Woollacott M. *Motor control: theory and practical application.* Baltimore: Williams & Wilkins, 1995:53.)

muscle spindle does not prevent lengthening of the muscle (4).

Another type of receptor within the muscle is the Golgi tendon organ (Fig. 12.2). The Golgi tendon organ, like the muscle spindle, is a somatic sensory receptor. These two receptor types have the ability to transmit information about muscle force, velocity, and length to the spinal cord, where the information is forwarded to the somatic sensory cortex or used in the spinal cord for reflex action (5). Unlike the muscle spindle, however, the tendon organ has no efferent innervation and it therefore cannot be controlled from the central nervous system. The construction of the Golgi tendon or tendon organ is not as complex as the muscle spindle. It consists of a sensory nerve fiber that follows a convoluted pattern among collagen fibrils near the musculotendinous junction. There are slightly fewer Golgi organs in a muscle than there are muscle spindles. The muscle spindle is influenced by muscle length and the Golgi tendon organ is influenced by tension.

In contrast to the muscle spindle, the tendon organ is coupled in series with the extrafusal muscle fibers. Active contraction of the muscle or passive stretch of the muscle and tendon increases the tension of the Golgi tendon organ. Experiments suggest, however, that the tendon organ is primarily concerned with signaling tension created when a muscle is contracting rather than when the muscle or tendon are being passively stretched (6).

THEORIES IN STRETCHING

The literature is replete with descriptions and photos of stretches. There is much information on the contractile nature of muscle fibers and the muscle spindle's involvement during an unwanted stretch. What happens physiologically when safe stretching is performed is not as well known or well documented.

An explanation of the therapeutic effects of stretching could be made based on factors that are known. During a muscle stretch, the origin and insertion of the muscle are pulled further apart. The sliding–gliding mechanism of actin and myosin filaments during contraction and relaxation may enhance circulation to surrounding tissue and muscle. Sliding and gliding of filaments through a particular range of motion may be necessary for normal healthy muscle. In addition, the lengthening and shortening of

these fibers with routine stretching and contraction may stimulate the dynamic processes including synthesis of adenosine triphosphate and other energy sources needed for movement.

Stretching may also be essential to maintain neural integrity between the afferent and efferent neurons of the CNS. The simple act of placing one's hands in position to do a stretch may be enough stimulus to excite the CNS. Studies by Brodal indicate the muscle spindle can be stimulated with vibration (6). This input to the CNS from receptor sites may interfere with pain as Melzack and Wall theorized with their gate control theory of pain. Stretching or lengthening a muscle from its origin to insertion sets up a chain of events that produces stimulation within the CNS and peripheral nervous system, and this stimulus, in addition to contributing to the obvious physiologic benefits we see, may be sensed by the brain and muscle as soothing or relaxing.

APPLICATIONS TO ATHLETICS

The influence of stretching on an athlete's physical performance can be significant. Basketball players should stretch prior to ballistic movements. Baseball, softball, and swimming coaches and trainers should encourage conditioning and stretching exercise for the shoulder. Track-and-field events should include prior stretching of most of the lower extremity muscles. It makes perfectly good sense that volleyball and tennis players do stretching of the upper extremity, lower extremity, and back muscles to provide movement that is needed during competition.

Warming up before stretching is strongly encouraged. This increases body temperature and circulation, which prepares muscles for activity, increases range of motion, and reduces stiffness. Heat may also be applied prior to stretching as long as the tissue is not acutely inflamed. Stretching after exercise is also advised as a cool-down, since this period is when the muscles are most flexible.

As long as serious pathology has been ruled out, stretching is indicated for muscles that are shortened or during muscle spasms. Stretching can be classified as therapeutic stretching or self-stretching. Stretching may be delivered by the health care professionals previously described or performed alone with little to no equipment.

TYPES OF STRETCHING

Static Stretching

Static or passive stretching is probably the most commonly used form of stretching. The muscle is gently lengthened to the point of resistance or slight discomfort and then is held in this lengthened position for a period of time. The stretching and lengthening should then be repeated several times for each muscle group. During this easy, held stretch, one relaxes and focuses attention on the muscles being stretched. The feeling of slight tension in the stretching muscle should slowly subside. Then one stretches a bit further, until the mild tension is again felt (never any pain), and held for 30 to 60 seconds.

Static stretching should be relaxing, and it should be done slowly. When static muscle stretching is performed correctly, it may provide immediate therapeutic benefits in a single session. Static stretching can be applied to most if not all sports. In addition to providing relief of muscle spasms that are healing after injury, static stretching can decrease muscle fatigue and soreness after a strenuous workout or competitive event. It is important to remember that this type of stretching should not be painful. Pain may suggest an improper stretch technique or a more serious medical condition that may warrant medical attention. Static stretching is most practical because it can be done independently, is less painful than proprioceptive neuromuscular facilitation techniques (PNF), and requires little time and assistance.

Dynamic Stretching

Dynamic stretching promotes dynamic flexibility and could be a useful warm-up for dynamic activities such as martial arts or dance. During dynamic stretching, the moving body parts

gradually increase in speed of movement or reach of movement, or a combination of both. The movements associated with dynamic stretches are not
stretches. Dynamic stretches do not include jerky or bouncing movements. Examples of this type of stretch are slow, controlled torso twists, arm swings, and leg swings.

Ballistic Stretching

Ballistic stretching uses the momentum of a moving body part in an attempt to force that part beyond its normal range of motion. Bouncing into or out of a stretched position uses the muscles as a spring, for example, repeatedly bouncing to touch your toes. The literature does not advocate this type of stretching, because it can lead to injury by repeatedly activating the stretch reflex.

Proprioceptive Neuromuscular Facilitation (the Contract/Relax Technique)

PNF is usually performed with a partner. A muscle group is stretched by first moving the joint to the end of its range of motion. Then the partner provides resistance as rhe same muscle group is statically contracted for about 5 to 7 seconds. Finally the partner moves the joint further into its range of motion. During this contract/relax technique, an isometric contraction of the muscle prior to stretching fatigues the muscle and this produces relaxation, which enhances a good stretch. PNF produces the greatest gains in flexibility, but according to the literatute it also causes more pain and stiffness than other techniques such as static stretching. PNF also requires a partner or health care professional to provide resistance for the contraction phase. Contract/relax is an excellent form of stretching, however, when there has been a chronic loss of movement or significant loss of movement. This is generally a technique that is used in rehabilitative services by a physical therapist or athletic trainer.

Active Stretching

During active stretching, the contraction of an agonist muscle assists in the lengthening of the antagonist muscle. Holding your leg up without any manual or mechanical assistance is an example of an active stretch. The hip flexors or agonists at the hip would actively contract, helping the hip extensors or antagonists to relax and lengthen. The meditational art of yoga generally applies such principles to its stretching techniques.

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13

CORE STABILIZATION IN THE ATHLETE

GWYNNE WATERS

OVERVIEW

The core is where the human body's center of gravity is located and where all movement begins. Core stabilization is particularly important for an athlete to achieve optimal performance. Whether the individual is competing at the school-age or an elite level, the athlete may experience pain symptoms only with functional overloading, while the pain may be absent or not disabling in normal daily activity. Additionally, injuries in other regions of the body can occur primarily because of poor core stability. However, treatment of injury is also different for the athlete, for complete functional recovery needs to take place in as short a period of time as possible (1).

All athletes are subject to the repetitive axial compressive and torsional forces required in athletic competition (1,2). Other factors involved include injuries due to collisions, the quality of the playing surface, the athlete's age and experience, and excessive physical demands of the sport (3). Many individuals have developed functional strength, power, neuromuscular control, and muscular endurance; however, few people develop the muscles required for spinal stabilization. Even with their peak conditioning, superior motor skills, and higher motivation, elite athletes have not only about the same incidence of back pain as the nonathlete population, but also have the same problems with activation of stabilizing muscles of the trunk (4). Athletes who train in one particular sport frequently, or who compete year-round without rest, may experience overtraining syndrome due to lack of definition of an optimal training zone and the limited ability of bone and connective tissue to quickly respond to match the demands of the sport. This has led routinely to arm, shoulder, and lumbar instability, chronic nonsteroidal anti-inflammatory (NSAID) use, and time loss injuries during the season (3).

THE CORE

The athlete's core is composed of the trunk, and the pelvic and shoulder girdles. The core operates as an integrated functional unit enabling the entire kinetic chain to work synergistically to reduce force load, dynamically stabilize, and generate force against abnormal forces. An efficient core allows for the maintenance of optimal length-tension relationships of functional agonists and antagonists, which makes it possible for the body to maintain optimum force-couple relationships.

All functional activities are multiplanar and require deceleration, dynamic stabilization, and acceleration. Movement may appear to be single plane dominant, but the other planes need to be dynamically stabilized to allow for optimum neuromuscular efficiency (1). Optimal articular range of motion, muscle strength and extensibility, stability, and the best automatic movement patterns possible must be present in these areas (5).

The trunk muscles must be able to hold the vertebral column in a stable position in order for independent upper and lower extremity movement to occur and to enable load to be transferred from the upper extremity to the ground. If extremity muscles are strong and the core is

weak, there will not be enough force created to produce efficient movements. A weak core is a fundamental problem inherent to inefficient movement that leads to predictable patterns of injury. The core musculature is an integral part of the protective mechanism that relieves the spine of the excessive forces during competition.

Athletes who participate in high-impact sports that require great physical strength need strong core musculature in order to generate sufficient force to play their position safely and absorb the impact of collisions. Football players and hockey players must be able to generate force quickly, while being able to perform highly coordinated movements. This is not possible without a strong base musculature, trained in a sport-specific manner. Throwing or racquet athletes require strength and neuromuscular coordination throughout their trunk, pelvic and shoulder girdles, and lower extremities to generate the needed force from their proximal to distal upper extremity. Golfers generate most of their power though the trunk and pelvic girdle, even though the successful golf swing is mediated through the upper extremities (2).

The core maintains postural alignment and dynamic postural equilibrium during functional activities, and relies on an efficient neuromuscular system. If the neuromuscular system is not efficient, it will be unable to respond to the demands placed on it during athletic endeavors. A strong and stable core can improve optimum neuromuscular efficiency by improving dynamic postural control. As the efficiency of the neuromuscular system decreases, the ability of the kinetic chain to maintain appropriate forces and dynamic stabilization decreases significantly. Decreased neuromuscular efficiency leads to compensation and substitution patterns, as well as poor posture during functional activities. These altered patterns lead to increased mechanical stress on the contractile and noncontractile tissue, and lead to repetitive microtrauma, abnormal biomechanics, and injury. Research has demonstrated that people with low back pain have an abnormal neuromotor response of the trunk stabilizers accompanying limb movement, as well as greater postural sway and decreased limits of stability (1).

Decreased dynamic postural stability in the proximal stabilizers has been demonstrated in individuals who have sustained lower extremity ligamentous injuries. It has also been demonstrated that joint and ligamentous injury can lead to decreased muscle activity. Articular or ligamentous injury can lead to joint effusion, which causes pain, which in turn leads to muscle inhibition and altered proprioception and kinesthesia. The result is altered neuromuscular control in other segments of the kinetic chain, destabilizing them and breaking down the kinetic chain (3).

CORE MUSCULATURE

The neuromuscular system must be able to stabilize the spine against shear in all directions (i.e., torque, traction, and compression) if the trunk is to remain stable during repetitive or forceful activities and be able to absorb the impact of collisions. Stability is dependent on three systems (4,5):

1. A control system (neurologic).
2. A passive or inert system (skeleral, including the spine, and pelvic and shoulder girdles).
3. An active system (spinal and trunk muscles).

Bergmark classified the lumbar muscles as either local or global, while Lee refers to these muscles as the inner unit and the outer unit (5,7,8).

The Local Stabilizers

Local stabilizing muscles tend to produce little movement due to their positioning, and their overall length changes very little during contraction. They tend to be monarticular and contract during both agonistic and antagonistic movements, especially during high-speed movement. They are deep muscles that attach to the inert structures of the joint (capsule and ligaments), and tend to be tonic rather than phasic muscles.

The main local spinal stabilizers are considered to be the following (5):

- Lumbar spine: transversus abdominis and multifidus

- Thoracic spine: sternocostalis and rotators
- Cervical spine: multifidus, rotators, longus capitis, longus colli, and semispinalis cervicis
- Pelvic floor: levator ani, puborectalis, iliococcygeus, ischiococcygeus

The inner unit is thought to be composed of the pelvic floor musculature, the lower multifidi, the transversus abdominis, and the diaphragm (5). The pelvic floor muscles have been shown to contract with the abdominals. If all of the abdominals are contracted, all of the pelvic floor muscles also contract. If specific abdominal muscles contract, the specific pelvic floor muscles they are paired with also contract. Pelvic floor musculature is capable of moving the sacrum into either flexion or extension, and can prevent sacral movement when co-contracting.

The Global Stabilizers

Global stabilizers are larger muscles that function primarily in an agonistic manner, providing for movement of larger joints and functional units. Excessive contraction of the global muscles may occur in patients with poor ability to activate their local stabilizers. The result can be low back pain, or symptoms elsewhere in the body, depending on the activity of the athlete. The global musculature includes (5):

Longissimus thoracis
Iliocostalis lumborum thoracis
Quadratus lumborum (lateral fibers)
Rectus abdominis
Internal obliques (some authors include the internal obliques as part of the innet unit)
External obliques
Erector spinae

Key hip musculature involved with core stabilization includes (6):

Gluteus maximus
Gluteus medius
Psoas
Adductor complex
Hamstrings
Quadriceps

Four global muscular subsystems are associated with movement of the trunk and limbs, and equalize external loads placed on the body. These muscles function as integrated functional units, and are important because they transfer and absorb forces from the upper and lower extremities to the pelvis. These subsystems are as follows (6):

1. *Deep longitudinal:* Erector spinae, biceps femoris muscles; also the thoracolumbar fascia and sacrotuberous ligament. This system allows for reciprocal force transmission longitudinally from the trunk to the ground.
2. *Posterior oblique:* Latissimus dorsi, gluteus maximus muscles; also the thoracolumbar fascia. This system works synergistically with the deep longitudinal subsystem.
3. *Anterior oblique:* Internal and external obliques, contralateral adductors, and hip external rotators. This system provides transverse plane stabilization and force transmission.
4. *Lateral:* Gluteus medius, gluteus maximus, tensor fascia latae, the adductor muscle complex, and quadratus lumborum.

The fundamental precept of core stabilization is that muscles function as an integrated unit. The central nervous system is designed to optimize the selection of muscle synergies, not isolated muscles. Muscles not only produce force (concentric contractions), in one plane of motion, but also reduce force (eccentric contractions) and provide dynamic stabilization in all planes of movement during functional activities (1,6).

Core Stabilization Mechanisms

Three mechanisms assist in providing core stabilization in the athlete (1):

Thoracolumbar stabilization mechanism
Intra-abdominal pressure mechanism
Hydraulic amplifier mechanism

The thoracolumbar stabilization mechanism relies on the thoracolumbar fascia, which is a network of noncontractile tissue that plays an essential role in the functional stability of the lumbar spine. Although these tissues are noncontractile, the fascia can be engaged dynamically because of the contractile tissue that attaches to it. These muscles include the deep

erector spinae, multifidi, transversus abdominis, internal oblique, gluteus maximus, latissimus dorsi, and quadratus lumborum. Contraction of the transversus abdominis and internal oblique creates a traction and tension force on the thoracolumbar fascia, which enhances the regional intersegmental stability in the lumbo-pelvic-hip complex. This contraction decreases translational and rotational stresses at the lumbosacral junction.

The intra-abdominal pressure mechanism decreases compressive forces in the lumbo-pelvic-hip complex. As the abdominal muscles contract, they push superiorly into the diaphragm and inferiorly into the pelvic floor. This results in elevation of the diaphragm and contraction of the pelvic floor musculature, and assists in providing intrinsic stabilization.

The hydraulic amplifier mechanism occurs at approximately 45 degrees of lumbar flexion when the electromyographic activity of the erector spinae decreases and "load-shifting" occurs to the noncontractile tissue and the eccentrically contracting gluteals and hamstrings. Potential energy is stored in these structures, which is then transferred into kinetic energy in the erector spinae during hip and trunk extension, reestablishing an upright posture. This is superimposed on an efficient thoracolumbar fascia mechanism. These stabilization mechanisms interdependently provide stabilization to the athlete's core.

Goals of Core Stabilization

Core stabilization is a concept that considers the integrated relationship between the legs, pelvis, trunk, and upper extremities. The aims of core stabilization are as follows (7):

1. Achieve localized segmental neuromuscular control.
2. Ability of the athlete to achieve and hold, isometrically, the position of power (neutral pelvis) or optimal stability.
3. Improve neuromuscular coordination between the trunk, pelvic, and shoulder girdles during changing movement patterns.
4. Improve the athlere's musculoskeletal and cardiovascular fitness and endurance.
5. Educate the athlete about what he or she can and cannot do, with regard to the particular injury or condition, if present.

Benefits of core stabilization training include improving dynamic postural control, ensuring appropriate muscular balance and joint arthrokinematics, allowing for the expression of functional strength, and improving neuromuscular efficiency throughout the entire kinetic chain. Manual therapy techniques are used to restore segmental and joint range of motion and decrease pain, reducing facilitation and inhibition of muscles and allowing better control of movement via appropriate concentric and eccentric work. If joint dysfunctions are present, they must first be addressed because they can inhibit the function of the surrounding muscles. Muscles can be inhibited due to pain, reflex inhibition, or disuse. Atrophy of multifidi muscles has been observed as a result of any of these causes. Pain and nonpain reflex inhibition must be reduced or eliminated before adequate activation and recruitment can occur. Core stabilization exercises provide for the education of intrinsic stabilizer muscles, thereby reducing stress on the anatomic restraints and providing a better base for the larger muscles to work from. Balance therapy reduces the amount of work the body must do to maintain stability over a constantly changing base of support. It is the clinician's job to ensure that the athlete has the following (or as optimal as possible) (5):

Pain-free status
Full segmental/articular range of motion
Segmental/articular stability
Normal muscle tone
Full muscle strength (isometric, eccentric, and concentric)
Full muscle extensibility
Normal balance
Normal movement patterns

Scientific Rationale

Most individuals, athletes included, do not adequately train their core stabilizers in comparison with other muscle groups. It is detrimental to perform exercises incorrectly or to perform

exercises that are too advanced for the athlete. Research has demonstrated the following (1):

- Decreased firing of the transversus abdominis, internal oblique, multifidi, and deep erector spinae has been noted in individuals with chronic low back pain.
- Abdominal training without proper pelvic stabilization increases intradiscal pressure and compressive forces in the lumbar spine.
- Hyperextension training without proper pelvic stabilization can increase intradiscal pressure to dangerous levels, cause buckling of the ligamentum flavum, and lead to narrowing of the intervertebral foramen.
- Individuals with chronic low back pain demonstrate decreased stabilization endurance.
- Individuals with low back pain demonstrate decreased cross-sectional area of the multifidus. The multifidus did not spontaneously recover following resolution of symptoms. Traditional curl-ups increase intradiscal pressure and increase compressive forces at L2-3.

The core stabilizers are primarily type I slow-twitch fibers that respond best to time under tension muscle contraction. The contraction lasts for 6 to 20 seconds and emphasizes hypercontractions at rhe end ranges of motion. This method improves intramuscular coordination, which improves static and dynamic stabilization. Core strength endurance must be trained appropriately to allow an athlete to maintain dynamic postural control for prolonged periods of time (1). It is important for the cervical spine to maintain a neutral position during core training as this will improve posture, muscle balance, and stabilization. Research has also demonstrated increased electromyographic activity and increased pelvic stabilization when an abdominal drawing-in maneuver is performed prior to initiating core training (1). This maneuver is described later in the chapter.

CORE STABILIZATION TRAINING GUIDELINES

Following a kinetic chain assessment, the clinician must address any muscle imbalances and arthrokinematic deficits that are discovered prior to beginning an aggressive core training program with any athlete. The following training guidelines are based on the optimum performance training method developed by the National Academy of Sports Medicine.

Program Design

The core stabilization program should be progressive, systematic, activity specific, integrated, proprioceptively challenging, and based on current science (1). During stabilization exercises, the athlete is taught to specifically recruit the trunk muscles isometrically and then to maintain this brace as he or she moves the upper and lower extremities independently. Initially, the base of support is very stable. The program is progressed by increasing the level of difficulty, by reducing the base of support, by making the base more unstable, and by increasing and changing the load that must be controlled. It is important to make the exercise program multiplanar and multidimensional, to use the entire muscle contraction spectrum, to use the entire contraction velocity spectrum, and to manipulate all acute training variables (sets, repetitions, intensity, rest intervals, frequency, and duration).

The core training program must address movements in the frontal, sagittal, and transverse planes, plus a combination of all three planes. Varying body positions can include supine, prone, side lying, sitting, kneeling or half-kneeling, and standing. The base of support can be varied, for example, a chair or exercise bench, a stability ball, or other balance modality. The lower extremity stance should be varied and progressed: two legs, two legs staggered stance, single leg, two legs unstable, staggered stance unstable, and single leg unstable. The upper extremity progression should also be varied: two arms, alternate arms, single arm, and single arm with rotation. Forms of external resistance can include barbells, dumbbells, cables, tubing, medicine balls, power balls, Bodyblade, and so on. Balance modalities that can be used to challenge the individual include progression from a stable surface (floor or chair) to

a sport beam, Airex Pad, Dyna Disc, BOSU, Proprio shoes, and sand, to name a few.

The athlete should begin working in the most challenging environment he or she can control. The athlete is progressed through the program as mastery of the exercises is achieved, maintaining stability and optimum neuromuscular control. The exercises the athlete performs should be safe, challenging, progressive, proprioceptively enriched, and sport specific. The core training program can be manipulated regularly by changing the plane of motion, range of motion, loading parameters (stability ball, ball, weight vest, dumbbell, tubing, etc.), body position, amount of control, speed of execution, amount of feedback, duration (sets, repetitions, tempo, time under tension), and frequency.

The athlete is progressed by advancing the exercise components from slow to fast, from known to unknown, from a stable environment to a controlled environment to a dynamic functional environment, from low force to high force, and by emphasizing correct exercise execution with increasing intensity. Again, the goal of the program is to develop optimal levels of functional strength and dynamic stabilization. Neural adaptations are the focus of a core stabilization program rather than achievement of absolute strength. Quality of movement is stressed over quantity. The clinician who allows the athlete to train with poor technique and poor neuromuscular control may cause the development of poor motor patterns and poor stabilization. The focus of the program must always be on function.

From Small to Large

It is important to ensure that the athlete is able to recruit the inner unit or local stabilizers in a neutral pelvic position prior to beginning a core stabilization program. If this is not done, reinforcement of faulty postures and facilitation of abnormal movement patterns may predispose the athlete to injury in the future. Activation of the multifidi and transversus abdominis muscles is the initial building block of any core strengthening program.

Transversus Abdominis

In the hook-lying position, the athlete is asked to perform a drawing-in maneuver to bring the navel toward the spine. The pelvis is optimally in a neutral position, but if pain is a consideration, an anterior or posterior tilt may be incorporated, especially in the early stages of exercise. The athlete must not demonstrate the following compensations (5):

- Lumbar kyphosis or lordosis, or pelvic or thoracic movement; this indicates the inability to activate the transversus abdominis independently of the other abdominals. If this global recruitment occurs later, then fatigue may be the issue.
- Failure to perform the maneuver, delayed global recruitment, or shuddering; this indicates abnormal fatigability.
- Loss of normal breathing pattern or loss of abdominal draw; this indicates the inability to use the transversus abdominis independently of the diaphragm.

The athlete may find it easier initially to perform contraction of the transversus abdominis in four-point kneeling. In this position, the weight of the abdominal contents provides a stretch to the abdominal wall and may facilitate the contraction by increasing the athlete's awareness of the muscle and its contraction. If the athlete is able to activate the transversus abdominis independently, he or she is asked to maintain a contraction for 5 to 10 seconds while continuing to breathe normally.

If the athlete has a difficult time understanding how to contract the transversus abdominis, a pressure feedback cuff may be used in either supine or prone position to assist in providing him or her with feedback. In the prone position, the cuff is placed under the abdominals, and in the supine position, the cuff is placed under the lumbar spine. In prone, the cuff is inflated to 70 mm Hg while the athlete is asked to lift the abdomen off the cuff while continuing to breathe normally. If the pressure is reduced 6 to 10 mm Hg successfully, contraction has occurred. In supine, the cuff is inflated to 40 mm Hg. Again, the athlete is asked to perform the

muscle contraction. He or she is asked to perform specific and progressive loading tasks while maintaining a steady pressure under the spine. Increases in cuff pressure (posterior pelvic tilt) or decreases in cuff pressure (anterior pelvic tilt) indicate a loss of stabilization.

Joseph Pilates developed a series of exercises that focus on increasing core strength, with emphasis on the individual's ability to perform the abdominal drawing-in maneuver to facilitate isolated contraction of the transversus abdominis. The cue "navel to spine" is frequently used while teaching exercises based on his method. Pilates classes are very popular today, and are used in both the rehabilitation and fitness arenas. Certified instructors teach exercises on the mat or the Reformer, and also use other aids such as the chair, the barrel, their Fitness Circle, and tubing.

Multifidi

The lumbar multifidi are important local stabilizers of the spine. The muscles are innervated segmentally. If the multifidi need to be re-educated in the athlete, palpation of the specific muscle in the paraspinal gutter will assist in determining if any atrophy has occurred, or if the area is tender. In prone, the athlete is asked to "swell" the muscle against the clinician's palpating finger. Facilitation by rapid stretching of the muscle by sudden, deep palpation may be necessary. The multifidi work synergistically with the transversus abdominis, so if the athlete is unable to activate these muscles volitionally, they may be activated in concert with the transversus abdominis. Electrical stimulation to the local muscle may also be of assistance (5).

Exercise Progression

Once the athlete is able to demonstrate isolated and sustained contraction of the local stabilizers with normal breathing and without overactivity of the global muscle system, and in any posture, he or she is ready to begin restoration of optimal movement patterns, or functional rehabilitation related to the specific sport. Volitional contraction of the local stabilizers must be transformed into an automatic load that is being put through the spine. Lighter loads and less complex movements are trained first.

Initially, the muscles are trained under light loads and static conditions. Isometric contractions in a variety of positions are designed to improve intrinsic stabilization and provide optimum neuromuscular control for the core. Gradually, the isometric stabilization exercises are replaced with dynamic concentric and eccentric activities throughout the full range of motion. The athlete is asked to move the arms and legs in functional directions while maintaining the abdominal draw in the pelvic neutral position. He or she must maintain control during active, and then resisted, concentric and eccentric muscle contractions. The specificity, speed, and neural demand of the exercises are progressed. Total kinetic chain neuromuscular efficiency is enhanced by providing maximum proprioceptive stimulation to the central nervous system during integrated functional movements while maintaining optimum stabilization of the entire core. Resistance from a variety of directions is initiated in supine hook lying, prone, and four-point kneeling positions. Once the athlete masters these positions, he or she assumes the sitting position and remasters the exercises. As previously mentioned, positions that may be used in training include

Supine lying
Side lying
Four-point kneeling
Two-point kneeling
Sitting
Standing
Walking
Running

Exercises that may be incorporated include

Supine trunk and pelvic rotation
Supine arm and leg
Supine double leg bridge
Four-point kneeling arm and/or leg extensions
Sitting leaning
Balance ball

Arm and leg extensions
Trunk rotations
Wobble board
Standing
Arm and leg extensions

Normal breathing and neutral spine posture must be maintained. The athlete can be challenged while walking or running, progressing to sport-specific activities. Swiss balls, wobble boards, inclined planes, discs, pads, and the like can all be incorporated into the program, thereby decreasing stability and increasing muscle recruitment to challenge the athlete as he or she masters successive activities.

Once the athlete demonstrates optimal neuromuscular control with a variety of movements, the training focus changes to the performance of sport-specific movement patterns. This incorporates more normal movement such as proprioceptive neuromuscular facilitation patterns. Sudden changes in direction, velocity, and contraction type (concentric, isometric, eccentric) are demanded from the trunk, arms, legs, and head. The athlete performs specific exercises at a similar intensity and similar rate of force production that is expected in his or her sporting environment.

CONCLUSION

All athletes should train their core through a functional exercise program to assist them in obtaining optimal physical performance as well as injury prevention. Specific functional exercise progressions are most beneficial, with the athlete mastering contraction of the inner unit muscles in a pelvic neutral position before progressing to more demanding exercises that incorporate the outer unit muscles and the entire body. Sport-specific core training exercises will provide the athlete an opportunity to use all body structures in a controlled environment before being subjected to the competitive environment. Faulty postures and poor mechanics should always be avoided. An athlete's core can nevet be too strong.

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14

THROWING PROGRAM

STEVE SCHER

OVERVIEW

When throwing athletes are ready to return to play after injury or time off, it is necessary to provide them with steps for a gradual return. Due to the torque and stress placed on the body during throwing, athletes cannot start throwing hard or long right away; there needs to be an acclimation period. This is the primary reason for the use and implementation of a throwing program.

Throwing distance, velocity, accuracy, and control can be effectively regulated and progressed when returning from an injury. Failure to do so may be the difference between successful return to throwing and failure of the athlete's recovery. Interval distance throwing progressions were designed to allow safe return through controlled increases in distance as the athlete's body adapts to the demands placed on it during the throwing motion. Interval distance throwing also allows for gradual increase of joint loads, thus protecting the joint from pathologic problems as a result of excessive strain too early in recovery.

DESCRIPTION

Throwing programs typically begin 4 to 6 months after a major surgical procedure and consist of interval distance throwing and specialized exercises designed specifically for throwers. For nonoperative injuries, the throwing program may begin earlier, based on physician approval, negative results from a clinical examination, sufficient range of motion, neuromuscular control, and demonstrated success with functional exercises (e.g., plyometrics). As an athlete returns from either surgery or injury, he or she must also exhibit normal strength of the rotator cuff and accessory muscles.

Over the years, various throwing programs have been developed and modified. At the professional, collegiate, and youth levels of sport, athletic trainers, therapists, and physicians have adapted their own specific exercises and versions of the throwing program progression. Many resources were used in the development of the throwing exercise program presented in this chapter, with a basic foundation of the Thrower's Ten exercises outlined by Kevin Wilk and James Andrews (1).

Modifications to the program were made to account for the skill level of the athlete and injury status, allowing for variation depending on the status of the throwing athlete. Commonly, the rehabilitation consultant or athletic trainer will change distances slightly and tailor the exercises to the needs of each individual athlete in accordance with physician consent. Because the sports medicine health care team will frequently add or subtract components of the protocol depending on the athlete's injury or rehabilitative status, frequent communication and consultation between the members of the sports medicine health care team is essential. This communication will ensure the athlete's optimal recovery and return to play.

Conditioning

One important element of the throwing program is the need for conditioning the entire athlete, not just the shoulder and scapular region. The time to rehabilitate an athlete after an elbow surgical procedure such as a medial collateral ligament reconstruction can go beyond

1 year, so that time is when the athlete should be doing all he or she can to condition the rest of the body to avoid breakdown and compensatory injury. Most throwing injuries occur when throwing while the body is fatigued (2), so improving the ability to avoid or lessen fatigue should be mandatory for such an athlete.

ELEMENTS

A throwing program should be functional and have a transitional approach involving game-simulated activities. Early rehabilitative exercise stages are often specific and direct in protecting the healing tissues. However, controlled functional patterns can be added effectively in rehabilitation to initiate crossover to the sport and transition for the throwing athlete. For example, the arm is often put in a sling for protection and healing after shoulder surgeries. Shoulder dumping is a great way to incorporate early throwing movement while keeping the arm in a sling. The shoulder dump is executed by dropping the shoulder across the body to the opposite leg.

Functional exercises are selected in order to facilitate a strengthening process with sport-specific movement patterns that approximate those occurring in the throwing activity. Basic static exercises (e.g., external rotation with the arm at the side) are useful in early rehabilitation stages, but they do not involve transition ro the dynamic requirements needed in throwing. For these reasons, distance throwing is an essential component for returning ro optimal performance.

To date, few research studies have evaluated the importance of proprioception (joint sense) of the shoulder for throwing athletes (3–6). Proprioception training is an important aspect of the throwing program and imperative to the success of any athlete. Proprioceptive and neuromuscular exercises are also necessary for an overhead athlete returning from injury. Proprioceptive receptors, called mechanoreceptors, are damaged with any soft tissue injury. These mechanoreceprors communicate joint position and motion to the brain. The throwing motion involves a substantial amount of movement secondary to the range of motion provided by the shoulder. Thus, the athlete needs to be able to sense the position of the arm, forearm, and hand before release of the ball in order to execute a successful throw.

Stabilization and plyometric exercises that provide change in tension, position, pressure, and rate of movement to the muscle-tendon junction are essential for proprioceptive training and complete return to sporr. Stabilization exercises of the shoulder allow the athlete to respond to change in movement and use cocontraction of the rotaror cuff muscles. For example, the clinician can modify the rhythmic stabilization of external and internal rotators by having the athlete perform the exercise with his or her eyes opened or closed.

A throwing program has three basic components:

1. Warm-up phase
2. Interval distance throwing regimen
3. Series of supplemental exercises to maintain shoulder, elbow, wrist, and core strength

The throwing program should always begin with a proper warm-up phase. Athletes should begin the warm-up phase with a short toss or light catch exercise. Athletes should always include cardiovascular activity for 5 to 15 minutes, such as a light run, bike ride, or use of an upper body ergometer. Due to the extreme load of decelerating the arm, the posterior rotator cuff and capsule become tauter during throwing activities. Therefore, the warm-up includes upper extremity stretching of the posterior rotator cuff, posterior capsule, pectoralis minor, and latissimus dorsi muscles. Shoulder instability should be ruled out before initiating posterior rotator cuff and shoulder capsule stretches.

Overall athletic conditioning should be a significant portion of any program (2). Throwing requires a large demand on the trunk and arm muscles to get the ball to its target. Proximal muscular control is essential for a baseball player to throw—"proximal stability for distal mobility." Conditioning should focus on core strength, as the trunk and gluteal muscles must be trained as the base of support for integrated arm movement. Once proximal control is achieved, then progression to distal muscle

control can be initiated. This concept is the foundation of the kinetic chain principle allowing a transfer of energy from the feet, to the legs, to the trunk, through the scapula and arm, and finally into the forearm and hand, resulting in ball release (7). It is essential that a good core program be developed in accordance with the athlete's exercise regimen.

BASIC PROGRAM

Both basic and advanced interval distance progressions are outlined in the following text and tables. Basic distance throwing progression (Table 14.1) starts with a shorter distance, is less rigorous, and is more suited for the youthful and occasional thrower. The advanced distance

TABLE 14.1. BASIC INTERVAL DISTANCE THROWING PROGRESSION

Throwing will be performed every other day.
Each stage should last approximately 1 week.
If pain occurs during any stage, return to the previous stage.
Continue pre- and postexercises on throwing days; allow time for recovery.
Distances may be adjusted for younger athletes (see youth interval section in text).

Stage 1 45-ft *Week 1* (M, W, F, and Sun)
1. Warm-up throwing
2. 45-ft (15 throws)
3. Rest 15 min
4. Warm-up throwing
5. 45-ft (15 throws)
6. Rest 15 min
7. Warm-up
8. 45-ft (15 throws)

Stage 2 60-ft *Week 2* (T, Thurs, and Sat)
1. Warm-up throwing
2. 60-ft (15 throws)
3. Rest 15 min
4. Warm-up throwing
5. 60-ft (15 throws)
6. Rest 15 min
7. Warm-up
8. 60-ft (15 throws)

Stage 3 90-ft *Week 3* (M, W, F, and Sun)
1. Warm-up throwing
2. 90-ft (15 throws)
3. Rest 15 min
4. Warm-up throwing
5. 90-ft (15 throws)
6. Rest 15 min
7. Warm-up
8. 90-ft (15 throws)

Stage 4 120-ft *Week 4* (T, Thurs, and Sat)
1. Warm-up throwing
2. 120-ft (15 throws)
3. Rest 15 min
4. Warm-up throws
5. 120-ft (15 throws)
6. Rest 15 min
7. Warm-up
8. 120-ft (15 throws)

Note: Each stage can be performed at 50%, 75%, and 100% intensity.

TABLE 14.2. ADVANCED DISTANCE PROGRESSION

Stage 1: 60-ft (50%–75%)	
a. Start with	60-ft 1 × 15 throws
b. Days 2/3	60-ft 2 × 15 throws
c. Days 4/5	60-ft 3 × 15 throws
Stage 2: 60-ft (on arc)	
a. Maintain	60-ft 3 × 15 throws
90-ft (50%–75%)	90-ft 1 × 15 throws
b. Days 9/10	90-ft 2 × 15 throws
c. Days 11/12	90-ft 3 × 15 throws
Stage 3: on arc	
a. Days 14/15	60-ft 3 × 15 throws
b. Days 15/16	90-ft 3 × 15 throws
120-ft (50%–75%)	120-ft 1 × 15 throws
c. Days 17/18	120-ft 2 × 15 throws
d. Days 19/20	120-ft 3 × 15 throws
Stage 4: 90-ft (on line)	
a. Days 21–23	90-ft 3 × 15 throws
b. Days 24–27	120-ft 3 × 15 throws

1. Days are estimates.
2. Pitchers can begin off-speed work after 120 ft.
3. Progress to 150 ft for catchers.

Note: (1) After 120-ft or stage 3, begin 60-ft or 90-ft on-line and increase intensity (80–100%).
(2) Mound progression—45- to 60-ft on level ground using wind-up. Allow more time for recovery when beginning from the mound.

throwing progression (Table 14.2) is longer in distance, more intense, and designed for the more avid or elite thrower. Both programs can be adjusted as necessary and tailored to the athlete's postinjury status and position played.

An interval distance based progression is designed to start the athlete with short distances and low throwing intensities. As the distance increases, so do the demands of endurance and strength. The position the athlete plays and injury status will determine the distance of the program; for instance, a first or second baseman may not need to throw 150 ft. The basic progression starts the athlete at 45 ft, while the advanced progression starts at 60 ft. Usually, the thrower is asked to start throwing the ball on an arc, rather than on a straight line, which allows less force and intensity to be placed on the shoulder and elbow. Typically, there are four stages based on distance. Each stage should last approximately 1 week. However, stage duration ultimately depends on injury status, pain, in-season versus off-season training, and any changes in throwing mechanics. During each stage, throwing is performed in three sets for both basic and advanced progressions.

The basic progression initiates the distance throwing with three sets consisting of 15 throws in each set, at 45 ft in length. A warm-up and a 15-minute rest period between each set are both part of the program. The first stage lasts for approximately 1 week, and the throwing is completed every other day. Pre- and post-thrower's exercises are also completed after throwing. In week 2, the thrower starts throwing at 60 ft. By the third week, the athlete throws from 90 ft, then 120 ft by week 4. The interval distance progression component of the throwing program provides excellent opportunity to critique the thrower's mechanics. Mechanics are vital to

throwing athletes returning from injury and should be assessed. It is advantageous to use a camera to provide visual feedback to the thrower. Mechanical changes made for injury prevention purposes should be determined after consultation with a pitching coach.

At the beginning of the first stage in the advanced progression, one set of 15 repetitions is completed followed by the pre- and postexercise routine. Each stage consists of a one day on and one day off rotation. The advanced progression has a buildup of three sets at each stage. Each stage increases distance and velocity of the ball. The velocity varies by throwing the ball in an arc or on a line. Day 1 consists of the first stage and pre- and post-throwing exercises. On day 2, the athlete is allowed to rest and recover. This cycle of every other day continues until completion of the program. Allowing recovery time yields an opportunity for the muscles to adapt to the stresses of throwing. On the third day of throwing, two sets of 15 repetitions, throwing the ball on an arc are completed. Between each set, the athlete is provided with 5 minutes of rest.

When progressing to the next distance or stage, the athlete starts each session with the previous distance. Hence, the thrower will complete three sets of 15 repetitions at 60 ft before completing one set of 15 repetitions at 90 ft. This means the thrower will throw 60 times in that session. Allow the athlete to throw at 50% to 75% of his or her maximum intensity at the first two distances (60 ft and 90 ft), which allows gradual increased demand on the athlete's arm. When the athlete reaches 120 ft, it is advised to discontinue the throwing repetitions at 60 ft. Simply continue the cycle at 90 ft with three sets of 15 repetitions before 120-ft sets are started.

For the advanced progression, once stage 3 is reached, the thrower will be physically challenged by eliminating the arc pattern throws and starting on-line throws. This progression increases the intensity and velocity demands of the thrower. The distance progression can reach up to 120 to 150 ft based on position played. It is advisable to have outfielders and catchers throw to 150 ft secondary to their total throwing distance demands. Once the athlete reaches 120 ft, the athlete is throwing a total of 75 to 90 times with the advanced progression.

When an athlete throws 120 ft on-line without complaints, game simulation can take place. Throwing athletes often have to throw off-balance and on the run, putting increased stress on joints. The game simulation drills will help the athlete with a variety of these types of plays and throws. It is recommended to provide reduced pitch count for pitchers returning from injury and gradually increase innings or total throws over many games.

Mound Progression

Returning to pitching is often challenging when the athlete has been away from the game for a while. Setting goals each day or week is helpful when returning from injury. Most pitchers who return to the mound from a surgical procedure are initiating throwing activities at 4 months and mound work at 6 month postoperatively. At 7 months, full velocity throwing can be initiated. This progression can last as long as 10 to 12 months before the athlete is game ready, depending on the diagnosis. An ulnar collateral ligament reconstruction can require up to 12 months of rehabilitation.

During the mound progression, there is an increase in distance each session. Each session, repetitions increase by 15 throws, allowing ample time for recovery. Before progressing to the mound, begin with fastballs at 15 to 30 ft from flat ground. It is optimal to allow the pitchers to feel where their arm, hand, and the ball are in space during the windup and delivery to reeducate form and proper mechanics. This proprioceptive input is mandatory for a successful return to sport. Have the pitcher complete his or her windup with good balance and slow pace to reproduce proper form. Wilk et al. (8) state that pitchers should start at 60 ft on level ground as they begin to increase their pitching velocity. The mound increases the forces on the body and the shoulder, and the flat surface allows the pitcher to avoid those forces.

The mechanics of a pitcher have additional components secondary to the windup. Pitchers have a longer delivery than other position players. Windup training should consist of practice in both full and stretched (or half of a full windup) positions. It helps to have a pitching coach present fot monitoring and tutoring on pitching mechanics. Warm-up throwing consists of 90 to 120 ft until the pitcher feels loose and ready. Often this warm-up phase is considered as a long toss. Progress distance from the mound after warm-up throwing is completed. Put the pitcher in gamelike situations (with a batter) as he or she progresses to full distance, from the rubber to home plate. In a recent American Sports Medicine Institute study, when pitchers attempted to throw at 50% and 75% of their maximum velocity, use of the radar gun revealed they actually were throwing at greater speeds than intended. Therefore, use of a radar gun will help to provide feedback of throwing intensities for a pitcher recovering from injury.

YOUTH INTERVAL THROWING

Little leaguer's throwing progression is different from the adult thrower's progression. The primary difference results from the different size and distances of the baseball diamond. Another difference is the consideration for the growth and strength development of young throwing athletes. One study (9) investigated the distances that a little leaguer needs to throw to improve arm strength and recover from injury, and described interval progression reasonable for young children. It also outlined the difference between short-toss and long-toss parameters, stating that short toss is used specifically for the demands that are game situational. The long-toss program is more suited to endurance and long-duration type training. Lyman et al. recommend a pitch type of fastball and change-up or off-speed pitches for young throwers (10). The curve ball and slider are too aggressive and have the potential to cause stress type injuries. Studies by Lyman (10) and *USA Baseball* (11) had similar results when looking at the appropriate age to start developing different pitches. It is recommended that the fastball should first be taught at the age of 8 ± 2 years, then the off-speed pitch at ages 10 ± 2 years, and finally, the curve ball and slider at 14 and 16 ± 2 years, respectively (10,11).

Besides type of pitch, the number of pitches thrown in a game influences the chance of injury for a young thrower. When evaluating the safety of pitching in young athletes, pitch limits should be examined rather than the amount of innings pitched regarding increased injuries for youth players. The same *USA Baseball* study (11) noted a pitch count limit by age: 8- to 10-year-olds, 52 ± 15 pitches; 11- to 12-year-olds, 68 ± 18 pitches; and 13- to 14-year-olds, 76 ± 16 pitches. Pitching mechanics also play a vital role in the prevention of shoulder injury at any age (12).

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SECTION

III

REGIONAL EVALUATION AND TREATMENT

15

PHYSICAL EXAMINATION OVERVIEW AND ESSENTIALS

STEVEN J. KARAGEANES

The physical examination is a crucial tool used by the sports medicine clinician to diagnose musculoskeletal injury. In skilled hands, the examination can confirm a diagnosis without standard imaging studies like magnetic resonance imaging, computed tomography, and musculoskeletal ultrasound. However, it is only one component of the athlete's evaluation, and it requires skill and experience to use effectively and confidently. Each part of the assessment is limited in scope; only when everything is put together does the most accurate diagnosis become clearer to see. Thus, an integrated approach is strongly advocated for any sports medicine professional.

HISTORY

All physical examinations in sports medicine start with the history. Exactly how one obtains the history has been debated for years and there is no consensus. Some follow an established set of questions covering predetermined content (1), while others follow a logical problem-solving approach (2). Other approaches fall in between, but there is no debate that the history is the most important part of the examination. It aids the sports medicine clinician in making the diagnosis, planning a therapeutic strategy, provides a basis for predicting the course of recovery, and establishes a baseline against which future progress is evaluated. Nowhere is the history taking more necessary than in the realm of athletic performance, where progress is assessed, qualified, and quantified with stunning swiftness and precision.

Gathering the history in athletics is different than in the office setting. Most athletes should already be assessed in the preseason during their pre-participation physical examinations. Any acute injury that occurs can be compared to a recent established baseline assessment of the athlete's health, and further history taking can target more detail. However, many times a baseline examination was not done, and the clinician must work from the beginning.

Even when an athlete is acutely injured, for example, on the football field, the physician and trainer should always try to get an accurate history as quickly as possible, even if they witnessed the event. History gathering should be direct and specific to the injury, in order to recognize and expediently treat any severe injuries. For an athlete injured on the field, time should be taken from the competition to ascertain the level of severity. Only when the severity of the injury is established should the athlete be removed from the playing field. In many cases, the diagnosis can be made before the examination or reduced quickly to two or three possibilities. In most cases, the physical examination is limited without a thorough history of the athlete on record.

In the acute setting, the sideline evaluation may be done in many different environments—football fields, hockey arenas, basketball courts. The decision whether to examine the athlete on the field, on the sidelines, or in the locker room is based on several factors. If there is any question about instability, particularly of the spine, it is appropriate to complete the examination on the field until the athlete is appropriately

stabilized or transferred to a location where he or she can be stabilized.

The sports medicine clinician should seek out answers to the following questions in the acute setting:

1. Is the athlete responsive and alert? (the ABCs—airway, breathing, and circulation—of advanced trauma life support)
2. Is the athlete in significant pain?
3. What was the mechanism of injury? (The clinician should ask the athlete this question, even if he or she witnessed the injury.)
4. Where is the pain or disability located?
5. Can the athlete move the injured body part?
6. Can the injured body part function properly?

The responses to these questions quickly indicate the severity of the injury and how aggressively the injury should be examined and treated. For example, a player is hit on the knee while falling down and cannot get up to return to play. During questioning, the athlete can quickly identify the trainer and physician, yells in pain when they talk with him, easily localizes the pain, and tells them about the injury. The athlete is barely able to lift the leg or bend the knee, and it hurts too much to walk.

In this example, the physician and trainer have quickly assessed that the athlete is stable, there are no obvious concussive effects from the trauma, but there is a significant knee injury that will limit participation in the event. From here, they can either examine the joint on the field or bring the athlete to the sideline for a more complete evaluation. In certain injuries, the immediate postinjury period is optimal for evaluation, such as in anterior cruciate ligament tears, so a physician may choose to perform an efficient knee examination on the field. Sometimes an athlete needs to be stabilized, and then fully evaluated later, after 12 to 24 hours of acute care depending on the situation. The initial examination should be efficient, but it must also help the clinician reach a decision quickly. A prolonged evaluation in the acute setting can affect outcome and morbidity. The examination should help establish a treatment plan, and appropriate follow-up is mandatory.

COMPONENTS OF THE PHYSICAL EXAMINATION

The athlete should disrobe to expose the injured body part, including its opposite joint. If the clinician is performing a complete physical, male athletes should just be in shorts, while female athletes should be in shorts and a sports bra. Missing a diagnosis because the athletes kept their clothes on is inexcusable.

When examining a specific joint, the uninjured joint should be examined first. This not only allows the examiner to feel an athlete's normal variations in laxity and anatomy as a comparison, but it also puts the athlete at ease by showing that the examination will proceed without provoking pain.

Each physical examination chapter in this book is designed to follow a similar template. There are many ways to perform examinations with even more variations, so this template is not a consensus statement on physical examination. It does, however, offer the reader a comprehensive and systemic approach to the examination of the athlete that can be modeled as well as referenced. The template is relatively consistent from chapter to chapter. The basic components are as follows:

1. *Appearance:* The examination begins by the clinician simply observing, comparing, and noting visual abnormalities, particularly when compared to the opposite unaffected body part. Typical symptoms to note include swelling, deformity, erythema, and the presence of skin lesions or wounds.
2. *Gross testing:* This is done with caution. The goal is to have the athlete perform a function that would hurt or be difficult in the face of injury to the joint being examined. If there is no difficulty in executing the test, the likelihood of any significant pathology is slim. If any pain or instability is produced by the gross test, then that body part undergoes a thorough examination. An athlete

who is apprehensive about trying a gross motion test is treated as a positive test, and then examined more carefully.

3. *Palpation:* Significant information is obtained by palpation. The examiner palpates using the TART system:
   - (T)issue texture changes
   - (A)symmetry of landmarks
   - (R)estriction of motion
   - (T)enderness to palpation

   Types of findings identified through palpation include edema, effusion, erythema, crepitus, bogginess, spasm, tenderness, and chronic dysfunction.
4. *Neurovascular:* Always assess circulation and neurologic function, especially in treating acute trauma.
5. *Active range of motion* (AROM): This is performed before passive range of motion to assess the athlete's comfortable range of motion. Moving an injured leg passively too far may accidentally trigger pain, worsen the injury, or increase muscle guarding. The AROM gives the examiner an idea of the depth of injury without provoking pain and spasm.
6. *Passive range of motion:* See no. 5, above.
7. *Provocative tests and maneuvers:* These special tests are designed to examine specific parts and structures of the anatomy. Clinician skill and practice are required.
8. *Joint play:* A normal joint always has a normal amount of play. If pathology occurs, play is restricted in a joint, which leads to dysfunction.

NEUROLOGIC EXAMINATION

The neurologic examination is extremely important, as it can help a clinician hone in on a difficult diagnosis. The basic elements of the neurologic evaluation apply to each region of the body and are presented later in this section. Each specific chapter on physical examination discusses the specific neurology germane to that region, such as dermatomes and myotomes.

Testing for deep tendon reflexes rarely elicits an abnormal response, yet doing so helps make a diagnosis in many cases, for example, in herniated discs and cerebrovasculat accidents. The tendon should be where the reflexes are elicited, and a proper reflex hammer should be used. A light tap on the tendon itself should be enough if the right location is struck. Tendon reflexes are graded according to the scale shown in Table 15.1.

TABLE 15.1. TENDON REFLEX GRADING SCALE

Grade	Description
0	Absent
1+ or +	Hypoactive
2+ or ++	"Normal"
3+ or +++	Hyperactive without clonus
4+ or ++++	Hyperactive with clonus

Muscle strength testing uses a rough standard for manual evaluation. The scale in Table 15.2 provides a rough meter for strength evaluation. For patients with more focal muscle weakness, nerve conduction studies (electromyogram) or specific strength testing (Cybex) that measure concentric and eccentric contractile force, peak torque, and muscle endurance can be used.

The joint should be stabilized by the examiner while the athlete listens carefully to the directions. The basic manual tests estimate isometric strength, such as the rotator cuff, so the examiner should securely stabilize the athlete's body part. On command, the athlete conttacts against the resistance offered by the examiner for roughly 2 to 3 seconds. The examiner may want a longer contraction to assess endurance, but that should be specified before the examination.

Assess muscle strength as described in Table 15.2, but also note the quality of contraction. Fasciculations, unsteadiness, and instability during the contraction may indicate a more subtle or insidious injury. Likewise, lack of effort on the athlete's part should be noted as well; some athletes with occupational injuries involving personal or financial gain may have less than optimal examinations. Other times, a neurologic problem may render the shoulder

TABLE 15.2. MUSCLE STRENGTH TESTING

Grade	Value	Muscle Strength
5	Normal	Complete range of motion (ROM) against gravity with full resistance
4	Good	Complete ROM against gravity with some resistance
3	Fair	Complete ROM against gravity with no resistance; active ROM
2	Poor	Complete ROM with some assistance and gravity eliminated
1	Trace	Evidence of slight muscular contraction; no joint motion evident
0	Zero	No evidence of muscle contraction

weak and flaccid. Quality of contraction should be considered with the strength of contraction.

A complete sensory examination is typically not done during routine screening or preparticipation physical examinations, but it should be done when significant injury or trauma is suspected. Many of the sensory tests are not needed if other tests are negative for deficit. However, a complete set of tools for evaluating the neurologic system through a sensory examination can pay dividends, particularly for naming difficult diagnoses.

Key points for the general evaluation of the sensorium are listed in Table 15.3. Perhaps the most important point is to always check the corresponding region on the opposite side for comparison. What may be empirically "abnormal" on a scale may be normal for that particular patient. A complete sensory examination is described as follows:

1. *Position sense*
   - Grasp the athlete's big toe and hold it away from the other toes to avoid friction.
   - Show the athlete "up" and "down."
   - With the athlete's eyes closed, ask the athlete to identify the direction you move the toe.
   - If position sense is impaired, move proximally to test the ankle joint.
   - Test the fingers in a similar fashion.
   - If indicated, move proximally to the metacarpophalangeal joints, wrists, and elbows.
2. *Dermatomal testing*
   Dermatomes are sensory neurologic paths that are innervated by specific nerve roots. They can be used to locate neurologic lesions. In order to test for dermatomes, the examiner must have knowledge of the regions of the body and the nerves that innervate them. By following the path of pain and sensory changes back to the spinal cord, the specific peripheral nerve and spinal root can be identified, as well as the spinal cord level from which the nerve emanated. If the dermatomal evaluation is within normal limits in the fingers and toes, the rest of this examination will likely be normal. In each physical examination chapter, the specific dermatomal patterns will be introduced and illustrated.
3. *Pain sensation*
   - Use a suitable sharp object to test "sharp" or "dull" sensation.
   - Test the following areas: (3, 4)

     Shoulders (C4)
     Inner and outer aspects of the forearms (C6 and T1)
     Thumbs and little fingers (C6 and C8)

TABLE 15.3 EVALUATING THE SENSORIUM

Explain each test to the athlete beforehand.
Athlete's eyes should be closed.
Compare symmetrical areas on both sides.
Compare distal and proximal areas of the extremities.
Map out sensory loss boundaries in detail.

Front of both thighs (L2)
Medial and lateral aspect of both calves (L4 and L5)
Little toes (S1)

4. *Vibration*

- Use a low-pitched tuning fork (128 Hz).
- Test with a *nonvibrating* tuning fork first to ensure that the athlete is responding to the correct stimulus.
- Place the stem of the fork over the distal interphalangeal joint of the athlete's index fingers and big toes.
- Ask the athlete to tell you if he or she feels the vibration. If vibration sense is impaired proceed proximally:

Wrists
Elbows
Medial malleoli
Patellae
Anterior superior iliac spines
Spinous processes
Clavicles

5. *Temperature*

- Often omitted if pain sensation is normal.
- Use a tuning fork heated or cooled by water and ask the athlete to identify "hot" or "cold."
- Test the following areas: (3, 4)

Shoulders (C4)
Inner and outer aspects of the forearms (C6 and T1)
Thumbs and little fingers (C6 and C8)
Front of both thighs (L2)
Medial and lateral aspect of both calves (L4 and L5)
Little toes (S1)

6. *Light touch*

- Use a fine wisp of cotton or your fingers to touch the skin lightly.
- Ask the athlete to respond whenever a touch is felt.
- Test the following areas: (3, 4)

Shoulders (C4)
Inner and outer aspects of the forearms (C6 and T1)
Thumbs and little fingers (C6 and C8)
Front of both thighs (L2)
Medial and lateral aspect of both calves (L4 and L5)
Little toes (S1)

7. *Discrimination*

Since these tests are dependent on touch and position sense, they cannot be performed when the previous touch and position sense tests are clearly abnormal. (3)

- Graphesthesia

With the blunt end of a pen or pencil, draw a large number in the athlete's palm.
Ask the athlete to identify the number.

- Stereognosis

Use as an alternative to graphesthesia. Place a familiar object in the athlete's hand (coin, paper clip, pencil, etc.).
Ask the athlete to tell you what it is.

- Two-point discrimination

Use in situations where more quantitative data are needed, such as following the progression of a cortical lesion.
Use an opened paper clip to touch the athlete's finger pads in two places simultaneously.
Alternate irregularly with one-point touch.
Ask the athlete to identify "one" or "two."
Find the minimal distance at which the athlete can discriminate.

VERTEBRAL MOTION TESTING

Before we begin the discussion of vertebral motion, it is important to review "normals" for range of motion, and *Fryette's laws of motion* for the spine. Range of motion for the vertebrae is measured in side bending, flexion, and extension in both active and passive movements. These limits represent gross motion, or how the entire spine performs. The examiner should also test for segmental motion, which is evaluation of the motion of each thoracic vertebra, primarily measured in rotation and side bending.

A restriction to a motion is a limit or barrier to its freedom of movement in the above-mentioned limits. These principles are echoed throughout this book, because they are the basis for using manual medicine. *Anatomic barriers*, formed by objects such as bones, ligaments, and tendons, are assessed through passive range of motion. Movement beyond the anatomic barrier would result in tissue damage. There are also *physiologic barriers*, which are limited by the normal neuromuscular function of the patient. They are assessed through active range of motion, which is less than the passive range. The *restrictive barrier* is a limit of motion within the anatomic barrier, and this represents the somatic dysfunction that responds well to manipulative treatment. Last, there is evidence of a *pathologic barrier*, which is a permanent restriction of motion due to an anatomic change caused by injury or disease (5).

When we consider the motion that the spinal segments display, it is helpful to understand Fryette's principles of vertebral motion, or simply, Fryette's laws (6). The *first principle* is that side bending of a vertebra will be opposite to the side of rotation of the same vertebra when the spine is in neutral position. In a clinical setting, this law would apply to a patient whose thoracic spine segments are side-bent in one direction, but rotated in the opposite direction. This is also called a type I, or group dysfunction (Fig. 15.1).

For example, if thoracic segments 5 through 9 (in neutral position) are side-bent right (identified by the concavity to the right), and the same segments are rotated left, then you would note this by writing T5-9 N SR RL. For example, an athlete has a group of paraspinal muscles on the right side that are in spasm, and the spinal segments involved (which attach to these muscles) gradually compensate from the induced side bending by rotating in the opposite direction. On examination, you would appreciate a palpable set of transverse processes on the left (or convex) side of the curve. Another example of this type of dysfunction is with scoliosis, which is addressed in later chapters.

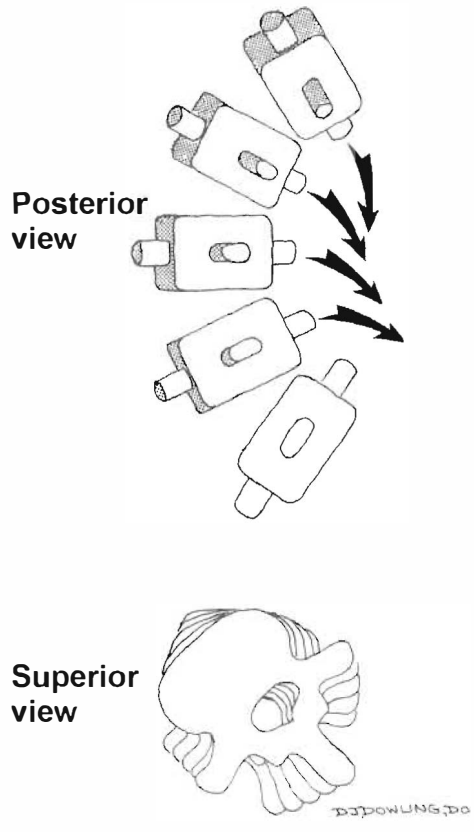

FIGURE 15.1. Side bending according to Fryette's first law; rotation and side bending occur in opposite directions. (Reprinted with permission from DiGiovanna EL, Schiowitz S. *An osteopathic approach to diagnosis and treatment*. Philadelphia: JB Lippincott Co, 1991:52.)

The *second principle* is that side bending of a vertebra will be to the same side of rotation of the same vertebra when the spine is in a flexed or extended position. This law usually applies to single-segment lesions, or type II dysfunctions, and more often involves vertebral rather than muscular restrictions. The side bending and rotation that occur on the same side often produce pain, tenderness, and restricted motion at the site of dysfunction when compared to the segment below it. This can occur from an abrupt movement, such as suddenly moving from one extreme range of motion to another. For example, a dysfunction at T4 that is flexed, side-bent right, and rotated right (T4 F SR RR) would

demonstrate a posterior transverse process on the right in extension. The examiner would feel restriction anteriorly and toward the left.

The *third principle* is that when motion of a spinal segment is initiated in any plane, the movement of this segment will be modified in other planes. In other words, motion in any one direction decreases motion in all other directions. This law more or less further characterizes the first two principles. For example, in type I mechanics, if the segment is rotated left and side-bent right, it will continue to move easier in those directions, but it will be harder to rotate the segment to the right or side-bend it to the left. Similarly in type II mechanics, if a segment is rotated right and side-bent right, it will continue to move easier in those directions, but it will be harder to move the segment to the left.

When differentiating between neutral, flexed, and extended lesions, each segment is palpated in all three positions. A posterior transverse process that gets worse in neutral or remains the same in all three positions is a neutral dysfunction. A segment that is more prominent or restricted in flexion, but disappears in extension, is called an extension dysfunction, and one that is more prominent or restricted in extension, but disappears in flexion, is called a flexion dysfunction. The restriction or dysfunction is named for its limitation to motion, which is what the clinician is trying to correct.

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16

HEAD AND NECK

16.1

Anatomy

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GAIL A. SHAFER-CRANE

The skull is the skeleton of the head, which encloses the brain and its coverings as well as the 12 cranial nerves connected to the brain; houses the organs of special senses (sight, hearing, taste, and smell); and surrounds the openings into the digestive and respiratory tracts. The cervical spine, consisting of seven cervical vertebrae, furnishes stability and support for the head, permits motion of the head and neck, and provides protection and housing for the cervical spinal cord and nerve roots as well as the vertebral artery (Figs. 16.1.1 and 16.1.2). The cervical spine is convex anteriorly. Anatomy of the head and neck is presented in detail in major anatomic textbooks (1–6).

Several important bony and cartilaginous structures can be palpated as one examines the head and neck. Beginning anteriorly, the horseshoe-shaped hyoid bone can be palpated in the crease of the neck superior to the thyroid cartilage at the level of the C3 vertebral body. Inferior to the hyoid bone in the midline of the neck is the thyroid cartilage. The superior notch can be palpated as well as the bulging central region, commonly referred to as the "Adam's apple." The thyroid cartilage lies at the level of the C4 and C5 vertebral bodies. The cricoid cartilage lies inferior to the thyroid cartilage at the level of the C6 vertebral body. It forms a complete ring, which is narrower anteriorly and considerably wider posteriorly. The cricothyroid membrane can be palpated between the thyroid and cricoid cartilages. Inferior to the cricoid cartilage is the first tracheal ring. The carotid tubercle lies about 2.5 cm lateral to the cricoid cartilage. This is the small anterior tubercle on the transverse process of the C6 vertebra and is covered by overlying muscles. The common carotid artery runs adjacent to the tubercle, and locating this tubercle is frequently used as a landmark for approaching the inferior cervical (stellate) sympathetic ganglion. The transverse process of the atlas (C1) can be palpated inferior to the ear between the angle of the mandible and the styloid process of the temporal bone.

On the lateral aspect of the skull, about 4 cm superior to the midpoint of the zygomatic arch, is the pterion. This is an H-shaped formation of sutures that unite the frontal, parietal, sphenoid, and temporal bones. The pterion is important clinically because it overlies the anterior branches of the middle meningeal artery, and a blow to this vulnerable area may rupture the underlying vessels. Posterior landmarks begin with the occipital bone (occiput). On the occipital bone one can palpate the external occipital protuberance (inion) on the midline and the superior nuchal line extending laterally from the external occipital protuberance. These structures mark the line of separation between the neck inferiorly and the head superiorly. At the lateral end of the superior nuchal line, the rounded mastoid process of the temporal bone can be palpated (Fig. 16.1.3). Beginning at the external occipital protuberance and proceeding inferiorly, the spinous processes of the cervical vertebrae can be palpated along the midline of the neck. The midline of the neck is indented due to the presence of the nuchal ligament lying immediately over the spinous processes and the laterally

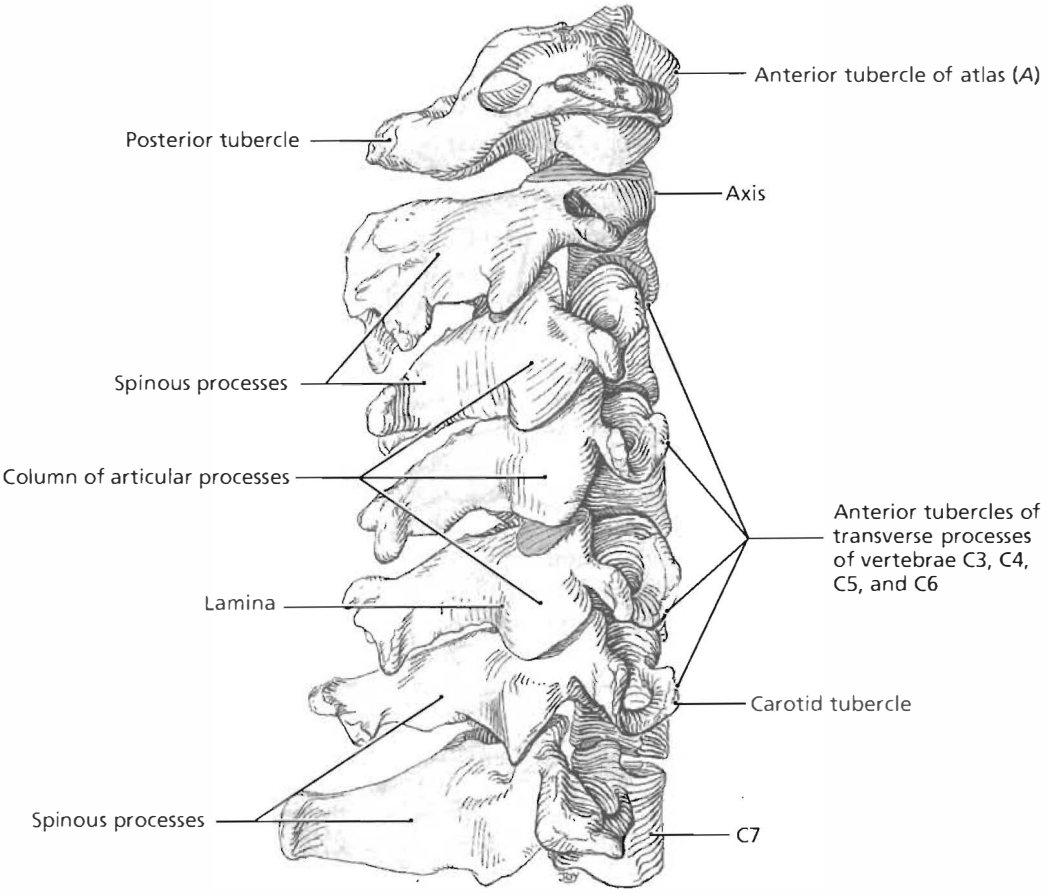

FIGURE 16.1.1. The cervical vertebrae. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

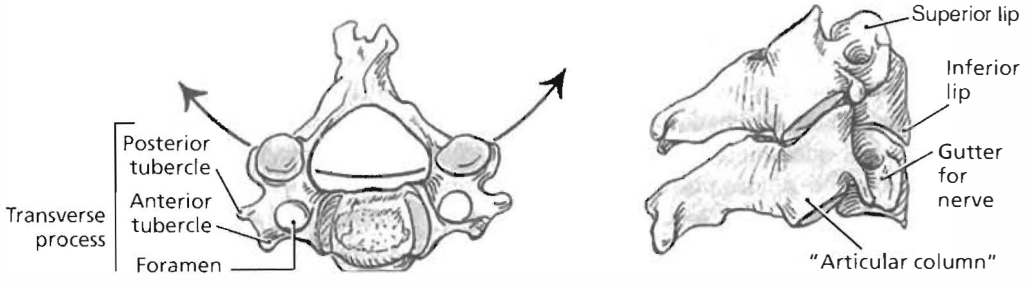

FIGURE 16.1.2. Cervical vertebrae, superior **(left)** and lateral **(right)** segmental views. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

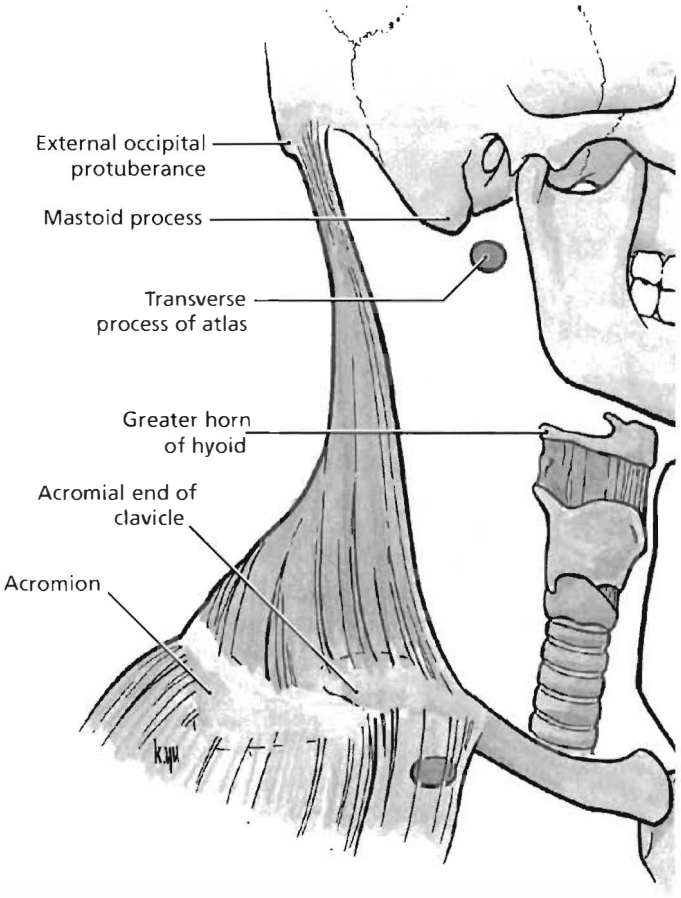

FIGURE 16.1.3. Bony landmarks of the neck, lateral view. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

located paraspinal muscles and the overlying trapezius muscle. The first cervical spinous process that can be palpated is C2 and the rest can be palpated sequentially to C7. The C7 spinous process, at the base of the neck, is the longest of the cervical spinous processes and serves as an important landmark for the end of the neck and the beginning of the thorax. Moving laterally from the spinous process of C2, the zygapophyseal joints of the cervical vertebrae, lying deep to the trapezius muscle, can be palpated sequentially as one proceeds inferiorly until the articulation between C7 and T1 is reached.

All seven cervical vertebrae are easily identified by the presence of a transverse foramen in each transverse process. These foramina transmit the vertebral artery and accompanying sympathetic nerve plexus. Cervical vertebrae C3-C7 share common anatomic features. Their relatively small cell body is wider side to side than front to back. The vertebral foramen is large in comparison to its size in other regions of the spine in order to accommodate the enlarged cervical spinal cord. The superior margin of the body curves up as an uncus (lip). The uncus articulates with beveled reciprocal facets on the inferior surface of the vertebral body above to form the uncovertebral joints (of Luschka). The pedicles are short and, along with the lateral aspect of the body, serve as the origin for the broad transverse processes. The superior and inferior vertebral notches are shallow, thus creating a very narrow intervertebral foramen. The superior and inferior articular processes are large, and their facets are oriented in the coronal plane. The facets of the superior articular

processes face posteriorly and slightly superior, while the facets of the inferior articular processes face anteriorly and slightly inferior. The spinous processes of C2-C6 may or may not be bifid. The C7 spinous process is the longest and most prominent cervical spinous process.

The first two cervical vertebrae are adapted for movements of the head. The atlas, C1, is attached to and "holds up" the skull. The axis, C2, provides the axis upon which the atlas and the attached skull can rotate. The atlas lacks a body and is composed of an anterior and posterior arch connected on each side to lateral masses. The lateral masses contain superior and inferior articular facets. The superior facets face superiorly and articulate with the occipital condyles of the occipital bone, while the inferior facets face inferiorly and articulate with the superior facets on the body of the axis. The internal surface of the anterior arch contains a small facet for articulation with the dens of the axis. Projecting off of the anterior arch is an anterior tubercle, while projecting off of the posterior arch is the posterior tubercle, which replaces the spinous process. The transverse processes are prominent and contain the vertebral foramina. On the superior surface of the posterior arch is a groove for the vertebral artery as it crosses the arch to enter the vertebral canal. The axis consists of a body and a vertical dens projecting superiorly off of the body. The dens has anterior and posterior facets for articulation with the facet on the anterior arch of the atlas and the transverse ligament of the atlas. The axis has no superior articular processes but does have superior articular facets on each side of the body. Its inferior articular processes, spinous process, and transverse processes resemble those of other cervical vertebrae.

In the cervical spine two types of joints, other than the uncovertebral joints described previously, unite cervical vertebrae C2-C7. These include facet joints (zygapophyseal joints) and intervertebral body joints (intervertebral discs). The facet joints are synovial joints between the articular processes of adjacent vertebrae. A thin, loose articular capsule surrounds each joint. The facet joints permit gliding movements between the vertebrae, and the shape of the articular surfaces limits the range of motion possible. Branches arising from the posterior primary rami of the spinal nerves innervate these joints. The intervertebral discs are fibrocartilaginous joints, designed for weight bearing and strength, situated between the bodies of adjacent vertebrae. Each intervertebral disc consists of an outer anulus fibrosus, composed of concentric lamellae of fibrocartilage, which surrounds a gelatinous nucleus pulposus. The anuli insert into the rounded rims on the articular surfaces of adjacent vertebral bodies. Branches arising from the anterior primary rami of the spinal nerves innervate the intervertebral discs. The intervertebral discs are avascular structures, which receive their blood supply by diffusion from the vertebral bodies.

Ligaments reinforce and stabilize the facet joints and the intervertebral discs between the C2-C7 vertebrae. The anterior and posterior longitudinal ligaments resist anterior and posterior displacement of the vertebral bodies on one another. The anterior longitudinal ligament limits extension of the vertebral column. The posterior longitudinal ligament runs within the vertebral canal beginning at C2 and limits flexion of the vertebral column. The strong, highly elastic, flattened ligamenta flava attach to the laminae of adjacent vertebrae to preserve the normal curvature of the vertebral column and straighten the vertebral column after it has been flexed. Short interspinous ligaments join adjacent spinous processes. In the cervical spine, the supraspinous ligament is replaced by the ligamentum nuchae, which separates the muscles on each side of the posterior neck and provides attachment for them. On its deep service, it attaches to the spinous processes of the cervical vertebrae, while superiorly it attaches to the external occipital protuberance and median nuchal line. Its posterior margin spans the distance between the external occipital protuberance and the spinous process of C7. This ligament helps to support the weight of the head when it is flexed.

The atlanto-occipital facets are obliquely positioned and permit nodding of the head and some lateral bending with practically no rota-

tion. The three atlantoaxial joints are specialized for head rotation. The median atlantoaxial joint is a pivot, with the atlas pivoting around the dens and carrying the head with it. Several strong ligaments provide the stability of the atlanto-occipital and atlantoaxial joints, most importantly the transverse ligament of the atlas, which converts the anterior arch of the atlas into a complete ring around the dens. The dens is connected to the occipital condyles of the occipital bone by the alar ligaments, which limit flexion and rotation. Extending superiorly and inferiorly from the transverse ligament of the atlas are superior and inferior crura, which together form a ligamentous complex referred to as the cruciform ligament. All of these ligaments lie deep to an upward extension of the posterior longitudinal ligament called the tectorial membrane.

The vertebral artery, which traverses the transverse foramina of the cervical vertebrae, typically arises from the subclavian artery at the base of the neck medial to the anterior scalene muscle. It runs superiorly and slightly posterior and usually enters the transverse foramen of the sixth cervical vertebra. Along with the artery are postganglionic sympathetic nerve fibers derived from the cervicothoracic or stellate ganglion and the vertebral vein. In the neck, small branches of the vertebral artery supply adjacent deep muscles of the neck and spinal branches enter the intervertebral foramina to supply the spinal cord and its coverings.

Muscles help to stabilize the spine and control the effects of gravity (Fig. 16.1.4). In the cervical spine two major functional groups of muscles are present: extensors and flexors. Each group is also capable of rotating and lateral bending the cervical spine. The extensor muscles are situated posterior to the laminae and transverse processes of the cervical vertebrae. These muscles also lie deep to the cervical portion of the thoracolumbar fascia, which separates them from more superficial muscles (trapezius and rhomboids) attaching the spine to the upper limb.

The extensor muscles can be divided into four groups: the splenius, erector spinae, trans-

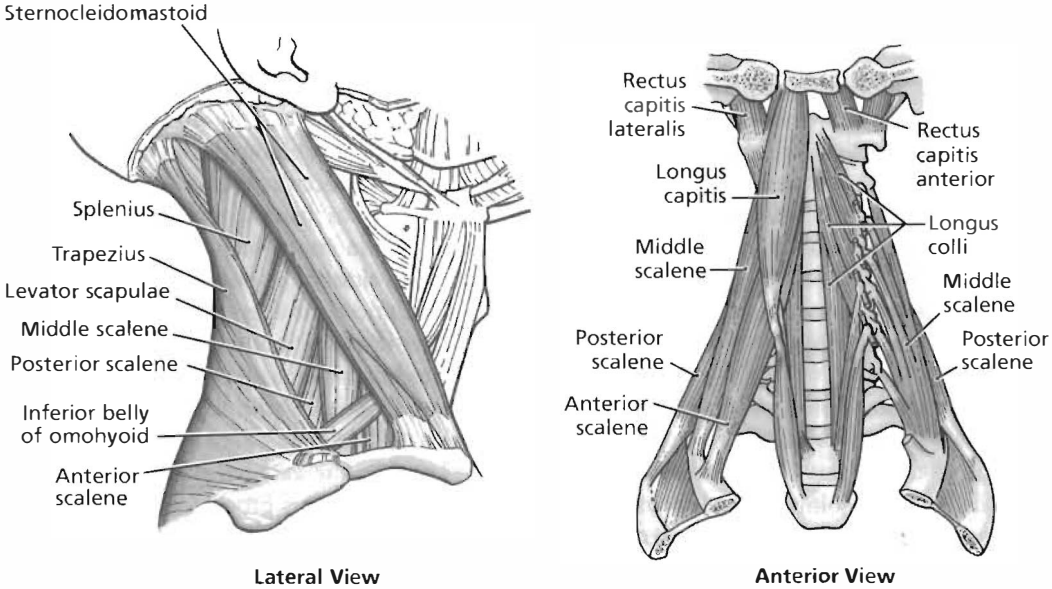

FIGURE 16.1.4. Superficial muscles of the neck. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

versospinalis, and suboccipital muscles. These muscles are arranged in three layers. The superficial layer contains the erector spinae and the splenius muscles. The intermediate layer contains the semispinalis and multifidus muscles of the transversospinalis group. The deep layer contains the interspinal, intertransverse, and rotator muscles of the transversospinalis group and the suboccipital muscles. The splenius muscles consist of the broad splenius capitis and the narrow splenius cervicis. The erector spinae muscles in the neck include the following from superficial to deep:

1. Iliocostalis cervicis
2. Longissimus capitis
3. Longissimus cervicis and capitis
4. Semispinalis cervicis and capitis

The multifidus muscles, lying deep to the semispinalis muscles, are thin in the cervical region. They arise from the articular processes of the lower four cervical vertebrae, run medially and superiorly and attach into the spinous processes of the axis and the upper two or three cervical vertebrae below the axis. The interspinous, intertransverse, and rotator muscles lie deep to the multifidus and are fairly well developed in the cervical region.

The suboccipital muscles connect the atlas and the axis to each other and to the skull and function in rotating and extending the head. This muscle group is composed of major and minor rectus muscles and superior and inferior oblique muscles. The rectus muscles produce extension of the atlanto-occipital joints, whereas the oblique muscles rotate the atlas and the skull on the axis. The major and minor oblique muscles and the major rectus muscle form the borders of the suboccipital triangle. This triangle contains the vertebral artery, suboccipital nerve, which supplies all of the suboccipital muscles and sensory greater occipital nerve.

The flexor muscles of the cervical spine are important in raising the head from a supine position. These prevertebral muscles in the cervical region are in close apposition to the vertebral column, directly flex the spine, and are represented by the longus colli and scalene muscles. The sternocleidomastoid muscle, attaching superiorly to the masroid process of the skull and inferiorly to the manubrium of the sternum and the clavicle, is a more powerful flexor of the cervical spine than the prevertebral muscles because of its more anterior position. Not only does it draw the head forward but it also flexes the cervical spine. The sternocleidomastoid is also the most powerful rotator of the cervical spine but it works in combination with other muscles. The prevertebral muscles are innervated by the anterior primary rami of the cervical spinal nerves, while the sternocleidomastoid muscle is innervated by the accessory nerve (CN XI).

The spinal cord and its coverings (meninges) are located in the vertebral canal of the cervical spine. The cervical spine contains eight cervical spinal cord segments along with their anterior and posterior nerve roots. Cervical spinal nerves (eight pairs) are formed just outside the intervertebral foramina by the union of the anterior and posterior roots. In the cervical spine, the spinal nerves ate formed above the vertebra they correspond to in number. Thus, the C1 spinal nerve is formed between the skull and the atlas, while the C8 spinal nerve is formed just outside the intervertebral foramen between the C7 and T1 vertebrae. After the spinal nerve is formed, it divides almost immediately into anterior and posterior primary rami. The posterior primary rami innervate segmentally skin and muscles of the back as well as the facet joints of the cervical spine. Communications between the anterior primary rami of the C1-C4 spinal nerves form the cervical plexus deep to the internal jugular vein and the sternocleidomastoid muscle in the neck. The phrenic nerve is the motor nerve to the diaphragm; it arises from the C3-C5 roots and descends through the neck on the anterior surface of the anterior scalene muscle down to the diaphragm.

The brachial plexus is derived from the anterior primary rami of the C5-T1 spinal nerves and provides motor and sensory innervation to the upper limb (Fig. 16.1.5). The

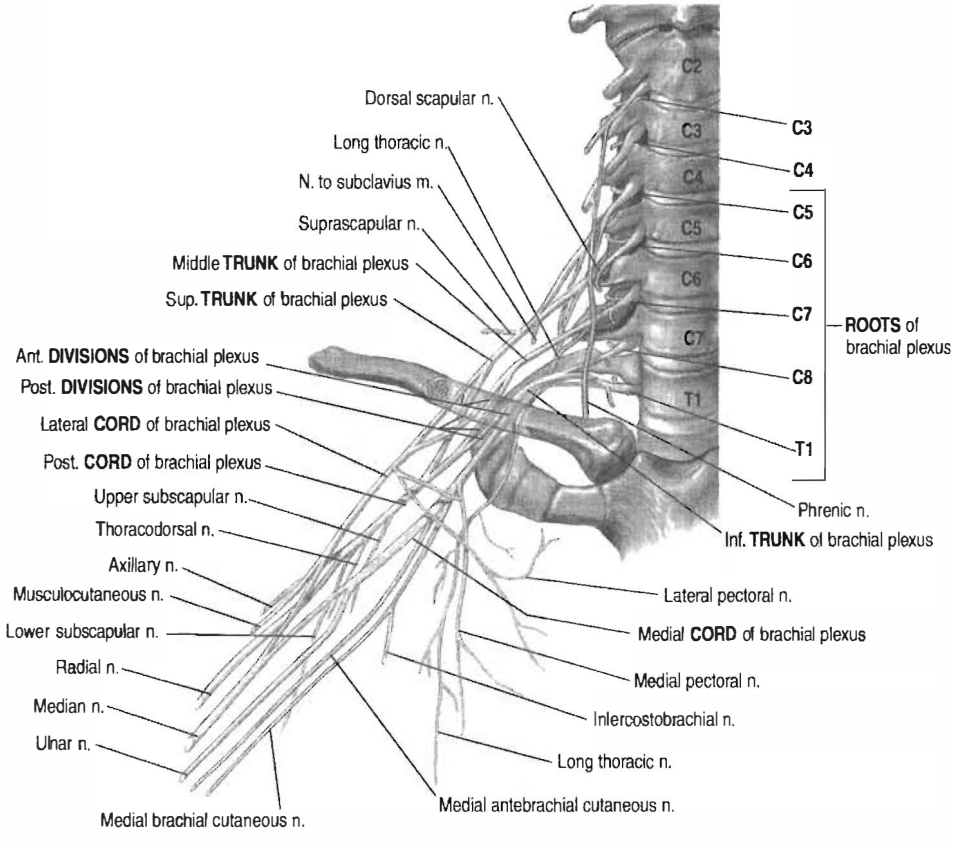

FIGURE 16.1.5. Brachial plexus, anterior view. (From Altcheck DW, Andrews JR. *The athlete's elbow: surgery and rehabilitation*. Baltimore: Lippincott Williams & Wilkins, 2001.)

plexus extends from the neck into the axilla and is composed of roots, trunk divisions, cords, and branches. The roots and trunks with their branches lie in the posterior triangle of the neck, with the roots situated between the anterior and middle scalene muscles along with the subclavian artery. Branches in the neck include the dorsal scapular nerve (C5) to the rhomboid and levator scapulae muscles, the long thoracic nerve (C5-C7) to the serratus anterior muscle, and the suprascapular nerve (C5 and C6) running laterally across the posterior triangle of the neck to supply the supraspinatus and infraspinatus muscles and the shoulder joint.

There are several important soft tissue landmarks in the neck (Fig. 16.1.4.) The sternocleidomastoid muscle extends from the mastoid process of the skull to the manubrium of the sternum and clavicle, and divides the neck into anterior and posterior triangles. The muscle is palpable along its entire length. A lymph node chain is situated along the medial border of the sternocleidomastoid muscle. The trapezius muscle extends from the external occipital protuberance to the T12 vertebra and attaches laterally into the clavicle, the acromion, and the spine of the scapula. The muscle can be palpated along its superior border from its origin to its distal attachment. The trapezius and the sternocleidomastoid share a continuous attachment along the base of the skull to the mastoid process as well as a common nerve supply by way of the accessory nerve (CN XI). The superior border

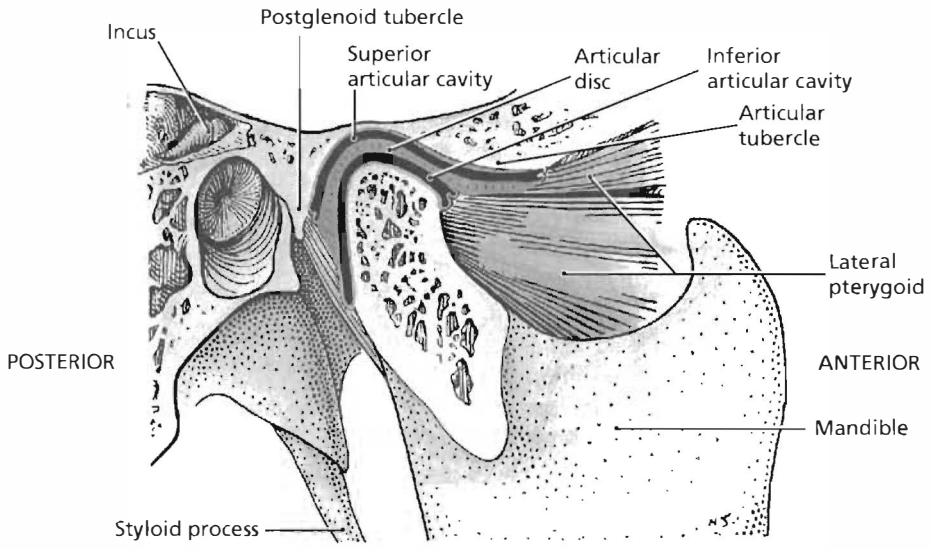

FIGURE 16.1.6. Temporomandibular joint. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

of the trapezius forms the posteriot boundary of the posterior triangle of the neck. The thyroid gland lies in the anterior triangle of the neck at the level of the C5-T1 vertebrae. It consists of right and left lobes located anterolateral to the larynx and trachea. An isthmus connects the lobes across the trachea on the midline of the neck at the level of the second to third tracheal rings. The esophagus, posterior to the trachea, begins at the inferior border of the thyroid cartilage and passes inferiorly through the superior thoracic aperture into the thorax. The carotid sheath is a fascial tube that extends from the base of the skull to the root of the neck deep to the sternocleidomastoid muscle in the anterior triangle of the neck. It contains the common and internal carotid arteries, internal jugular vein, and vagus nerve (CN X). At the level of the superior border of the thyroid cartilage, the common catotid artery divides into the internal and external carotid arteries. The root of the neck (supraclavicular fossa) is at the junction between the neck and the superior thoracic aperture. It is bounded anreriorly by the manubrium of the sternum, laterally by the first rib, and posteriorly by the body of the T1 vertebra. The sternocleidomastoid muscle, as it approaches its distal attachment, and covered by the platysma muscle, can be palpated.

The temporomandibular joint (TMJ) is a synovial joint, anterior to the auricle of the ear, where the head of the condyle of the mandible articulates with the articular tubercle and the mandibular fossa of the temporal bone (Fig. 16.1.6). An articular disc divides the joint cavity into superior and inferior compartments each lined by a synovial membrane. The fibrous capsule attaches superiorly to the margins of the articular area on the temporal bone and around the neck of the condyle. The capsule is thickened laterally to form the lateral ligament, which strengthens the TMJ. Two additional ligaments connect the mandible to the skull but add little strength to the TMJ. The stylomandibular ligament extends from the styloid process of the temporal bone to the posterior aspect of the angle of the mandible. The sphenomandibular ligament, medial to the TMJ, runs from the spine of the sphenoid bone to the lingula of the mandible.

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16.2

Physical Examination

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OBSERVATION

The head examination begins with inspection. The athlete's disposition alerts the examiner to the level of distress of the athlete. Note any lacerations, lesions, or swelling. Patterns of bruising should also be noted, although bruising may take up to 24 hours to appear. Bruising of the eyelids and orbital region is typically associated with an orbital fracture, basilar skull fracture, or fracture of the base of the anterior cranial fossa. Battle's sign is demonstrated by bruising behind the ear and along the hairline, which is associated with a basilar skull or temporal bone fracture (1). Evaluate for asymmetry of the athlete's facial features. Although many people do not have perfectly aligned eyes, pronounced asymmetry may be a sign of a zygomatic bone fracture.

Evaluate the athlete's eyelids for significant ptosis, which may be associated with cranial nerve damage. Normally, the athlete's upper eyelid should not cover the pupil. If the athlete has significant swelling of the eyelids that prevents him or her from opening the eye, assume the globe is ruptured and get an ophthalmologic referral immediately. Palpate and inspect the rest of the skull, noting any hematoma, deformity, and areas of pain to palpation.

While examining the oral cavity, ask the athlete to close the teeth together and examine the bite, noting alignment and pain with biting. Malaligned teeth are common, so that is not necessarily diagnostic for a fracture. Check to see if there are any teeth missing, and tap them to see if they are loose or if there is any pain. Evaluate the integrity of the gingiva, buccal mucosa, and tongue. While you are evaluating the athlete's bite, examine the temporomandibular joint (TMJ) by placing your index fingers over the joint and noting motion and position. Crepitus or pain over the TMJ is a sign of degeneration or significant injury of the joint (1). The TMJ is often the cause of severe headaches or earaches, so examine it carefully, particularly when you find evidence of mandibular trauma.

A common deformity seen in wrestlers is "cauliflower ear," in which the auricular cartilage hypertrophies and thickens from subcutaneous bleeding due to recurrent friction and twisting of the ear. An otoscopic examination

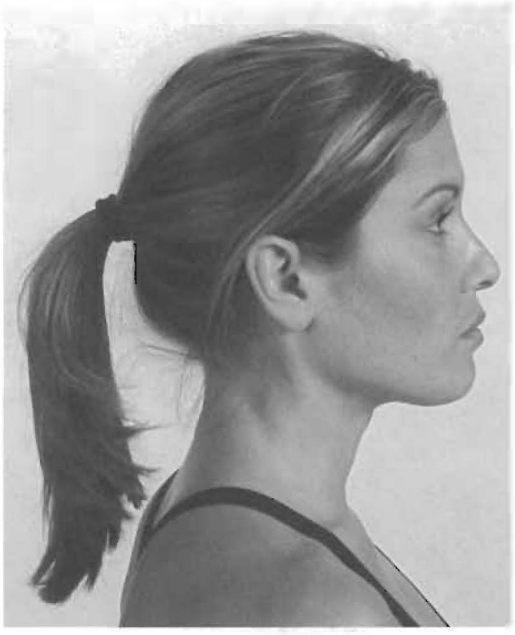

FIGURE 16.2.1. Neck, lateral view.

can visualize the ear canals and tympanic membranes. Evaluate for both clear and bloody discharge. As with the nose, clear discharge may be the sign of a cerebrospinal fluid (CSF) leak and skull fracture. Also, the ear tips should align with the corner of the eyes, so any discrepancy should be evaluated further.

Inspection of the cervical spine should reveal sufficient cervical lordosis, or a backward curve (Fig. 16.2.1). Loss of this curve may be a clue to the source of pain, such as degenerative disc disease or spondylosis. Poor posture, particularly in more mature athletes with early spinal osteoarthritis or disc degeneration, leads to increased "humping" at the C6-T2 region (2). The head should sit between the shoulders, not in front of them. Note any abnormal rotation or side bending. When inspecting the athlete from the front, the nose should be in line with the sternum. From the side, the earlobe should be in line with the acromion process. The muscle bulk of the trapezius, deltoid, and sternocleidomastoid muscles should be symmetrical; however, often the athlete's dominant shoulder will lie lower than the nondominant shoulder.

PALPATION

Starting at the anterior cervical region superiorly, palpate the hyoid bone, thyroid cartilage, first cricoid cartilage ring, and carotid tubercle. The thyroid gland is just inferior and lateral to the thyroid cartilage (Fig. 16.2.2), while the sternocleidomastoid lies one fingerbreadth laterally from the thyroid gland. Palpate these structures, noting pain or deformity.

At the base of the neck anteriorly, palpate the sternoclavicular (SC) joints and the length of the clavicles bilaterally, noting position, joint play, and motion. The position of the SC joints can be gauged by resting the index finger on the anterior and superior surfaces and comparing position. Passively elevate, rotate, and extend the athlete's arm while monitoring the SC joint to further evaluate joint motion (Fig. 16.2.3). The sternocleidomastoid inserts here as well, so palpate its enthesis for pain or disruption.

The cervical spine examination can be done with the athlete supine to allow the neck muscles to relax, although the examination can also be done with the athlete seated. The examiner should palpate the suboccipital fossae and note tenderness and somatic dysfunction. Traveling superolaterally from the fossae are the greater occipital nerves, which may be tender in athletes with chronic headaches and in those with atlantoaxial and occipitoatlantal dysfunctions.

FIGURE 16.2.2. Palpating the thyroid gland, with the athlete sitting up. The examiner's fingers reach around the sternocleidomastoid.

FIGURE 16.2.3. Palpating the sternoclavicular joint with the athlete's left arm abducted.

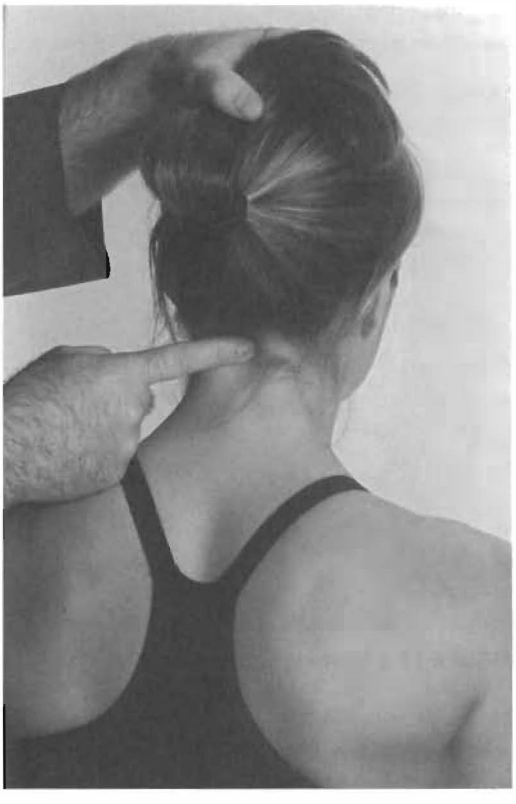

FIGURE 16.2.4. Palpating the C2 spinous process just below the occiput.

The skull should be examined for tenderness, depressions, or bony movement, particularly with any evidence of blunt trauma. An athlete with an obvious open skull fracture should be immediately transported to emergency services. Prominent posterior landmarks to examine include the occiput, mastoid processes, inion, and superior nuchal line. Anteriorly, palpate the orbital rim and zygomatic bone for crepitus, pain, or a stepoff, which can be associated with a fracture. Care must be taken to palpate gently throughout this examination in case an unstable fracture exists, lest the examiner displaces any fracture into a disadvantageous position.

Inspect and palpate the athlete's nose for deviation, depression of the nasal bridge, or nasal discharge. Plug one nostril by pressing on the side of the naris and ask the athlete to gently blow out through his or her nose, noting the ease and amount of air flow. If the athlete does have discharge, determine if the discharge is CSF secondary to an ethmoid fracture. If the discharge is bloody, use a piece of white gauze to soak up the discharge. If there is a yellowish-orange halo surrounding the central bloody discharge, then a CSF leak is likely. Athletes often complain of a salty taste in the mouth when they have a CSF leak; also ask the athlete if his or her sense of smell has changed, because fracture of the frontobasal or nasoethmoid bones can result in an altered sensation of smell.

The first palpable vertebral structure below the occiput is the spinous process of C2. Palpate along the spinous processes of C2 through C7, noting pain, swelling, stepoffs, and any differences in interspinous distance (Fig. 16.2.4). Note the superior nuchal ligament and the interspinous ligaments during palpation, especially in injuries involving sudden hyperflexion of the neck. Palpate with the neck flexed as well.

The spinous processes of C6 and C7 should be more prominent than those of C3-C5, but the C6 prominence disappears when the neck is in extension. Any cervical spine with spinous processes that show either a stepoff or a significant difference in interspinous distance needs radiographs taken to rule out instability or fracture. The transverse process of C1 can be palpated inferior and anterior to the mastoid process.

Move laterally and palpate along the facet joints, noting any prominent pillars or tender

FIGURE 16.2.5. Palpating the cervical pillars with the athlete supine.

points (Fig. 16.2.5). Note whether one facet is more prominent than the one on the contralateral side, for this can help identify a somatic dysfunction. Finally, continue to move laterally and palpate the musculature in the posterior neck.

RANGE OF MOTION

Active

The examiner asks the athlete to perfotm four motions:

1. Bend the head backward.
2. Bring the chin to the chest.
3. Look over the left and right shoulders.
4. Bring each ear to each shoulder.

Normal range of motion of the cervical spine is 60 to 90 degrees in flexion, 70 degrees in extension, 20 to 45 degrees in side bending, and 70 to 90 degrees in rotation. Cervical flexion primarily occurs at the occipitoatlantal joint and centers on the C5 and C6 vertebrae, while cervical extension centers around the C6 and C7 vertebrae. Rotation occurs primarily at the atlantoaxial joint, and normal range of motion is between 70 and 90 degrees. Side bending occurs primarily at the occipitoatlantal and atlantoaxial joints. Vertebrae C3 through C7 move in flexion and extension as one continuous group. While the athlete is flexing, pay close attention to the spinous processes and note if any vertebral body is more prominent than the others (3).

Passive

Passive range of motion (PROM) should be evaluated and compared with active range of motion. By placing the athlete in a supine position, the cervical postural muscles relax, and PROM can be more accurately assessed. Both of the examiner's hands should be on the athlete's head as it is moved through the ranges of motion. Painful barriers, muscle guarding, and anatomic restrictions should be noted.

Intersegmental Motion Testing

Occipitoatlantal Motion

The athlete lies supine with the examiner sitting at the head of the table. The examiner's hands hold the athlete's head while the index and middle fingers are placed on the suboccipital space. The examiner introduces lateral translation to the left and right, noting ease of motion or restriction (Fig. 16.2.6A). Testing is repeated in flexion and extension of the head. Note any motion restriction or palpable dysfunction in the occipital articulation.

Atlantoaxial Motion

With the athlete supine, the examiner flexes the head while monitoring the lateral masses of the atlas with the index finger pads (flexion locks out the C2-C7 vertebrae). The head is rotated left and right, while the examiner notes any restriction or pain (Fig. 16.2.6B).

C2 through C7 Vertebrae

Intersegmental motion is more difficult to assess in the C2-C7 vertebrae, and the examiner will have more success palpating for joint play and tissue spring in each level. The examiner places the pads of the index fingers along the lateral side of the posterior pillars of the specific segment, while the rest of the hand supports the athlete's head. The fingers introduce side bending into that segment, translating it left and right. Note spring, tenderness, and amount of play. Translation is alternated between the fingers, even lightly "bouncing" the segment

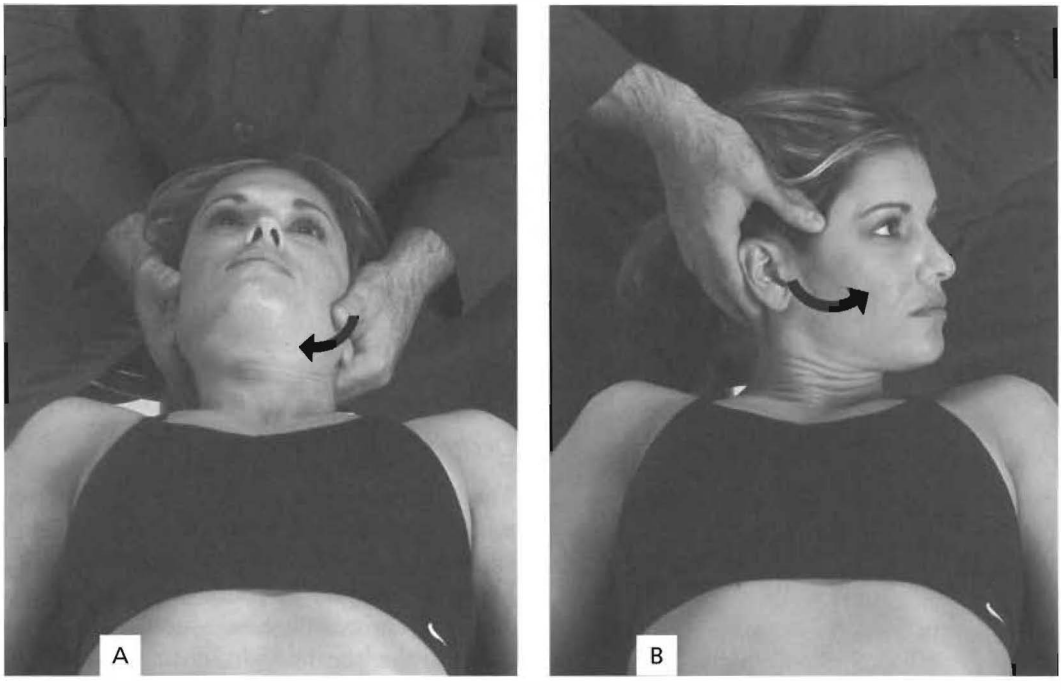

FIGURE 16.2.6. Upper cervical motion with athlete supine: occipitoatlantal motion **(A)**; atlantoaxial motion **(B)**.

four or five times before the examiner can get a feel for the restriction. This helps to identify whether a segmental lesion is restricted in left or right side bending (Fig. 16.2.7).

The examiner then moves the index finger pads to touch the posterior aspect of the facets and introduces anterior translation, noting spring, tenderness, and amount of play. This helps to identify whether a segmental lesion is restricted moving in extension. The vertebrae follow type II mechanics, so side bending and rotation are on the same side. Therefore, if a segment resists flexion and side bending to the left, it is classified as a vertebral segment that is positionally extended, and side-bent and rotated to the right.

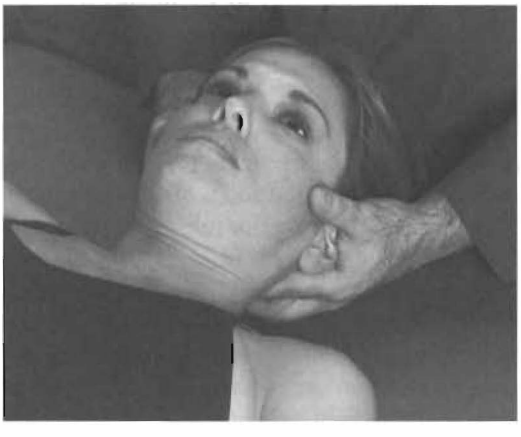

FIGURE 16.2.7. C4 vertebral motion testing.

STRENGTH

Evaluate strength with resisted isometric movements. The examiner can evaluate flexion, extension, side bending, and rotation by resisting the athlete's head with a stabilizing hand while the athlete is seated. This is usually done with minimal spine motion in order to minimize any opportunity for injury (1, 4).

TEMPOROMANDIBULAR JOINT

Inspect the athlete opening and closing his or her mouth. This motion should be smooth,

and there should be no deviation of the jaw to the left or right. If the athlete's jaw does deviate, it usually will deviate to the side of TMJ pathology. Movements of the TMJ include opening of the mouth; closing of the mouth; and protrusion, retrusion, and lateral deviation of the mandible. Normal range of motion on opening of the mandible is from 35 to 50 mm. Functionally, only 25 to 35 mm of opening is needed for activities of daily living. To assess normal range of opening, have the athlete place two or three flexed proximal interphalangeal (PIP) joints into the mouth. An inability to perform the PIP test may indicate a TMJ problem.

To palpate the TMJ, place your fingers either anterior to the tragus over the mandibular condyles or directly over the external auditory meatus. Ask the athlete to open and close his or her mouth while you palpate for clicking, grinding, or popping, noting any pain with this movement (Fig. 16.2.8).

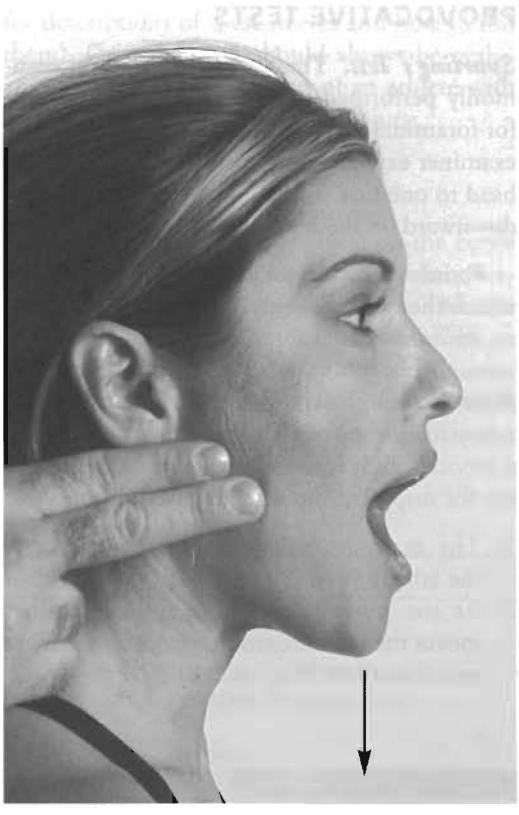

FIGURE 16.2.8. Temporomandibular joint palpation with jaw opened.

Normal range of motion on protrusion of the mandible is from 3 to 6 mm and on retrusion it is from 3 to 4 mm; however, this may vary greatly for the athlete with a significant overbite or underbite. Normal lateral deviation is from 10 to 15 mm to each side.

Test TMJ strength by resisting mandibular movement. While testing these movements, note strength and any pain that is referred to the TMJ itself. Place your fingers under the athlete's mandible and have him or her open the mouth against your resistance to test depression of the jaw. To test occlusion of the mandible, place your fingers on the athlete's lower teeth and have him or her close the mouth against your resistance. Make sure you tell the athlete that you are testing strength and that full closing of the jaw is not necessary so that he or she does not bite your fingers. To test lateral deviation, place one hand on the temple of the athlete's head opposite the side that you want to test and place the other hand on the athlete's jaw on the side you want to test and ask the athlete to move the mandible toward the hand that is on his or her jaw.

Jaw Reflex. This tests cranial nerve V, which is important in TMJ motion. The athlete gently opens his or her mouth while the examiner places his or her thumb on the athlete's chin. The examiner uses a reflex hammer to tap the thumb. A normal reflex response is closing of the mouth (4).

Positive test: Absence or hyperreactivtity is considered loss of the jaw reflex.

Indicates: Fifth cranial nerve injury.

Chvostek's Test. This test evaluates for seventh cranial nerve (facial) pathology. Irritation to the seventh cranial nerve will often refer pain to the area of the TMJ. The examiner taps on the parotid gland and observes for a reaction (4).

Positive test: The facial muscles twitch as a result of the tapping.

Indicates: Seventh cranial nerve palsy or injury.

PROVOCATIVE TESTS

Spurling's Test. This is one of the most commonly performed neck tests. It is used to test for foraminal compression on a nerve root. The examiner extends the athlete's neck, rotates the head to one side, and applies axial compression downward on the head (Fig. 16.2.9).

Positive test: Pain radiating down the arm to which the head is rotated.

Indicates: Cervical radiculopathy (2).

Adson's Test. This test evaluates the subclavian artery for compression by the scalene muscles or a cervical rib. It is an especially important test to use for suspected thoracic outlet syndrome.

1. The examiner palpates the radial pulse of the athlete.
2. As the pulse is monitored, the examiner moves the athlete's arm in abduction and external rotation (Fig. 16.2.10) (2).

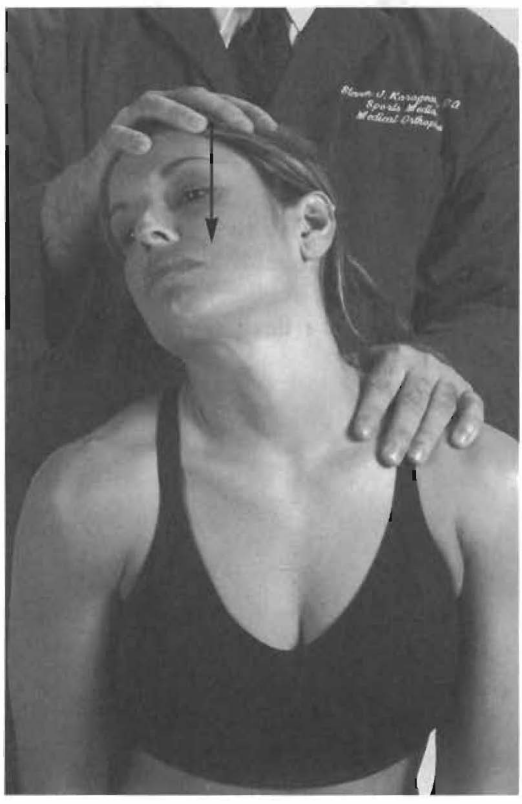

FIGURE 16.2.9. Spurling's test.

FIGURE 16.2.10. Adson's test.

3. The athlete takes a deep breath and turns his or her head toward the side of the arm.

Positive test: Marked diminution or loss of pulse.

Indicates: Subclavian artery compression.

NEUROVASCULAR EXAMINATION

Carotid Pulse. Palpate just medial to the midportion of the sternocleidomastoid muscle belly anteriorly. Note strength and rate of pulsation.

Jugulovenous Distention. Lay the athlete back at 30 degrees and 60 degrees to observe jugular distention and pulsation. Pulses should be seen upon contraction of the heart, but the jugular veins should not be distended in either position after the upstroke. Evidence of distention should be investigated for causes such as fluid overload or heart failure.

Lymph Nodes. Palpate the nodes in the head and neck region, noting pain, enlargement, or erythema. The following regions should be evaluated for lymphadenopathy (1):

a. Occipital nodes (skull base)
b. Postauricular nodes superficial over the mastoid processes
c. Preauricular nodes in front of the ear
d. Patotid and retropharyngeal (tonsillar) nodes at the angle of the mandible
e. Submaxillary nodes halfway between the angle and tip of the mandible

f. Submental nodes behind the tip of the mandible
g. Superficial cervical nodes near the sternocleidomastoid (SCM) muscle
h. Posterior cervical nodes along the anterior border of the trapezius muscle
i. Deep to the SCM
j. Supraclavicular nodes in the region marked by the SCM and clavicle

Cranial Nerves

The cranial nerves can be tested during a normal neurologic examination (see Table 16.2.1 for descriptions of these nerves and how to test them). Cranial nerves should always be evaluated during the examination of an athlete with head trauma or traumatic brain injury.

Spinal Nerves

The spinal nerve roots that come off the cervical spine can be tested by knowing their dermatomal and myotomal distributions. This can give insight into the neurologic status of an athlete with whiplash or other cervical trauma. A nerve root is labeled according to the vertebral level just below it, so the root in between

TABLE 16.2.1. CRANIAL NERVE FUNCTION AND TESTING

Cranial Nerve	Function	Tests
I—Olfactory	Smell	Aromatic testing
II—Optic	Vision	Visual acuity
III—Oculomotor	Eye movements, accommodation, and pupillary constriction	Eye gaze, light reactivity, eyelids, accommodation
IV—Trochlear	Eye movements: depression and intorsion	Eye gaze
V—Trigeminal (motor)	Muscles of mastication	Bite-down test
V—Trigeminal (sensory)	Sensation to face, scalp, and teeth	Sensory to maxillary, mandibular
VI—Abducens	Eye movements: elevation and extorsion	Sensory testing, corneal reflex, jaw clenching, facial muscle atrophy
VII—Facial	Muscles of facial expression; taste; sensation to the palate and external ear; secretion of the lacrimal, submandibular, and sublingual glands	Eye gaze; smiling, frowning, squinting, tongue sensory tests
VIII—Vestibulocochlear	Hearing and balance	Hearing test, Romberg test
IX—Glossopharyngeal	Elevates palate; taste; sensation of the ear and pharynx; secretion of the parotid gland	Swallowing study, observe palate action, gag reflex
X—Vagus	Swallowing; phonation; taste; sensation of ear, larynx, and pharynx; parasympathetic to heart and abdominal viscera	Gag reflex
XI—Spinal accessory	Shoulder, head, and neck movements; phonation	Shoulder shrug
XII—Hypoglossal	Movement of the tongue	Tongue movement, speech

Adapted from Seidel HM, Ball JW, Dains JE, et. al, eds. Neurologic system and mental status. *Mosby's Guide to Physical Examination,* 3rd edition. St. Louis:Mosby-Year Book, 1995;p716, and Bates B, ed. The nervous system. *A guide to physical examination and history taking,* 5th edition. Philadelphia:Lippincott, 1991;510–519.

C4 and C5 would be identified as the C5 root. The root between C7 and T1 is labeled C8, despite there being only seven cervical vertebrae. The next root down is T1, and from that point the labeling shifts to naming after the vertebra above the root.

There are two important sets of nerves that come off the cervical cord (2, 4):

1. *Diaphragmatic nerve roots: C3-C4-C5.* Injury to the neck in this location affects diaphragmatic contraction, and consequently, breathing. Ease of respiration should be noted if the clinician is suspicious of cervical trauma in this region.
2. *Brachial plexus: C5-C6-C7-C8-T1.* This complex intersection of five nerve roots controls the upper extremity.

Dermatomal Testing. Each brachial plexus nerve root innervates a portion of the skin sequentially through the arm and hand. Testing for changes in sensation in specific regions, or dermatomal distributions, can isolate the nerve root that is damaged or injured. Table 16.2.2 describes the innervation and dermatomal distribution of the brachial plexus.

Myotomal Testing. Due to the physiology of the brachial plexus, the myotomes have some overlap with innervation (Table 16.2.2). However, specific movements have been identified that best correlate with a specific nerve root.

CONCUSSION EVALUATION

Concussions are as common as ankle sprains in certain sports. A *concussion* is defined as a "complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces" (5). Football, soccer, rugby, and hockey are sports with higher levels of physical contact during competition; thus the risk for traumatic brain injury is higher. Second-impact syndrome is a potentially fatal phenomenon that can occur when an athlete is not fully recovered from a previous concussion and receives another impact. The second-impact phenomenon underscores the importance of diagnosing and treating concussions quickly and comprehensively. More importantly, athletes with a history of concussion have a sixfold increase in the likelihood of suffering another concussion (16).

Symptoms

Concussions can manifest themselves in a variety of ways. Table 16.2.3 lists the common symptoms consistent with a concussion. Subjective findings alone, however, do not suffice for concussion management.

The Glascow Coma Scale is a standard neurologic trauma test that scores trauma victims on their ability to respond to pain and verbal prompts. Three categories are assessed: eye

TABLE 16.2.2. BRACHIAL PLEXUS INNERVATION, DERMATOMES, AND MYOTOMES

Nerve Root	Nerve	Reflex	Dermatome	Myotome
C5	Axillary	Biceps	Lateral deltoid	Shoulder abduction
C6	Musculocutaneous	Brachioradialis	Lateral forearm, thumb, index and half of middle finger	Wrist extension
C7	Radial	Triceps	Middle finger	Wrist flexion, elbow and finger extension
C8	Median/ulnar		Ulnar side of little finger	Finger flexion, hand intrinsics
T1	Medial brachial cutaneous		Medial upper forearm and distal arm along elbow	Finger abduction

TABLE 16.2.3 PHYSICAL SIGNS OF CONCUSSION

Vacant stare (befuddled facial expression, glassy eyed)
Poor concentration, easily distracted
Delayed verbal and motor responses (slow to answer questions or follow instructions)
Confusion and inability to focus attention (easily distracted and unable to follow through with normal activities)
Disorientation (walking in the wrong direction; unaware of time, date, and place)
Slurred or incoherent speech (making disjointed or incomprehensible statements)
Gross observable incoordination (stumbling, poor balance, inability to walk tandem/straight line)
Emotions out of proportion to circumstances (distraught, crying for no apparent reason)
Memory deficits (exhibited by the athlete repeatedly asking the same question that has already been answered, or inability to memorize and recall 3 of 3 words or 3 of 3 objects in 5 min.
Loss of consciousness (paralytic coma, unresponsiveness to arousal) or impaired conscious state

Adapted from American Academy of Neurology. Report of the Quality Standards Subcommittee. Practice parameter: The management of concussion in sports (summary statement). *Neurology* 1997;48:581–585; Aubry M, Cantu R, Dvorak J, et al. Summary and agreement statement of the First International Symposium on Concussion in Sport, Vienna 2001. *Clin J Sport Med* 2002;12(1):6–11.

opening (1–4 points), verbal response (1–5 points), and motor response (1–6 points). A total score of 13 to 15 is considered good, while a score below 7 is considered poor. Although helpful in significant trauma with unconscious athletes, the scale is too blunt a tool for the average athlete, in whom a concussion rarely causes prolonged loss of consciousness.

McCrea et al. developed the Standardized Assessment of Concussion (SAC), which has proved helpful in tracking the course of concussions, as well as aiding trainers and physicians in returning athletes to play (7–10). The SAC does not clear an athlete for competition; even if an athlete achieves a perfect score, it does not mean that the athlete is cleared to play, particularly if symptoms still exist. It is merely a tool to assess baseline and postinjury levels of cognitive function in a quantifiable and more meaningful way (9).

The SAC is quick to administer and has several measures of cognitive function. The sideline examination is shown in Table 16.2.4. These components can be printed on cards for

TABLE 16.2.4 SIDELINE EXAMINATION OF THE STANDARDIZED ASSESSMENT OF CONCUSSION

Orientation (time, place, person, and circumstances of injury)
Immediate memory (recall of 5 words in 3 separate trials)
Neurologic screening
▪ Loss of consciousness (occurrence, duration)
▪ Post-traumatic amnesia (either retrograde or anterograde)
▪ Strength
▪ Sensation
▪ Coordination
Concentration (reciting numbers backward; months in reverse order)
Exertional maneuvers (jumping jacks, sit-ups)
Delayed recall (5 words)

Adapted from McCrea M, Kelly JP, Randolph C, et al. Standardized assessment of concussion (SAC): on-site mental status evaluation of the athlete. *J Head Trauma Rehabil* 1998;13(2):27–35; American Academy of Neurology. Report of the Quality Standards Subcommittee. Practice parameter: The management of concussion in sports (summary statement). *Neurology* 1997;48:581–585.

ease of use on the sidelines. The noise and action of the game may be too distracting to perform the entire test accurately, so if the clinician has any question about the mental status of the athlete, he or she should take the athlete into the locker room or a sequestered area to administer the test. If the athlete is symptomatic, he or she will likely not be returning to play, so that decision would allow time to properly administer the test.

Classification scales for concussion are numerous, yet no single standard has been adopted. The most commonly used ones have been published by Robert Cantu, the Colorado Medical Society, and the American Academy of Neurology (AAN) (11,12,13). These sources have also established return-to-play criteria, but no consensus exists on those standards either. Research suggests that loss of consciousness (LOC) is not an accurate predictor of concussion severity, and the grading scales differ on the exact weight LOC carries.

The First International Symposium on Concussion in Sport was held in Vienna in November 2001, and the consensus was that neuropsychological testing is the cornerstone of concussion treatment, particularly since research indicates that symptoms can resolve before the cognitive function deficits (5). Neuropsychological testing is becoming more popular, both in evaluating baseline function and assessing cognitive deficits after concussion. Newer neuropsychological testing can evaluate an athlete within 30 minutes, whereas the older tests took hours and lost their effectiveness as the test progressed over time. Cognitive function can be followed throughout the course of an injury, giving the clinician some tangible quantification of an athlete's recovery. This is important for return to play, as most athletes will likely want to return if symptoms are minimal. By helping to identify periods of glycolysis/perfusion mismatches (which may be responsible for second-impact syndrome in high school athletes), neuropsychological testing can help keep athletes safe after traumatic brain injury. Although far from perfect, these tests help in the athlete's recovery, most notably in staging and following recovery. Their usefulness is much higher when baseline tests exist for the injured athlete, but executing preseason testing can be logistically difficult.

Return-to-play criteria are noted in the AAN, Cantu, and Colorado guidelines (12, 13, 14). The Vienna conference recommended the following stepwise approach (5):

1. Complete rest
2. Light aerobic exercise (stationary cycling, walking)
3. Sport-specific training (skating in hockey, running in soccer)
4. Noncontact training drills
5. Full contact training after medical clearance
6. Game play

Athletes progress to the next level only when they are asymptomatic at the current one.

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16.3

Common Conditions

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The cervical spine is the source of many common complaints in athletes, with trauma as the most common cause of athletic cervical spine injury. Due to the vulnerability of this region to traumatic injury, great care must be used in applying manual medicine techniques to standard treatment. A proper diagnosis should be obtained before progressing with treatment. If done correctly, manual techniques can greatly speed recovery and return to play.

ATHLETES

Football players have received the most attention from epidemiologists and researchers on cervical spine injury. For years, the use of a tackling technique called "spearing" caused many catastrophic injuries from direct axial impact on a flexed cervical spine. Since the outlaw of this tackling technique and emphasis on proper tackling, cervical neurotrauma injuries have decreased.

However, the size and speed of the athletes playing football today at all levels are greater than at any time in the history of sports. The potential for catastrophic injury is increased by the high levels of kinetic energy generated and transferred at the point of collision. Along with the high number of players on the field and the inherently physical nature of the game, the risk for inadvertent cervical trauma is significant.

The axial loading injury mechanism is the most common cause of cervical spine injury in other sports such as rugby, soccer, diving, ice hockey, and wrestling (1). The force generated in collisions is even greater in today's athletes due to their increased speed, strength, and confidence. Improved training techniques and increased supplementation are just two reasons for high force generation, but improvements in protective equipment also help make athletes more fearless in initiating contact. Certain modifications, such as the cervical roll bar behind the neck, may help prevent whiplash injuries but also may increase the athlete's confidence that his or her neck is protected, and therefore increase the propensity of the athlete to absorb a cervical spine axial force.

This section focuses on common neck injuries, their subtle differences, and the application of manual medicine to their standard treatment. High-energy cervical-spine injuries, such as vertebral fractures, cord contusions, transient quadriplegia, and problems such as congenital cervical stenosis, are not discussed, as these are beyond the scope of this text.

WHIPLASH INJURY

The term *whiplash* refers to the motion of the head and cervical spine when the head is suddenly accelerated or decelerated. This "lash of the whip" action occurs when head acceleration or deceleration occurs relative to the body, resulting in an extension or flexion deformation of the spine. Many different interpretations exist for whiplash injury, broad to narrow, so in this discussion, we will refer to whiplash injuries as those that arise from the aforementioned mechanism.

In acceleration injuries, kinetic energy is accumulated into the mechanical system by an outside force that introduces force and inertia. For comparison, imagine holding a whip. With a flick of the wrist, an energy wave can be sent down the free end of the whip until the end, where a snap can be heard. This is the shock wave arriving at the end from propagation of the small impulse from the flick. Now, replace the length of the whip with seven cervical vertebrae, and the tail end of the whip with the skull. As energy accumulates at each segment from acceleration, the energy reaches the head as a shock wave, and the snap occurs.

The two common scenarios for whiplash are as follows:

1. A body at rest suddenly put in motion (e.g., a quarterback in the pocket getting hit from behind)
2. A moving body suddenly stopped (e.g., a hockey player absorbing a big hit in open ice)

According to Barral and Croibier, four types of whiplash can be distinguished, which do not necessarily occur in isolation (2).

Posteroanterior (back to front)
Anteroposterior (front to back)
Lateral (right to left)
Lateral (left to right)

The key element in whiplash scenarios is the unpreparedness of the body to defend against and absorb the impact. The cervical musculature can offer resistance to such force when an athlete expects the contact. Blind-side or unexpected trauma does not allow the musculature to recruit enough muscles to slow down the shock wave propagating through the spine. In an athlete prepared for an impact, the muscles will contract in support of the spine, but if the force magnitude is greater than the defensive mechanism, it overwhelms the system.

Athletes encounter high impact in such contact sports as hockey and football, where physical contact is an integral and often encouraged aspect of the game. For example, hockey players in competitive leagues learn to skate with the head up so they can see oncoming players and collisions. Quarterbacks in football are often standing vulnerable and unprepared for a blind-side hit by defensive linemen when they are looking to pass downfield.

Symptoms

There is no definitive description of whiplash injury, which is why it is often referred to as "whiplash syndrome." Because multiple systems can be affected from one insult, the manifestations of pathology are numerous and varied.

Neck pain is the classic sign of whiplash injury. In benign whiplash, the athlete can go hours before soreness, stiffness, and fatigue ensue, sometimes with nausea. In the following days, muscle spasm and soreness, stiffness, suboccipital headaches, and pain around the trapezius and shoulder regions can develop if edema to the injured soft tissues evolves. Sterling and co-workers found that 62% of victims of whiplash injuries still had ongoing pain after 3 months, a finding seen in various other studies (3). Vestibular dysfunction causing dizziness and vertigo can develop as well.

The picture becomes more serious when evidence of structural damage is found. The more severe injuries are seen in the intervertebral discs, apophyseal joints, and ligaments. Pain is

usually sharper, quicker in onset, and may radiate anywhere. Range of motion can be severely limited. Depending on the damage sustained, symptoms can be as severe as spinal cord injury.

OCCIPITAL NEURALGIA AND CEPHALGIA

Post-traumatic headaches are not always caused by a concussion. Frequently, the occipitoatlantal (OA) joints are pain generators in sports activities. The OA joints may become restricted as the result of a forceful head trauma or from a straining mechanism with the neck in an awkward posture, for example, in wrestling and weightlifting.

Anatomically, the occiput rests comfortably in the two cups of the C1 atlas vertebra. In neutral posture the skull is balanced between the two sides. However, head and neck movements that deviate from neutral may permit one or both of the occipital condyles to slip off their respective cups and possibly lock. The pain is not always immediate; rather, it may develop hours later as the surrounding musculature of the occipital triangle tightens up and goes into spasm, further restricting joint motion. The greater occipital nerve courses through this area and may be irritated by trauma or pressure placed upon it. Because nerves carry various sensory and motor fibers, noxious stimuli may induce other sensations besides pain such as nausea and dizziness.

Occipital neuralgia has a straightforward presentation in nontraumatic situations. However, in athletes who present initially with a history of an acute force applied to the head, it may be difficult to differentiate the symptoms from those of a concussion. In both conditions, athletes often complain of headache, nausea, dizziness, and possibly photosensitivity. Headache pain does not necessarily have to be present; many patients present with only post-traumatic dizziness or nausea that is aggravated by changes in head movement.

History

Anyone with a history of head trauma needs to be worked up for an intracranial injury. If the workup is negative, avoid the tendency to immediately tag the athlete with a diagnosis of concussion. Obtain a pertinent history and perform appropriate neurologic testing. A concussion prognosis has more conservative return-to-play criteria than occipital neuralgia, so a wrong diagnosis could force an athlete to needlessly wait days, weeks, or months before being cleared for return to play. On the other hand, athletes can sustain both a concussion and occipital neuralgia from the same injury, so the examiner should be thorough in his or her evaluation.

Physical examination reveals tenderness and restrictions involving one or both of the OA joints. Palpation of these joints often reveals a subtle fullness on the symptomatic side. Motion testing of the OA joints is helpful. Concussion patients may experience dizziness at rest and with head movement. Conversely, occipital neuralgia athletes tend to feel better with the head still, but they often complain of woozy sensations with OA motion, particularly with flexion or extension maneuvers. In other words, occipital neuralgia produces a mechanical headache.

The neurologic examination in occipital neuralgia is unremarkable, including mental status testing, which is not often the case in concussions. Imaging studies are not needed in atraumatic cases. They are used, however, in cases of trauma for the obvious reasons of excluding fractures and bleeds.

Standard Treatment

The pain from occipital neuralgia does not respond well to anti-inflammatory agents or narcotics. These medications alter perception of pain signals, but the pain remains. Multiple medications are often used to treat the various symptoms until the condition resolves spontaneously. In more severe cases, oral prednisone may alleviate the pain from an inflamed greater occipital nerve. However, the root of the problem is OA joint dysfunction, and patients continue to complain of pain until resolution of symptoms.

Manual treatment of occipital neuralgia is aimed at unlocking the mechanical restrictions present. Reduction of the OA restrictions is often associated with rapid, significant

pain relief. Most athletes describe a sensation like a pressure valve having been released.

Mobilization of the OA joints is best performed in a gentle manner, as movements that are too quick or forceful may induce a protective reflex in the athlete, making reduction more difficult. Counterstrain and muscle energy techniques may be employed first in order to fatigue the affected muscles in preparation for the mobilization (the joints may even reduce themselves during the muscle energy maneuvers). High-velocity, low-amplitude (HVLA) thrust techniques can be used at the end of treatment if other direct or indirect techniques fail. At the very least the muscles and soft tissue will be more lax for the next treatment. Following release of the OA restrictions, a nonsteroidal anti-inflammatory medication and an ice pack may be given to hasten resolution of local tissue edema.

CERVICAL STRAINS

Muscle strain, by definition, implies a stretch injury to a muscle or its tendon. Cervical strains may involve the anterior strap muscles, such as the sternocleidomastoids, or the posterior paraspinal groups. Strain injuries typically do not induce immediate discomfort; rather, they tend to become symptomatic hours, or even days, later. They intensify quickly over the course of the first few days and then gradually subside over time (how long depends on the intensity of the injury).

The history can be similar to a whiplash injury, in which the head and neck are suddenly decelerated or accelerated. If the muscles support the spine in time before the trauma and if the overall kinetic energy is low, then cervical strain is more likely than whiplash. In either case, the clinician must examine thoroughly for a whiplash-type injury mechanism.

Strains are perceived as dull, aching pain and are often associated with sensations of swelling and fullness of the affected muscle(s). Pain can be reproduced through resisted movement of the affected muscle(s). Spasm may be noted, but not all paravertebral muscle spasm is due to strain injuries. In fact, most of the time the spasm is due to reflexes initiated by acute somatic dysfunctions, such as facet joint restrictions.

Treatment

Strains respond quickly to conservative treatment that includes initial use of cold packs (1–3 days), nonsteroidal anti-inflammatory agents, and gentle stretching. Moist heat replaces the ice after the effects of the initial insult have been halted. In some severe cases, physical medicine modalities (electrical stimulation and ultrasound) and the use of muscle relaxants may be required.

CERVICAL SPRAINS

Sprains are injuries to a ligamentous structure that connects adjacent bones. Most often, the combined term *sprain/strain* is used to desctibe many cervical injuries including whiplash (flexion-extension) injuries. In this instance, the injury is often to the anterior or posterior longitudinal ligaments, the ligamentum flavum, or the interspinous ligaments.

Whiplash injuries often have a cervical sprain component. The cervical spine has a lordotic curve that is maintained by the ligaments to absorb excessive or unexpected forces (along with intervertebral discs) without causing significant damage. When the spine is in neutral with natural curves intact, side bending of a region is well tolerated and without ill effects as the individual vertebral segments accommodate to the movement as a group. The segments return to their neutral positions once the movement has been completed.

Whiplash-type injuries result in a loss of these regional curves; the head and neck extend backward, creating a straight spine along the cervical and upper thoracic regions. Intervertebral segmental motion is disrupted and side bending becomes restricted. To visualize this better, fold an 8 × 11 in. piece of paper in half so that it becomes 4 × 11 in. Grasp the folded edge using the thumbs of both hands and attempt to bend the spine of the paper. Observe that the paper must kink in order to promote bending. This kink represents specific somatic dysfunctions.

Participants in many sports are vulnerable to regional straightening of the cervicothoracic junction: football, gymnastics, and wrestling to name a few. These injuries result in a restriction of one or more facet joints of one of the cervicothoracic junction vertebrae. The nerves innervating these joints detect the restrictions and initiate reflexes that ultimately result in the development of increased paravertebral muscle tension, and possibly spasm. Note that this protective spasm is the result of the pinched facet joint(s) reflex mechanism, and is not usually due to a sudden stretch of the tissues.

The diagnosis of cervical sprain with somatic dysfunction is confirmed by the physical examination. Restrictions are noted in one or all of the three planes of cervical range of motion. Palpation reveals tender vertebral segmental dysfunctions. It is interesting to note that most of the restrictions affecting cervical range of motion originate from the upper and midthoracic spine. Alleviating neck problems by treating the upper back is welcomed by athletes afraid of cervical mobilizations.

Sprains may alter the cervical curvature permanently if plastic deformity occurs in the connective tissue. Chronic unresolved cervicothoracic dysfunction can exaggerate this condition, so these lesions should be identified and treated. Changes in curvature can be seen best on lateral radiographs. Without treatment and exercise, the altered spinal mechanics may become longer term.

Radiographs are not necessary in most outpatient circumstances; however, they should be performed to exclude cervical fracture, dislocation, or instability if suspected based on the mechanism of the injury. Neither magnetic resonance imaging (MRI) nor computed tomography (CT) is particularly helpful under routine circumstances. However, if radicular symptoms persist despite conservative management, then imaging should be done.

Treatment

The initial treatment consists of cold/ice pack applications to the affected region. Athletes tend to apply heat because heat feels better initially, but they should be informed that it will result in increased stiffness and discomfort in the following days. An anti-inflammatory medication is often helpful to control discomfort. On the other hand, if there is extensive regional pain and spasm, the use of opiate analgesics and muscle relaxants should be considered.

Soft cervical collars can be used initially in the acute setting, particularly if significant spasm is noted. Longer-term use is discouraged due to deconditioning of the supportive musculature. Physical therapy rehabilitation can add modalities such as iontophoresis, phonophoresis, and electrical stimulation to stretches, traction, and exercise to treat the muscular and ligamentous injuries. In industrial cases, physical therapists implement work-hardening programs to acclimate the whiplash victim to the stresses of his or her job.

Manual Medicine

Before considering the use of manual medicine on an athlete with a history consistent with whiplash injury, the clinician must rule out fracture or derangement of the head and neck. An index of suspicion for intervertebral disc injury should be high, and appropriate CT or MRI testing is warranted. Direct manual medicine techniques should not be used until the clinician is certain that there is no vertebral fracture, neurologic loss, or cervical instability.

Whiplash injuries involve more than musculoskeletal trauma. The damage can involve neurologic, vascular, respiratory, and craniosacral disruption (2,3). One insult can strain the dural layers, alter spinal motor reflexes, disrupt the flow of cerebrospinal and lymphatic fluid, sprain the intervertebral ligaments, have an impact on cerebral tissue and functions, and affect the primary respiratory mechanism (2,3).

With that being said, manual manipulative techniques can play a valuable role in treating whiplash injury and restoring equilibrium to the multiple affected systems. The workup for whiplash injury should be thorough, and the techniques performed carefully and sequentially; shotgun techniques have no place here, particularly if a workup is not complete.

Various techniques can be applied to whiplash, depending on the presentation. The appropriate initial treatments include counterstrain, myofascial release, and craniosacral medicine (4). Muscle energy and deep tissue massage are more effective in the subacute phase. HVLA thrust techniques may be productive toward achieving recovery based on the presentation, but they should be reserved for later in the course when most of the soft tissue injury has recovered. However, because of the apprehension athletes may have with aggressive manipulative techniques in whiplash injuries, we suggest avoiding HVLA if possible, and consider its use only after indirect and muscle energy techniques have been utilized.

Muscle energy and counterstrain techniques are effective in treating muscle strains (5), particularly in the cervical region. Initial evaluation may require treatment of any dysfunctions in the spine and pelvis as well, but after the first treatment, follow-up sessions can usually focus on improving the muscle length and tension in conjunction with stretches. Initial treatments should be less intense, the clinician using only counterstrain and mild muscle energy, then progressing as tolerated by the athlete.

Clinical Pearl: As noted earlier, restricted cervical range of motion is most often due to restrictions of the cervicothoracic junction (particularly the upper thoracic spine). Mobilizations can be achieved with less force if more flexion is achieved with the initial positioning.

Occipital Release

Rationale: The force transmitted through the neck causes a whiplike action of the head. The suboccipital musculature fires to help stabilize the occipitoatlantal and atlantoaxial articulations, and these muscles usually are traumatized. Spasm, strain, and dysfunction are often seen on palpatory examination. Postwhiplash cephalgia can emanate from this region as well.

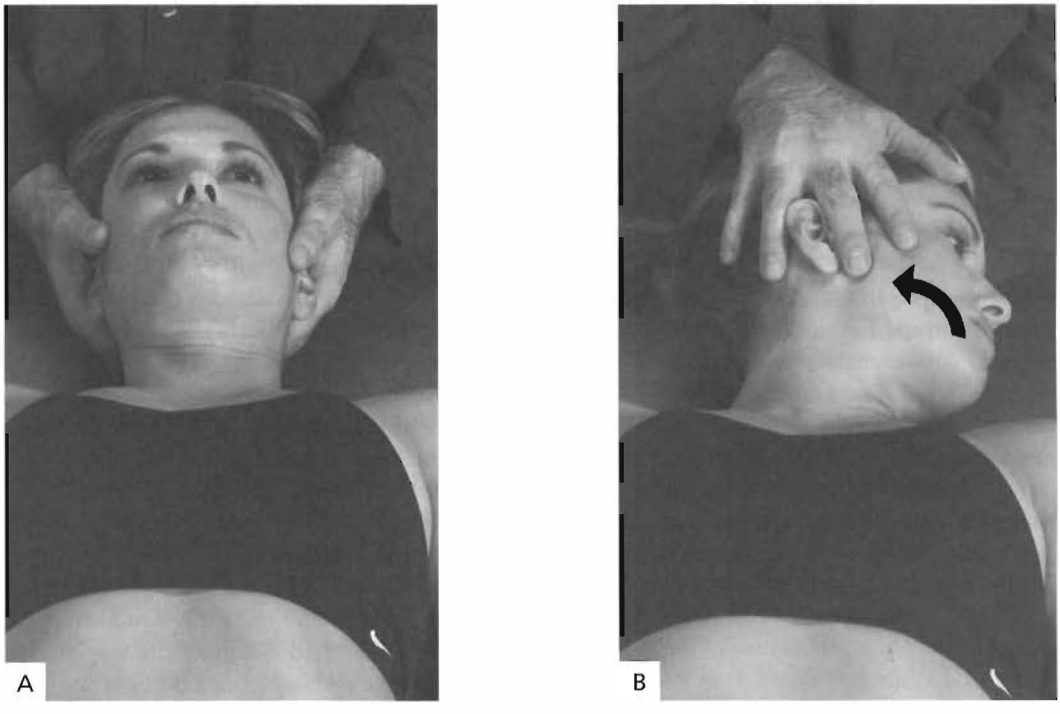

FIGURE 16.3.1. Occipitoatlantal joint muscle energy. **A,** The athlete's head is in neutral position, with the clinician's hands on the head. **B,** The athlete's head is rotated to the left, then the athlete gently turns the head right (arrow).

By releasing this region without HVLA thrust, motion can be restored and tension headaches may resolve. We discourage the use of HVLA technique to the occiput for athletes because the trauma of the technique may trigger flares of the soft tissue injuries from the whiplash itself.

Muscle Energy: Occipitoatlantal Joint

1. The athlete is supine with the clinician at the head of the table (Fig. 16.3.1A).
2. The clinician rotates the athlete's head to the left and right with the cervical spine held in neutral to check for restriction.
3. The clinician engages the restrictive barrier and asks the athlete to gently turn against resistance (Fig. 16.3.1B).
4. Relax, reposition, and repeat three to five times.

Muscle Energy: Atlantoaxial Joint

1. The athlete is supine with the clinician at the head of the table.
2. The clinician flexes the athlete's head and spine to a comfortable barrier (usually at least 45 degrees; Fig. 16.3.2A).
3. The clinician rotates the athlete's head to the left and right in the flexed position.
4. The clinician engages the restrictive barrier and asks the athlete to gently turn against resistance Fig. 16.3.2B).
5. Relax, reposition, and repeat three to five times.

Note: With this technique, the clinician should have the athlete's head in control at all times. If flexion is painful secondary to painful muscle spasm, stop the technique and treat the spasm. Do not perform this technique if the spasm cannot be resolved.

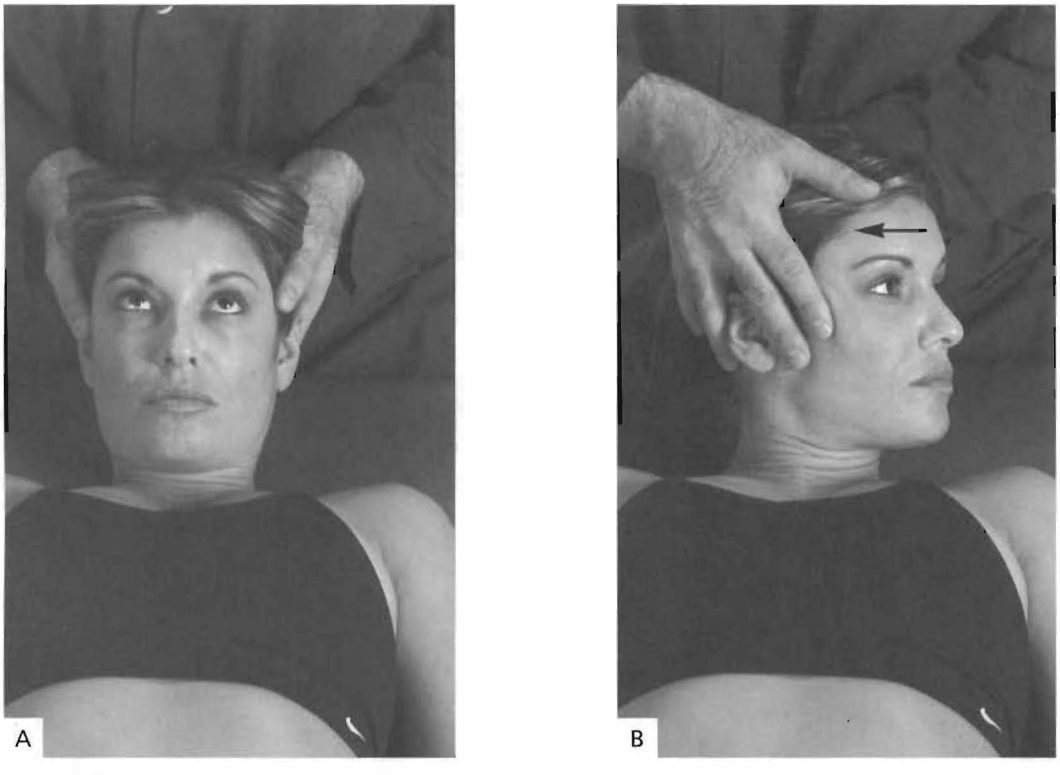

FIGURE 16.3.2. Atlantoaxial joint muscle energy. **A,** The athlete's head is flexed and in neutral. **B,** The athlete's head is rotated to the left, while the athlete gently turns the head right.

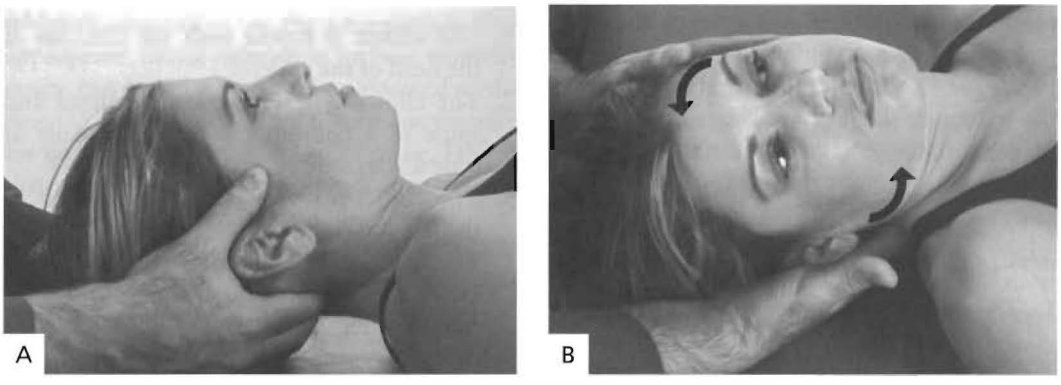

FIGURE 16.3.3. Occipitoatlantal counterstrain, suboccipital region. **A,** With the clinician's index finger on the right suboccipital fossa. **B,** The athlete's head is side-bent at the occipitoatlantal joint.

Counterstrain, Suboccipital Region: Occipitoatlantal Joint

1. The athlete is supine with the clinician at the head of the table.
2. The clinician palpates and diagnoses the occipitoatlantal dysfunction (Fig. 16.3.3A).
3. With the index finger the clinician monitors the lesion and gently moves the athlete's head into a position of ease (i.e., away from the barrier) (Fig. 16.3.3B).
4. Hold the position of ease and put firm pressure on the lesion for 90 seconds or until the lesion resolves.

Note: Move the athlete's head with small movements into flexion-extension, side bending, and rotation, asking the athlete how painful the dysfunction is. The tissue should relax under the index finger pad when you find the position of ease.

Traction Stretch

1. The athlete is supine with the clinician at the head of the table.
2. The clinician places one hand around the mastoids and cranial condyles and the other hand gently on the chin (Fig. 16.3.4).
3. The clinician pulls with the bottom hand in a cephalad direction. The stabilizing hand on the chin merely keeps the head in neutral alignment.
4. Hold the stretch for 30 seconds, making sure the athlete is not uncomfortable.

Note: The athlete can perform deep breathing through the nose during this technique to take advantage of respiratory facilitation and relax the tissues more.

FIGURE 16.3.4. Traction stretch of the cervical spine.

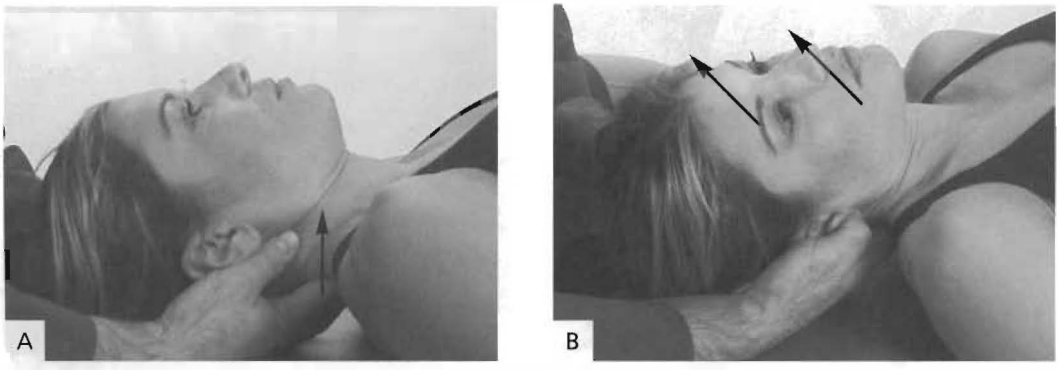

FIGURE 16.3.5. C7-T1-first rib muscle energy. The clinician's right index and middle fingers are placed on the right T1 pillar. **A,** Anterior translation of segment. **B,** The athlete is side-bent right with the clinician's left hand against the temple.

C7-T1-First Rib Complex

Rationale: When treating the occiput, the cervicothoracic junction must be addressed, as it accounts for a good portion of cervical range of motion. An elevated first rib can limit rotation and irritate the trapezius and splenius muscles that insert into the occipital region. Always look at this complex with any cervicothoracic pain.

Muscle Energy: C7-T1-First Rib Dysfunction

1. The athlete is supine and the clinician sits at the head of the table.
2. The clinician places the index finger on the dysfunctional segment pillar into the restrictive barrier while his or her contralateral hand is placed against the contralateral side of the athlete's face (Fig. 16.3.5A).
3. The clinician side-bends the athlete's neck to introduce the dysfunctional segment into the barrier (Fig. 16.3.5B).
4. The athlete pushes his or her head gently against resistance from the clinician for 3 to 5 seconds.
5. Rest, reposition, repeat, and retest.

HVLA Technique: C7-T1-First Rib Dysfunction (Right)

1. The athlete is supine and the clinician sits at the head of the table.
2. The clinician places his or her right index finger PIP joint against the lesion (Fig. 16.3.6).
3. The clinician introduces right side bending (into the restriction) and left (wrong-way) rotation of the athlete's head (moving into the restrictive barrier).

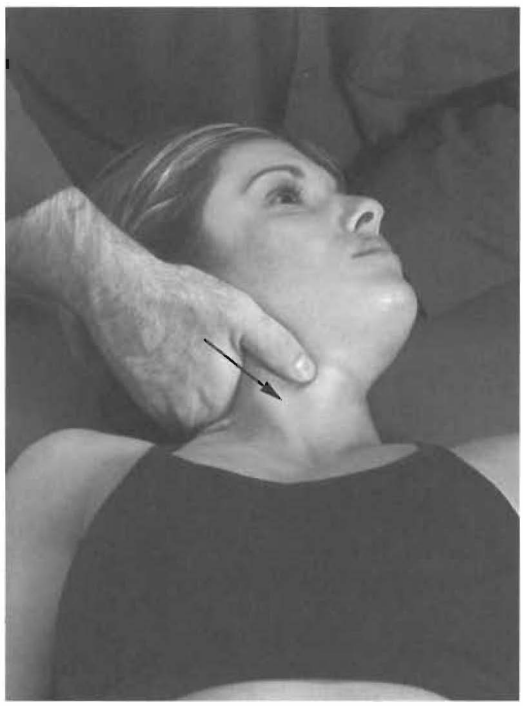

FIGURE 16.3.6. C7-T1 high-velocity, low-amplitude technique setup.

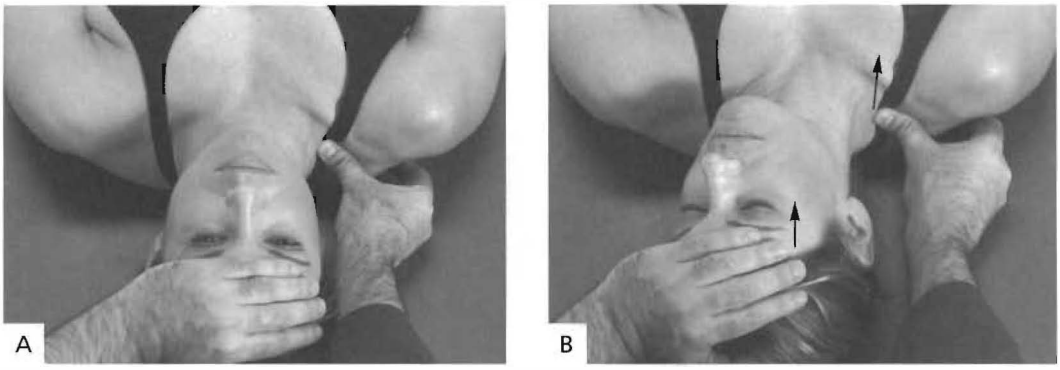

FIGURE 16.3.7. Elevated first rib (left); muscle energy. **A,** Setup with the athlete's head in neutral. **B,** The athlete's head is rotated away, with the athlete lifting the head up.

4. The clinician introduces a gentle thrust impulse directed toward the left hip pocket.

Muscle Energy: Elevated First Rib (Right)

1. The athlete is supine with the clinician at the head of the table.
2. The clinician places the right thumb over the first rib shaft in the supraclavicular fossa. The dysfunction may be tender, so be careful with palpation (Fig. 16.3.7A).
3. The clinician's left hand lies on the forehead of the supine athlete.
4. The clinician rotates the athlete's head away from the dysfunctional first rib until the scalenes tense up and pull upon the rib shaft. The athlete's head is held at this position (Fig. 16.3.7B).
5. The athlete gently lifts his or her head up off the table against the clinician's resistance for 5 seconds.
6. Relax, reposition, and repeat three to five times.

Articulatory Mobilization: C7-T1-First Rib Complex (Right)

Note: If the athlete has too much discomfort during the setup to the point where he or she cannot relax, choose another technique.

1. The athlete is prone with the clinician at the head of the table.
2. The clinician side-bends the athlete's head to the left far enough to engage the C7 vertebra and rests the head on the chin.
3. The clinician crosses his or her hands so that the right palm is against the first rib angle and the left hand is against the right occipital condyle (Fig. 16.3.8A).
4. The athlete inspires deeply and expires; the clinician gently follows the expiration with pressure directed into the table (taking up the slack) (Fig. 16.3.8B).
5. The clinician introduces a gentle impulse at the end of expiration, more with the right hand, less with the left, in a scissors-like maneuver.

Visceral Layers of the Neck

Rationale: These tissues can be damaged in whiplash injury, and tissue reaction can alter myofascial, vascular, neurologic, and lymphatic function. Relieving congestion and restoring mobility and elasticity of the soft tissues will help overall cervical function and recovery.

Global Release: Visceral Layers of the Neck

1. The athlete is supine with the clinician at the head of the table.
2. The clinician places his or her index finger on the athlete's lower anterior cervical region, immediately above the clavicle, in the

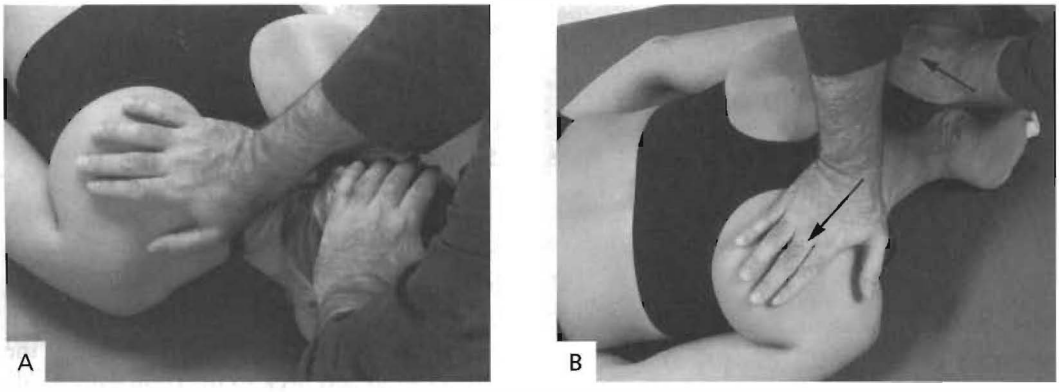

FIGURE 16.3.8. C7-T1-first rib (right): articulatory mobilization. **A,** Setup with the athlete's head in neutral prone position, and side-bent left. **B,** End of technique follow-through.

gliding spaces between the retropharyngeal and retroesophageal space. The middle finger and little fingers go on the middle to upper part behind the gonion.

3. The clinician draws the viscera in transverse and longitudinal directions (Fig. 16.3.9).
4. To treat specific restrictions in the tissue, the clinician brings the tissue toward the restriction, rhythmically repeating this four to five times until a release is achieved.
5. Observe the athlete's response carefully during this technique and back off or stop when the athlete feels discomfort.

Longus Colli (Deep Neck Muscle) Release. This technique is applicable to any cervical trauma as long as an acute process is not occurring simultaneously (Fig. 16.3.10).

1. The athlete is supine while the clinician is at the head of the table.
2. The clinician places a stabilizing hand on the athlete's head.
3. The stretching hand reaches behind the neck and holds the athlete's head just off the table and against the clinician's chest. The cervical spine should be slightly flexed on the thoracic vertebrae.
4. The clinician's palm and index fingers are placed on the cervicothoracic junction, palpating the anterior transverse processes.
5. The clinician's thumb then probes the region until an indented mass of muscle (longus colli) is felt lying on the anterior column.

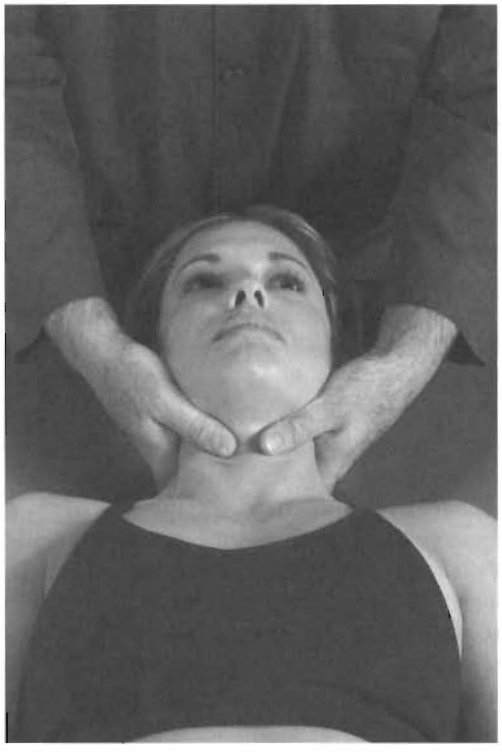

FIGURE 16.3.9. Visceral layers of the neck, global release.

FIGURE 16.3.10. Longus colli release.

6. The thumb compresses and holds pressure on areas of tension while the cervical spine is gently and progressively side-bent and moved into lordosis. The thumb should be moved along the length of the muscle until it relaxes.

Prevention

Teaching proper technique to athletes in such sports as football and soccer, and wearing the appropriate equipment properly, best accomplish avoiding whiplash injury in sports. Football and hockey players should make sure the helmets and shoulder pads fit appropriately. Using the head in tackling should be discouraged, while proper techniques in tackling and hitting, even in hockey, should always be practiced.

Many recreational-level skiers ski only a few times during the winter and their fitness level falls off during the rest of the year.
who do not acclimate properly when traveling to a higher altitude or condition properly are at risk for injury, especially if they ski slopes above their ability or continue too long after their legs fatigue. Falling and wiping out can cause whiplash if the head is snapped back and hits firm packed snow. Encourage these types of skiers to do a 3- to 4-week training camp program before their trips to get their strength and endurance up, particularly if the trip extends for several days.

Any athlete in a contact sport should also work on cervical muscle and trapezius muscle strengthening to support the spine in the event of an acceleration-deceleration force transmission. A stronger and more flexible spine can handle and disperse more force without injury.

BRACHIAL NEURAPRAXIA (STINGERS)

Brachial neurapraxia, also called "stingers" or "burners," is a transient neurologic injury to the upper trunk of the brachial plexus involving the C5 and C6 nerve roots. The most common mechanism of injury is the side bending of the athlete's head and neck to the contralateral side of a forcefully depressed shoulder. Arm pain is present on the ipsilateral side of the depressed shoulder. Compression of the upper plexus between the scapula and a shoulder pad is also common (6).

These injuries are relatively common in such contact sports as football. Skiers whose poles catch on trees, diving soccer goalies, and mountaineers and hikers who carry heavy backpacks are other athletes who may suffer upper extremity injuries (7).

The overall incidence of brachial neurapraxia injuries is unknown. However, an in-depth study of the 1974 Naval Academy and 1976 University of Wisconsin football teams showed that over 50% of the players on each team had sustained at least one such injury during their careers. Defensive backs and defensive linemen had the highest incidence of stingers; quarterbacks, running backs, and receivers suffered few or none (7,8).

Etiologic Factors

Intrinsic. An underlying cervical sagittal stenosis may be present in an athlete who suffers recurrent stingers in a high-velocity collision sport. This condition is usually asymptomatic until a hyperextension injury or vertebral subluxation occurs, or until spondylolysis develops. These conditions may lead to transient or permanent quadriplegia. Flexion and extension lateral cervical spine films should be taken if cervical sagittal stenosis is suspecred (these can be used to rule out instability and congenital

anomaly as well). The cervical canal width is measured from the middle of the posterior surface of the vertebral body to the nearest point on the ventral surface of the spinous process. From C3 through C6, a sagittal diameter ranging from 14.5 mm to 20 mm is within normal limits (6,9).

Extrinsic. Protective gear should be checked, especially football shoulder pads. They should be in good condition and have a proper fit, be worn separately, and protect from side bending beyond safe limits (9).

History and Physical Examination

The classic presentation is sharp, burning shoulder pain that radiates down the arm. Findings directly related to brachial neurapraxia include involvement of one arm and noting that side bending of the neck away from the painful arm aggravates symptoms. Weakness of the affected arm can be present along with numbness or tingling. Trauma to the neck or shoulder precipitates symptoms, particularly with sudden cervical side bending or scapular depression.

A cervical spine field examination should be performed to rule out spinal cord injury. The affected arm should be examined for nerve damage. The athlete may be holding the affected arm, letting it hang limply at his or her side. Side bending the neck away from the involved side may aggravate symptoms.

Neurologic evaluation should include testing for sensation to light touch, pinprick, and deep tendon reflexes. Map out deficits by nerve root and peripheral nerve distribution. The rhomboid muscle receives innervation from the dorsal scapular nerve (C5), so observe for weakness in acute cases and wasting in recurrent ones. Strength testing is done by resisting adduction to the side with a flexed elbow. The serratus anterior muscle receives innervation from the long thoracic nerve (C5-C7). To check for serratus anterior activity, ask the athlete to perform a wall push-up. Check for atrophy of the supraspinatus and infraspinatus. To evaluate the muscles supplied by the brachial plexus, ask the athlete to abduct the shoulder, flex and extend the elbow, and pronate and supinate the forearm against resistance (6,9).

Examination of the cervical spine and shoulder is warranted if suspicion is present for associated injuries such as cervical spine fractures; disc herniations; clavicular, scapular, or humeral fractures; or scapulothoracic dissociation. Since spasticity or weakness in the ipsilateral leg suggests a concomitant spinal cord injury, examination of the lower extremities is included.

Standard Treatment

Acute. Ice and rest are prescribed in most athletic settings. After 5 minutes, the clinician reassesses the athlete for pain, range of motion, and strength. If all three are normal after the neurologic symptoms have subsided and if no other problems are noted, then the athlete is usually cleared to return to play. If pain constantly persists, the athlete should be sent to an emergency room setting to be more closely evaluated for neurovascular damage, such as a fracture compressing the brachial plexus or a nerve root avulsion.

Subacute. Physical therapy modalities can treat soft tissue injury related to the trauma. Stretching and strengthening exercises for the shoulder and neck muscles, particularly the scalene and levator scapulae muscles, can be applied to help prevent type II (non-neutral) cervical somatic dysfunctions from recurring. Strengthening exercises for the neck, shoulders, and upper back are important for prevention. Protective gear should be checked and implemented if not already in place.

Manual Medicine

The goal of manual medicine techniques is to restore function to the cervicothoracic region quickly so that full recovery can occur without decompensation occurring. Direct techniques should not be applied if any of the relative or absolute contraindications are present, particularly nerve root avulsion, vertebral disc herniations, and vertebral body fracture.

Muscle Energy: Anterior and Middle Scalene Muscles

Rationale: If tightness is present, it can contribute to multilevel extension restrictions (flexed, rotated, and side-bent) [FRS] dysfunctions that are rotated and side-bent contralateral to the hypertonic muscles).

Muscle Energy: Hypertonic Levator Scapulae Muscle

Rationale: If present, tightness can contribute to multiple flexion restrictions (extended, rotated, and side-bent [ERS] dysfunctions that are rotated and side-bent to the ipsilateral side of the hypertonic muscle) affecting C2-C4. It also limits scapular depression as well as shoulder function.

Bilateral-lateral Stretch: Cervical Soft Tissue

Rationale: This technique applies a stretch to help relax the cervical paraspinal soft tissue.

Direct Myofascial Release: Thoracic Inlet. Boundaries include anterior: clavicles and first ribs; lateral: first ribs; posterior: cervicothoracic [CT] junction.

Rationale: Allows range of motion of the clavicles, first ribs, and CT junction to be as physiologic as possible. This also allows for optimal lymphatic drainage.

Muscle Energy: Typical Cervical Vertebrae (C2-T1)

Rationale: Directly improves range of motion through the restrictive barriers of extension, rotation, and side bending to the left (this can relieve tension on cervical nerve roots).

Muscle Energy: Typical Cervical Vertebrae (C2-T1)

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A functional indirect technique can be used for either of the above two techniques.

1. With the index and middle fingers over the dysfunctional segment, the clinician supports the athlete's head and upper cervical spine.
2. The clinician seeks the dynamic balance point (point of maximum ease) in the following planes: anterior-posterior translation, left to right/right to left translation, rotation bilaterally.
3. Distraction/compression is attempted at the dysfunctional segment. Continue that which maintains the dynamic balance point.
4. Motion is initiated to the direction of ease and followed until tissue tension releases.
5. Retest (5).

Prevention

Proper blocking and tackling technique should be taught to football players instead of spearing or head tackling. Ensure that shoulder pads and neck roll, if used, are in good condition and fit properly (10). The neck can be strengthened with cervical isometric exercises in flexion, extension, side bending, and rotation. Progressive resistive exercises can benefit the rotator cuff muscles as well (internal-external rotation and abduction) (11). The shoulders and upper back can be strengthened with the following exercises.

1. *Upper back exercises*
   - *Seated rows* are performed with the back straight and the shoulder blades set in a squeezed-together position (11).
   - *Lateral pull-downs* are usually done seated. The athlete leans back slightly at the hips, grips the bar slightly wider than shoulder width, and pulls the bar down in front of the head, touching the bar to the chest. Avoid behind-the-neck lateral pull-downs, as they can increase cervical strain (11).
2. *Top of shoulder exercise. The athlete performs shoulder shrugs* with a palm-down hold on the barbell (or a dumbbell in each hand). Keep hands and feet 16 in. apart. Hold the bar at arm's length, shrug the shoulders up, and rotate them backward (11).

3. *Chest and shoulder blade exercise* (cable-weight machine or tubing). *Cable crossovers* can strengthen the pectoral muscles as arms are extended, and the serratus anterior muscles as wrists cross each other (11).

Stretches for the Head and Neck

1. *Upper trapezius, sternocleidomastoid, anterior and medial scalenes* (Fig. 16.3.11A). The athlete holds the table with one hand while

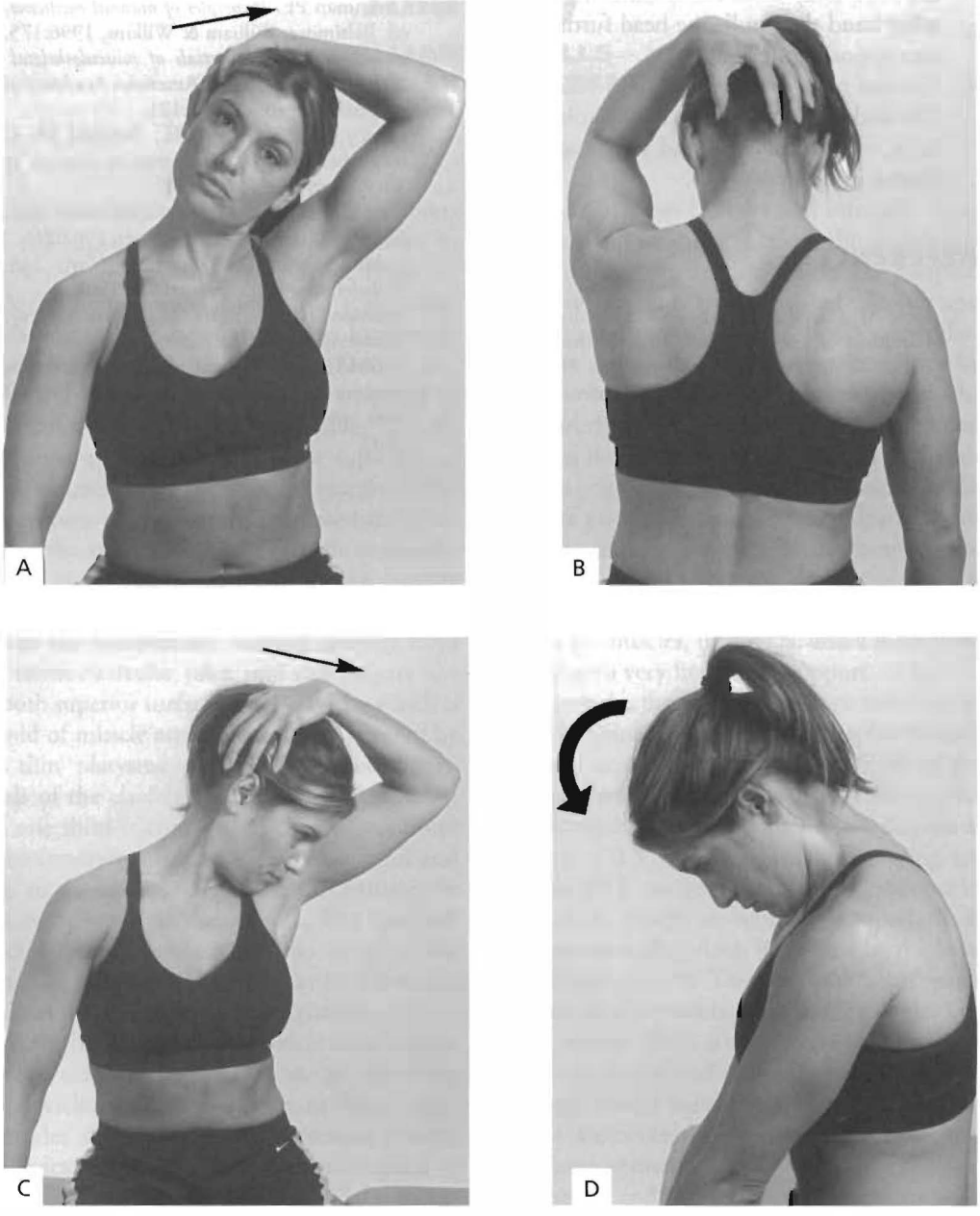

FIGURE 16.3.11. Stretches. **A,** Upper trapezius, sternocleidomastoid, anterior and medial scalenes. **B,** Lower trapezius. **C,** Levator scapulae. **D,** Cervical paraspinal muscle.

the other gently side-bends the head to the left or right.

2. *Lower trapezius* (Fig. 16.3.11B). The athlete sits with the head down, pushing down the head into flexion while sitting up.
3. *Levator scapulae* (Fig. 16.3.11C). The athlete holds the table with one hand while turning the head away from the ipsilateral side. The other hand then pulls the head further away into opposite side bending.
4. *Cervical paraspinal muscles* (Fig. 16.3.11D). The athlete sits with both hands holding the table, chin tucked in, and the head gently moved into flexion.

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17

THE SHOULDER

17.1

Anatomy

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The shoulder girdle is composed of three joints and one articulation, which work together to permit movement of the upper limb. These include the sternoclavicular joint, the acromioclavicular joint, the glenohumeral (shoulder) joint, and the scapulothoracic articulation (1–6).

Several bony structures can be palpated as one examines the shoulder girdle (Fig. 17.1.1). Beginning on the midline is the suprasternal notch situated along the superior margin of the manubrium of the sternum. Immediately lateral to the suprasternal notch is the sternoclavicular joint. The medial end of the clavicle participates in this joint and lies slightly superior to the manubrium. Moving laterally from the sternoclavicular joint one can palpate the smooth superior surface of the clavicle, which is devoid of muscle attachment and is covered by the thin platysma muscle. The medial two thirds of the clavicle are convex, while the lateral one third is concave. At the deepest point of the concavity of the clavicle and inferior and deep to the anterior edge, one can palpate the coracoid process of the scapula. The coracoid process faces anterolaterally, lies deep to the pectoralis minor muscle, and may be felt as one palpates into the deltopectoral triangle.

At the lateral end of the clavicle is the subcutaneous acromioclavicular joint. At this joint the clavicle, which has begun to flatten out, protrudes slightly above the acromion process of the scapula. The acromion is rectangular in shape, contributes to the contour of the shoulder, and can be palpated on its superior and anterior surfaces.

As one palpates laterally and inferiorly from the lateral lip of the acromion, the greater tubercle of the humerus can be felt. The bicipital groove of the humerus is situated anterior and medial to the greater tubercle. It is bordered laterally by the greater tubercle and medially by the lesser tubercle and is best palpated with the arm laterally rotated. Within the bicipital groove is the tendon of the long head of the biceps muscle. Inferior to the acromion is the shoulder joint, which cannot be palpated. This is a very mobile joint where the head of the humerus fits into a very shallow glenoid cavity. As a result, the humerus is suspended from the scapula by muscles, ligaments, and a loose joint capsule with very little bony support.

Posteriorly, the acromion tapers and continues as the spine of the scapula. The spine extends obliquely across the superior four fifths of the posterior surface of the scapula and ends as a flat triangle on the vertebral border of the scapula at the level of T3. The vertebral border, lying approximately 5 cm from the spinous processes of the thoracic vertebrae, terminates superiorly at the superior angle, which is covered by the levator scapulae muscle. The vertebral border ends inferiorly at the subcutaneous inferior angle. The lateral border, which is covered by the latissimus dorsi, teres major, and teres minor muscles, runs obliquely toward the glenoid.

The sternoclavicular joint is divided into two compartments by an articular cartilage. The medial end of the clavicle articulates with the manubrium of the sternum and the first costal cartilage, reinforced by the anterior and

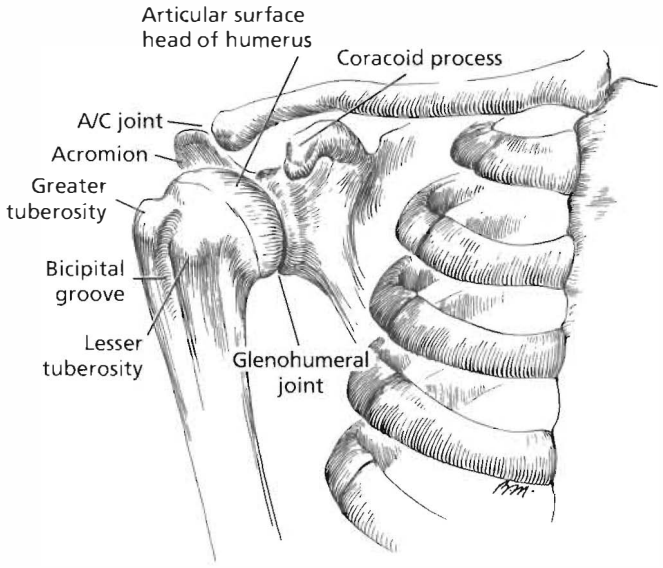

FIGURE 17.1.1. Bony anatomy of the shoulder. (From Rockwood, Green. *Fractures in Adults*, 4th ed., vol 2. Baltimore: Lippincott Williams & Wilkins, 1996.)

posterior sternoclavicular ligaments. The costoclavicular ligament, medial to the joint, attaches the inferior surface of the clavicle to the first rib and costal cartilage. The sternoclavicular joint moves in anterior, posterior, superior, and inferior directions. The joint is innervated by the medial supraclavicular nerve and the nerve to the subclavius.

The acromioclavicular joint is a synovial joint, surrounded by an articular capsule, where the lateral end of the clavicle articulates with the acromion of the scapula. The superior and inferior acromioclavicular ligaments strengthen the joint superiorly and inferiorly, and the coracoclavicular ligament attaches the clavicle to the underlying coracoid process of the scapula. The acromion rotates on the clavicle from scapulothoracic movements. The acromioclavicular joint is innervated by the supraclavicular, lateral pectoral, and axillary nerves.

The glenohumeral joint is a synovial joint where the head of the humerus articulates with the shallow glenoid cavity of the scapula, which is deepened by the fibrocartilaginous glenoid labrum (Fig. 17.1.2). The loose joint capsule is attached medially to the margin of the glenoid cavity and laterally to the anatomic neck of the humerus. The synovial lining of the capsule forms a sheath for the tendon of the long head of the biceps muscle, which runs through the joint cavity.

The three glenohumeral ligaments strengthen the anterior aspect of the joint capsule, and the coracohumeral ligaments strengthen the joint superiorly. The capsule is also strengthened by the transverse humeral ligament, which helps to hold the tendon of the long head of the biceps muscle in the bicipital groove. The coracoacromial ligament extends across the superior aspect of the joint, separated from the joint capsule, and together with the coracoid process and the acromion forms the strong coracoacromial arch, which protects the joint superiorly. The shoulder joint receives its blood supply from the suprascapular artery and branches of the anterior and posterior humeral circumflex arteries from the axillary artery and is innervated by the suprascapular, axillary, and lateral pectoral nerves.

The rotator cuff muscles attach the scapula to the humerus (Fig. 17.1.3A and B). The supraspinatus, infraspinatus, and teres minor are palpable at their attachments onto the greater tubercle of the humerus. The supraspinatus is an abductor of the humerus, while the infraspinatus and teres minor are lateral rotators. With the arm hanging at the side, the supra-

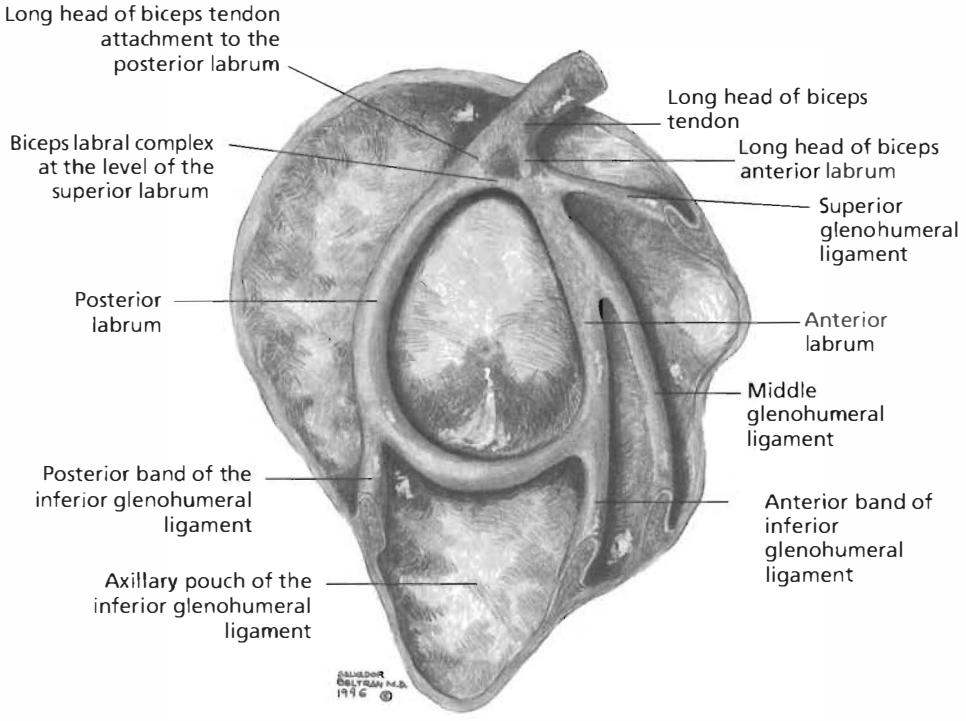

FIGURE 17.1.2. Glenohumeral ligaments and labrum, lateral view. (From Stoller DW. *MRI, arthroscopy, and surgical anatomy of the joints.* Baltimore: Lippincott Williams & Wilkins, 1999.)

spinatus lies directly over the head of the humerus deep to the acromion (Fig. 17.1.4). The infraspinatus is posterior and inferior to the supraspinatus, and the teres minor is posterior and inferior to the infraspinatus. The fourth cuff muscle, the subscapularis, lies anterior to the humeral head, attaches to the lesser tubercle of the humerus, and acts as a medial rotator of the humerus. The supraspinatus is innervated by the suprascapular nerve (C4, C5, C6); the infraspinatus by the suprascapular nerve (C5, C6); the teres minor by the axillary nerve (C5, C6); and the subscapularis by the upper and lower subscapular nerves (C5, C6, C7).

Bursae surround the shoulder lying between the tendons of the rotator cuff muscles and the shoulder joint capsule. They reduce friction on the tendons passing over bones and other areas of resistance. The subacromial bursa lies between the tendon of the supraspinatus muscle and the overlying acromion process of the scapula. Several portions of this bursa are palpable. A lateral extension of the subacromial bursa, the subdeltoid bursa, extends deep to the deltoid muscle, separating it from the rotator cuff below and allowing each to move more freely.

Several other muscles attach to the shoulder girdle and can be palpated. The sternocleidomastoid muscle arises from the manubrium of the sternum and the medial third of the clavicle, bisects the neck obliquely, and attaches superiorly to the mastoid process of the temporal bone. This muscle laterally flexes the neck and rotates the head to the opposite side and is innervated by the spinal accessory nerve (CN XI).

The fan-shaped pectoralis major muscle forms the anterior wall of the axilla, arises from the medial two thirds of the clavicle and the anterior surface of the sternum, and inserts into the lateral lip of the bicipital groove of the humerus. It adducts and medially rotates the humerus, and draws the scapula anteriorly and inferiorly. The pectoralis major receives innervation from the lateral and medial pectoral

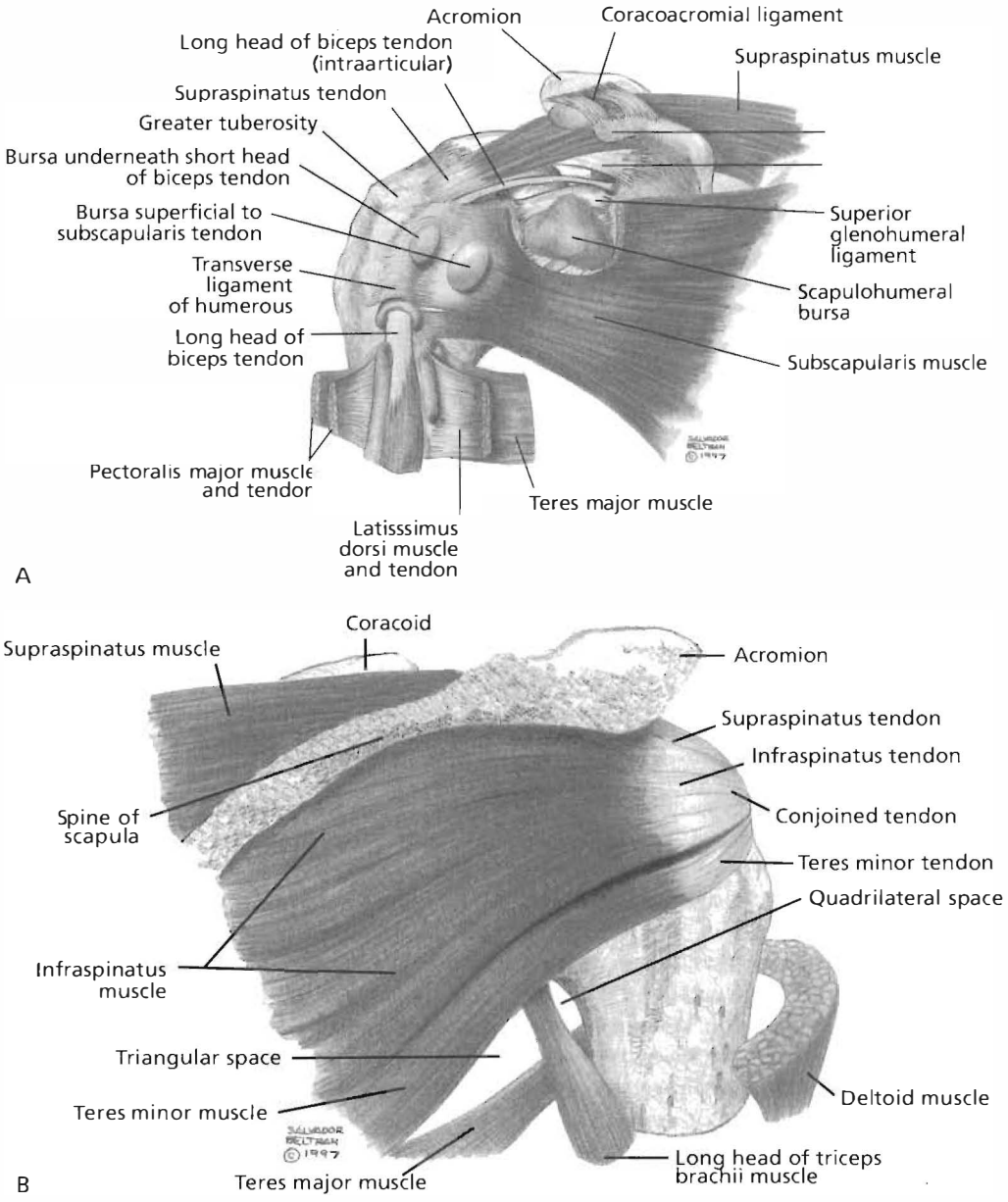

FIGURE 17.1.3. A, Rotator cuff, anterior view. **B,** Rotator cuff, posterior view. (From Stoller DW. *MRI, arthroscopy, and surgical anatomy of the joints.* Baltimore: Lippincott Williams & Wilkins, 1999.)

nerves (clavicular head [C5, C6] and sternal head [C7, C8, T1]).

The biceps muscle, located in the anterior compartment of the arm, is composed of two heads, the long head and short head. The long head runs through the bicipital groove of the humerus and the shoulder joint cavity to attach to the supraglenoid tubercle of the scapula. The short head attaches to the coracoid process. Both heads attach distally to the radial tuberosity in the forearm. The muscle supinates the forearm and flexes a supinated forearm. It receives its innervation from the musculocutaneous nerve (C5, C6).

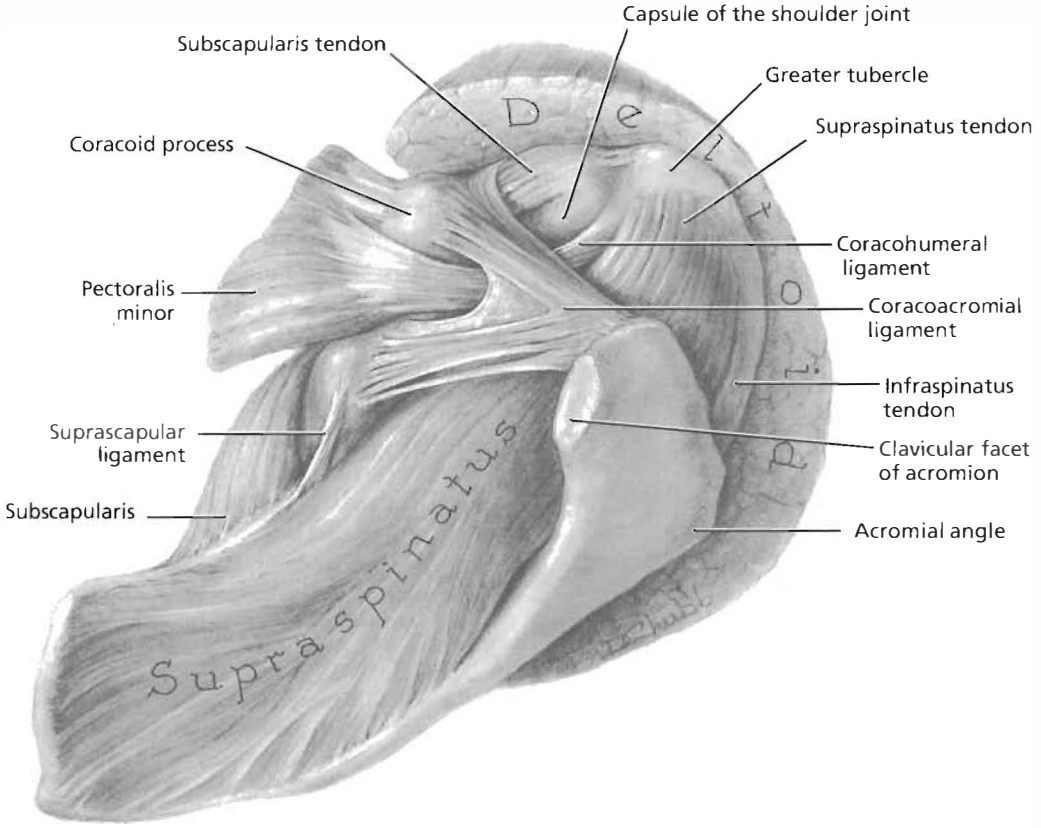

FIGURE 17.1.4. Superior view of the supraspinatus anatomy relevant to impingement. Note the coracoacromial ligament forming the anterior roof of the subacromial space. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy*, 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

The deltoid muscle contributes to the full contour of the shoulder. It has a broad, curved origin from the lateral one third of the clavicle, acromion, and spine of the scapula. The fibers converge to attach halfway down the lateral surface of the humeral shaft on the deltoid tuberosity. The anterior part of the muscle flexes and medially rotates the arm, the middle part abducts the arm, and the posterior part extends and laterally rotates the arm. The deltoid muscle is innervated by the axillary nerve (C5, C6).

The trapezius muscle is prominent in the cervical region. It arises from the medial one third of the superior nuchal line, external occipital protuberance, nuchal ligament, and the spinous process of the C7-T12 vertebrae. Its fibers attach onto the shoulder girdle at the lateral one third of the clavicle, acromion, and spine of the scapula. The main actions of this muscle are scapular elevation-depression, retraction, and rotation. The trapezius muscle is innervated by the spinal accessory nerve (CN XI).

The rhomboid (major and minor) muscles are postural muscles, that originate from the nuchal ligament and the spinous processes of the C7-T5 vertebrae. They extend laterally and attach to the vertebral border of the scapula from the spine to the inferior angle. These muscles retract the scapula and rotate it to depress the glenoid cavity as well as fixing the scapula to the thoracic wall. The rhomboid muscles are innervated by the dorsal scapular nerve (C4, C5).

The latissimus dorsi muscle has a broad origin from the spinous processes of the lower six thoracic vertebrae, thoracolumbar fascia, iliac crest, and the lower three or four ribs. The muscle is

directed toward the shoulder where its tendon twists upon itself before attaching into the floor of the bicipital groove of the humerus. The muscle forms the posterior wall of the axilla. The latissimus dorsi extends, adducts, and medially rotates the humerus and is innervated by the thoracodorsal nerve (C6, C7, C8).

The serratus anterior muscle can be palpated along the medial wall of the axilla. It originates from the external surfaces of the lateral aspects of the first to eighth ribs and attaches along the length of the anterior surface of the vertebral border of the scapula. The serratus anterior muscle is important in holding the scapula against the thoracic wall as well as in helping in scapular protraction and rotation. This muscle is innervated by the long thoracic nerve (C5, C6, C7). The attachments of the rhomboid and serratus anterior muscles from thoracic spinous processes and ribs, respectively, to the vertebral border of the scapula form the scapulothoracic articulation.

The axilla is a pyramidal area at the junction of the arm and the thorax. The apex lies between the first rib, clavicle, and superior edge of the subscapularis muscle. Arteries, veins, lymphatics, and nerves pass through the apex connecting the arm and the base of the neck. The pectoralis major muscle forms the anterior wall of the axilla; the posterior wall is formed chiefly by the latissimus dorsi muscle. The second through sixth ribs and the overlying serratus anterior muscle define the medial wall. The lateral wall is the bicipital groove of the humerus containing the tendon of the long head of rhe biceps muscle. The brachial plexus arises from C5-T1 nerve roots and forms the origin of the major nerves to the upper limb. It runs in the axilla along with rhe axillary artery and its branches, the axillary vein and its tributaries, and axillary lymph nodes.

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17.2

Physical Examination

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The examination always begins with a thorough history, which leads to specific physical examination findings and provocative tests/maneuvers for the suspected condition. Shoulder pain can be primary or referred from the cervical or thoracic areas, even from the heart, as with angina or myocardial infarction. The history leads the clinician to areas that require evaluation.

Therefore, the history should include questions about the onset of the pain. Was it acute or gradual? If the pain was acute, was there an associated pop? If so, inquire about hearing more than one pop (a popping-out and popping-back-in sensation) that would suggest subluxation or dislocation. Ask about associated numbness, tingling, or burning pain distally along the upper

extremity. Also ask about falls or trauma onto the tip of the shoulder or onto a flexed elbow or an outstretched arm.

If the athlete is involved in a throwing or racquet sport, learn the phase of each activity when the pain occurs, such as the cocking phase, acceleration phase, release phase, or follow-through. Ask about any crepitance or a feeling of looseness since the injury. The clinician should assess the quality of pain: sharp or dull, radiating or localized, intermittent or constant, worse with certain ranges of motion or activities such as throwing or raising the arms.

When starting the physical examination of the shoulder, always examine the cervical spine as well. Even if the history is classic for an isolated shoulder injury, cervical pathology may be implicated, especially if the shoulder is targeted for treatment and the neck is not.

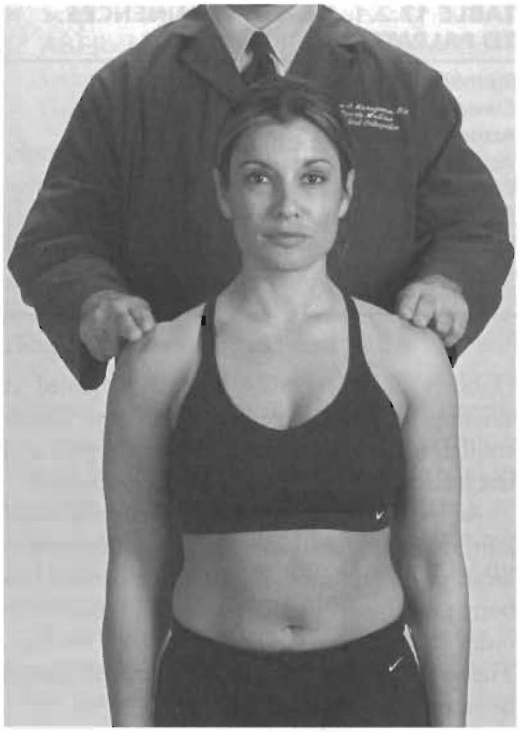

FIGURE 17.2.1. The clinician stands behind the athlete with fingers on the acromion to evaluate shoulder heights.

OBSERVATION

Observe the athlete entering the examination room. Observe the athlete's movement of the arms as he or she walks, sits on the examination table, or possibly removes a jacket or other outer clothing. Many times, the examiner's first encounter with the athlete is when he or she is seated on the examination table. Watch how the athlete extends his or her hand to shake and disrobe.

Exposing both shoulders, the examiner should observe the anterior view with close attention to the acromion heights, the sternoclavicular and acromioclavicular joints, and the deltoid, pectoral, and anterior trapezius muscles (Fig. 17.2.1). The bicipital groove and biceps muscle belly should be observed for deformity and swelling. In a lateral view, look for an increase in thoracic kyphosis, uneven scapulae, and/or winging of the scapulae. Also check proper alignment of the head with the acromion process to rule out a cervical process.

Posteriorly, look for a low shoulder or uneven scapulae by checking the acromion processes and the inferior angles of the scapulae. Note any hypertrophy or atrophy of the posterior shoulder girdle musculature. It is not uncommon to find hypertrophy in the dominant extremity, especially in throwing or racquet sport athletes. Examine the trapezius, levator scapulae, and rhomboid muscle groups for symmetry and strength, particularly for symmetry with the scapula. Note any scapular winging.

On inspection, also note indentations, nodules, or skin changes such as discoloration, hematomas, abrasions, and erythema. Scars, either postsurgical or otherwise, should be noted. Look for deformities from such conditions as glenohumeral dislocations, clavicular fractures, and acromioclavicular sprains. The scapulothoracic relationship should be noted. The scapula may be either retracted, protracted, or exhibit uneven heights.

PALPATION

Begin by systematically palpating the bony prominences, and the muscles and tendons of the shoulder and the scapulothoracic region

TABLE 17.2.1. BONY PROMINENCES TO PALPATE

Sternoclavicular (SC) joint
Clavicle
Acromioclavicular (AC) joint
Acromion process
Coracoid process
Scapular spine
Proximal humerus
Bicipital groove
Greater tuberosity

(Table 17.2.1). Palpation should be aimed at eliciting tenderness. Be sure to palpate deeply enough where the athlete complains of pain and test bilaterally so the athlete has a comparison.

An indentation posterior to the glenohumeral joint may suggest an anterior glenohumeral dislocation. Significant palpable tenderness on bony prominences should cause the clinician to order radiographic studies to rule out fracture. The greater tuberosity of the humerus should be easily palpated (Fig. 17.2.2).

Palpation of the muscles should target the pectoralis, biceps brachii, deltoid (anterior portion, midportion, and posterior portion), supraspinatus, infraspinatus, teres minor, trapezius, and rhomboid posteriorly, and the latissimus dorsi, which comprises the posterior axilla. The sternocleidomastoid muscle should also be evaluated, as it is the most superior muscle involved in shoulder motion.

Palpate the tendons of the shoulder, specifically, the supraspinatus and the bicipital tendons. The subacromial bursa can also be palpated in most athletes. This can be done seated or standing. To palpate the supraspinatus tendon at the greater tubercle, internally rotate the arm with the humerus at the side and elbow flexed at 90 degrees (Fig. 17.2.3). The biceps tendon can be palpated between the greater and lesser tubercles of the humerus (Fig. 17.2.4). External rotation of the humerus allows the subscapularis to be palpated at the lesser tubercle (Fig. 17.2.5). Palpate all muscle bellies and attachments at the scapula, acromion, crest of the scapula, and thoracic wall.

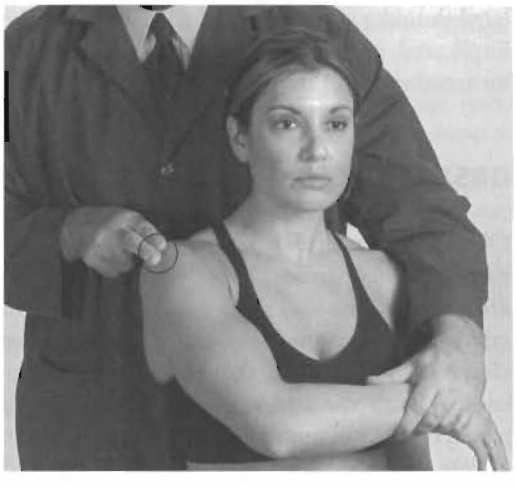

FIGURE 17.2.3. Supraspinatus palpation.

FIGURE 17.2.2. Greater tuberosity palpation.

FIGURE 17.2.4. Biceps tendon palpation.

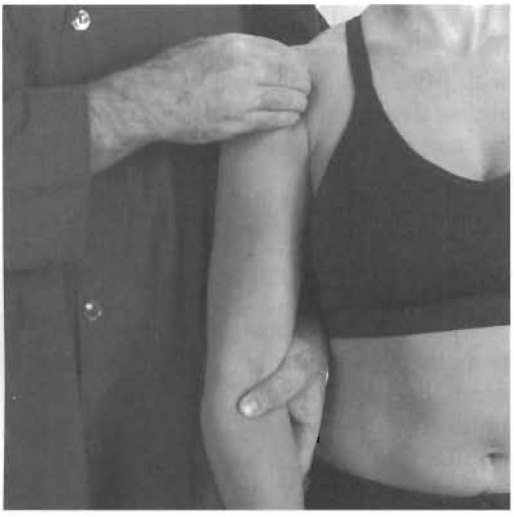

FIGURE 17.2.5. Subscapularis palpation at the lesser tuberosity.

RANGE OF MOTION

The shoulder should be actively and passively assessed in the following ranges:

a. Abduction—arms at side, out at side, and above the head
b. Adduction—arm at 90-degree elevation, brought across the chest
c. Flexion—arms at side, lifted in front of the athlete over the head
d. Extension—arms at side, brought straight back behind the athlete
e. Internal rotation—arm at side, elbow flexed 90 degrees, rotate arm in and bring behind back
f. External rotation—arm at side, elbow flexed 90 degrees, rotate arms out
g. Scapular retraction—squeeze scapula together
h. Scapular protraction—reach forward with both arms while observing scapular motion
i. Scapular elevation—shoulder shrug
j. ABER (ABduction, External Rotation)—same position as for the apprehension sign (see Fig. 17.2.18A).

Note range of motion and end-feel, as well as symptoms of instability, apprehension, and pain. If the active range of motion is limited, the examiner should assess the passive ranges of motion carefully. For instance, "sticking points" in a passive range of motion may identify a mechanical barrier such as a labral tear, rotator cuff tear, or adhesion formation. Pain can limit an athlete's active range of motion, but if there is no mechanical barrier, then the examiner should be able to passively move the shoulder past that end point. Make sure the athlete is relaxed during passive range of motion. Note any popping, clicking, or crepitus perceived by the athlete or noted by the examiner.

There is quite a range of normal variance in the population. Some athletes may have flexibility that would suggest possible capsular laxity but is normal for that particular athlete. As with most "loose-jointed" individuals, this could predispose the athlete to muscular injury. Always compare both shoulders to distinguish general laxity from specific instability.

Look for glenohumeral motion isolated from scapulothoracic motion. Test the first 30 to 40 degrees of movement by having the athlete raise both arms from his or her side overhead with straight elbows. Normally, the scapula will not start to rotate until the arm is abducted beyond 30 degrees. If abduction occurs with the shoulders shrugging, the shoulder is using scapulothoracic motion to compensate for glenohumeral restriction. If abduction is not nored, glenohumeral motion is likely independent and normal (Fig. 17.2.6).

FIGURE 17.2.6. Elevation of both arms to 90 degrees.

Glenohumeral restriction is best seen in cases of adhesive capsulitis. Usually the scapula rotates 1 degree for every 2 degrees of abduction at the glenohumeral joint. In a frozen shoulder, glenohumeral motion is markedly reduced, so abduction comes almost exclusively from scapular motion.

Apley's Scratch Test

There are two Apley's scratch maneuvers, lower and upper. The lower test grossly checks internal rotation, adduction, and extension. The upper test checks for external rotation, abduction, and flexion. It helps to determine range of motion early in rehabilitation and then to continually assess range of motion as rehabilitation progresses. It should be noted that most athletes have inequality with this maneuver, for most athletes normally have one shoulder that is somewhat tighter than the other.

Lower test. The athlete puts the arm behind his or her back with the palm facing outward and the thumb pointed cephalad. The athlete should then elevate the thumb as high as possible on the back while the examiner notes the level on the spine at which the thumb can reach (Fig. 17.2.7A). From here, the liftoff test can be done.

Upper test. The athlete abducts the arm, placing the palm of the hand behind the neck with palm facing toward his or her body. The athlete should then attempt to scratch the lowest possible vertebrae with the index finger (Fig. 17.2.7B).

NEUROVASCULAR EXAMINATION

Sensorium. The dermatomes of the upper extremity are shown in (Fig. 17.2.8). Sensation is tested by pinprick and followed by a small brush. The two-point discrimination test is also helpful.

Reflexes

1. *Biceps (C5 nerve root):* The examiner places his or her thumb on the insertion of the biceps tendon. A small hammer then strikes the thumb of the examiner.
2. *Triceps (C6 nerve root):* The athlete's arm is at 90 degrees of vertical abduction and the elbow is flexed to 90 degrees with 90 degrees of internal rotation. The examiner then holds the upper arm in one hand and taps the triceps tendon, which provokes elbow extension.

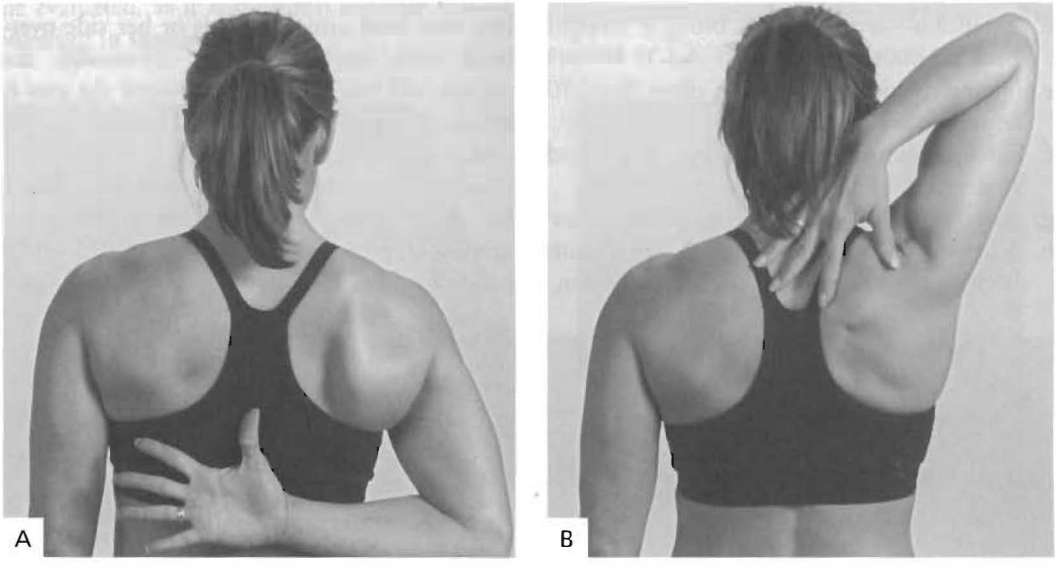

FIGURE 17.2.7. Apley's scratch test. **A,** Lower. **B,** Upper.

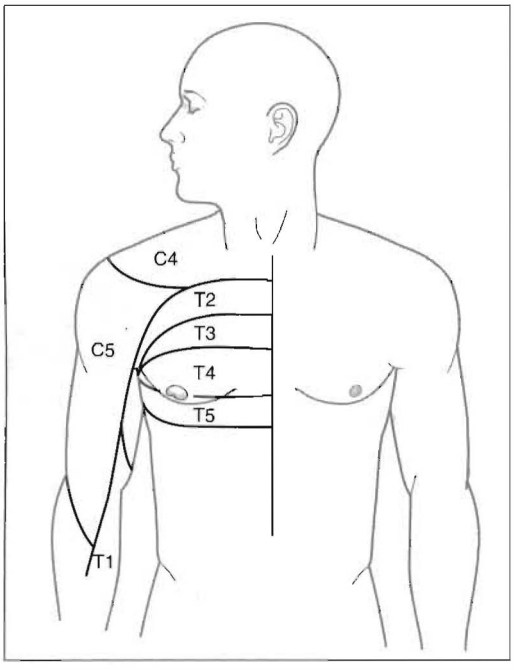

FIGURE 17.2.8. Dermatomal patterns of the shoulder and arm.

3. The *brachial plexus* traverses the glenohumeral joint on its way down the arm, and shoulder pain can emanate from any plexopathy. Any neurologic deficits can cause pain radiating down the nerves themselves, or create muscle weakness that can lead to shoulder pathology such as rotator cuff impingement and scapulothoracic dysfunction. These nerves can be tested through manual muscle testing.

Manual Muscle Testing

The list below demonstrates the maneuvers used to test specific muscles for strength. Table 17.2.2 shows the vertebral levels comprising upper extremity strength(1,2).

Specific Strength Testing

- Deltoid (C5 and C6—axillary nerve):
  - Anterior deltoid: arm at 90 degrees of abduction, full external rotation and supination
  - Middle deltoid: arm as for anterior deltoid, but with some internal rotation and palm facing down
  - Posterior deltoid: arm as for anterior deltoid, but with full internal rotation and palm facing backward
- Teres minor: arm at side, elbow at 90 degrees of flexion, athlete pushes hands out
- Infraspinatus: arm at 90 degrees of abduction, elbow partially bent, resistance applied downward
- Subscapularis: arm at 90 degrees of abduction, resistance directed upward
- Supraspinatus: empty can or full can test
- Trapezius: shrug shoulders against inferior resistance
- Serratus anterior: arm flexed 90 degrees, palm down, resistance downward
- Rhomboids: hand on hip with elbow at 90 degrees, resistance anterior against elbow

TABLE 17.2.2. UPPER EXTREMITY STRENGTH

C5: Deltoid abduction at shoulder
C6: Biceps flexion at forearm
C6: Wrist extension (extensor carpi radialis)
C7: Wrist flexion
C7: Elbow extension (triceps)
C7: Finger extension
C8: Finger flexion, middle finger (flexor digitorum profundus)
T1: Small finger abductors (abductor digiti minimi)
T1: Spread fingers (interossei)

Supraspinatus (C4-C6, Suprascapular). Set up athlete with arms abducted 90 degrees and flexed 45 degrees with the thumbs up (full can test). If the arm does not drop, then the examiner can assess strength by the athlete's resistance against inferior directed force.

Subscapularis (C5-C7, Upper and Lower Subscapular). The athlete rests the arms at the side with elbows flexed at 90 degrees. The examiner places his or her hands inside the athlete's wrists to resist internal rotation.

Infraspinatus (C5-6, Suprascapular). The athlete sets up as for the subscapularis, and the examiner resists external rotation by placing

the hands on the outside of the wrist, grading strength (+1 to +5) and noting any elicited pain.

Teres Minor (C5-6, Suprascapular). Same as for infraspinatus.

Levator Scapulae (C5, Dorsal Scapula; C3-4, Cervical). Either sitting or standing, the athlete elevates the scapula while the examiner resists with the hands on top of the shoulder, pushing inferiorly.

Serratus Anterior (C5-C7, Long Thoracic). Use the wall push-up test for scapular winging (see Fig. 17.2.12). Another way to test scapulothoracic strength is to ask the athlete to put both hands on the hips with the examiner behind him or her. The examiner then offers pressure to the medial aspect of the elbow and asks the athlete to push the elbows together posteriorly against the examiner's resistance, noting weakness or scapular winging.

PROVOCATIVE TESTS AND MANEUVERS

Cervical Spine

Spurling's Test. This is the first test to perform when evaluating the cervical intervertebral discs as a source of shoulder pain. The examiner extends the athlete's neck and rotates it to one side with corresponding axial compression on the athlete's head (see Fig. 16.2.9).

Positive sign: pain elicited down the ipsilateral arm from the neck.

Indicates: cervical disc disease.

Acromioclavicular Joint

Cross-arm Test. The examiner passively adducts the athlete's arm across the chest wall with the humerus parallel to the ground so that the free hand of the examined elbow rests on the opposite shouldet (Fig. 17.2.9). The athlete then pushes the elbow superiorly against the clinician's resistance.

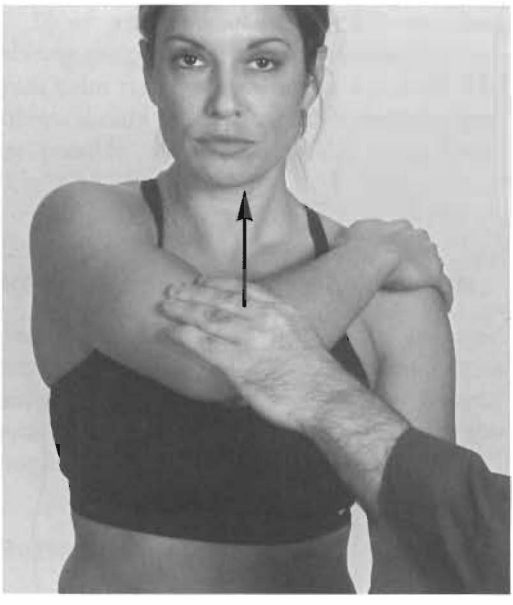

FIGURE 17.2.9. Acromioclavicular crossover test.

Positive sign: Pain with end-range adduction or with pushing arm up.

Indicates: Acromioclavicular joint pathology (3).

Active Compression Test. With the examiner behind the athlete, the athlete vertically abducts the arm to 90 degrees and adducts 10 to 15 degrees with internal rotation (thumbs down) (Fig. 17.2.10A). The athlete resists the examiner's downward force. This maneuver is then repeated with the arms supinated (Fig. 17.2.10B).

Positive test: Pain that improves or resolves with the second maneuver.

Indicates: Acromioclavicular (AC) pathology if the pain is in the AC joint, or labral pathology if the pain feels more internal in the shoulder. (4)

Scapulothoracic Motion

Range of Motion. The examiner observes scapular rhythm as the athlete abducts the shoulder over the head. Note any difference between the scapulae in elevation and depression, external and internal rotation, retraction, and protraction.

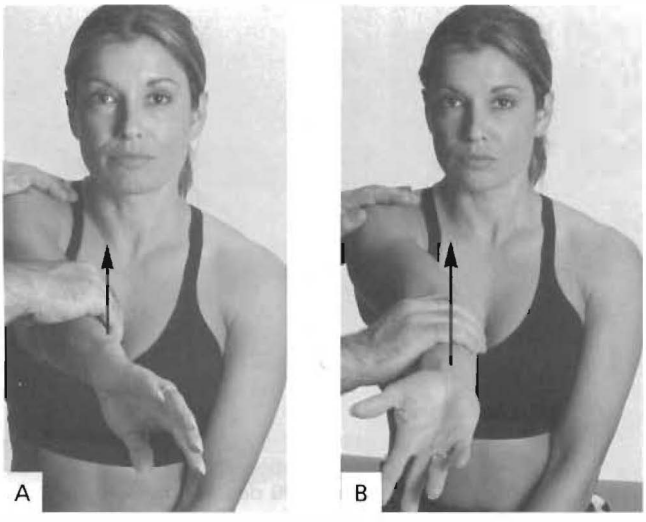

FIGURE 17.2.10. Active compression test. **A,** The athlete's arm is internally rotated. **B,** Then the arm is externally rotated.

Scapular Winging. This is often related to weakness of the serrratus anterior, which is innervated by the long thoracic nerve (Fig. 17.2.11).

Test 2: The athlete stands in a relaxed position or with the hands on the hips. Have the athlete hang the arms at the side with palms out, then bring them up to a wall or door and perform a push-up off the wall. Observe the scapular behavior (Fig. 17.2.12B).

Test 1: The athlete forward flexes the arms at 90 degrees against clinician's or tubing resistance (Fig. 17.2.12A).

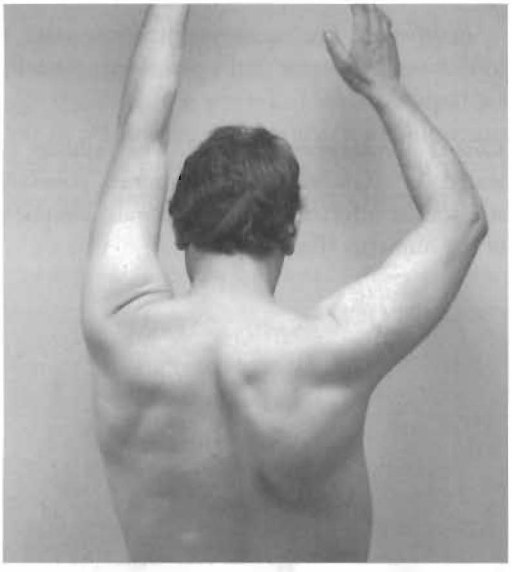

FIGURE 17.2.11. Scapular winging in a 38-year-old man is demonstrated by a wall push-up and forward flexion. This is the classic picture seen when a long thoracic nerve palsy weakens the serrratus anterior muscle.

Bicipital Tendon

Yergason's Test. The athlete is in the seated position with the elbow at his or her side and flexed to 90 degrees. The examiner holds the elbow with one hand while holding the wrist with the other hand. The examiner then tests the stability of the biceps tendon by externally rotating the athlete's arm against resistance while simultaneously pushing downward on the elbow (Fig. 17.2.13A).

Positive test: Tendon will pop out of the groove and cause significant pain (1).

Indicates: Unstable bicipital tendon and subluxation, subscapularis damage.

Speed's Test. This test also looks for bicipital groove pathology. The athlete's shoulder is in 90 degrees of forward flexion, elbow extended and hand supinated, with applied resistance to cephalad motion (Fig. 17.2.13B) (5).

Positive test: Pain in the bicipital groove.

Indicates: Bicipital tendon pathology, usually tendinitis.

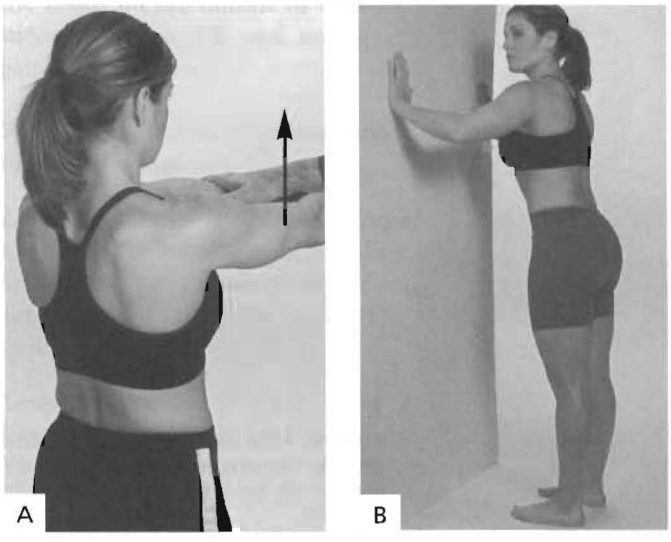

FIGURE 17.2.12. Scapular winging provocation, **A,** arms resisting flexion at 90 degrees. **B,** Doing a push-up off the wall

Rotator Cuff Impingement

Full Can Test. The athlete abducts both arms to 90 degrees and forward flexed 45 degrees with the thumbs pointing to the ceiling (Fig. 17.2.14). The athlete resists downward pressure.

Positive test: Weakness, pain, or dropping of the arm, which occurs in significant tears of the supraspinatus muscle with even a gentle tap to the forearm (6).

Indicates: Supraspinatus tendon tear.

Note: Recent studies suggest that the full can test is more sensitive for supraspinatus pathology than the empty can test, which has been the standard for years (5).

Empty Can Test. Same as the full can test, but with thumbs down.

Hawkins Test (Coracoid Impingement Test). The examiner passively rotates the humerus into internal rotation while forward flexing to 90 degrees in the sagittal plane. This opposes the rotator cuff against the coracoacromial ligament and acromion (Fig. 17.2.15B).

Positive test: The maneuver produces pain.

Indicates: Rotator cuff pathology (usually supraspinatus) (3).

Classic Impingement Sign. The athlete is seated or standing. The examiner passively stresses the affected atm into forward elevation of the humerus (Fig. 17.2.15C).

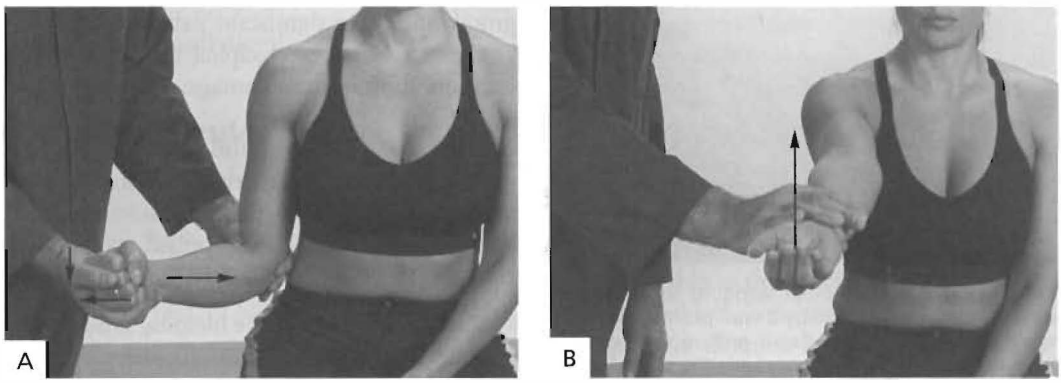

FIGURE 17.2.13. Biceps testing. **A,** Yergason's test. **B,** Speed's test.

FIGURE 17.2.14. Full can test.

Positive test: Pain is elicited as the impinged rotator cuff opposes the anterior acromial arch.

Indicates: Rotator cuff pathology (3).

Neer Impingement Test. The examiner stabilizes the athlete's shoulder on the top with his or her off hand, forward flexes the humerus to 90 degrees, then abducts the arm to about 80 degrees with the arm internally rotated (Fig. 17.2.15A).

Positive test: Provokes pain.

Indicates: Rotator cuff impingement pathology (8). If pain is provoked with forward flexion to 90 degrees, it is primary impingement. Pain that is provoked when the arm is moved into abduction is secondary impingement (3).

Injection Test. The examiner injects the subacromial space with 1% lidocaine (approximately 5–10 mL) and then repeats the impingement test. If no pain is produced, the examiner knows that the problem is in the subacromial area.

Painful Arc. The athlete abducts the arms overhead as far as they can go, bringing them out laterally (Fig. 17.2.16).

Positive test: Pain with shoulder abduction between 80 and 100 degrees in the coronal plane.

Indicates: Rotator cuff impingment.

Note: Pain starting after 100 degrees of abduction often suggests acromioclavicular joint

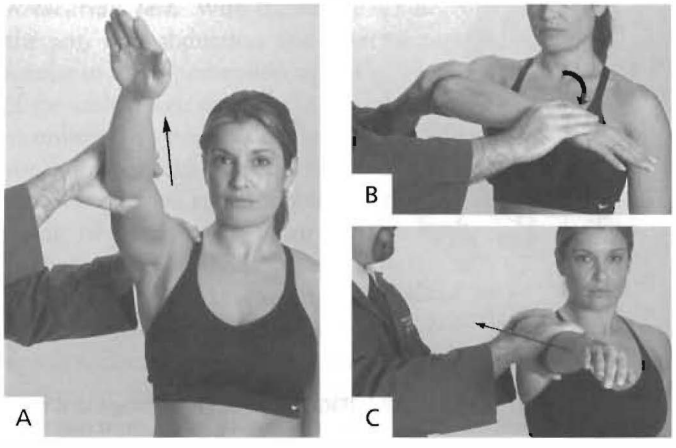

FIGURE 17.2.15. Impingement testing. **A,** Neer impingement. **B,** Hawkins test. **C,** Classic impingement.

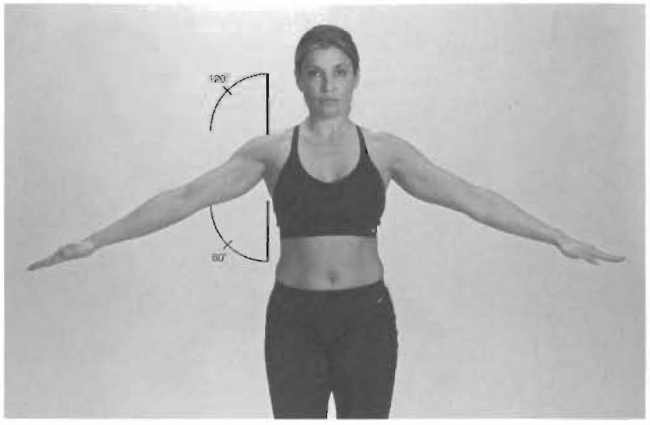

FIGURE 17.2.16. Painful arc. The athlete abducts the arms overhead.

pathology, while pain immediately with abduction may indicate adhesive capsulitis, shoulder trauma, or neurapraxia.

Subscapularis Injury

Napoleon Sign. If the athlete cannot fully internally rotate, place the athlete's hand on his or her stomach and have the athlete push against it (Fig. 17.2.17A).

Positive test: The elbow will drop backward.
Indicates: Subscapularis injury or weakness.

Gerber's (Liftoff) Test. This evaluates subscapularis muscle strength by limiting pectoralis major firing. The athlete puts his or her hand behind the lumbar spine and attempts to lift the hand away from the back (Fig. 17.2.17B).

Positive test: If the athlete cannot accomplish the liftoff (3).
Indicates: Subscapularis injury or weakness.

Glenohumeral Instability

Apprehension Sign (Crank Test). The athlete is in the sitting or supine position, with the arm vertically abducted to 90 degrees and the elbow flexed to 90 degrees. The forearm is then forced into external rotation past 90 degrees (Fig. 17.2.18A).

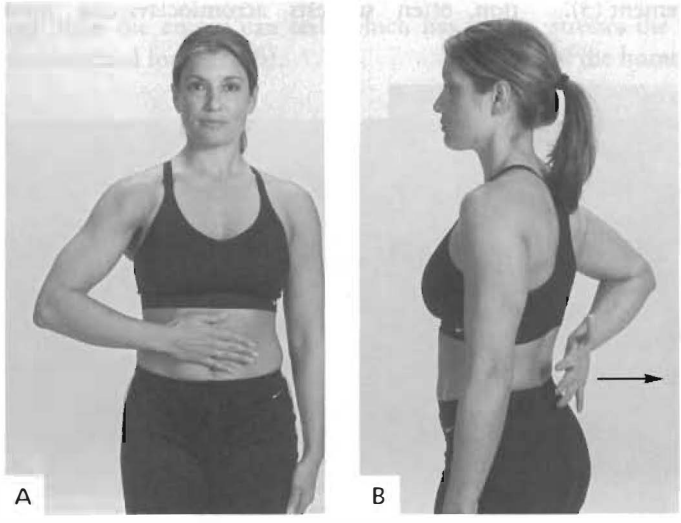

FIGURE 17.2.17. Subscapularis tests. **A,** Napoleon sign. **B,** Liftoff test.

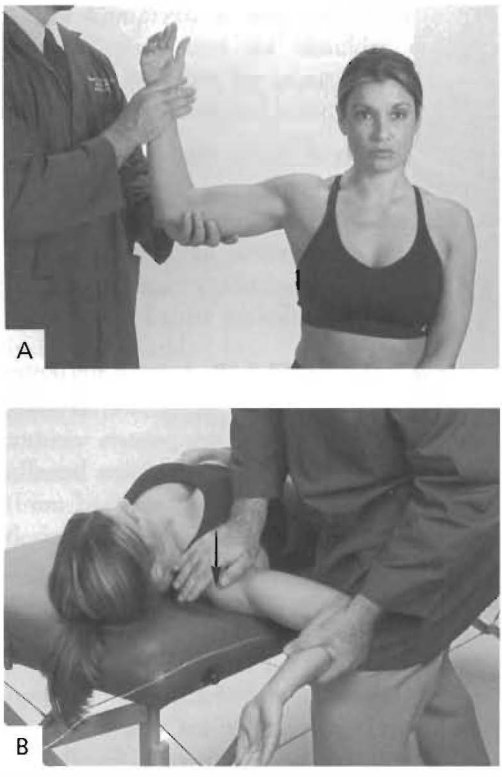

FIGURE 17.2.18. Glenohumeral instability signs. **A,** Apprehension sign. **B,** Relocation test.

Positive test: The athlete will be very apprehensive and ask the examiner to stop for fear of a repeat dislocation.

Indicates: Glenohumeral instability, previous glenohumeral dislocation or subluxation.

Relocation Test. With the athlete supine, rotate the arm into abduction and external rotation, similar to the apprehension sign (Fig. 17.2.18B). If the athlete feels unstable or apprehensive, the examiner then presses on the anterior aspect of the humerus with the other hand toward the table. A positive relocation test is an improvement of symptoms. From here, bring the humerus into further external rotation while still stabilizing the anterior shoulder. If the athlete's shoulder pain becomes worse when the arm is suddenly released, this is called a positive release, or surprise, test and corroborates the apprehension sign.

Note: A positive test on the apprehension, relocation, and surprise examinations is highly specific for glenohumeral instability (7). Additionally, Ian et al. found the surprise test was significantly more specific than the relocation test. In fact, the relocation test failed to identify instability in a significant portion of subjects tested (7).

Load Shift Test. The examiner stands behind the seated athlete and places one hand over the shoulder and scapula to steady the shoulder girdle. The opposite hand grasps the humeral head, fingers upon the medial edge, and pushes it into the glenoid fossa (Fig. 17.2.19). The examiner then shifts the head anteriorly, then posteriorly, noting pain and clicking, and measuring translation in each direction by a +1 to +3 scale. In a +3 load shift the humerus can be subluxed out of the glenohumeral joint.

Posterior Subluxation Test. The examiner moves the athlete's arm into internal rotation, adduction, and flexion. The examiner gently but firmly pushes posteriorly (Fig. 17.2.20).

Positive test: Pain, clunk, apprehension, or reproduction of symptoms.

Indicates: Posterior instability.

Sulcus Sign. The athlete's elbow is grasped and inferior traction is applied.

Positive test: An indentation appears in the area beneath the acromion.

Indicates: Inferior instability, multidirectional instability.

Labral Pathology

Clunk Test. The athlete is supine, and the examiner's hand is posterior to the humeral head. The examiner applies an anterior force to the humeral head, while the opposite hand holds the distal humerus and rotates it. The athlete's arm is then brought overhead into a fully abducted position (Fig. 17.2.21).

Positive test: A clunk or grinding in the shoulder.

Indicates: Labral pathology.

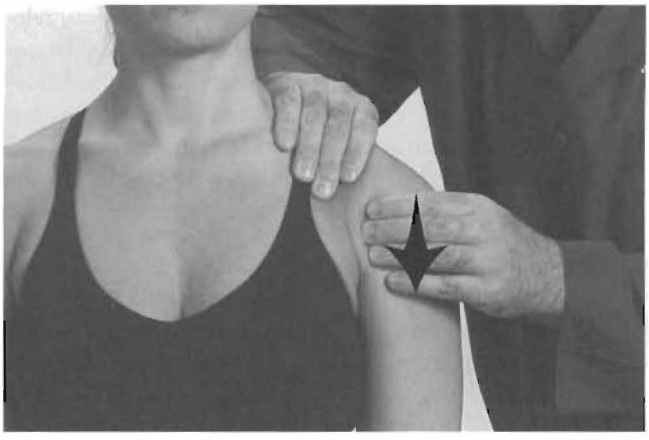

FIGURE 17.2.19. Anterior and posterior load shift, with the clinician standing behind the athlete.

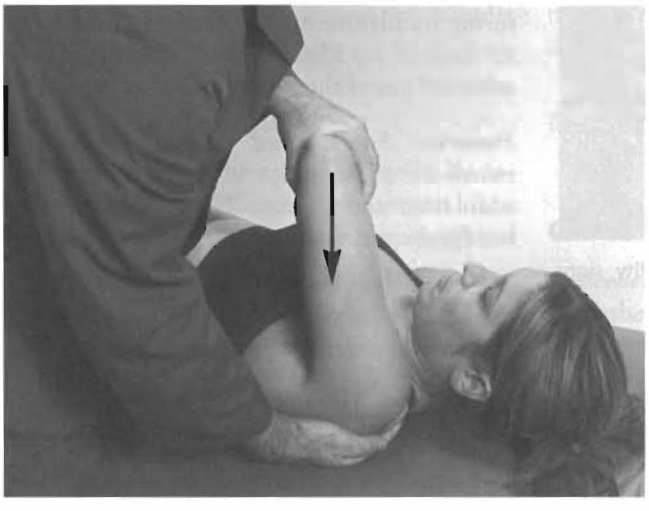

FIGURE 17.2.20. Posterior subluxation test.

FIGURE 17.2.21. Clunk test.

Active Compression Test. See the section on cervical spine tests for shoulder etiologies (Fig. 17.2.10): Pain for labral damage is deep inside the glenohumeral joint.

Positive test: Pain occurs in the first maneuver but improves or resolves with the second maneuver. Pain is located deep in the shoulder, as opposed to an acromioclavicular injury, which also causes a positive test.

Indicates: Labral pathology (if pain is deep inside the shoulder) (4).

Anterior/Posterior Drawer Tests. For the *anterior drawer*, the athlete is supine with the affected arm at 80 to 120 degrees of abduction (from horizontal), 0 to 20 degrees of forward flexion, and 0 to 30 degrees of external rotation. The stabilizing hand holds the scapula by the spine and coracoid process, while the mobilizing hand grasps the humerus just below the neck. The examiner translates the humeral head anteriorly, similar to Lachman's test of the knee. The *posterior drawer test* requires the arm to be forward flexed 20 to 30 degrees without abduction, and the force is directed into the table.

Positive test: Grinding with translation, humeral head easily subluxed.

Indicates: Glenohumeral instability, possible labral pathology.

Note: Relocation and apprehension tests evaluate for instability, which can be caused by damage to the glenoid labrum (7). These two tests should be included, therefore, when evaluating the labrum. Instability and labral testing can go hand in hand; however, instability can occur without labral pathology, and labral pathology can occur in a stable shoulder. This accentuates the need for a thorough shoulder examination to make an accurate diagnosis.

JOINT PLAY

Movements in the Glenohumeral Joint

1. *Lateral movement of the humeral head from the glenoid cavity* (Fig. 17.2.22). The examiner places the left (mobilizing) hand medial to the proximal humerus near the axilla. The right (stabilizing) hand holds the posterolateral elbow, while the athlete's forearm rests across the chest. The right hand holds the elbow at the athlete's side while the left hand thrusts away from the body, distracting the humerus laterally from the glenoid cavity. (8)

2. *Anterior movement within the glenoid cavity* (Fig. 17.2.23). The setup is the same as for no. 1 above, except that the examiner's mobilizing hand is on the posterior proximal humerus. The examiner exerts downward force with the stabilizing hand toward the table, while the mobilizing hand lifts upward, translating the humeral head anteriorly. In cases of significant anterior capsular laxity, take up the slack anteriorly before introducing the mobilizing force. (8)

3. *Posterior shear within the glenoid cavity* (Fig. 17.2.24). The examiner places the left

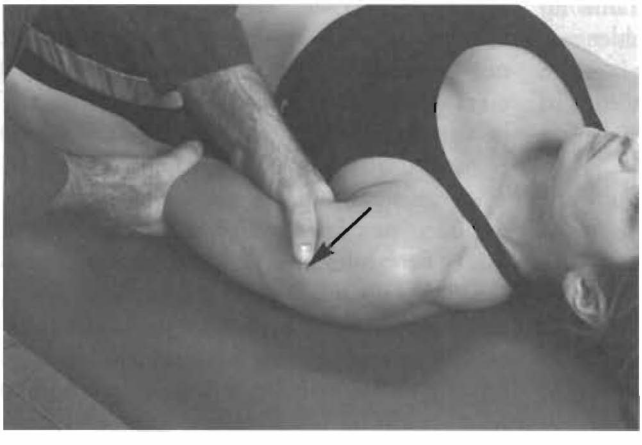

FIGURE 17.2.22. Lateral movement of the humeral head from the glenoid cavity.

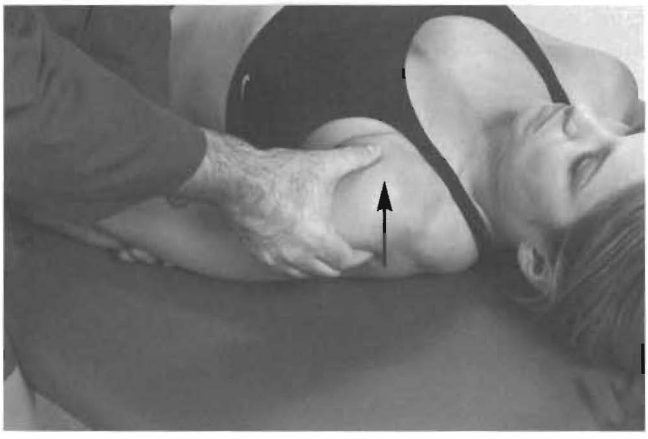

FIGURE 17.2.23. Anterior movement within the glenoid cavity.

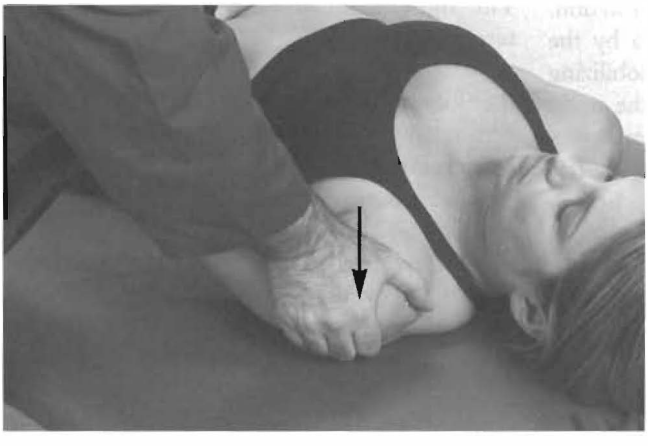

FIGURE 17.2.24. Posterior shear within the glenoid cavity.

hand on the posterior elbow of the athlete, while the right hand has its thenar eminence over the greater tuberosity. The left mobilizing hand raises the elbow, flexing the shoulder and extending the elbow, so that the scapula is secure against the table. Then, the right hand thrusts in a posterior direction, forcing the humeral head backward in the glenoid cavity. (8)

4. *Down-and-back movement within the glenoid cavity* (Fig. 17.2.25). The examiner is positioned so the left shoulder can be a fulcrum against the athlete's elbow. Both hands are clasped over the anterior humeral head near the surgical neck. The athlete's arm is flexed 45 degrees, or up to the pain barrier. The humeral head is pulled down and back within the glenoid cavity. At the same time, the examiner thrusts upward with the shoulder to counterbalance the arm movement. The arm should not be flexed; rather, the examiner's shoulder takes up the slack produced by mobilizing the humeral head. (8)

5. *Out-and-back movement* (Fig. 17.2.26). The examiner now turns so that the right shoulder is a fulcrum for the athlete's distal third of the humerus. The examiner grasps the proximal humerus around the medial side and translates the head away from the glenoid and back. The examiner thrusts the right shoulder forward to counterbalance the arm movement.

FIGURE 17.2.25. Down-and-back movement within the glenoid cavity.

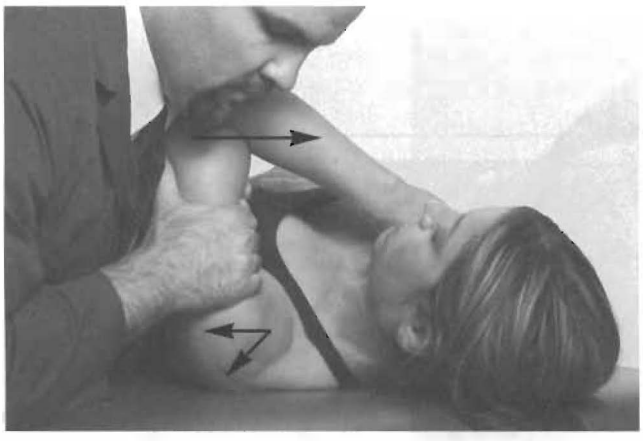

FIGURE 17.2.26. Out-and-back movement.

6. *Direct posterior movement with the arm forward flexed at 90 degrees* (Fig. 17.2.27). The athlete's arm is flexed at 90 degrees, elbow flexed, and hand resting on opposite shoulder. The examiner places both hands directly over the elbow and thrusts into the table, thus moving the humeral head posteriorly (8).

7. *External rotation within the glenoid cavity* (Fig. 17.2.28). The athlete's arm rests at his or her side, elbow flexed. The examiner's left hand picks up the elbow, moving the shoulder into neutral position. The examiner's right hand clasps the athlete's upper arm posteriorly near the surgical neck. The athlete's forearm should be under the examiner's right arm. Start the maneuver with the arm already in the end range of voluntary external rotation. The right hand rotates the humerus with the left hand stabilizing the elbow, going to the anatomic barrier (8).

Movements in the Sternoclavicular Joint

Anteroposterior Glide. The play is along a vertical plane. Hold the medial third of the clavicle between the thumb and index finger. Glide the clavicle up and down through the anteroposterior range of motion (Fig. 17.2.29) (8).

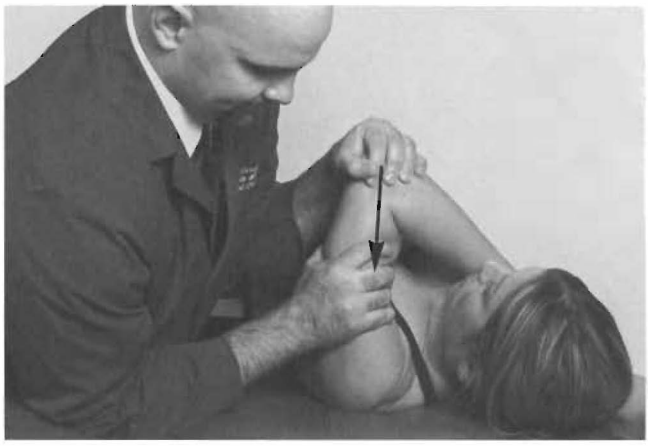

FIGURE 17.2.27. Direct posterior movement with the athlete's arm forward flexed at 90 degrees.

FIGURE 17.2.28. External rotation within the glenoid cavity.

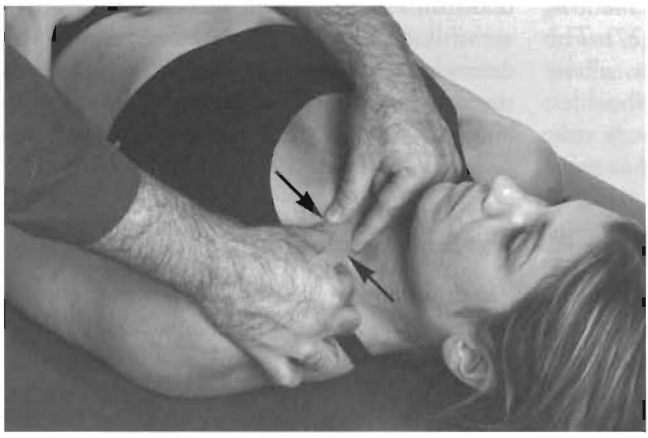

FIGURE 17.2.29. Anteroposterior glide of the sternoclavicular joint.

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17.3

Common Conditions

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SICK SCAPULA SYNDROME

Description

Scapular dyskinesia describes the abnormal biomechanics of the scapula that alter normal shoulder function (1–3). The athlete who has repetitive microtrauma of the throwing motion, in particular, is susceptible to muscle strain, fatigue, and breakdown. Craig Morgan coined this syndrome the "SICK (scapula infera coracoid dyskinesia) scapula syndrome" (1). These imbalances can produce painful conditions about the shoulder, further inhibiting muscles such as the serratus anterior and lower trapezius (4). This dysfunction does not allow full scapular retraction and the resultant elevation of the acromion with arm elevation, which can lead to impingement symptoms (5).

Kinematics

The scapula is the foundation for normal shoulder biomechanics, as outlined by Rubin and Kibler (6). The scapula links the trunk to the arm, provides a stable articulation for the glenohumeral joint, and creates clearance of the rotator cuff during upper extremity elevation. Rubin and Kibler (6) classify shoulder muscles based on their group function. The serratus anterior, the trapezius, the rhomboids, and the levator scapulae are the "scapular pivoters." The "glenohumeral protectors" are the rotator cuff

muscles that provide concavity and compression and resist translation. The "propellers" are the pectoralis major and latissimus dorsi, which connect the trunk to the upper extremity (Table 17.3.1). These groups are defined by the agonist and antagonist muscles working together, known as 'force couples' (7). Stability of the scapulothoracic articulation is dependent on the balanced function of these force couples and their fascia that attach to the scapula.

The scapula is the kinetic-chain link between the proximal segments of the shoulder and energy released through the arm and hand (7). Its mobility serves to allow the humerus to perform overhead activities and move in extreme ranges of motion that no other joint can achieve. Its role as a stabilizing base for the glenohumeral joint allows it to perform repetitive actions and absorb higher levels of stress. The ratio of glenohumeral to scapulothoracic rotation is 2:1 throughout arm elevation, although in the first 30 degrees of abduction and 60 degrees of flexion, the ratio becomes 4:1(7). The scapula tilts posteriorly during arm elevation, more so during the first 90 degrees of abduction. A lower posterior tilting angle in the sagittal plane was seen clinically in patients with impingement (8).

TABLE 17.3.1. MUSCLE GROUPS OF THE SHOULDER COMPLEX

Scapular pivoters
Serratus anterior
Trapezius
Rhomboids
Levator scapulae
Glenohumeral protectors
Rotator cuff muscles
Supraspinatus
Infraspinatus
Teres minor
Subscapularis
Propellers
Pectoralis major
Latissimus dorsi

Adapted from Kibler WB. The role of the scapula in athletic shoulder function. *Am J Sports Med* 1998;26: 325–337.

During the first 100 degrees of humeral elevation, several events occur in the normal shoulder complex:

a. The deltoid fires to elevate the arm.
b. The upper trapezius fires to oppose the lateral pull of the deltoid.
c. The upper serratus anterior fires to oppose the deltoid pull, protract the scapula, and maintain a close articulation between the scapula and thoracic cage.
d. The rotator cuff muscles fire to maintain humeral position in the joint (often referred to as the instantaneous center of rotation, or ICR). The firing causes lateral traction against the scapula.
e. The levator scapulae, rhomboid, and lower trapezius fire to counterbalance the lateral torque of the serratus anterior and rotator cuff.
f. The lower serratus anterior pulls the inferior scapular angle laterally to achieve upward scapular rotation.
g. The axis of upward scapular rotation is at the root of the scapular spine.

During humeral elevation above 100 degrees, the following changes occur:

a. The costoclavicular ligament tightens, restricting upward rotation at the root of the scapular spine.
b. The trapezial ligament becomes taut, restricting further scapular excursion.
c. The root now glides inferolaterally as the scapula glides along the thoracic cage.
d. Therefore, the axis of scapular rotation now occurs around the acromioclavicular joint.
e. The inferior scapular angle now moves inferolaterally, pulled by the lower serratus anterior.
f. The lower trapezius fires more actively to stabilize the scapula.
g. The humeral head now lies just superior to the glenoid fossa.

Pathophysiology

There are several factors involved with scapular dyskinesia that lead to rotator cuff impingement.

1. *Scapular orientation (protraction).* Improper scapular positioning influences rotator cuff impingement. The scapula needs to be able to retract fully to form a stable base, particularly for throwing. If the serratus anterior and lower trapezius are unable to retract the scapula, its position will be more protracted than normal. Magnetic resonance imaging (MRI) studies have shown decreased subacromial space with passive protraction of a normal shoulder (9). Less space means more pressure on the coracoacromial arch. Protraction also results in glenoid anteversion and excessive anterior strain in the anterior band of the inferior glenohumeral ligament (10).

2. *Pain.* Any pathology causing pain, such as labral tears, blunt trauma, osteoarthosis, and soft tissue injury, can disrupt the scapulothoracic rhythm by biofeedback inhibition of the muscles. Such trauma to tissue can produce poor coordination of the shoulder girdle muscles and alter the force couples. This disruption can allow the humeral head to abut the inferior acromion at 90 degrees of abduction instead of holding it down in the glenoid fossa, thus causing impingement without any secondary pathology (e.g., acromioclavicular arthrosis, labral tears). Needless to say, the longer these dysfunctional mechanics remain, the higher the likelihood of creating secondary pathology.

3. *Posterior scapular tilt.* The scapula lies in the thoracic cage with a subtle posterior tilt. Patients with impingement syndrome have been found to have excessive anterior tilt of the scapula (7). The mechanism relates to the force couple of the serratus anterior and trapezius. When the force couple is disrupted, the inferior angle floats away from the thoracic cage, tilting the acromion toward the greater tuberosity. If the serratus anterior is not strong enough to hold the angle down, a functional stenosis of the coracoacromial joint develops, leading to impingement. Symptoms are typically worse with increased anterior-posterior tilt (7).

4. *Muscle fatigue.* Endurance is as important as strength in muscle function. Fatigue of the lower trapezius and serratus anterior will let the scapula become more protracted with repetitive use. This condition is more difficult to diagnose, and the clinician may have to exercise the athlete to fatigue before examining to make the diagnosis.

5. *Spinal curvature.* Thoracic kyphosis leads to protraction, while cervical lordosis can limit retraction, depending on the cause of the lordosis (7).

In most of these situations, scapulothoracic dyskinesia precedes the onset of impingement, and usually provokes it. Thus, scapulothoracic dyskinesia may be a risk factor for impingement and should be rreated aggressively to prevent symptoms.

Athletes

Scapular biomechanics play a crucial role in the throwing athlete (11). The act of throwing involves the transfer of energy from the lower extremities to the upper extremity, and the shoulder sustains high stress across the soft tissues. It is estimated that the speed of ball acceleration is 7,000 degrees per second (12). This feat requires a stable retracted scapular base and functional force couples to allow the distal extremity to propel a ball at high speed.

Shoulder pain itself can disrupt the throwing mechanics and scapulothoracic rhythm. Soreness and discomfort is a part of pitching, which is why ice treatments and strength training are done aggressively after throwing during the season. Persistent pain leads to alterations in muscle strength and firing, starting the cascade of scapular dyskinetic behavior that will lead, if unchecked, to subacromial impingement.

Poor pitching mechanics are well known for causing upper extremity injuries, either directly through impingment or by scapular dyskinesia. Several key flaws are identified that disrupt scapulothoracic rhythm.

1. The humerus should never go above 90

degrees of elevation during the windup, acceleration, release, and follow-through. If elevation goes up to and beyond 100 degrees, the scapular mechanics change, as previously described, and the humeral head is no longer stable in the glenohumeral joint.

2. Lack of trunk flexibility does not allow proper dispersion of energy during follow-through and transmits more stress through the anterior capsule.
3. A throwing shoulder that "opens up" too quickly will have the elbow lagging behind the glenohumeral joint. It often appears as if the shoulder is leading the elbow, which stretches and strains the anterior capsule and humeral restraints on every pitch.
4. Poor muscle endurance of the rotator cuff and scapulothoracic muscles leads to destabilization of the scapula with repetition. Once a pitcher reaches the point of fatigue, throwing efficiency decreases, full scapular retraction to the stable cocking point is more difficult, and compensatory changes alter energy dissipation (3). From that point, the shoulder is at risk for injury.

Scapular protraction and retraction are especially important in the throwing athlete. The humeral head already experiences an anterior translation force during the normal cocking phase. Added stress on the anterior shoulder caused by a protracted resting scapular position places the shoulder at considerable risk for injury. Without the ability to fully retract the scapula, the throwing athlete loses the stable cocking point that dissipates the energy transfer from the lower to upper extremity in the kinetic chain (3). This instability reduces throwing efficiency and promotes an overuse muscular fatigue syndrome of the scapula as well. Tightness of the shoulder girdle, glenohumeral posterior capsule, and myofascial layers increases shoulder protraction in the cocking and follow-through phases of throwing (13).

History

Questions to ask the athlete include the date of onset of symptoms, phase of throwing when pain occurs, severity of symptoms (able to throw or not), duration of symptoms, change in performance level, prior injury history, and treatments and their outcomes. Most cases are insidious and gradual, but a traumatic insult can instigate this problem if the athlete tries to compete before recovery.

The symptomatic athlete generally presents with vague anterior shoulder pain but may also complain of lateral, posterior, superior medial scapular angle, and medial scapular border pain. The paraspinal muscles of the neck on the symptomatic side may also be affected with radicular or thoracic outlet symptoms in the involved extremity (1). Morgan reported on 39 overhead-throwing athletes, 80% of whom had anterior (coracoid process) pain, 70% had both anterior and posterior pain (superior medial scapular angle), 10% had isolated anterior coracoid pain, 20% had isolated superior medial scapular angle and neck pain, 20% had lateral (impingement) pain, and 5% had mild radicular pain in the involved extremity (1). Acromioclavicular joint pain may also occur but is less frequent (1).

Physical Examination

The physical examination includes a thorough biomechanical evaluation of the cervical, thoracic, and lumbosacral spine and the pelvis from the front, side, and back, looking for postural dysfunction. This finding is not unusual with scapular dyskinesia. A forward head and neck, cervical lordosis, thoracic kyphosis, lumbar lordosis, and protracted scapulae can be seen (14). Weakness of core trunk muscle strength is also a cause of scapular dyskinesia in the throwing athlete (15). The assessment of leg-length discrepancy and hip rotation asymmetry should be noted during the examination of the lower extremities. Deficiencies in these areas can prevent efficient transfer of energy from the lower extremities to the throwing arm.

The evaluation of the scapula should be performed initially from posterior at a distance to allow complete visualization. The scapular position is evaluated by assessing scapular height,

the distance from the superior angle of the scapula to the spine, and the distance from inferior angle of the scapula to the spine. The scapular position is assessed with the athlete's arms relaxed at the sides, with hands on hips and with the arms at or below 90 degrees of abduction with the shoulders internally rotated and the forearms pronated (6). A side-to-side comparison is performed. A difference from side to side equal to or greater than 1.5 cm is significant (6).

The examination proceeds to the anterior aspect to assess the position of the scapula, the coracoid process of the scapula, and its relationship to the clavicle. Observe the scapula in 90 degrees of flexion and then in 90 degrees of abduction. Observing motion in concentric and eccentric phases makes subtle cases of scapular dyskinesia more obvious. Scapular "muscular assistance" can be used to see if impingement is due to lack of shoulder protraction (3). The examiner pushes laterally and upward on the inferior medial border of the scapula; impingement symptoms are significantly abated or resolved in cases with muscle inhibition.

Athletes with scapular dyskinesia and coracoid pain lack full forward flexion versus the noninvolved shoulder. An attempt to passively flex the extremity increases the coracoid pain. The scapular relocation test (1) is performed with these patients in the supine position, manually reducing or "relocating" the scapula, allowing full flexion. Strength is assessed by performing manual muscle testing of the periscapular muscles in protraction, rerraction, and elevation; depression; and lateral and medial rotation. A wall push-up is a simple way to assess scapular muscle strength by asking rhe athlete to perform ten repetitions. Scapular winging may be seen in individuals with scapular weakness.

Palpate for localized tenderness of the coracoid process of the scapula and the tendons that originate from it, acromioclavicular joint, superior medial angle of the scapula (levator scapulae), medial border of the scapula, and subacromial area. Assess the local tissue tension of these areas as well as the corresponding fascia, especially the clavipectoral fascia.

Scapular winging needs to be evaluated carefully. The classic presentation is medial winging from serratus anterior weakness and long thoracic nerve palsy. Lateral winging occurs from trapezius weakness and usually accompanies an elevated acromion. Kibler et al. described three specific patterns of scapular dyskinesia (16):

Type I: Winging occurs at the inferior medial border.

Type II: The entire medial border is involved.

Type III: The superior medial border is prominent.

Morgan (1) described a 20-point clinical rating scale to assess the factors found in scapular dyskinesia at initial presentation and to monitor the treatment course (Table 17.3.2). Healthy throwing shoulders exhibit almost no abnormalities (1).

Assess for an internal rotation deficit. Morgan (1) refers to this as a glenohumeral internal rotation deficit (GIRD), defined as the difference in degrees of internal rotation between the throwing shoulder and the nonthrowing shoulder. For accuracy, this should be measured with a goniometer and recorded. Pitchers with type II SLAP (superior labrum anterior-posterior) lesions requiring operative treatment had GIRDs greater than 25 degrees, the average being 53 degrees (1). A complete evaluation of the shoulder and the upper extremity should follow the scapular examination.

TABLE 17.3.2. FACTORS IN SICK SCAPULA THAT LEAD TO SHOULDER IMPINGEMENT

Scapular orientation
Pain from trauma or overuse
Posterior scapular tilt
Muscle fatigue
Spinal curvature

Adapted from Morgan CD. Thrower's shoulder: two perspectives. A–perspective 1. In McGinty JB, ed. *Operative Arthroscopy*, 3rd ed. Baltimore: Lippincott Williams & Wilkins, 2003.

Standard Treatment

Rehabilitation (1,3,5,14,15) is divided into three stages: stability exercises, closed-chain exercises, and open-chain exercises (7). Stability exercises involve isometrics such as scapular pinch and shrugs. External assistance may be used to help get the scapula into normal position and stimulate coordination of muscle firing patterns (3). The athlete also strengthens the cervical retractors and periscapular muscles with emphasis on scapular co-contraction during frontal and sagittal plane exercise.

Closed-chain exercises physiologically reinforce the muscle firing patterns to a greater extent. These include wall and floor exercises moving the scapula through elevation, depression, protraction, and retraction. Lower trapezius and rhomboid exercises are introduced as well. Once these are mastered, open-chain exercises can be instituted, such as rowing, push-ups, press-ups, and scaption (humeral elevation in the scapular plane) (7). At this point, proprioceptive and plyometric activities are done to condition the scapular stabilizers to return to sport.

Stretching the tight tissue usually involves the clavipectoral fascia and pectoralis minor. Care must be taken not to overload the anterior shoulder capsule with stretching techniques; sometimes the only capsular stretching done is in the posterior capsule. Core (trunk) strengthening and correction of postural dysfunction are done throughout all three phases of rehabilitation. Throwing is restricted until the scapular position is at least 50% improved (1).

Throwing athletes should progress into their sport with a complete throwing program (see Chapter 14). No athlete should return to his or her sport until the scapulothoracic mechanics are fully restored.

Manual Techniques

To facilitate rehabilitation and recovery of the SICK scapula syndrome, manual medicine can assist in several ways:

1. *Treat ligamentous articular strain.* Specifically, this describes the somatic dysfunction that occurs in the ligamentous structures surrounding the acromioclavicular joint (17). Injury disrupts the balanced tension of the soft tissues about the joint. Treat scapular malposition through treatment of these dysfunctions in the shoulder girdle according to what is seen on examination. If there is anterior scapular tilt, specifically check the levator scapulae and upper trapezius muscles. If the scapula is unable to fully retract, target the pectoralis minor, biceps brachii, anterior cervical musculature, and fascia. The regions to target include the following:
   - The cervical fascia and anterior scalene muscles
   - The middle and posterior scalene muscles and levator scapulae
   - The clavicle
   - The shoulder and teres minor
   - The pectoralis minor, coracobrachialis, and short head of biceps

   Muscle energy, myofascial release, and Spencer techniques are also effective.
2. Treat posterior capsular restriction with Spencer techniques, joint play, or muscle energy.
3. Massage treatments are effective in mobilizing the soft tissues of the shoulder girdle and improving blood flow. Deep tissue techniques can be applied to the dysfunctional muscles in conjunction with the other techniques.

Manual medicine can assist in other regions:

1. Somatic dysfunction of the spine, pelvis, and lower extremities should be evaluated and treated.
2. Balance the pelvis and treat any static leg-length discrepancy.
3. Treat the upper and lower lumbar dysfunction. The upper lumbars are the origin of the iliopsoas, so check hip flexor flexibility as well.
4. Treat and improve any thoracic kyphosis and cervical lordosis as best as possible.

Consider radiographs to evaluate degenerative changes.

5. Note any dysfunction of the C7-T1-first rib complex. Direct techniques such as high-velocity, low-amplitude (HVLA) thrust and muscle energy work well. If this region is significantly painful, use functional, counterstrain, or deep massage techniques as tolerated.

Prevention

Prevention involves maintenance of strength and flexibility of the lower extremity, core (trunk), and upper extremity. The last mentioned especially involves scapular strengthening, stretching of the coracoid origin muscles, rotator cuff strengthening, and internal rotation stretching. Throwing athletes should be on a routine that incorporates these exercises and stretches, but overstretching the anterior capsule can be detrimental.

Throwing athletes need to know when their scapular stabilizers are dysfunctional. If the girdle cannot loosen up during warm-ups, avoid throwing. Coaches and trainers should keep videotapes of their athletes throwing when their mechanics are sound and pain-free. This gives a comparison for when shoulder pain occurs during pitching. Keep control of pitch counts and watch for signs of periscapular muscle fatigue. Changes in mechanics will help to identify moments of fatigue and breakdown.

SHOULDER IMPINGEMENT SYNDROME AND BICEPS TENDINITIS

Introduction

The term *impingement* is most commonly used to describe a painful shoulder syndrome. This is a structural abnormality (primary impingement), which may be a congenital, acquired, or mechanical dysfunction (secondary impingement). It may be the result of musculotendinous overuse, internal derangement, or inflamed tendons and bursae (18). The impingement interval (or rotator interval) is the term used to describe the space between the undersurface of the acromion and humeral head (20). There are a number of classification systems that have been described and adopted into clinical practice. One such classification divides the syndrome into extrinsic and intrinsic etiologies of impingement (18). *Extrinsic/external* causes are located outside the rotator cuff and are influenced by the shape of the acromion, shoulder instability, acromioclavicular joint degeneration or spurring, coracoacromial ligament calcification, postural dysfunction (protracted shoulders), poor humeral head depression, and posterior impingement (overhead athletes).

Intrinsic/internal causes are located within the rotator cuff itself. These causes are muscle weakness or imbalance, partial tears secondary to overuse, and/or degenerative tendons, such as the long head of the biceps (19).

In 1972, Charles Neer devised the classic theory of impingement based on cadaveric dissections and clinical as well as surgical experience. He described the stages of impingement following a pattern of severity. Stage I involves edema and hemorrhage. Stage II presents with cuff fibrosis, thickening, and partial tearing. Stage III presents with partial or full-thickness tears, bony changes, and tendon rupture. Current thinking now separates the impingement syndromes into two general categories: impingement occurring in patients older than 35 years of age, and impingement occurring in patients younger than 35 years of age (18).

The cycle of impingement is described as extrinsic/external causes leading to narrowing of the subacromial space (rotator interval), which causes irritation and swelling of the rotator cuff, which in turn leads to impingement with overhead activity, which leads to rotator cuff tendinitis and the cycle continues until the end stage of partial or complete rupture of the rotator cuff. Intrinsic/internal causes

or combinations of internal and external factors can also create this impingement cascade (19).

A classic inrrinsic/internal etiology of impingement that occurs distal to the acromioclavicular joint is instability of the glenohumeral joint. Conditions associated with glenohumeral joint instability include Bankart lesions, SLAP lesions, or a Hill-Sachs deformity (19).

The shoulder complex includes both static and dynamic stabilizers. The static stabilizers are the glenoid and its labrum, capsule, ligaments (superior glenohumeral, middle glenohumeral, and inferior glenohumeral), joint cohesion, and intra-articular negative pressure.

The dynamic stabilizers are the rotator cuff muscles along with the long head of the biceps. The scapulothoracic stabilizers are the rhomboids, trapezius, serratus anterior, and pectoralis minor muscles.

The shape of the acromion influences the subacromial space for the rotator cuff during shoulder motion. The three types ate as follows: type I (normal): 17% occurrence; type II (curved): 43%; and type III (hooked): 40%.

A type III acromion narrows the subacromial space and increases the propensity for impingement, and thus is a risk factor.

History and Physical Examination

With any evaluation of a shoulder problem, it is important to first obtain a thorough history. The mechanism of injury is a key component of this initial evaluation. Was the pain of gradual or sudden onset? Are the symptoms brought on by a particular activity? What sport does that athlete participate in and at what position? A detailed history including exacerbating activities, length of symptoms, and history of trauma are an important part of arriving at an accurate diagnosis.

Hallmark primary impingement symptoms are pain exacerbated with overhead activities and pain with lying on the involved shoulder. Secondary impingement typically involves pain with throwing activities, with pain less likely to be symptomatic at night. The pain is also more localized posteriorly (21).

Following a detailed history, the shoulder should be examined by inspection, palpation, range of motion (a painful arc of elevation between 70 and 120 degrees indicates generalized impingement), and muscle and neurologic testing as well as special testing when indicated. As with any physical examination, a detailed examination of the joint above and below the affected joint should be done for discovery of other potential pain generators as well as a careful vascular examination.

Special testing includes the Hawkins, Neer, and painful arc tests. The sensitivity, specificity, and accuracy of these tests, respectively, are as follows: Hawkins: 92.1%, 25%, 72.8%; Neer: 88.7%, 30.5%, 72.0%; and the painful arc: 32.5%, 80.5%, 46.5% (24).

Stability testing must be accomplished to assist in distinguishing between primary and secondary impingement. The standard stability tests include the apprehension test, the anterior/posterior glide test (also called the load shift test), and the relocation test.

Biceps Tendinitis

Another common etiology for anterior shoulder pain and possible impingement is biceps tendinitis. Since the long head of the biceps extends intra-articularly beneath the acromion through the rotator cuff to insert at the superior portion of the glenoid, it is a possible site of impingement. Impingement is brought on by inflammation of the tendon or subluxation of the tendon from the bicipital groove (18,21).

The biceps plays an important functional role in rotator cuff impingement. It compresses the humeral head in the glenoid and supports the anterior capsule and rotator cuff to resist anterior humeral translation. Biceps tendinitis usually occurs secondary to other

pathology, usually from rotator cuff impingement. When the rotator cuff is unable to keep the humeral head seated properly in the glenoid, either from weakness or tendon damage, the long head of the biceps tendon is excessively loaded, leading to inflammation and tendon breakdown.

The long head of the biceps tendon is also the muscle origin that allows elbow and shoulder flexion, but the short head is able to compensate adequately in the event of long head rupture. Many times, long head rupture does not require surgery, and strength recovery is functional. Concomitant subscapularis damage can destabilize the biceps tendon within its groove and lead to subluxation.

Factors leading to biceps tendinitis include tendinosis or degeneration of the rotator cuff, especially the subscapularis and supraspinatus, wear and tear from the acromion with the humerus in full abduction, anterior acromial decompression due to external humeral rotation, hyperlaxity of the shoulder in younger athletes, labral tears disrupting the biceps anchor, and weakness of the transverse humeral ligament (22).

Symptoms of biceps tendinitis include pain over the anterior aspect of the shoulder, especially with activities of lifting. Various combinations of forceful shoulder or elbow flexion or forearm supination may also cause pain in this area. It may, for example, be commonly seen in a softball pitcher who begins to throw breaking balls that require rapid forearm pronation or supination at the end of the underhand throwing motion. Pain and snapping accompany the biceps tendon subluxing from the bicipital groove.

Evaluating the biceps tendon involves Speed's test and Yergason's test. The addition of resisted external rotation can increase the sensitivity of Yergason's test for biceps pathology (23) to assess stability in the bicipital groove. Radiographic examination including MRI and MR arthrography may be helpful in evaluating both bony and soft tissue pathology, rotator cuff overuse injury, and tears and labral pathology, as well as acromioclavicular joint degenerative disease. These tests should be ordered to further establish a diagnosis obtained by history and physical examination (23).

Standard Treatment

The essential goal in the management of patients with impingement syndrome is to relieve the pain and to correct the cause in order to prevent further advancement of the pathologic processes. Nonsurgical rehabilitation management strategies for impingement syndromes are designed based on the specific diagnosis of the disorder. The initial treatment is relief of pain and inflammation by restricting the aggravating activities in combination with comprehensive multimodal pain management techniques.

These may include simple analgesics, nonsteroidal medications, and physical therapy modalities (25). It is important to evaluate and discuss relevant sport-specific technique issues along with modification of activities and sport mechanics to prevent further impingement complications. Including the sports coach or trainer in the evaluation can be invaluable. Motions should initially be restricted to below the horizontal plane of the shoulder in order to reduce mechanical impingement until pain and inflammation have subsided.

Once the acute inflammatory period has subsided, a specific rehabilitation program of rotator cuff and periscapular strengthening is started with clinically directed functional rehabilitation techniques based on the specific biomechanical diagnosis and injury pattern. The end result of the rehabilitative process is to have the rotator cuff become a more effective humeral head depressor and anterior stabilizer as well as reestablish the synchrony between the shoulder girdle complex, thorax, spine, and pelvis reacting to the loads and motions transmitted through the lower extremities.

Treatment of this condition is geared to improve the biomechanics of the shoulder so that impingement does not occur. The acute phase involves relative rest, ice, electrical stimulation, and anti-inflammatory medication. The subacute phase emphasizes physical therapy rotator cuff strengthening and postural stabilization.

Once this is achieved, a gradual return to throwing and sport-specific tasks can be initiated. If the athlete fails a conservative course of care including physical therapy, joint injections, and relative rest, then surgery may be indicated.

Impingement is treated by the following approach:

Stage 1: Goal: decrease inflammation and pain

a. Modalities (ice and electrical stimulation)
b. Restore range of motion (ROM) in pain-free range (passive to toleration)
c. Home management program consisting of anteroposterior couple strengthening (subscapularis, infraspinatus, teres minor) and scapular facilitation exercises
d. Inferior and posterior glenohumeral glides
e. Closed-chain exercises

Stage 2: Goal: full pain-free ROM and initiate strength

a. Active ROM to 70 to 90 degrees only, active-assisted ROM and passive ROM to tolerance
b. Restore proper muscle and static stabilizer length (stretch posterior cuff and capsule)
c. Improve the loss of internal rotation
d. Cuff strengthening below 70 degrees
e. Scapular stabilization exercises

Stage 3: Goal: restore function

a. Initiate sport-specific programs
b. Pain-free diagonal patterns
c. Upper extremity plyometric and glenohumeral stabilization

Diagnosis-driven osteopathic manipulative techniques can be helpful in maintaining the most efficient muscle balance, neuromuscular firing patterns, and flexibility in the shoulder complex. The Spencer technique is commonly employed in this regard (26).

Sport-specific programming can be incorporated during the later stages of the rehabilitative process and progressed functionally based on the response of the athlete's symptom complex to comprehensive management.

Patients refractive to nonsurgical treatment may be referred for orthopedic intervention. A number of factors influence the decision for surgery, including but not limited to stage of the pathology, age and underlying diagnosis, response to treatment, and goals for returning to play. Surgery should not be considered for any athlete who has not undergone a formal structured rehabilitation program (18).

Manual Techniques

More than any other body part, the shoulder can be a victim of dysfunction elsewhere in the body, and breakdowns in function are often traced to regions outside the shoulder. This requires a thorough history and examination, and attention must be paid to the body as a whole. Athletes can develop shoulder pain from changes in the gait, pain in the foot, cervical disc pathology, thoracic dysfunction, first rib dysfunction, elbow and wrist tendinitis, and many others. The trick is to identify the pathology in the shoulder and elsewhere, so that when the shoulder improves with treatment, other areas of dysfunction do not set it off again.

The following are effective techniques in treating rotator cuff impingement:

Seven stages of Spencer
HVLA thoracic mobilization
Scapular lift and rotation
Seated muscle energy for the thoracic spine
Prone counterstrain for the thoracic spine.
Muscle energy for the latissimus dorsi muscle
Muscle energy for biceps extension
Myofascial release for biceps flexion
Muscle energy for biceps pronation
Muscle energy for the latissimus dorsi

Seven Stages of Spencer. These are a series of proprioceptive neuromuscular facilitation techniques for the humerus. They treat early adhesive capsulitis, healed fractures, subacute dislocations, and any other degenerative or traumatic condition in which restrictions in glenohumeral motion are present (23). By decreasing muscle

spasms in the shoulder stabilizing muscles, the techniques are applicable and useful in many scenarios.

1. The athlete lies lateral recumbent with the affected shoulder up, while the clinician stands at the side of the table facing the athlete.
2. The athlete's knees are bent and the unaffected arm is abducted to 180 degrees for stabilization.
3. The clinician places one hand over the athlete's glenohumeral joint to monitor motion, while the other hand grasps the humerus at midshaft.
4. In each stage, the joint is gently moved into the restrictive barrier. Muscle energy can be applied at the barrier to further treat the dysfunction, but the Spencer techniques move the shoulder within the limits of pain. Each motion can be repeated between four and six times.

Stages

1. *Extension*
   a. The athlete's elbow is flexed to approximately 90 degrees.
   b. The clinician extends the humerus of the athlete to the perceived barrier in the range of motion (Fig. 17.3.1A).
2. *Flexion.* This is the same as extension but the clinician flexes the perceived humeral barrier in the range of motion and begins contract-and-relax cycles (Fig. 17.3.1B).
3. *Circumduction with compression:*
   a. The athlete's elbow is now fully flexed.
   b. The clinician grasps the elbow of the patient with the palm of one hand and uses the opposite palm to monitor motion at the glenohumeral joint.
   c. The clinician then exerts a gentle rotational force at the glenohumeral joint through the elbow in first a clockwise, then counterclockwise direction (Fig. 17.3.1C).
4. *Circumduction with traction.* This is similar to circumduction without traction. The difference is that instead of using the flexed elbow of the athlete as a fulcrum, the clinician uses the wrist as the athlete has his or her elbow fully extended. Traction is also applied to athlete's wrist. The arc of motion is generally in the opposite direction of the motion used for circumduction without traction (Fig. 17.3.1D).
5. *Abduction without traction.* The athlete's elbow is flexed to 90 degrees, while the operator grasps the medial aspect of the elbow to abduct it to the perceived barrier in the range of motion. Muscle energy can be applied at the end range of abduction (Fig. 17.3.1E).
6. *Internal rotation*
   a. The athlete moves toward the edge of the table so that the top arm can be dropped off the posterior edge of the table.
   b. The elbow of the side being treated is flexed to approximately 90 degrees, but the hand is placed posterior to the athlete's side.
   c. The clinician stabilizes the scapula while the mobilizing hand exerts anterior and inferior force on the olecranon toward the restrictive barrier. The clinician pushes the olecranon downward (inferiorly) to the petceived barrier (Fig. 17.3.1F).

Note: To move into an internal rotation muscle energy technique, move the elbow to the barrier and ask the athlete to gently resist for 3 to 5 seconds, then repeat. This stage can also be used for external rotation by reversing the positioning and vector force of the mobilizing hand.

7. *Humeral traction*
   a. The clinician grasps the proximal humerus of the athlete with both hands while the rest of the arm is supported against the clinician's side.
   b. The clinician performs a gentle but firm pumping action directly toward the clinician (Fig. 17.3.1G).

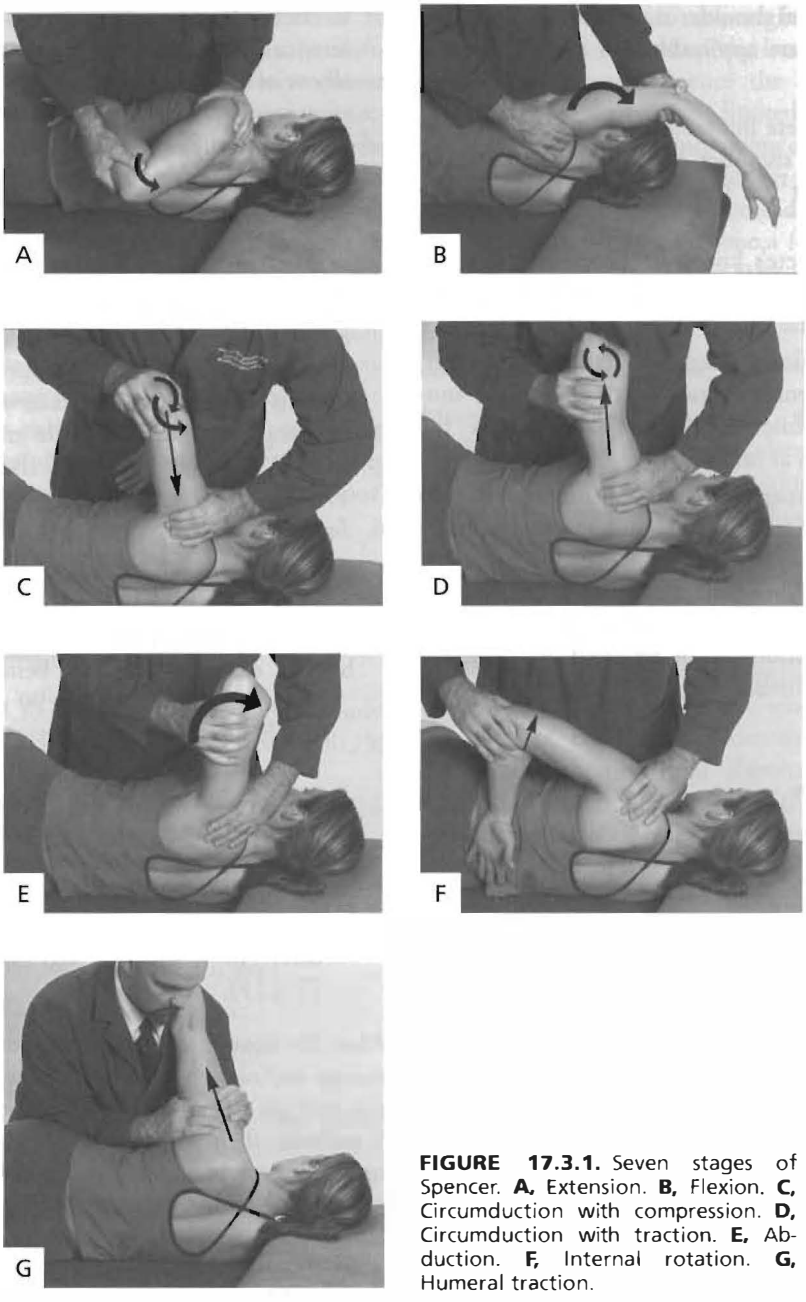

FIGURE 17.3.1. Seven stages of Spencer. **A,** Extension. **B,** Flexion. **C,** Circumduction with compression. **D,** Circumduction with traction. **E,** Abduction. **F,** Internal rotation. **G,** Humeral traction.

High-Velocity, Low-Amplitude Thoracic Mobilization. This not only improves thoracic motion, but it also aids in treating scapular motion restriction, particularly when the restrictions stem from the costothoracic origins of the scapular stabilizing muscles such as the rhomboids and lower trapezius.

Rationale: A freely mobile scapula will allow for increased motion and decreased tension on the glenohumeral joint. Plus, thoracic dysfunctions can radiate pain that influences the firing of the scapular stabilizers, including the rhomboids and lower trapezius.

Example: Right T4 on T5 Dysfunction, Extended

1. The athlete is supine while the clinician places the thenar eminence of the right hand on the pillar of the left T5 vertebra (opposite and one level below the dysfunction) (Fig. 17.3.2).
2. The athlete crosses the arms over the chest while the clinician cradles the athlete's head with his or her left arm, moving the athlete's thoracic spine into flexion.
3. The clinician leans on the athlete's arms with the chest and asks rhe athlete to inspire deeply and expire.
4. On expiration, the clinician introduces a downward impulse onto the athlete's arms and chest. The fulcrum effect on the left hand should allow the T4-5 segment to resolve. A release and popping sound may be heard.

Myofascial Release: Scapular Lift. This targets the rhomboids and associated scapular restrictions.

Rationale: Analogous to the double arm thrust, the increase in scapular motion subsequently increases glenohumeral motion.

1. The athlete lies on the side opposite the dysfunctional scapula.
2. The clinician places his or her finger pads under the medial border of the affected scapula.
3. The clinician applies gentle superior traction to the medial scapula, working along the entire medial border to release restriction in the soft tissue (Fig. 17.3.3).

Modification: Scapular Rotation

1. The clinician uses the same position as for the scapular lift, but he or she uses one hand to cup the superior shoulder with the thumb anterior and fingers posterior, palm ovet the acromioclavicular joint, while the other hand cups the fingers around the inferior scapular angle (Fig. 17.3.4).
2. The clinician introduces a steady deliberate rotational force to the scapula, alternating from internal to external rotation, working into the restrictive barriers. Reassess.

Seated Muscle Energy: Thoracic Spine. This technique also targets the rhomboids. It treats restrictions in the scapula and its stabilizers. The increase in scapular motion decreases strain on glenohumeral motion.

FIGURE 17.3.2. High-velocity, low-amplitude thrust for thoracic mobilization.

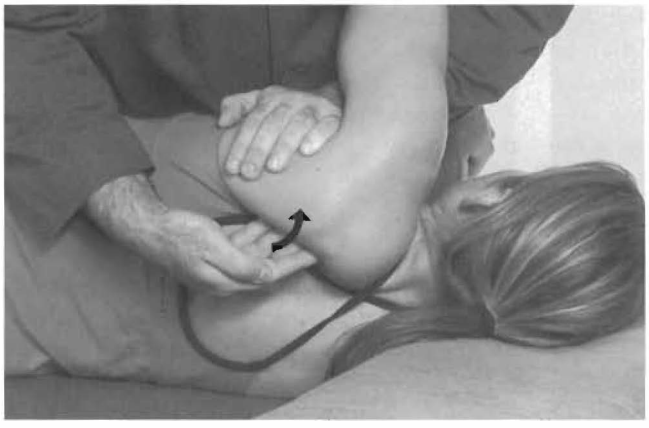

FIGURE 17.3.3. Myofascial release for scapular lift.

1. The athlete is sitting with the clinician standing behind.
2. The clinician places the palm of one hand over the medial scapular musculature and associated transverse processes of the affected side (Fig. 17.3.5).
3. The clinician identifies restrictions in side bending, rotation, and flexion or extension.
4. The clinician side-bends, rotates, and extends or flexes the athlete to engage those barriers in motion.

Prone Counterstrain: Thoracic Spine. This is a myofascial release technique for the latissimus dorsi and/or the erector spinae muscle mass.

Rationale: This treats restrictions in scapular motion, thereby improving glenohumeral motion.

1. The athlete is lying prone while the operator identifies points of tenderness along the spinous processes of the thoracic vertebrae and monitors the point with the index finger of one hand.
2. The clinician's opposite hand grasps the lateral pelvis on the side away from the clinician. The pelvis is rotated toward the tender point until the point is no longer tender (Fig. 17.3.6). This position is maintained for 90 seconds or until a fascial release is attained.

Variations include grasping the glenohumeral joint of the affected side and moving it away from the tender point as well as performing seated counterstrain from an anterior approach.

Muscle Energy: Biceps Extension. This treats restrictions in biceps motion along the flexion-extension plane.

FIGURE 17.3.4. Myofascial release for scapular rotation.

FIGURE 17.3.5. Muscle energy for the thoracic spine.

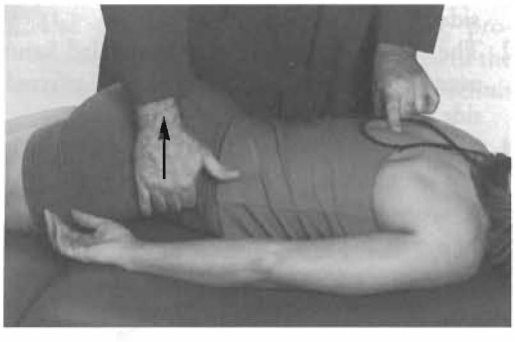

FIGURE 17.3.6. Prone counterstrain for the thoracic spine.

Rationale: Improved biceps motion allows for more functional biceps use and less tension, decreasing stress on the biceps tendon.

1. The athlete is seated with the elbow flexed to approximately 90 degrees and the hand fully supinated.
2. The clinician monitors the intertubercular groove with one hand while grasping the athlete's supinated hand with the opposite hand (Fig. 17.3.7).

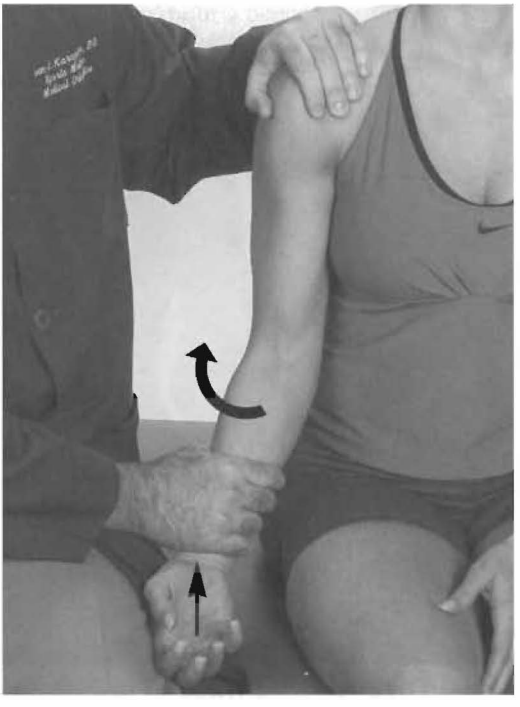

FIGURE 17.3.7. Muscle energy for biceps extension.

3. The clinician extends the elbow to the perceived motion barrier.
4. The athlete flexes the elbow against resistance for 3 to 5 seconds against the clinician's resistance. The athlete relaxes and the clinician repeats two to four times.

Myofascial Release: Biceps Flexion. This treats restrictions in biceps motion in the flexion-extension plane and allows for more functional biceps muscle use.

Rationale: Improved biceps motion allows for more functional biceps use and less tension, decreasing stress on the biceps tendon.

1. The setup is the same as for muscle energy biceps extension.
2. The clinician flexes the elbow to a position of ease (alleviation of pain) (Fig. 17.3.8).
3. This position is held for 90 seconds or until a release is detected by the clinician.

Muscle Energy: Biceps Pronation. This treats restrictions in biceps motion along the pronation and supination plane.

FIGURE 17.3.8. Myofascial release for biceps flexion.

FIGURE 17.3.9. Muscle energy for biceps pronation.

Rationale: The biceps is a wrist supinator, meaning that wrist pronation would be restricted if the biceps was restricted or shortened. This can aid in restoring normal biceps function.

1. The setup is the same as for the muscle energy biceps extension and the myofascial release biceps flexion.
2. The clinician pronates the forearm of the patient to a perceived barrier in the range of motion (Fig. 17.3.9).
3. The athlete supinates the forearm against the resistance of the clinician for 3 to 5 seconds.
4. Relax, reposition, repeat, and reassess.

Muscle Energy: Latissimus Dorsi. This technique for the latissimus dorsi muscle works in the abduction and adduction plane, as the latissimus dorsi attaches to the intertubercular groove of the humerus.

Rationale: Freedom of this muscle allows the humerus to flex more easily, thus requiring less flexion contractile force from the biceps.

1. The athlete is lying prone while the clinician places the caudal knee at the greater trochanter of the athlete on the unaffected side (Fig. 17.3.10).
2. The clinician then places the cephalad hand over the proximal humerus on the affected side.
3. The clinician then stabilizes the pelvis with the knee and side-bends the body of the athlete toward him or her with the proximal humerus as the fulcrum.
4. Once the barrier is reached, the athlete side-bends against the clinician's resistance for 3 to 5 seconds.
5. Relax, reposition, repeat, and reassess.

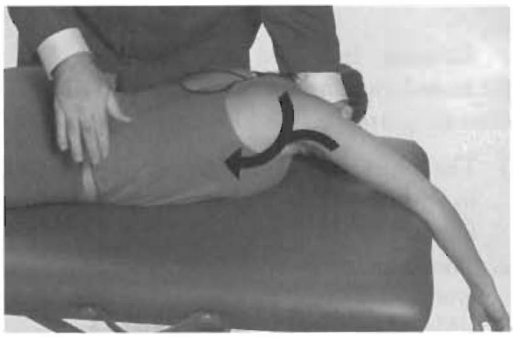

FIGURE 17.3.10. Muscle energy for the latissimus dorsi.

Manual medicine techniques useful for treating biceps tendinitis include, but are not specifically limited to, muscle energy biceps extension, myofascial release biceps flexion, muscle energy biceps pronation, and muscle energy for the latissimus dorsi.

Prevention

Rotator cuff impingement may be prevented by doing appropriate exercises, stretches, and modification of the athlete's activity. Exercises helpful for impingement include, but are not specifically limited to, rotator cuff strengthening exercises using "Therabands" or small (5 lb or less) hand weights for internal rotation, external rotation, and abduction. Scapular stabilization exercises such as "wall push-ups" can also be useful, as well as activities such as rowing.

Physical contact cannot be avoided in many sports, so traumatic injuries will be an inherent risk. The following are modifications to decrease the risk of rotator cuff impingement:

1. Use proper mechanics, particularly with swimming and throwing.
2. Use proper in-season strengthening programs in sports with overuse potential of the shoulder. This includes volleyball and tennis as well as throwing sports.
3. Stop the activity *before* the onset of pain. For instance, many pitchers have poor form, throwing with the arm instead of their body. Enabling the kinetic chain throughout the whole body saves wear on the shoulder and helps the athlete perform more effectively.
4. Young throwers should have pitch counts and time off in between pitching. Curve balls and other trick pitches should not be tried until high school at the earliest, and that requires proper coaching. Pitch counts have been encouraged to help prevent "little leaguer's elbow," among other arm injuries (27).
5. Allow for recovery. Swimmers should have laps tracked in practice so as to prevent overuse. If shoulder pain occurs, laps should be reduced to allow healing. Rest time should be instituted in order for the shoulder to heal from the repetitive stress. Athletes who move from one sports season to the next without a break may not have time to recover from injuries and may need to have their workouts and practices modified.
6. Do a proper warm-up before practice and play, particularly with "little leaguer's elbow" (27). Athletes with biceps tendinitis particularly need a good warm-up to loosen up the muscles and joint, thus limiting strain on the shoulder. With decreased stress, the biceps tendon, and the entire shoulder joint work much more smoothly and allow for better athletic performance.

In-season Rotator Cuff Stabilization Program

Rotator cuff programs are now commonplace among in-season professional baseball programs, and many nonthrowing athletes who repetitively load their shoulders, such as tennis players and swimmers, also derive significant benefit. The rationale is that by prehabilitating the joint, breakdown is less likely to occur and stress limits would be reached less often. As an athlete progresses through a season, the muscles fatigue and break down from repetitive use. Since the rotator cuff keeps the humeral head in the "dynamic socket" as the humerus rotates, any decrease in strength could allow the humeral head to migrate superiorly or sublux anteriorly, which stresses the biceps tendon. One of the reasons why coaches work so much with athletes on their mechanics is because the inefficiency from poor mechanics increases strain and leads to fatigue and breakdown.

Such in-season programs should be designed to maintain muscle endurance and strength from beginning to end. Attempts to build muscle strength and mass *during* a season may increase risk of injury. Those strength gains should be made in the off-season and coupled with flexibility training. Athletes would then use preseason training to acclimate to the changes and improve proprioception, sharpening their skills. Building muscle means inflicting enough damage to the muscle where microtears occur and heal, leading to hypertrophy and increased strength. This is not the ideal situation for an athlete who is

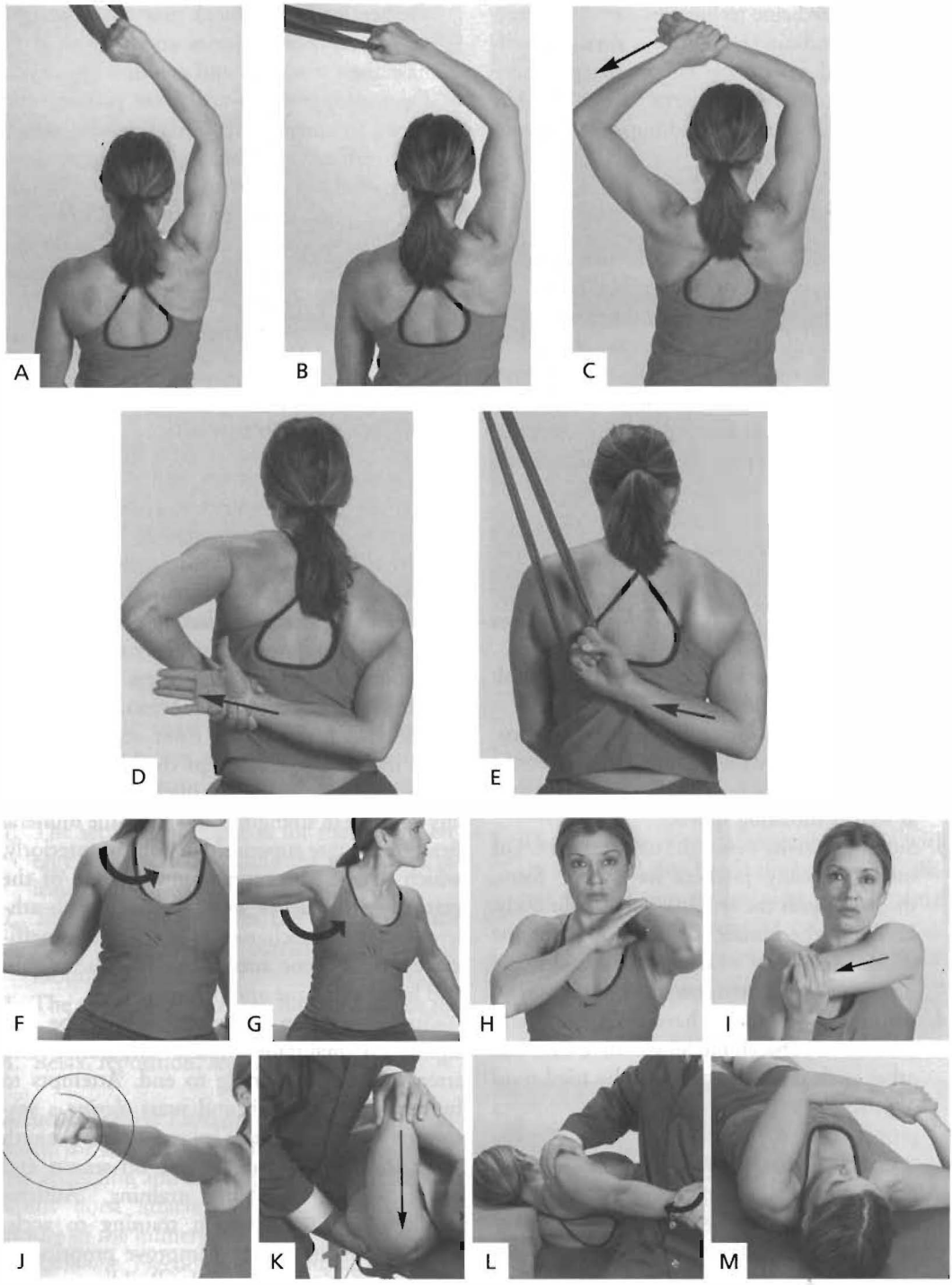

FIGURE 17.3.11. Stretches for the shoulder. **A,** Latissimus dorsi/inferior capsule stretch with Theraband. **B,** Variation of latissmus dorsi/inferior capsule Theraband. **C,** Teres minor. **D,** Supraspinatus. **E,** Variation of supraspinatus stretch with Theraband. **F,** Subscapularis. **G,** Biceps and pectoralis major. **H,** Triceps. **I,** Posterior capsule. **J,** Lateral pendulum. **K,** Pectoralis minor. **L,** Biceps tendon–long head. **M,** Infraspinatus.

playing and practicing daily throughout a long season. Any program trying to build strength through a season needs to have rest built into the schedule, something that many athletes do not get.

STRETCHES FOR THE SHOULDER AND UPPER EXTREMITY

1. *Trapezius.* The athlete places his or her hand on top of the head and gently distracts the head to either side and holds for 30 seconds, breathing throughout the exercise. (See Fig. 16.3.11.)
2. *Latissimus dorsi muscle/inferior capsule with Theraband* (Fig. 17.3.11A). The athlete holds Theraband in the right hand with the opposite end of Theraband firmly attached over the top of a door frame. Keep the right hand straight or extended. Stand with the back to the door. Step forward. The Theraband should elevate the right arm. A stretch may be felt in the lateral portion of the trunk and inferior capsule of the right shoulder if the muscle/capsule is tight or shortened. Hold to tolerance (30–60 seconds is ideal).
3. *Latissmus dorsi muscle/inferior capsule variation* (Fig. 17.3.11B). The athlete stands holding Theraband in the right hand with the opposite end of Theraband firmly attached over the top of a door frame. The left side of the trunk and the left shoulder are closest to the door. Step to the right side as necessary. You may feel a stretch in the right side of the trunk and inferior capsule of the right shoulder if the muscle or capsule is tight.
4. *Teres minor muscle* (Fig. 17.3.11C). The athlete raises the right arm toward the right ear with the forearm overhead and palm facing forward. Using the left hand, move the right forearm forward and to the right while the right arm remains against the right ear. You should feel a stretch along the outside border of the right shoulder.
5. *Supraspinatus, anterior and clavicular portions of deltoid, teres minor, infraspinatus, and pectoralis* (Fig. 17.3.11D). The athlete reaches backward with the right hand as if to reach in the right back pocket. Use the left hand to pull the right hand up toward the left shoulder blade.
6. *Supraspinatus—variation of stretch with Theraband* (Fig. 17.3.11E). The athlete holds Theraband in the right hand behind the back with the opposite end of Theraband firmly attached over the top of a door frame. Step forward. Stretch is felt in the anterior shoulder. Hold to tolerance (ideally 30–60 seconds).
7. *Subscapularis muscle* (Fig. 17.3.11F). The athlete keeps the right elbow in by the side of the trunk. The right forearm is against the door frame with the thumb facing upward. Turn to the left by moving the feet to the left. A stretch may be felt in the right armpit/axilla area or under the right shoulder blade.
8. *Biceps and pectoralis major* (Fig. 17.3.11G). The athlete places the right forearm on a door frame, keeping the right elbow straight. Turn the palm down and forearm inward. Turn the body to the left by moving the feet to the left.
9. *Triceps muscle* (Fig. 17.3.11H). The athlete flexes the right elbow. Using the left hand, grab the right arm near the elbow and raise the arm so the elbow is pointing in the direction of the ceiling. The palm of the right hand should bend so it approximates the shoulder. A stretch should be felt in the posterior aspect of the right arm.
10. *Posterior capsule* (Fig. 17.3.11I). As the athlete sits or stands, position the left hand on the right forearm near the elbow. Pull the right arm across the chest toward the left shoulder and hold when tension is felt. A stretch should be felt in the posterior aspect of the right shoulder.
11. *Lateral pendulum* (Fig. 17.3.11J). The athlete first abducts the arm to approximately 90 degrees, then swings his or her arms in smaller circles that gradually increase in size over time. The improved shoulder motion generated by this stretch can allow the entire shoulder mechanism to work more efficiently, including the biceps.

12. *Pectoralis minor muscle* (Fig. 17.3.11K). The athlete is lying with the right shoulder/scapula off of the table. The clinician supports the scapula with his or her left hand and positions the right shoulder of the athlete in flexion, adduction, and external rotation. The athlete's right elbow is flexed. The clinician holds the athlete's right forearm and moves the shoulder upward and back. Stretch should be felt near the coracoid process of the right shoulder blade. Good stretch if shoulder appears more anterior/forward while lying supine.
13. *Biceps muscle–long head* (Fig. 17.3.11L). The athlete is lying on the left side with the right shoulder extended and the elbow flexed. The palm is turned down and the forearm is turned inward or pronated. The clinician stands between the athlete's body and arm, and extends the elbow. Stretch should be felt in the volar aspect of the upper arm.
14. *Infraspinatus* (Fig. 17.3.11M). The athlete lies supine with the right elbow away from the body (shoulder abducted 90 degrees) and knuckles of the right hand facing toward the ceiling. Use the left hand to push the right forearm toward the table, keeping the right scapula against the table.
15. *Rhomboid muscles and paraspinal muscles* (see head and neck stretches in Chapter 16). While sitting, the athlete reaches across the body with the right upper extremity. Fasten right hand to front portion of the left side of chair. Turn neck to look in the direction of left hip. You should feel stretch between the right shoulder blade and the spine, as well as the right paraspinal muscles of the thoracic region.

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18

THE ELBOW

18.1

Anatomy

GAIL A. SHAFER-CRANE
WILLIAM M. FALLS

Three bones participate in the elbow joint, the humerus, radius, and ulna. The humerus widens and flattens as it approaches the elbow joint, where it articulates with the long bones of the forearm, the radius and ulna. The lateral and medial epicondyles are formed as the humerus broadens proximal to the elbow. The distal humerus is rounded into two articular surfaces, the trochlea and capitulum. The medial surface forms the cylindrical trochlea, and the lateral surface is the rounded capitulum. The ulnar notch, or groove for the ulnar nerve, is a hollow trench formed along the posterior aspect of the medial epicondyle. Flexion and extension of the elbow are permitted at the humeroulnar joint. The humeroradial joint increases elbow stability while permitting flexion and extension (Fig. 18.1.1A and B). Pronation and supination of the forearm occur at the proximal radioulnar joint (PRUJ). Anatomy of the elbow is presented in detail in major anatomic textbooks (1–8).

The most stable joint of the elbow is the humeroulnar joint. The distal humerus is a cylindrical trochlea that fits exactly into the trochlear notch of the ulna. The coronoid process of the volar ulna deepens the trochlear notch and reinforces the articulation. The olecranon process is a rectangular extension of the ulna that fits into a hollow on the posterior humerus, the olecranon fossa, during full extension of the elbow. The precise fit between these articular surfaces permits smooth extension and flexion of the elbow while preventing extreme hyperextension and dislocation. Slight hyperextension and a lateral carrying angle vary between the genders. Women tend to have up to 15 degrees of hyperextension with a carrying angle of 15 to 30 degrees. Men often do not demonstrate hyperextension of the elbow, and the carrying angle is usually from 10 to 15 degrees.

A fibrous capsule encloses this articulation. Folds form in the capsule as the attachments of the olecranon and coronoid processes stretch and slacken as the ulna rotates around the humeral trochlea. Fat pads partially fill the olecranon and coronoid fossae of the humerus.

The ulnar (medial) collateral ligament forms the medial joint capsule. This broad ligament is divided into three bands: the anterior, posterior, and oblique. The anterior band is cordlike with a proximal attachment of the medial epicondyle and the distal attachment on the tubercle of the coronoid process. This is the most important medial stabilizer of the elbow. The posterior band is triangular, with the narrow, proximal attachment along the inferior medial epicondyle deep to the anterior band, and the broad distal attachment along the olecranon process. The oblique band is superficial to the distal attachment of the posterior band from the proximal olecranon to the proximal coronoid process (Fig. 18.1.2A and B).

The humeroradial joint is between the fovea of the proximal radius and the capitulum of the humerus. The humeral head rotates about the capitulum during elbow flexion and extension, providing lateral stability to the elbow. The radial head articulates with the radial notch of the

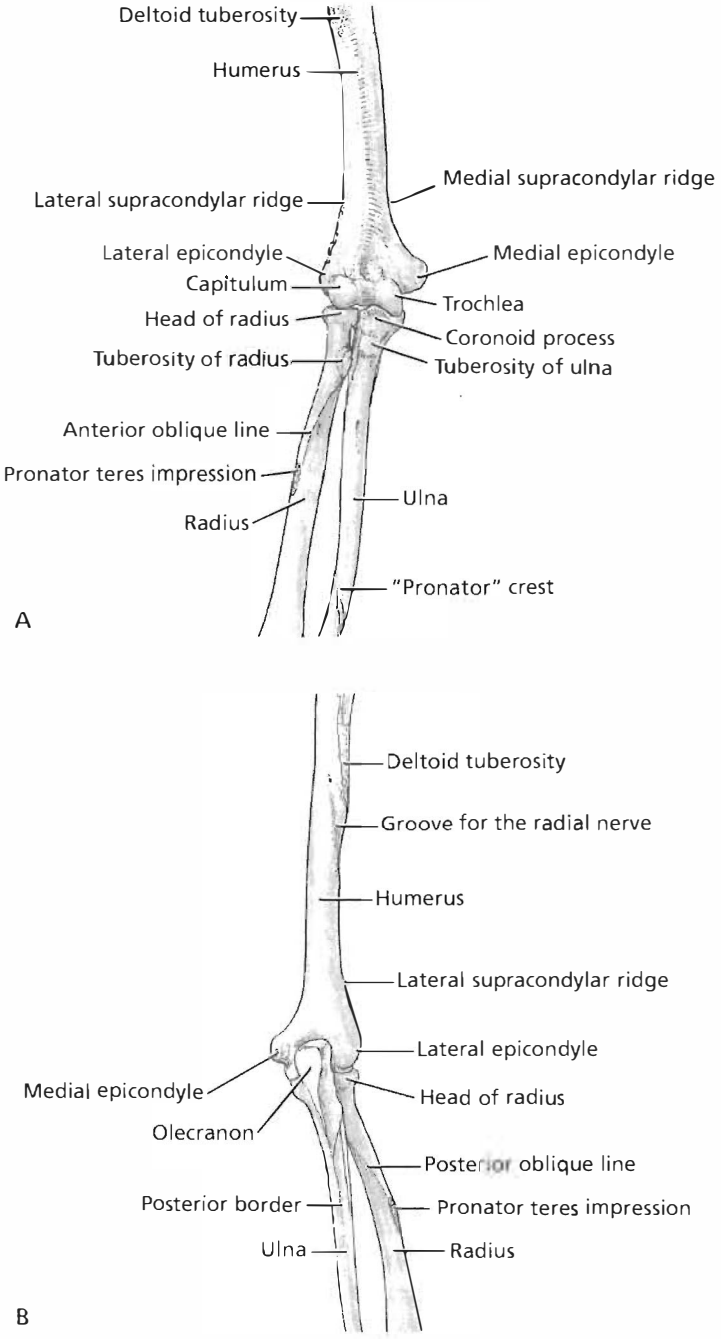

FIGURE 18.1.1. A, Bony anatomy of the elbow, anterior view. **B,** Bony anatomy of the elbow, posterior view. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

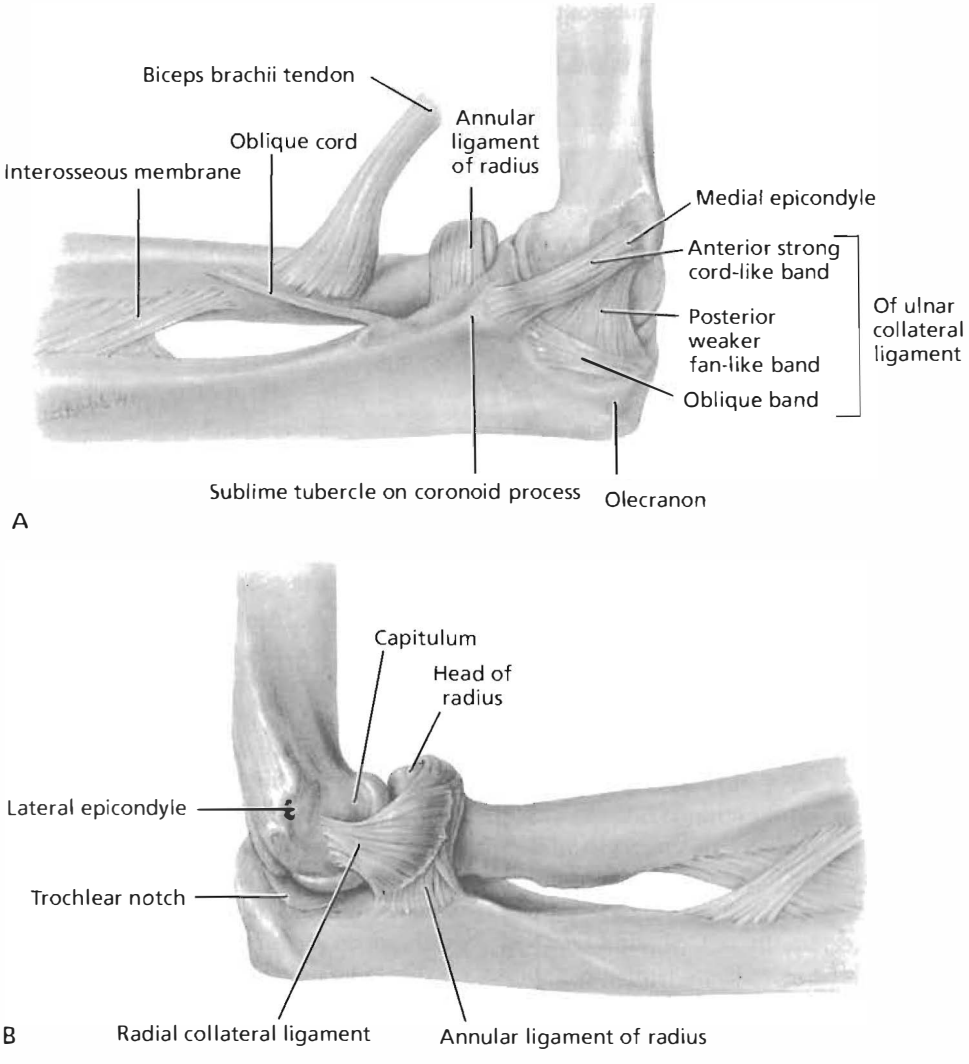

FIGURE 18.1.2. A, Ligaments of the medial elbow. Note the different divisions of the ulnar collateral ligament **B,** Ligaments of the lateral elbow. (From Agur AMR, Lee ML. *Grant's atlas of anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

proximal ulna, and is held into this pivot joint by the annular ligament. The radial (lateral) collateral ligament is triangular with its narrow proximal attachment on the lateral epicondyle, and the broader distal attachment into the annular ligament. Some superficial fibers have attachments on the lateral ulna.

Blood is supplied to the elbow joint by an anastomosis of branches of the brachial artery crossing the elbow. These include the brachial, superior and inferior ulnar collateral, the anterior and posterior branches of the deep brachial, and the recurrent radial arteries. The anterior and posterior interosseous arteries provide blood to the proximal radioulnar joint. The musculocutaneous (C5-C7), radial (C5-C8, T1) and ulnar (C7-C8, T1) nerves innervate the elbow joint.

Primary flexors of the elbow include the biceps brachii, brachialis, and brachioradialis

muscles. Proximal attachment of the long head of the biceps is into the supraglenoid tubercle of the scapula, and the short head attaches into the coracoid process of the scapula. The biceps brachii is the strongest supinator of the forearm because of its distal attachment on the radial tuberosity. The brachialis arises from the distal half of the anterior humerus, and inserts upon the coronoid process and tuberosity of the ulna. The brachioradialis takes its proximal attachment from the proximal two thirds of the lateral supracondylar humeral ridge and inserts upon the lateral surface of the distal radius. The biceps brachii and brachialis are within the anterior or flexor compartments of the arm, and are innervated by the musculocutaneous nerve (C5-C6). Although an elbow flexor, the brachioradialis muscle lies within the extensor compartment, while the radial nerve (C5-C7) supplies its innervation (Fig. 18.1.3A and B).

Muscles that flex and extend the wrist and digits also act as secondary flexors and extensors of the elbow. These muscles are arranged by function within fascial compartments in the forearm. The flexor muscles arise from the medial epicondyle, and are housed within the anterior forearm compartment. The extensors occupy the posterior forearm compartment, and arise from the lateral epicondyle.

The flexor carpi radialis (FCR), palmaris longus (PL), and flexor carpi ulnaris (FCU) attach to the medial epicondyle by forming a common flexor attachment. The FCU has two heads, the humeral head arising from the medial epicondyle, and the ulnar head, which arises from the olecranon and posterior border of the ulna. The FCR, PL, and FCU with the pronator teres muscle occupy the superficial forearm compartment, and cross the elbow joint. The flexor digitorum superficialis (FDS) is the largest of the forearm muscles. It divides into two heads, the humeroradial head, which arises from the medial epicondyle, ulnar collateral ligament, and coronoid process of the ulna, and the radial head, which arises from the superior half of the anterior border of the radius. The flexor digitorum profundus (FDP) arises from the anterior surfaces of the ulna and interosseous membrane, covering the proximal three fourths, and is considered to lie in the deep anterior compartment. The flexor pollicis longus (FPL) lies parallel to the FDP, and arises from the anterior surface of the radius and anterior interosseous membrane.

The extensor wad lies along the posterior, medial forearm. The extensor carpi radialis longus (ECRL) arises from the proximal attachment to the supracondylar ridge of the humerus. The common extensor tendon forms the proximal attachment for the muscles that arise from the lateral epicondyle. The four muscles in this group include the extensor carpi radialis brevis (ECRB), the extensor carpi ulnaris (ECU), which also attaches to the posterior border of the ulna, the extensor digitorum (ED), and the extensor digiti minimi (EDM).

The triceps and anconeus muscles extend the elbow. Proximal attachments of the triceps muscle include the long head, which attaches to the infraglenoid tubercle of the scapula; the lateral head, which attaches above the radial groove to the posterior humerus; and the medial head, which attaches to the posterior humerus distal to the radial
the proximal olecranon process of the ulna and the forearm fascia. The anconeus, an important valgus load stabilizer, attaches proximally to the lateral epicondyle, distally to the lateral olecranon, and superiorly to the posterior ulna. The radial nerve innervates both muscles (triceps C6-C8, anconeus C7-C8, T1).

The biceps brachii and the supinator muscles perform supination of the forearm. The supinator muscle attaches laterally to the lateral epicondyle, radial collateral and annular ligaments, and the supinator fossa and crest of the ulna, then wraps around the radius where it attaches to the posterior, lateral, and anterior proximal one third. As the supinator contracts, it "unwraps," rotating the forearm into supination. The deep branch of the radial nerve innervates the supinator muscle (C5-C6). The extensor pollicis longus and extensor carpi radialis longus muscles act as secondary supinator muscles.

Primary pronator muscles are the pronator quadratus and pronator teres. The pronator teres is the proximal muscle, with a proximal attachment at the medial humeral epicondyle and the

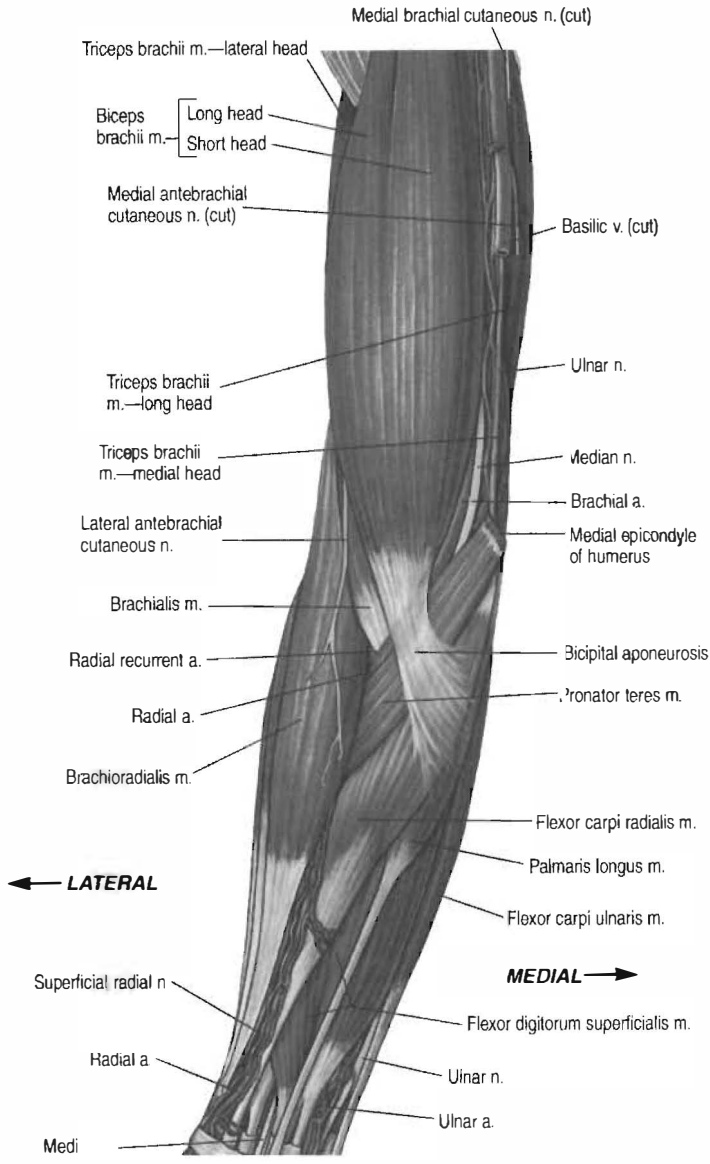

FIGURE 18.1.3. A, Muscles of the elbow, anterior view. **B,** Muscles of the elbow, posterior view. (From Altcheck DW, Andrews JR. *The athlete's elbow.* Baltimore: Lippincott Williams & Wilkins, 2001.)

coronoid process of the ulna. It then crosses the dorsum of the proximal radius to its distal attachment on the lateral radius. The median nerve (C7-C8) innervates the pronator teres. The pronator quadratus, as the name implies, is a rectangular muscle that lies across the dorsum of the distal radius and ulna. The anterior interosseous nerve, a branch of the median nerve (C8-T1), innervates the pronator quadratus.

The extrinsic finger flexor and extensor muscles, and the long wrist extensors cross the elbow and assist in elbow motion. They are organized into flexor and extensor compartments. Muscles of the extensor compartment

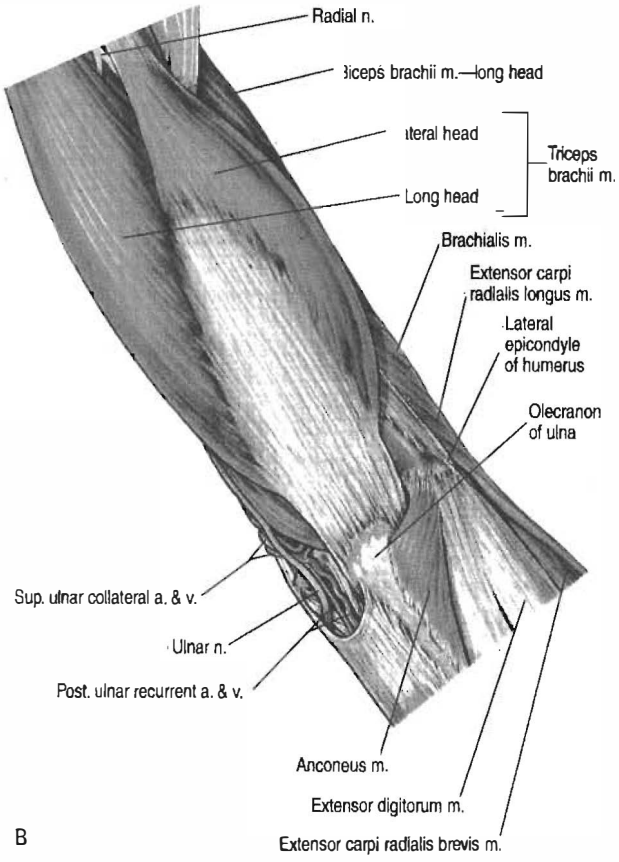

FIGURE 18.1.3. B (*continued*)

have proximal attachments on the lateral epicondyle or supracondylar ridge. The extensor carpi radialis brevis (ECRB), extensor digitorum (ED), extensor digiti minimi (EDM), and extensor carpi ulnaris (ECU) form a common extensor tendon that attaches to the medial epicondyle. The ECU also has a proximal attachment to the lateral proximal ulna. The extensor carpi radialis longus (ECRL) has a proximal attachment to the supracondylar ridge. The radial nerve and its branches innervate these muscles. The deep branch of the radial nerve (C7-C8) innervates the ECRB. The posterior interosseous nerve (C7-C8) provides innervation to the ED, EDM, and ECU.

The flexor/pronator compartment has deep and superficial divisions. The deep muscles, flexor digitorum profundus, flexor pollicis longus, and pronator quadratus do not cross the elbow joint. The superficial muscles, pronator teres (PT), flexor carpi radialis (FCR), palmaris longus (PL), flexor carpi ulnaris (FCU), and flexor digitorum superficialis (FDS) attach in part through a common flexor tendon to the medial epicondyle. The median nerve innervates the PT, FCR (C6-C7), FDS (C7-C8, T1), and palmaris longus (C7-C8). The ulnar nerve innervates the FCU (C7-C8).

Several bursae are located in association with the tendons at the elbow. The three olecranon bursae have clinical significance because of the frequency of inflammation or infection of these structures. The infratendinous olecranon bursa is sometimes present in the triceps tendon as it inserts into the olecranon process. The subtendinous bursa is located between the triceps tendon and the olecranon process, and the subcutaneous bursa may be found superficial to the triceps tendon in the subcutaneous connective tissue (1).

Major structures that cross the volar elbow are located within the cubital fossa. This is a triangular space bounded proximally by the horizontal flexion skin crease, medially by the pronator teres muscles, and medially by the brachioradialis muscle. Following the boundaries are the major veins of the arm, the basilic medially and the cephalic laterally. The intermediate cubital vein runs medial to lateral connecting the basilic and cephalic veins along the distal border of the cubital fossa. The supinator and brachialis muscles form the deepest part of the fossa. The deep and superficial branches of the radial nerve are located with these deep structures. The brachial fascia and the bicipital aponeurosis enclose the fossa superficially. The brachial artery forms its terminal branches within the cubital fossa. The biceps brachii tendon passes through the cubital fossa on its way to insert on the radial tuberosity, and the median nerve is located just medial to the biceps brachii tendon.

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18.2 Physical Examination

NANCY WHITE

When it comes to athletics, the elbow is best seen and not heard. It is a crucial link in transmitting force from the shoulder to the hand and it puts the hand in position to act and perform. Though injuries are relatively rare in athletics, breakdown of elbow function can disrupt the kinetic chain and quickly lead to subsequent injury and disability. The flexion-extension and rotatory components of the elbow allow for the appropriate positional placement of the hand.

An adequate clinical history includes assessing acute versus chronic pain, events surrounding the pain (i.e., throwing or racquet use), history of catching or locking, and history of paresthesias. Throwing athletes should be able to identify at which phase of throwing the elbow pain occurs. Referral pain is common to and from the elbow, so a keen eye along the kinetic chain is crucial.

OBSERVATION

Examination of the elbow begins with inspection of both elbows. Appropriate dress to allow for full view of both elbows is essential. Note any discrepancy in muscular definition, atrophy, surgical scars, or abnormal angulation at the elbow.

Initial evaluation of the elbow should also include presence or absence of swelling. Generalized effusion could signal a supracondylar fracture, or radial or olecranon fracture or dislocation. Focal swelling over the olecranon generally resembles a goose egg and represents olecranon bursitis. In this case, monitor for any signs of erythema or calor, which represent a secondary infection.

Alignment and mobility should be examined in the anteroposterior (AP) and lateral planes. In the AP view with the elbow extended, the carrying angle can be visualized. This is the valgus angle between the shaft of the humerus and the center line of the forearm (Fig. 18.2.1). Normal carrying angle is between 5 and 10 degrees and is usually 5 degrees greater in women than in men (2). Note any discrepancy in the carrying angle between the two elbows. Fractures of the distal humerus can alter the carrying angle.

Another way to examine alignment is to note the position of the epicondyles and the olecranon tip. At 90 degrees of flexion, the epicondyles should be equidistant to the tip of the olecranon. If not, a fracture or dislocation should be suspected in the face of an acute injury.

From the lateral position, note the alignment of the olecranon with the humeral shaft. Posterior and posterolateral dislocations result in an obvious deformity, due to the location of the olecranon process posterior to the humeral shaft.

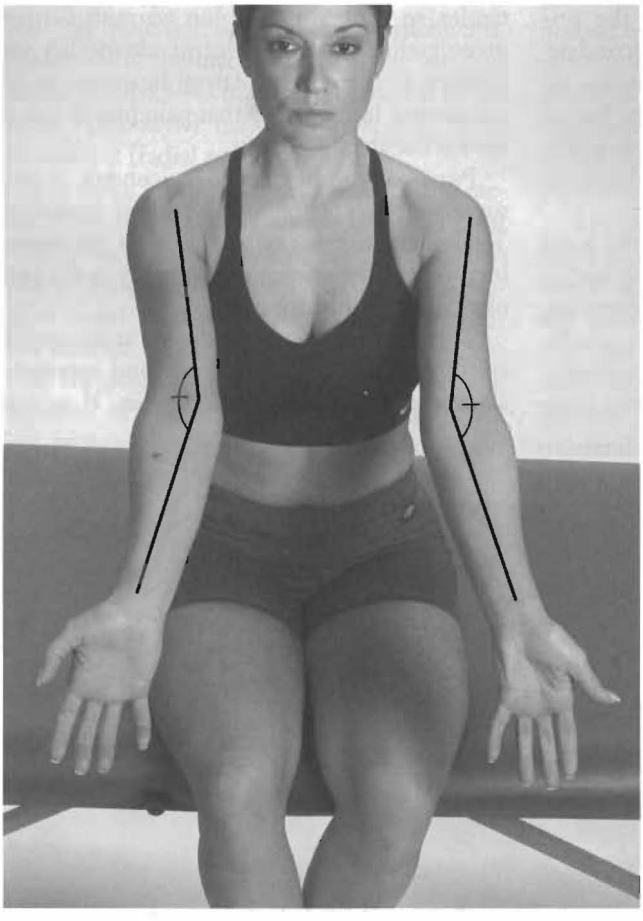

FIGURE 18.2.1. Carrying angle.

FIGURE 18.2.2. Medial epicondyle.

PALPATION

In the medial elbow compartment, the primary bony structure is the medial epicondyle (Fig. 18.2.2). Pain over the bony prominence, especially in conjunction with resisted wrist flexion and pronation, is indicative of medial epicondylitis. If pain is present in a young athlete, the examiner should consider a growth plate injury.

The medial collateral ligament is the most important stabilizer of the elbow, and its stability should be evaluated in every elbow examination. Palpate its epicondylar and ulnar attachments, noting tenderness, thickness, irregularity, and bogginess (Fig. 18.2.3). The anterior bundle is important to palpate, as it resists valgus load in throwing. Normal ligaments are never tender, so pain in this region warrants further investigation. The flexor carpi ulnaris lies superficial to the ulnar collateral ligament, so do not assume immediately that pain here is a torn medial collateral ligament.

Proximal to the medial epicondyle is the supracondylar region of the distal humerus. Tenderness in this area should alert the examiner to consider a fracture, especially in the face of a traumatic history.

The ulnar nerve can be palpated at the medial epicondyle and followed distally and anteriorly along the wrist flexor muscle bellies. If pain is

FIGURE 18.2.3. Palpation of the medial collateral ligament.

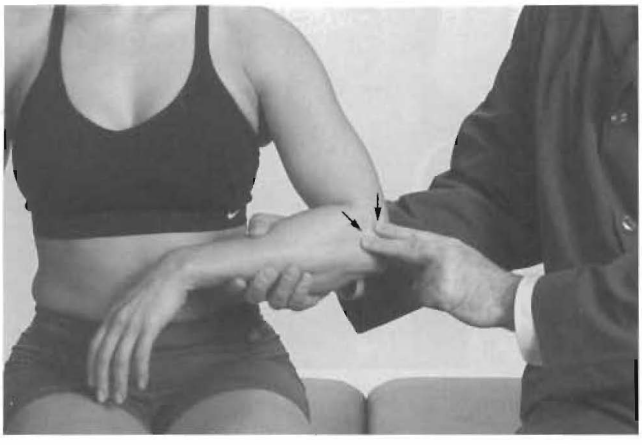

FIGURE 18.2.4. Radial head and lateral epicondyle.

elicited with gentle palpation of the nerve, the examiner should consider a nerve irritation injury.

In the lateral aspect, the radial nerve crosses the lateral supracondylar line as it traverses from the posterior to anterior compartments. Tenderness of the medial aspect of the extensor muscle bellies (3–4 cm distal and medial to the lateral epicondyle) may indicate radial nerve entrapment (radial tunnel syndrome).

The radial head is palpated posterior and medial to the wrist extensor muscle bellies and just distal to the lateral epicondyle (Fig. 18.2.4). Palpation, along with rotation, aids in the diagnosis of radial head subluxation or proximal radius injuries.

In the posterior compartment of the elbow, palpation of the olecranon bursa is relatively easy, as it overlies the distal triceps tendon and olecranon (Fig. 18.2.5). Note any swelling, pain, or thickening of the bursa or bony fragments, particularly after a traumatic injury. The tip of the olecranon is best noted with the elbow flexed beyond 60 degrees. The fossa, just superior to the olecranon tip, is also best palpated with the elbow in the flexed position. Note any pain, crepitus with flexion or extension, or bony fragments that would require further radiographic studies to evaluate for bony impingement.

The primary structure in the volar compartment of the elbow is the distal biceps tendon, which inserts onto the proximal radius. Note any pain in this area, which is common for overhead throwing athletes who sustain either acute trauma or overuse injury. Also palpate the brachial artery and compare it with the contralateral elbow.

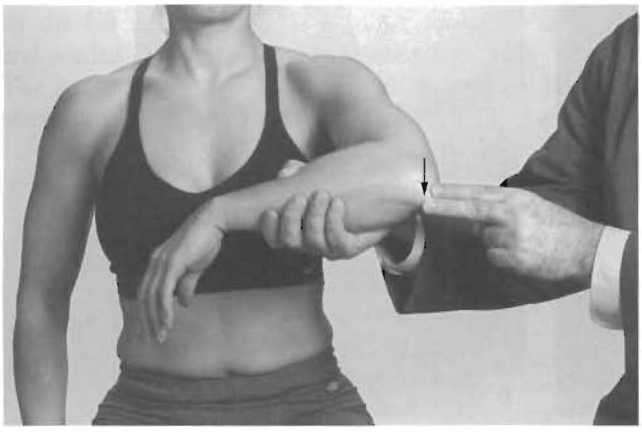

FIGURE 18.2.5. Palpation of the olecranon and bursa.

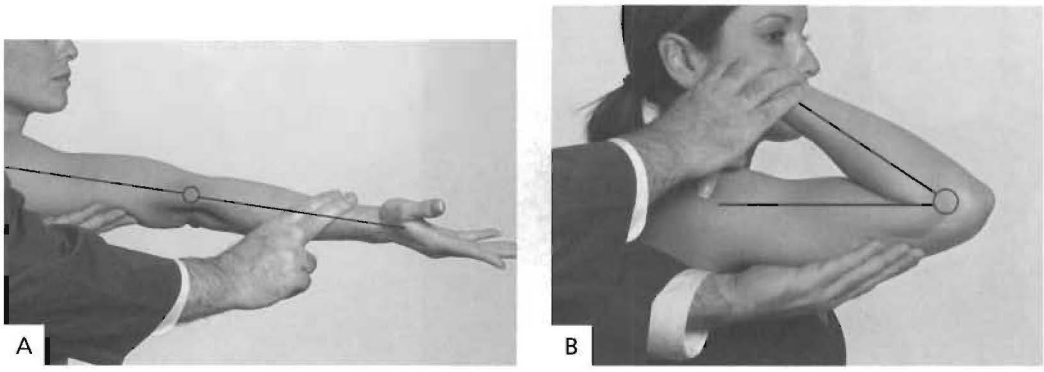

FIGURE 18.2.6. A, Forearm supination. **B,** Forearm pronation.

RANGE OF MOTION

Active motion should be examined first. Full extension is 0 degrees of flexion, and full flexion is approximately 140 to 150 degrees. Flexion and extension are primarily a function of the humeral-ulnar component of the elbow joint (Fig. 18.2.6A and B). Pronation and supination are primarily a function of the proximal radial-ulnar articulation (2). It is easiest to evaluate pronation and supination with the elbow flexed to 90 degrees. Neutral position is with the thumb up. From here, normal pronation is 90 degrees palm down, while supination is 90 degrees palm up (Fig. 18.2.7A and B).

Restriction or pain in motion should be evaluated further. Restricted motion may signal arthropathy, loose bodies, or muscular restriction. In particular, limited extension should signal the examiner to look for possible posterior humeral-ulnar joint arthropathy or posterior joint loose bodies. Loss of extension may also result from restriction of the distal biceps tendon. Athletes such as baseball pitchers may develop flexor contractures, yet have no loss of function or skill. (1)

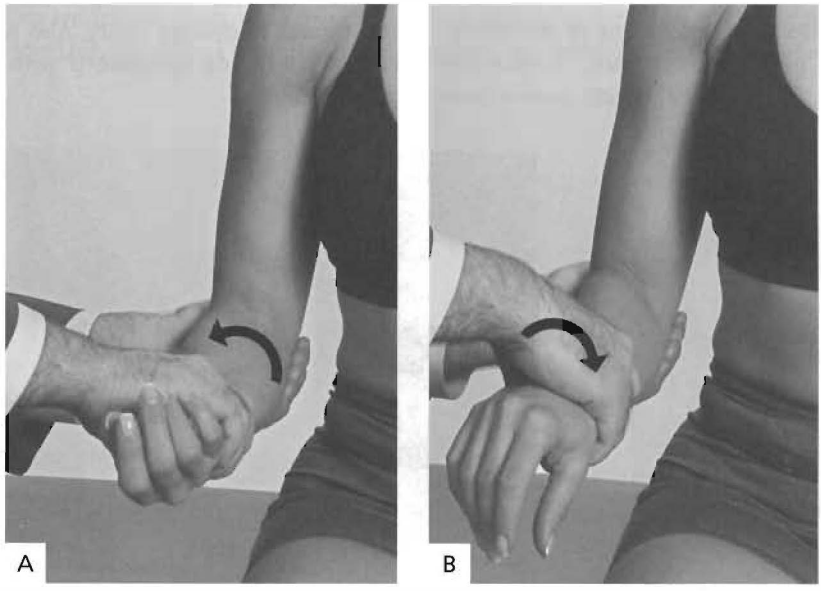

FIGURE 18.2.7. A, Elbow extension range of motion (ROM). **B,** Flexion ROM.

TABLE 18.2.1. PASSIVE RANGE OF MOTION FOR THE ELBOW

Motion	Range (degrees)
Flexion	135–150
Extension	0–5
Pronation (wrist)	90
Supination (wrist)	90

Similarly, limited flexion may represent anterior humeral-ulnar joint pathology or capsulitis. Any loss of motion should first be investigated with appropriate imaging. Normal ranges of elbow passive motion are listed in Table 18.2.1 (3).

NEUROVASCULAR EXAMINATION

Motor Strength

The muscles of the elbow are listed in Table 18.2.2. The primary motions for motor testing are elbow extension and flexion, and wrist pronation and supination. Testing is on a +5 scale. Testing biceps strength is best done with the forearm in full supination, while brachialis strength can be best assessed with the forearm in full pronation (1).

Sensory

The lateral epicondyle is in the C5 dermatome. The radial forearm, thumb, and index fingers are in the C6 dermatome. The C7 dermatome can be checked by testing the sensation of the long finger. The ring and fifth fingers, along with the hypothenar eminence, are innervated by the C8 sensory nerve. The ulnar forearm and medial epicondyle are innervated by T1. The sensory nerve for the proximal arm near the axilla is T2.

Tinel's Sign. The ulnar nerve may sometimes sublux or dislocate from the ulnar groove causing pain for the athlete. This is particularly troublesome for the throwing athlete. To test, the examiner uses a reflex hammer to gently tap the ulnar nerve in the ulnar groove.

Positive test: Paresthesias in the ulnar aspect of the forearm and hand.

Indicates: Ulnar nerve irritation at the ulnar groove.

Vascular

The brachial artery, as described in the anatomy section, 18.1, courses alongside of the distal biceps tendon. Pulses can be easily palpated in the cubital fossa. Trauma to the elbow joint causing marked swelling and ecchymosis can disrupt blood flow. Check flow to the wrist at the ulnar and radial arteries with the Allen test, pulse palpation, and capillary refill to ensure proper arterial supply. Disruption of flow at the elbow would stop flow to both radial and ulnar arteries.

STABILITY

The soft tissues of the elbow play a significant role in elbow stability. The most functional stabilizing ligament of the elbow is the (MCL) (4). The stability of this structure should be evaluated during every elbow examination. Other elbow problems are often caused by a weakness in this structure. The most important aspect of the MCL is the anterior bundle. Special tests have been designed to help determine laxity of the MCL structure.

Valgus Stress Test. The athlete is placed in a supine position with the shoulder abducted and

TABLE 18.2.2. MUSCLES OF THE ELBOW

Muscle	Function	Nerve
Brachialis	Primary flexor	Musculocutaneous
Biceps brachii	Primary flexor Supinator	Musculocutaneous
Brachioradialis	Secondary flexor	C6
Supinator	Primary supinator	C6
Triceps	Primary extensor	Radial
Anconeus	Secondary extensor	

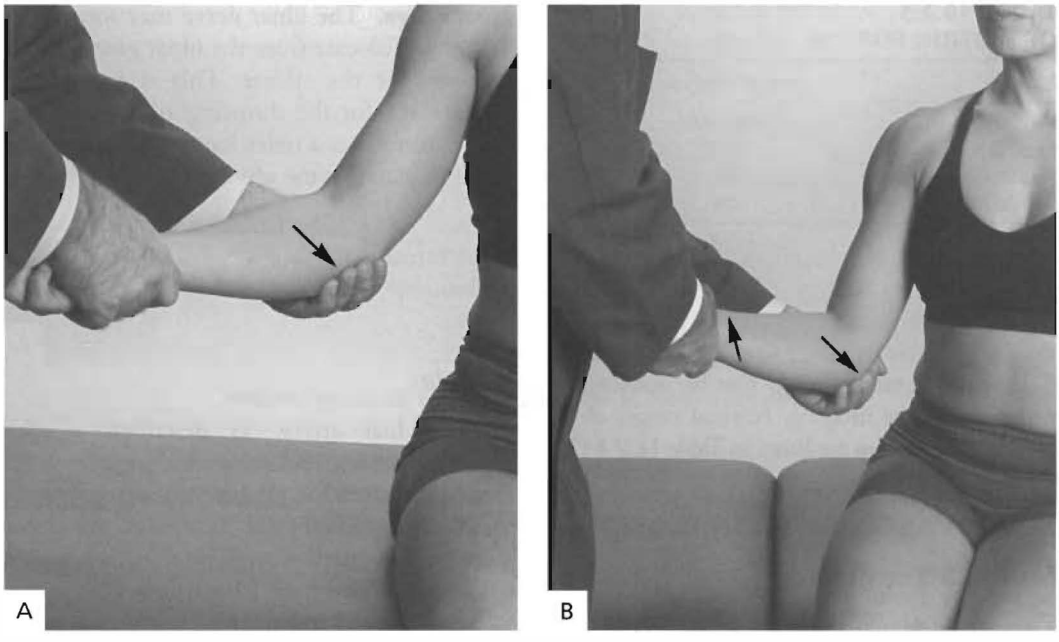

FIGURE 18.2.8. Valgus stress of the elbow at 30 degrees **(A)** and at 90 degrees **(B)**.

externally rotated, then valgus stress is applied to the elbow with the forearm pronated. The elbow should be tested at 0, 30, and 90 degrees of flexion (Fig. 18.2.8A and B). This test can be performed sitting as well (1).

Positive test: Pain, a perceived increase in medial joint gapping or opening compared with the contralateral elbow.

Indicates: Sprain of the medial collateral ligament with or without tears.

Varus Stress Test. The vatus stability of the elbow is primarily provided by the radial collateral ligament complex (radial collateral ligament and the lateral ulnar collateral ligament). These ligaments are tested with varus stressing at 15 degrees of flexion and the upper arm internally rotated (Fig. 18.2.9) (1).

Positive test: Pain or laxity along the lateral joint of the elbow.

Indicates: Lateral collateral ligament pathology.

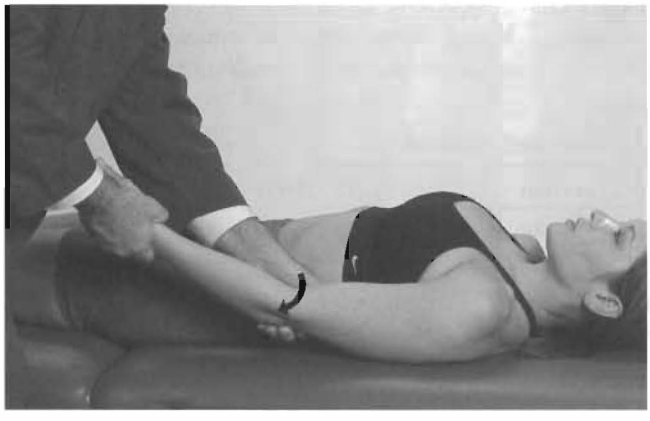

FIGURE 18.2.9. Varus stress of the elbow at 15 degrees.

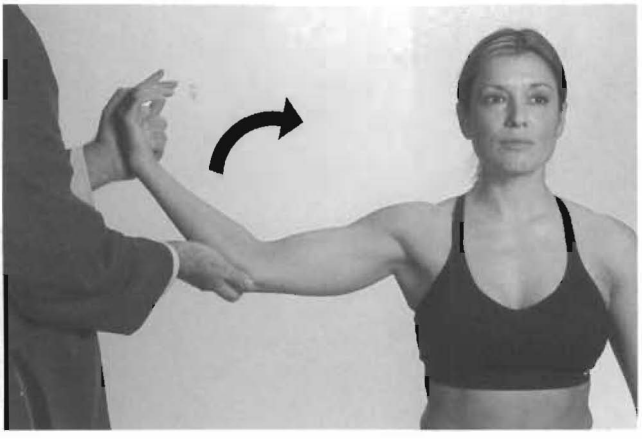

FIGURE 18.2.10. Milking sign.

Milking Sign. The athlete is sitting with the affected arm held in 90 degrees of abduction and elbow flexion. The examiner clasps the athlete's hand, index finger through the thumb-index finger web space and thenar eminences adjacent to each other. The examiner's stabilizing hand holds the elbow on its lateral side. The examiner then flexes the elbow while holding valgus load on the medial collateral ligament from flexion to extension (Fig. 18.2.10) (3,4).

Positive test: Pain, particularly between 30 and 60 degrees.

Indicates: Medial collateral ligament pathology, valgus instability.

Lateral Pivot Shift Test. An injury to the medial collateral ligament complex can result in posterolateral instability. This can lead to posterolateral subluxation of the radial head with a rotational subluxation of the humeroulnar joint.

To perform this test, the athlete is in the supine position with the arm forward flexed and the forearm supinated (Fig. 18.2.11). The examiner applies axial compression and valgus stress simultaneously to the elbow. Note any dimpling or indentation of the skin at the radiocapitellar joint secondary to subluxation of the radial head. This test is best performed with the athlete under anesthesia (1).

Positive test: Palpable radial subluxation or noted skin changes.

Indicates: Posterolateral instability of the elbow.

Note: When the elbow is flexed to approximately 40 degrees or more, reduction of the radius and ulna occurs, causing a palpable, visible clunk. This is further evidence of instability.

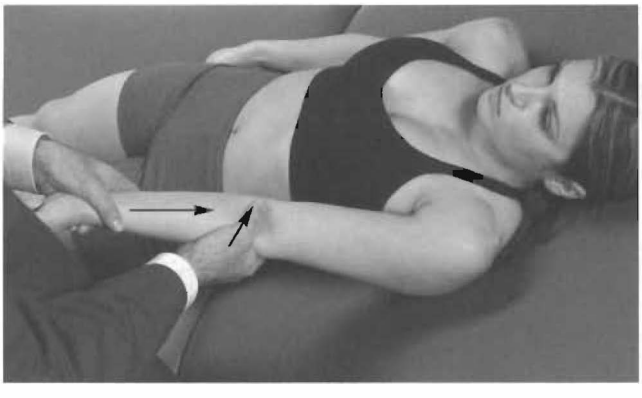

FIGURE 18.2.11. Lateral pivot shift test.

FIGURE 18.2.12. Maudsley's test.

SPECIAL TESTS

Maudsley's Test. The examiner has the athlete sitting and holds the elbow with the stabilizing hand so that the forearm is extended and parallel to the ground. The athlete is asked to extend the middle finger against the examiner's counterforce. When palpating the athlete's lateral epicondyle while exerting force against the middle finger extension with the elbow in full extension, pain can be elicited (Fig. 18.2.12).

Positive test: Pain provoked by the maneuver.
Indicates: Lateral epicondylitis, radiocapilellar dysfunction.

Mill's Test. The examiner places the athlete in a position to flex the elbow and flex the wrist and note any pain at the lateral epicondyle (Fig. 18.2.13).

Positive test: Pain at the lateral epicondyle in this position.
Indicates: Lateral epicondylitis, extensor muscle strain.

Ulnar Nerve Instability. The examiner places the athlete's arm abducted and externally rotated. While palpating the ulnar nerve at the ulnar groove, the examiner flexes and extends the elbow repeatedly (Fig. 18.2.14). The athlete can also be tested in the supine position.

FIGURE 18.2.13. Mill's test.

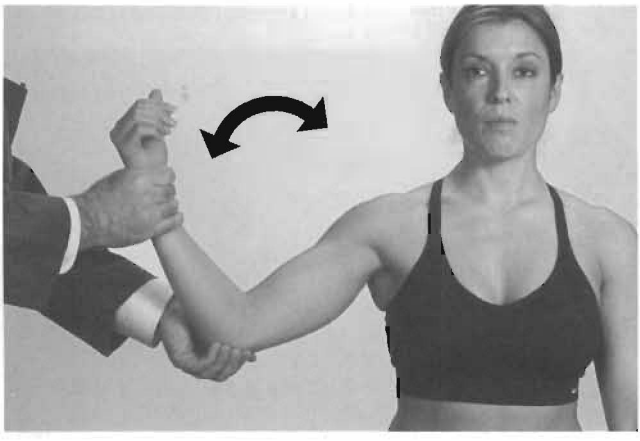

FIGURE 18.2.14. Ulnar nerve instability test.

Positive test: The examiner is able to palpate the ulnar nerve as it subluxes or dislocates in and out of the ulnar groove.

Indicates: Ulnar nerve instability.

JOINT PLAY

This is evaluated at the following articulations (5):

1. Superior radioulnar joint.
2. Radiocapitellar joint.
3. Humeroulnar joint.

Superior Radioulnar Joint

1. *Upward glide of the radial head*
   - The examiner flexes the athlete's elbow to 90 degrees and places the dorsiflexed right hand against the athlete's posterior distal humerus.
   - The examiner clasps the athlete's hand with the left hand, placing his or her thumb in the first web space between the thumb and index finger, thenar eminences together (Fig. 18.2.15).
   - Stabilizing the elbow with the right hand, the examiner thrusts with the left hand along the long axis of the athlete's radius.
2. *Downward glide of the radial head*
   - The examiner holds the athlete's arm around the wrist with the left hand.
   - The right hand holds the distal humerus in its first web space with the athlete's elbow at 90 degrees of flexion.
   - Stabilizing the athlete's humerus with counterforce from the right hand, the ex-

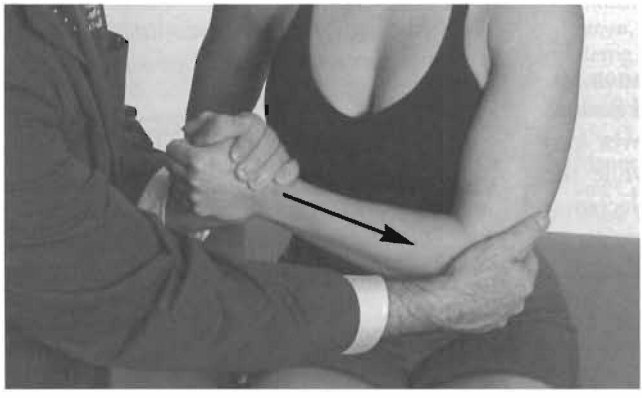

FIGURE 18.2.15. Joint play, superior radioulnar joint: upward glide.

FIGURE 18.2.16. Joint play, superior radioulnar joint: downward glide.

aminer uses the left hand to pull along the radial axis, distracting the radial head (Fig. 18.2.16).

Radiocapitellar Joint

The articulation between the radius and capitellum often becomes dysfunctional from secondary pathology such as lateral epicondylitis and radial nerve entrapment (6). The capitellum can absorb significant stress in throwing athletes and gymnasts, leading to orthopedic pathology such as osteochondritis dissecans, osteophytosis from degenerative osteoarthritis, and Panner's disease in children (1,3,6). Additionally, restricted radiocapitellar motion affects wrist rotation and elbow flexion, which are intrinsic in many athletic skills. Function at the radiocapitellar joint depends on proper function at the superior radioulnar joint, so treatment should be directed at the latter first, then the former.

Humeroulnar Joint

The trochlea is the seat for the olecranon, allowing for smooth elbow extension and flexion. The coronoid process of the ulna moves into the coronoid fossa during flexion, while the olecranon process moves into the olecranon fossa during extension. Joint play consists of medial and lateral tilt off the longitudinal axis of the humerus (5). Any joint dysfunction affecting articulations in either fossa can restrict range of motion and cause pain. Loss of joint play and a shift in ulnar tilt can cause the olecranon to enter the fossa offline during extension and impinge the synovial fat, a significant problem in throwing athletes and golfers.

1. *Medial tilt of the olecranon*
   - The examiner holds the athlete's elbow in the position used to valgus-load the medial collateral ligament. The athlete can be sitting, but control of the elbow is optimal when the athlete is supine.
   - The examiner flexes the elbow 15 degrees.
   - With the stabilizing hand holding the athlete's wrist, the examiner uses his mobilizing hand on the humerus to introduce a valgus thrust. At the same time, the examiner extends the elbow, sliding the olecranon process into its fossa (Fig. 18.2.17). Pain or crepitus could indicate pathology, such as the aforementioned impingement.
2. *Lateral tilt of the olecranon*
   - The test is the same as for the medial tilt, except a varus force is loaded at 15 degrees of extension.
   - The examiner also moves the elbow into extension during the varus load to rub

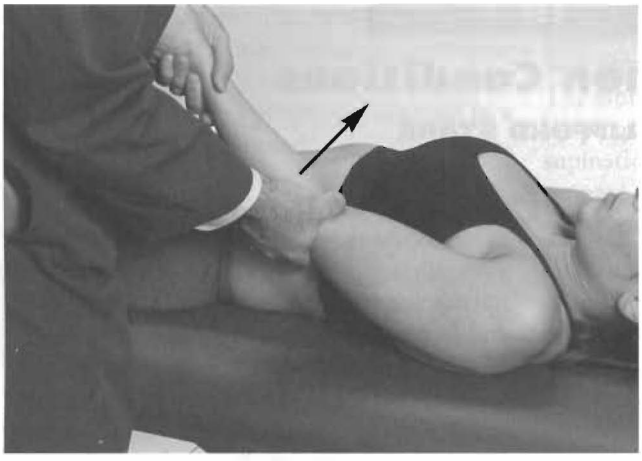

FIGURE 18.2.17. Joint play, humeroulnar joint: medial tilt of the olecranon.

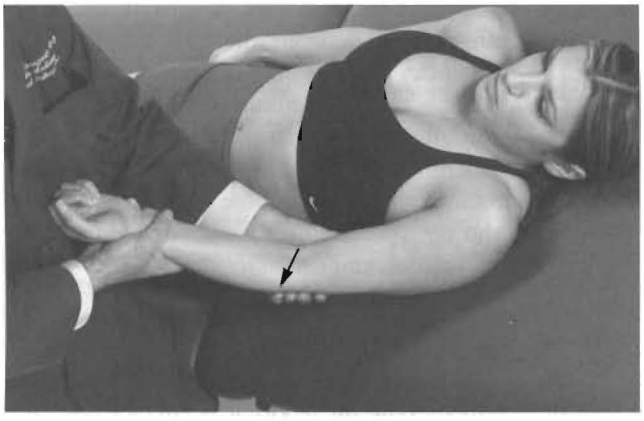

FIGURE 18.2.18. Joint play, humeroulnar joint: lateral tilt of the olecranon.

the olecranon process off the capitellum and into the fossa (Fig. 18.2.18). Note for pain and joint play.

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18.3 Common Conditions

CLIFFORD STARK

The history should always include the suspected mechanism of injury, timing, occupation, physical/athletic activity (including ability level, physical condition, and changes in activity), modifying factors, and associated symptoms. A history of neck, back, or shoulder pain or injury should raise heightened suspicion of contributing factors involving the entire kinetic chain in the trunk and upper extremity. The elbow is the intermediary between the stable shoulder and the mobile hand, so the elbow examination demands an investigation into the proximal and distal regions.

LATERAL EPICONDYLITIS (TENNIS ELBOW)

Lateral epicondylitis is the most common overuse elbow injury (1). It results from repetitive use of the wrist extensors in about 90% of the cases (2). This specifically involves the extensor carpi radialis brevis tendon at or near its origin on the lateral epicondyle, where it often undergoes degenerative changes (tendinosis), particularly if the process is more prolonged.

Athletes

Lateral epicondylitis is common in racquet sports as well as many occupations involving repetitive overuse of the wrist extensors (e.g., typing, clerical work). The condition is frequently seen in novice tennis players, who often use improper mechanics resulting in excessive strain on the wrist extensors. A notorious example is the late backhand, which, in addition to poor leverage, results in increased wrist extension in an effort to hit the ball straight. Poor trunk mechanics, scapular dyskinesia, decreased strength, and loss of motion in the neck and upper extremity compromise the kinetic chain, and therefore predispose to this injury.

Weightlifters can develop lateral epicondylitis from repetitive lifting, more so if heavy weights or dumbbells are involved. The large muscle groups are usually much stronger than the wrist flexors and extensors, so repetition can fatigue the forearms faster. Plus, many weightlifters do exercises that work the wrist flexors more than the extensors. A strength imbalance can develop between the two wrist muscles, which can lead not only to extensor breakdown but also to carpal tunnel symptoms. The no-pain, no-gain mentality of weightlifting can sometimes be a factor in epicondylitis, especially in chronic cases. The more intense weightlifters who do repetitive lifting without proper recovery, balance, and flexibility are at risk.

History and Examination

Athletes typically report achy lateral elbow pain and tenderness, with occasional sharp pain that radiates down the lateral forearm. Symptoms worsen by use of the involved upper extremity, and in many cases, athletes report pain and weakness while lifting a weight or turning a doorknob.

Examination shows tenderness over the extensor carpi radialis brevis tendon, typically just anterior, medial, and distal to the lateral epicondyle. Pain in this area is reproduced with resistance against wrist or long finger extension, particularly with the elbow in extension. Tinel's test over the radial tunnel should be negative, as a positive test would suggest *radial tunnel syndrome*, an entrapment neuropathy that mimics lateral epicondylitis (3).

Standard Treatment

Acute-phase treatment first involves stopping the provocative activities. Subacute treatment involves relative rest, protection, ice, and analgesics. A rehabilitation program is initiated in a stepwise fashion as tolerated, using strengthening exercises, stretches, and modalities such as iontophoresis or phonophoresis. A counterforce brace (tennis elbow brace) is often used to displace the forces from the injured area. In more severe cases, or those recalcitrant to conservative treatment, a local steroid injection is often used. Surgical intervention is reserved for the most severe cases unresponsive to prolonged treatment and usually involves débridement of tendinosis or repair (if possible) of significant tissue damage (4).

Manual Medicine

Lateral epicondylitis tends to be a chronic problem, and many times treatment failure is due to missed breakdowns in the kinetic chain. The first place to look and treat in the kinetic chain is the cervical spine. Look for evidence of radiculopathy, discitis, or facet injury. Identify and treat somatic dysfunctions in the neck, thorax, scapula, and ribs, most notably the C7-T1-first rib complex, and in the trapezius and levator scapulae muscles. The anterior cervical musculature (scalenes, sternocleidomastoids, platysma) pulls the cervical spine and head forward when the muscles are chronically restricted or in spasm, which increases stress in the C7-T1 region, restricting cervical extension and locking up the proximal link in the kinetic chain to the wrist. This should be evaluated and treated.

Examine the shoulder for glenohumeral pathology. Look for a tight posterior capsule with loss of adduction. For example, if the arm cannot cross over the body for a backhand, the extensor muscle bundle will activate more to compensate.

Most epicondylitis is related to restriction of the radial head at the radiohumeral and proximal radioulnar joint. Manual techniques should target the radial head and the associated articulations. Posterior radial head dysfunction is the most common type of dysfunction, and it results in restricted forearm supination. This may be demonstrated by having the athlete flex and bring the elbows to the chest, with the medial aspects of both forearms approximated. The athlete then attempts to extend the elbows, keeping the forearms together and maintaining supination. The dysfunctional forearm will pronate during extension.

When choosing a method of treating dysfunctions in the elbow, it is helpful to identify not only the dysfunction present but also the type of restriction. Myofascial restrictions are typically over a diffuse area, and usually respond well to myofascial release and/or muscle energy techniques. In contrast, fibrotic barriers are often best approached with high-velocity, low-amplitude (HVLA) techniques. Tender points, such as the lateral epicondyle, often improve dramatically with counterstrain treatments, which often help to break the feedback cycle of pain.

The following techniques are useful in treating some of the more commonly encountered dysfunctions associated with lateral epicondylitis.

Muscle Energy: Posterior Radial Head (5)

Rationale: To restore proper motion of the radial head in the anterior direction and to improve forearm supination.

1. The examiner flexes the elbow to 90 degrees while monitoring the posterior aspect of the radial head using the index finger. The forearm is supported by the stabilizing hand, while the mobilizing hand holds the wrist firm.
2. The athlete's forearm is rotated into the supination barrier (Fig. 18.3.1).
3. The athlete pronates gently against resistance for 3 to 5 seconds.
4. Relax, reposition, repeat, and reassess.

HVLA: Posterior Radial Head (6)

Rationale: A variation of the previously mentioned technique that may be useful in restoring anterior motion of the radial head, particularly when a fibrotic barrier (often chronic) exists.

1. The clinician grasps the athlete's proximal forearm with the index finger overlying the posterior aspect of the athlete's radial head.

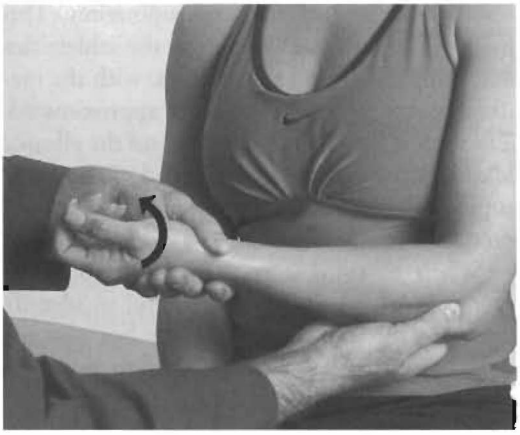

FIGURE 18.3.1. Muscle energy technique for a posterior radial head.

2. While stabilizing the athlete's distal forearm, hand, and wrist between the clinician's elbow and chest wall, the barrier of extension, supination, and slight adduction is engaged.
3. The clinician introduces a thrust on the radial head in an anterior and lateral direction.

HVLA: Anterior Radial Head (6)

Rationale: To restore normal posterior motion of the radial head, improving forearm pronation.

1. The clinician grasps the athlete's forearm with the thumb over the anterior aspect of the radial head (Fig. 18.3.2).
2. The clinician flexes and pronates the forearm while the radial head is held posteriorly.
3. Upon engaging the barrier, the clinician performs a thrust by increasing elbow flexion.

Counterstrain: Lateral Epicondyle or Radial Head

Rationale: To decrease spasm and tenderness in these areas by placing the arm in a position to minimize local tension. These techniques are well tolerated and easy to perform, and may be extremely useful in treating tender points on the lateral epicondyle or radial head.

1. The clinician holds the elbow in full extension, preferably with the use of a fulcrum such as the edge of the table.
2. While monitoring the tender point, the arm is supinated and abducted with varying degrees of force, until a position of minimal tenderness is encountered (Fig. 18.3.3).
3. This position is maintained for 90 seconds, then the arm is returned slowly to neutral position.

Myofascial Release: Forearm Extensors and Flexors (7)

Rationale: To help reduce restrictions in the soft tissue of the forearm and elbow region which lead to improper force distribution and excessive strain on the lateral epicondyle.

1. This technique can be accomplished in many different manners, depending on individual preference, and may be tailored to each athlete.
2. The clinician places both hands around the proximal and distal aspects of the athlete's forearm.
3. A steady twisting force may be applied with each hand, in either the same or opposite directions.

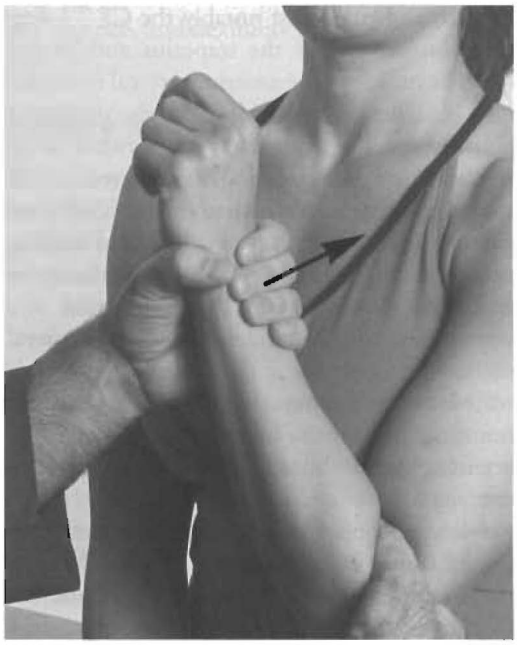

FIGURE 18.3.2. High-velocity, low-amplitude technique for an anterior radial head.

FIGURE 18.3.3. Counterstrain for the lateral epicondyle or radial head.

4. A concomitant axial force may be applied with either distraction or compression between the two hands (Fig. 18.3.4).
5. The direction of ease should be noted and used to approach new barriers.

Preventive Measures

Mechanics. Investigate the athlete's mechanics and adjust as deemed appropriate. Coaching support is strongly encouraged. In particular, look at the mechanics of a tennis or golf stroke, tennis serve, cycling position, or baseball pitch, depending on the athlete's particular sport.

Home Exercise Program. This consists of flexibility for the kinetic chain before activity and isokinetic and isometric strengthening exercises. The athlete should stay on this type of program if he or she is prone to lateral epicondylitis and overuse. Weightlifters should not neglect hand and wrist strength, because the endurance of these muscles enables the athlete to work out harder in developing the larger power muscles.

Lifting. Palms should be facing up whenever possible, and the arms at the side or in front of the body. Knees should be bent and the lumbar spine should be slightly extended to support the upper body and properly position the center of gravity over the spine.

Ergonomics. In the workplace, poor ergonomics must be evaluated and corrected to minimize recurrence. Those who work with a computer and mouse may develop problems with too low a chair, no padding around the front of the mouse pad, sitting too far from the desk, or having comfortable arm rests. Laborers using power tools are at higher risk as well.

The athletic setting has ergonomic issues as well. The grip width of a golf club or tennis rac-

FIGURE 18.3.4. Myofascial release for the forearm extensors and flexors.

quet affects this condition, particularly if the racquet is too narrow. The athlete should see a golf or tennis pro that knows the weight and width of clubs and their grips. Measure the hand, fit the grip correctly, and make sure the club or racquet is not too heavy, wide, or big.

MEDIAL EPICONDYLITIS

Often called golfer's elbow, little leaguer's elbow, and medial tennis elbow, medial epicondylitis is similar to lateral epicondylitis but involves pain in the medial elbow and proximal forearm that results from repetitive activities that require wrist flexion (often rapid wrist snapping) and forearm pronation. The primary pathology lies in the tendon origin of the pronator teres and forearm flexor muscle bundle.

Symptoms are usually worsened by activity. This injury is often seen in golfers and tennis players. In contrast with lateral epicondylitis, medial epicondylitis is often seen in more advanced tennis players who impart a lot of top spin on their forehand shot, or snap their wrist during the serve.

On examination, there is usually tenderness from the medial epicondyle to the pronator teres and the flexor carpi radialis muscles. Pain is elicited with resisted wrist flexion and forearm pronation, as well as passive wrist and elbow extension with forearm supination.

Standard Treatment

Standard treatment in all phases involves conservative measures similar to lateral epicondylitis. Local steroid injections may also be indicated in recalcitrant or severe cases. Surgery is rarely indicated, except in issues of ligament or tendon tears.

Manual Medicine

As with any other elbow condition, the entire kinetic chain needs to be addressed. Restrictions or imbalances in the pectoralis, scapulothoracic/shoulder, and even the low back and pelvic regions may result in poor mechanics and predispose to this injury by causing excessive wrist pronation (i.e., with racquet sports). These should be evaluated and treated using appropriate manual techniques.

Radial head dysfunction is again quite common in medial epicondylitis, as the usual mechanism of injury involves repetitive forceful wrist pronation. A posterior radial head will often result, with a supination restriction. See the manual medicine section under lateral epicondylitis for the diagnosis and treatment of this dysfunction.

Preventive Measures

Preventive measures are similar to those for lateral epicondylitis, with activity modification and flexibility and strengthening exercises that will improve biomechanics. Improved trunk and even lower extremity mechanics during sports often help prevent excessive use of the wrist for the desired result.

MEDIAL COLLATERAL LIGAMENT INJURY

In simple terms, injury to the medial collateral ligament usually occurs from repetitive abnormal transmission of force from the shoulder to the hand. If the transfer of energy is not efficient, then the ligament is overloaded and begins to fail. The approach to this injury in the athlete should always include discovering the breakdown in the mechanical link and correcting it, if possible. In situations where the ligament fails completely, a surgical approach is often needed to return the athlete to competition.

Pathophysiology

Medial collateral ligament injuries occur as a result of chronic valgus extension overload; acute injuries are typically traumatic and are rare. This injury is often associated with throwing, particularly in baseball pitchers and javelin throwers. The tremendous valgus force from

throwing stresses the ligament, which is the main valgus stabilizer, and the ligament develops microtrauma. Incomplete recovery of this damage first leads to inflammation of the ligament. Attenuation, scarring, calcification, and occasionally rupture ensues as the condition progresses.

Mechanics

Repetitive valgus stress occurs from overhead throwing. Softball pitchers do not have a similar incidence of medial collateral ligament injury because their windmill pitch mechanics avoids valgus stress. Baseball pitchers have a variety of throwing positions, or slots, in which they deliver the ball, such as overhead, three quarters, sidearm, or underarm (least common) (8).

Valgus overload is often a product of poor throwing biomechanics. Throwers who open up too soon (become front-on prematurely in the throwing motion) are at particular risk for developing this injury. Altered throwing mechanics due to restrictions in the neck, thorax, chest, and scapular region, including hypertonic latissimus dorsi and levators, as well as weakness in the scapula stabilizers and rotator cuff, may predispose to this injury (8).

Trauma from sudden valgus overload in sports like football and hockey can disrupt the fibers much like a collateral ligament injury in the knee. Immediate pain and disability are common.

History and Examination

History involves insidious onset of vague medial elbow pain, worse with activity. Symptoms are usually relieved with rest, but return with resumption of throwing at greater than 70% of normal velocity.

On examination, localized tenderness is present over the medial collateral ligament. Valgus stress with the elbow flexed to between 30 and 45 degrees reproduces the pain, and some laxity might also be noted when comparing to the contralateral side. The milking sign will be positive (8,9).

Standard Treatment

Standard treatment in the acute phase involves modification of activity, correction of technique toward proper biomechanics of throwing, ice, and analgesics as appropriate. Physical therapy usually includes local modalities and massage to the medial collateral ligament, as well as specific muscle strengthening, concentrating on the forearm flexors and pronators (4). The entire kinetic chain should be addressed to allow the best possible throwing mechanics. Treatment in the neck, trunk, scapula, and shoulder regions should be aimed at improving range of motion and strength, including scapular stabilization. A thrower with MCL injury should complete a throwing program before returning to competition. However, it should be noted that in one study, only 42% of throwing athletes has success recovering from a medial collateral ligament injury without surgery (8).

Manual Medicine

Because improper throwing mechanics often predispose to this injury, all components of the kinetic chain must be addressed. Restrictions in the neck, thorax, and scapula, including the levators and latissimus dorsi muscles should be evaluated and treated. Restricted external rotation of the shoulder may also allow increased valgus stress during the throwing motion. Dysfunctions in the elbow region may additionally alter the mechanics and result in increased stress on the ligament. The basic goal of manual treatment is to reverse any dysfunction that may compromise biomechanics and allow increase stress on the medial collateral ligament.

An elevated scapula may result from hypertonic levators, and is best evaluated by comparing with the contralateral side.

Muscle Energy: Elevated Scapula

Rationale: To reduce restrictions around the scapula (particularly the levators) to allow propet position and function in the kinetic chain.

1. The clinician positions the involved arm in 90 degrees of abduction, 45 degrees of forward flexion, and external rotation (Fig. 18.3.5).

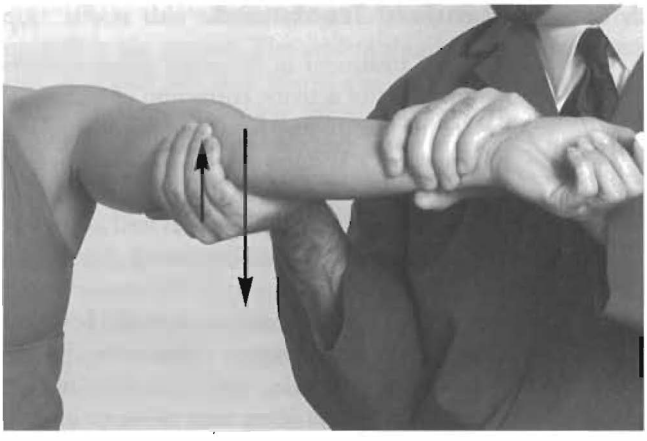

FIGURE 18.3.5. Muscle energy for an elevated scapula.

2. The athlete adducts (pushing the elbow downward) against the clinician's inferior pressure and resistance for 3 to 5 seconds.
3. Relax, reposition, repeat, and reassess.

Muscle Energy: Acromioclavicular Joint, Restricted External Rotation

Rationale: To allow greater external rotation of the shoulder, thereby decreasing the valgus load placed on the forearm during the throwing motion (10).

1. The clinician abducts the athlete's arm to 90 degrees and externally rotates to the barrier.
2. The athlete gently contracts against external rotation resistance from the clinician for 3 to 5 seconds (Fig. 18.3.6).
3. Relax, reposition, and repeat.

In the elbow region, myofascial treatments, counterstrain treatment to tender points, and muscle energy or HVLA for any dysfunctions may be performed, as described in the previous sections and throughout Chapter 18.

Preventive Measures

Preventive measures are aimed at decreasing repetitive valgus loading of the elbow. In addition to activity modification, it is important to develop strength and flexibility of surrounding muscles in the arm, as well as the neck, trunk, and shoulder region. The anconeus muscle serves as a valgus load stabilizer of the elbow, and should be addressed during rehabilitation.

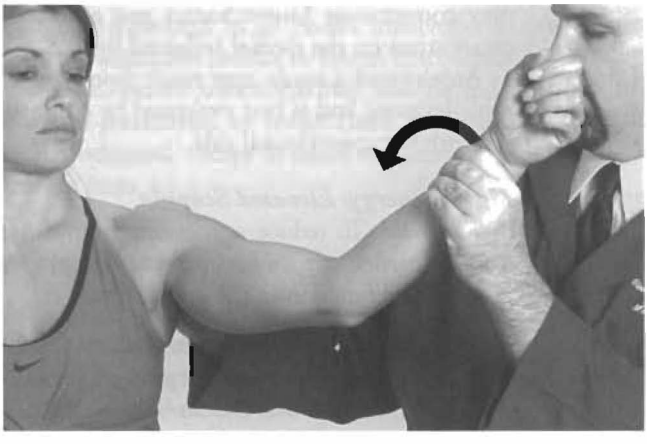

FIGURE 18.3.6. Muscle energy for restricted external rotation at the acromioclavicular joint.

Return to the level of activity prior to the injury should be very gradual.

OLECRANON IMPINGEMENT

Olecranon impingement is characterized by posterior elbow pain with clicking, locking, or occasionally crepitus in terminal extension. It typically results from repetitive valgus extension loading of the elbow, as in the throwing or serving motion, which causes the olecranon process to be forced against the medial wall of the olecranon fossa (1,3,8).

History and Examination

History typically includes pain and clicking or catching that occurs with full elbow extension. Loss of pitching or serving velocity or control is often reported. Physical examination is usually positive for tenderness and/or swelling posteriorly, and reproduced pain and clicking during full extension of the elbow. Maximum pain is usually elicited during forced extension with valgus stress. A subtle valgus laxity may also be noted. Loose bodies are occasionally palpable on examination.

Standard Treatment

Standard treatment in the acute phase involves relative rest and analgesia as appropriate. Physical therapy is aimed at improving flexibility and strength throughout the entire kinetic chain. In recalcitrant cases, surgical removal (via arthroscopy or posterior arthrotomy) of the olecranon tip, osteophytes, and loose bodies may be performed.

Manual Medicine

Due to the nature of this injury, restrictions from full extension often develop in the soft tissues in the anterior aspect of the arm, elbow, and forearm. Abduction-adduction is primarily a joint play movement at the ulnohumeral joint, which, when dysfunctional, may limit flexion-extension range of motion. In general, adduction restriction is more common than abduction. Hypertonicity in the elbow flexors, such as the biceps, may also occur, which further limits elbow extension. Joint play is an excellent choice to treat dysfunctions with medial or lateral olecranon tilt without significantly aggravating symptoms.

HVLA: Abduction-adduction Dysfunctions

Rationale: To reverse abduction-adduction restrictions in the elbow, allowing proper function of the elbow, particularly in extension.

1. The clinician grasps the athlete's proximal radioulnar region using two hands, while the athlete's hand and wrist are stabilized between the clinician's trunk and elbow.
2. The clinician translates the radioulnar region both medially and laterally, checking for restriction (Fig. 18.3.7).
3. The abduction or adduction barrier is engaged with the elbow locked in extension, and a short thrust is made in adduction or abduction, as appropriate. Slight flexion is occasionally helpful to avoid extension locking of the elbow.
4. A slight variation may be done, where the adduction-abduction barrier is engaged, and the elbow is then thrusted into full extension.

Muscle Energy: Restricted Ulnar Extension

Rationale: To improve elbow extension by reducing restrictions in the elbow flexors.

1. With the elbow supported and held in supination, the clinician engages the extension barrier (Fig. 18.3.8).
2. The athlete resists an isometric resistive effort, holding for 3 to 5 seconds.
3. Relax, reposition, repeat, and reassess.

Preventive Measures

Preventive measures most importantly involve minimizing repetitive valgus extension loading of the elbow. Proper biomechanics in throwing and serving is critical. Maintenance of strength and flexibility throughout the entire kinetic chain is extremely important in achieving ideal biomechanics.

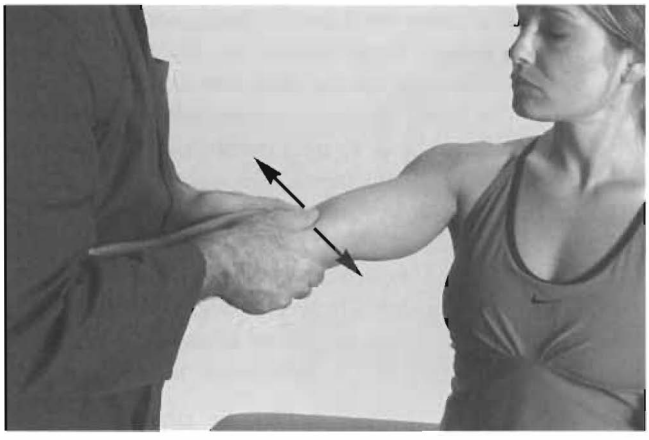

FIGURE 18.3.7. High-velocity, low-amplitude technique for abduction or adduction dysfunction.

PRONATOR TERES SYNDROME

The pronator teres syndrome involves entrapment of the median nerve between or just distal to the two heads of the pronator teres muscle. Symptoms are typically sensory, with pain, paresthesias, and decreased sensation in the median nerve distribution. Impaired pronation and grip are quite rare. This condition is often misdiagnosed as carpal tunnel syndrome, so the history and examination need to be performed carefully (1,3).

Athletes at risk for carpal tunnel syndrome (i.e., those who use racquets) are also prone to pronator teres syndrome.

History and Examination

History typically includes some form of repetitive use of the forearm in pronation, though often no mechanism is identified. The previously mentioned sensory symptoms in the median nerve distribution are usually teported. The athlete may complain of soreness or cramping in the forearm or the medial epicondylar region where the muscle originates, but this is less common.

On examination, symptoms are often reproduced with resisted forearm pronation, or resisted flexion of the flexor digitorum superficialis (FDS) tendon of the long finger. Symptoms may also be elicited by compressing the median nerve at the arch of the FDS origin.

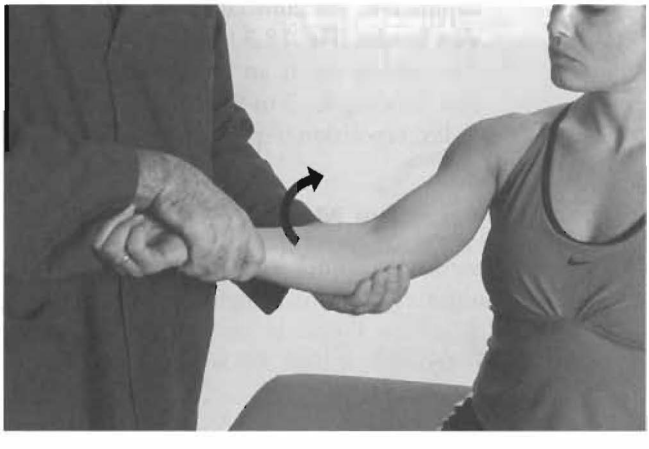

FIGURE 18.3.8. Muscle energy for restricted ulnar extension.

Standard Treatment

Standard treatment in the acute phase is conservative, with activity modification to avoid offending motions. A splint is often used for 3 to 6 weeks. Physical therapy, including stretches, soft tissue treatments, ultrasound, and other modalities may be helpful in relieving some of the restrictions in the pronator teres muscle. Surgical intervention is typically reserved for recalcitrant cases.

Manual Medicine

Because this condition involves entrapment of a nerve between the two heads of the pronator teres muscle, manual treatment should be aimed at relieving some of the restrictions in this region. Muscle energy and myofascial techniques are both very appropriate means of accomplishing these goals.

Muscle Energy: Restricted Elbow Supination

Rationale: To relieve restrictions in the forearm pronators, particularly to lengthen the pronator teres muscle in order to decrease tension on the medial nerve.

1. The clinician stabilizes and flexes the athlete's elbow to 90 degrees.
2. The clinician assesses and engages the supination barrier with comparison to the contralateral side (Fig. 18.3.9).
3. The athlete then provides gentle isometric resistance against supination for 3 to 5 seconds.
4. Relax, reposition, and repeat.

Myofascial Release. These manipulations would target the muscle belly and the overlying fascia. If the etiology of peripheral neuropathic symptoms to the hand is unclear, myofascial release can help differentiate between the pronator teres or the carpal tunnel as the source of neural compression. Restriction or dysfunction should be palpable in the pronator teres if it is hypertonic or inflamed to the point where it traps the median nerve. As myofascial release is performed, symptoms may be precipitated initially before the tissue relaxes. If there is no trigger point, hypertonicity, restriction, or positive response to myofascial release, the entrapment is less likely to be at the pronator teres than the carpal tunnel (7).

The technique may be performed in the same general manner described in the manual medicine section under lateral epicondylitis. Special focus should be made on relieving the myofascial restriction in the pronator teres muscle.

Preventive Measures

Preventive measures include activity modification to avoid excessive use of the forearm pronators. As in other conditions, particularly medial epicondylitis, repetitive forearm use in

FIGURE 18.3.9. Muscle energy for restricted elbow supination.

pronation should be avoided; flexibility in this region should be maintained. Ergonomics are important as well.

STRETCHES FOR THE WRIST AND FOREARM

Volar Forearm (includes flexor carpi radialis, flexor carpi ulnaris, flexor digitorum superficialis and profundus, palmaris longus). The athlete turns the right palm upward toward the ceiling, keeping the right elbow fully extended. Position the left hand on the right fingers and bend the wrist toward the floor (Fig. 18.3.10A). This is useful for golf, hand/racquetball, archery, and discus throw.

Dorsal Forearm (includes extensor carpi radialis longus and brevis, extensor digitorum, extensor

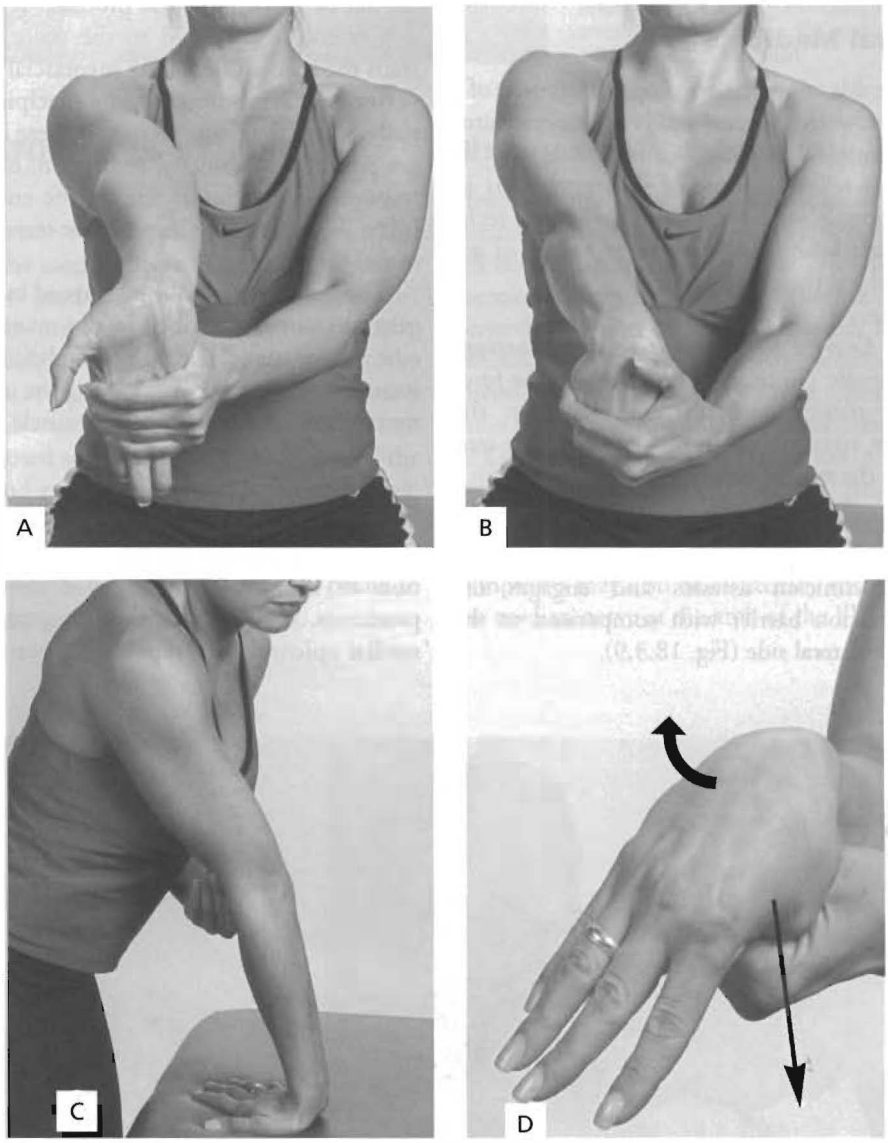

FIGURE 18.3.10. Stretches for the elbow and forearm. **A,** Volar forearm. **B,** Dorsal forearm. **C,** Pronator teres-quadratus. **D,** Extensor pollicis longus, extensor pollicis brevis, abductor pollicis longus.

indicis, extensor digiti minimi). The athlete makes a fist with the right palm down, keeping the right elbow extended. Bend the right wrist toward the floor, then ulnar-deviate the right wrist, noting tension on the dorsal right forearm. Reinforce this stretch by placing the left hand on the right hand and ulnar-deviating the right wrist (Fig. 18.3.10B). These stretches are useful for tennis, baseball/softball, hand/racquetball, discus throw, and archery. A variation of this can be done with the hand against the wall.

Pronator Teres Muscle/Quadratus Muscle. The athlete's right palm is on the table with the elbow flexed. The forearm should be turned outward. The left hand should be positioned on the proximal forearm of the right extremity. The left hand is used to straighten/extend the right elbow (Fig. 18.3.10C). Stretch should be felt in the volar/anterior aspect of the forearm.

Extensor Pollicis Longus, Extensor Pollicis Brevis, Abductor Pollicis Longus. The athlete sits or stands with the right forearm pronated and the elbow flexed. The elbow may be supported on the table. Use the left hand to bend the thumb toward the little finger, then ulnar-deviate the wrist (Fig. 18.3.10D). A stretch should be felt along the radial side of the forearm.

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19

THE WRIST AND HAND

19.1

Anatomy

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WILLIAM M. FALLS

Osteology of the wrist and hand is complex, and designed to maximize manipulative ability. The intricacy of the articulations sacrifices weight-bearing power in favor of fine motor manipulation and prehensile strength. Twenty-nine bones articulate with each other to form the distal radioulnar joint DRUJ), radiocarpal joint, intercarpal joints, carpometacarpal (CMC) joints, metacarpophalangeal (MCP) joints, and interphalangeal joints (Fig. 19.1.1). Anatomy of the wrist and hand is presented in detail in major anatomic textbooks (1–9).

The distal radius and ulna articulate, forming the most proximal joint of the wrist, the DRUJ. Pronation and supination of the hand are permitted at this synovial joint. The distal ulna rests in the ulnar notch of the radius, allowing the radius to swivel about the ulna, which is fixed by its proximal articulation with the humerus by the triangular fibrocartilage complex (TFCC). The TFCC attaches medially to the ulnar styloid process and laterally just distal to the ulnar notch of the radius. This contributes to the joint capsule and allows the joint to pivot. The fibrocartilaginous disc of the TFCC fills a hollow in the distal ulna (ulnar fovea). This contributes to the ulnar proximal articular surface. A superior redundancy in the synovial membrane of the articular capsule forms the sacciform recess. This connective tissue tether stretches as the radius pivots about the ulna. Transverse bands of the palmar and dorsal radioulnar ligaments complete the articular capsule of the DRUJ (4).

The radiocarpal joint includes the articulation of the distal radius and articular disc of the DRUJ with the proximal carpal row. These bones include the scaphoid, lunate, and triquetrum, with the pisiform, a sesamoid bone, anterior to the triquetrum. This complex synovial condyloid joint permits flexion and extension, circumduction, and radial-ulnar deviation of the wrist. The proximal carpal row glides as a unit within the axial concavity of the DRUJ. The carpals translate anteriorly to permit wrist extension, and posteriorly to allow wrist flexion. Radial and ulnar deviation are permitted by lateral and medial glides. Circumduction is a combination of these motions. The articulations of the distal carpal row (trapezium, trapezoid, capitate, and hamate) combine anterior and posterior glide on the proximal carpal row to contribute to full wrist flexion and extension.

Palpation of each carpal bone is performed using surface landmarks. The pisiform carpal bone is most prominent, and can be palpated on the medial volar surface of the wrist at the proximal flexion crease. The hook of the hamate bone is less easily palpated just distal to the pisiform, deep to the hypothenar eminence.

Proceeding laterally on the volar wrist, a depression is noted between the pisiform and hamate. The carpal bones form the floor and sides of the carpal tunnel, an arch with a volar concavity that houses the tendons of the finger and thumb flexors and the median nerve (Fig. 19.1.2). The transverse carpal ligament

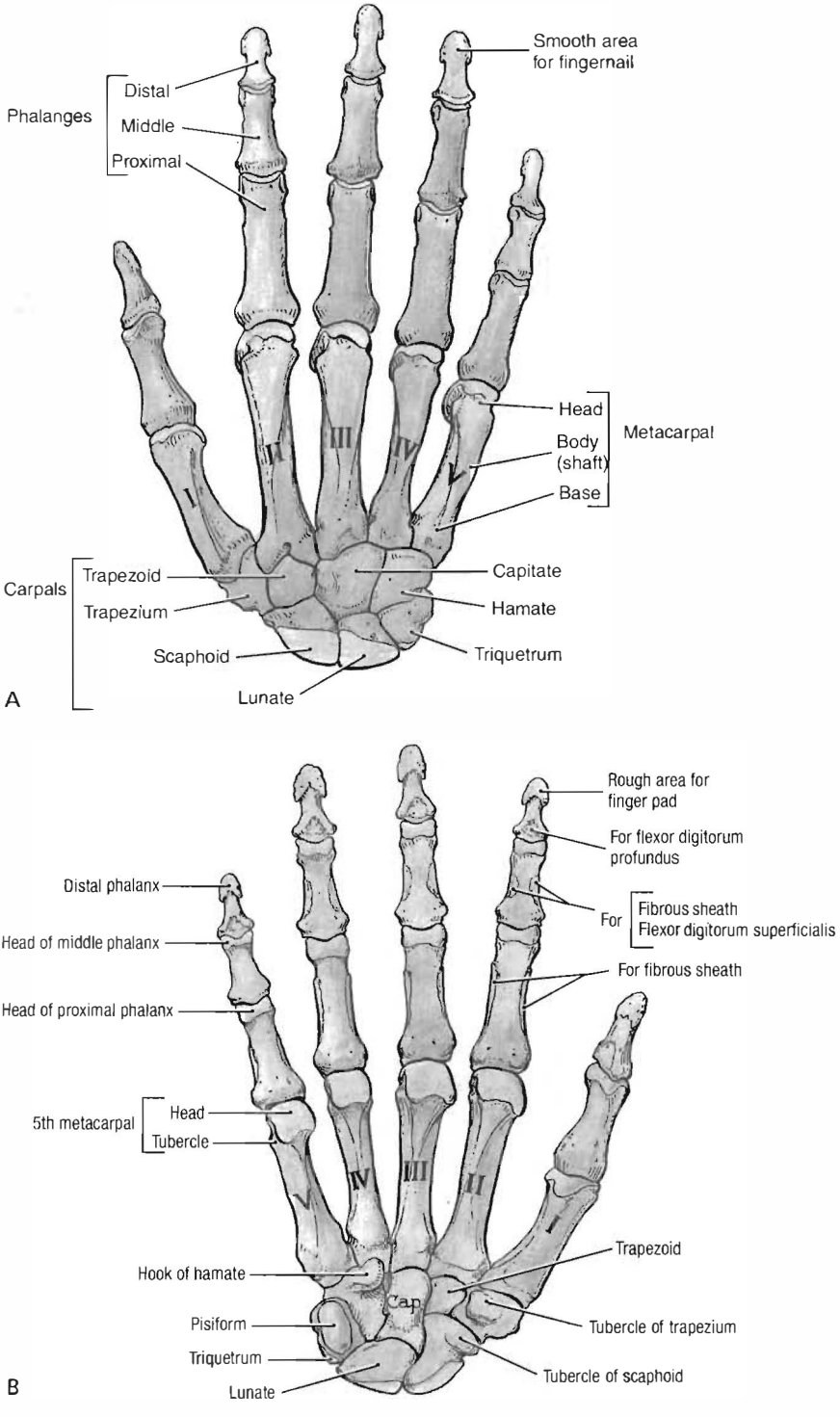

FIGURE 19.1.1. A, Bony anatomy of the hand, dorsal view. **B,** Bony anatomy of the hand, palmar view. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

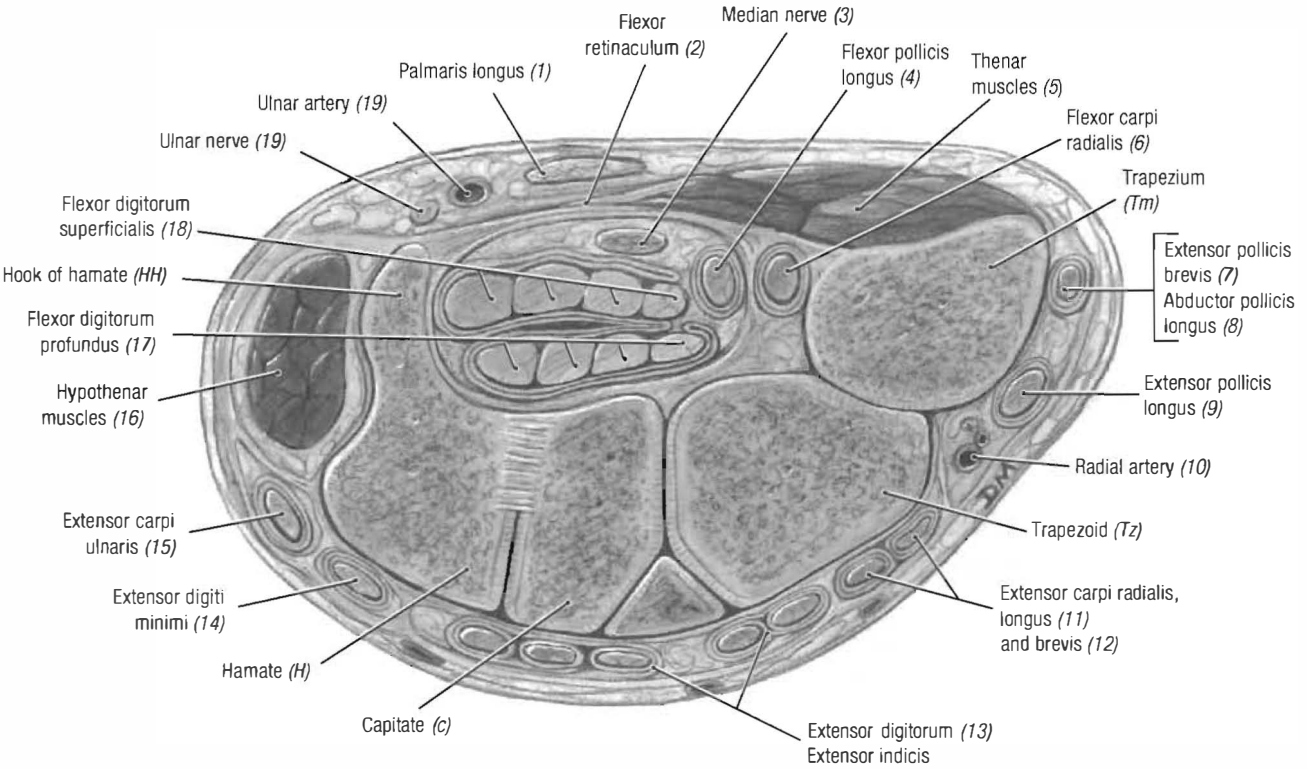

FIGURE 19.1.2. Section through the distal carpal tunnel. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

forms the roof by attaching to the pisiform and hamate medially and the scaphoid and trapezium laterally. The trapezium is palpated at the base of the thumb, distal to the distal volar flexion skin crease of the wrist.

Landmarks of the lateral wrist include the anatomic snuffbox, a small depression between two prominenr tendons where the scaphoid bone can be palpated dorsolarerally (Fig. 19.1.3). The medial dorsum of the wrist is marked by a small, visible depression where the lunate can be palpated as it articulates with the radius. The capitate bone is palpated just distal to the lunate during wrist flexion.

Much of the stability of the wrist is provided through the numerous ligaments securing the carpal bones to the DRUJ. These include the palmar radiocarpal and ulnocarpal ligaments, dorsal radiocarpal and ulnocarpal ligaments, and radial and ulnar collateral ligaments. Intercarpal ligaments permit gliding of the carpals upon each other as described earlier (2).

Continuing distally, the CMC joint of the thumb can be palpated distal to the radial styloid. Because of their limited motion, the CMC joints of the fingers are less easily palpated, but are located across the midpalm. These condyloid joints allow very little motion, with the exception of the CMC joint of the thumb. This is a saddle joint, named for the concavity of the trapezium as it articulates with the first metacarpal bone. The saddle joint permits opposition, circumduction, flexion, and abduction of the thumb. Without this versatile articulation, prehension and fine manipulation would not be possible.

The MCP joints ate the most proximal joints of the digits. They are formed by the articulation of the bases of the proximal phalanges with the metacarpal bones. These hinge joints allow flexion and extension of more than 90 degrees, and small medial and lateral movements that permit abduction of the digits away from the third digit, or middle finger. Radial (lateral) and ulnar (medial) collateral ligaments support the MCP joints.

Extrinsic muscles that are prime movers of the wrist and hand originate at the elbow. Muscles that act to move the wrist arise from the medial and lateral epicondyles of the humerus and forearm. Long tendons, with few exceptions, skip the carpals and attach these fusiform muscles to the proximal metacarpals (Fig. 19.1.4). The flexor carpi ulnaris (FCU) is the exception. It inserts on the pisiform bone, at the hook of the hamate bone, and finally on the base of the fifth metacarpal bone. The flexor carpi radialis (FCR) inserts on the anterior aspect of the base of the second metacarpal bone, the distal flexor retinaculum, and into the palmar aponeurosis. The extensor carpi radialis longus (ECRL) inserts on the posterior aspect of the base of the second metacarpal; the extensor carpi radialis brevis (ECRB) inserts onto the third metacarpal bone; and the extensor carpi ulnaris (ECU) inserts onto the base of the fifth metacarpal bone. More information on the compartments and proximal attachments of these muscles can be found in Chapter 18.

The five muscles that are primary movers of the wrist—the FCU, FCR, ECRB, ECRL, and ECU—work in synchrony. If all five muscles contract equally, they increase the stability of the wrist by compressing the intracarpal articulations, and the radiocarpal articulation (1). Wrist flexion is the action of both FCU and FCR rogether. Wrist extension is the action of the ECRL, ECRB, and ECU working evenly together. Radial and ulnar deviation require shortening of the corresponding pair of muscles, and circumduction of the wrist requires coordination of all five muscles shortening and lengthening progressively. The FCR has a close association with the carpal tunnel, but traverses the wrist in its own compartment along the radial side of the carpal tunnel. Guyon's canal houses the ulnar nerve and is found in a groove formed along the medial aspect of the triquetrum-pisiform joint proximally, and medial to the hook of the hamate bone distally in the wrist.

The extrinsic forearm finger muscles and intrinsic hand muscles perform the motion of these joints. The tendons of the extrinsic finger flexors, the flexor digitorum superficialis (FDS) and the flexor digirorum profundus (FDP), traverse the wrist in close proximity through the

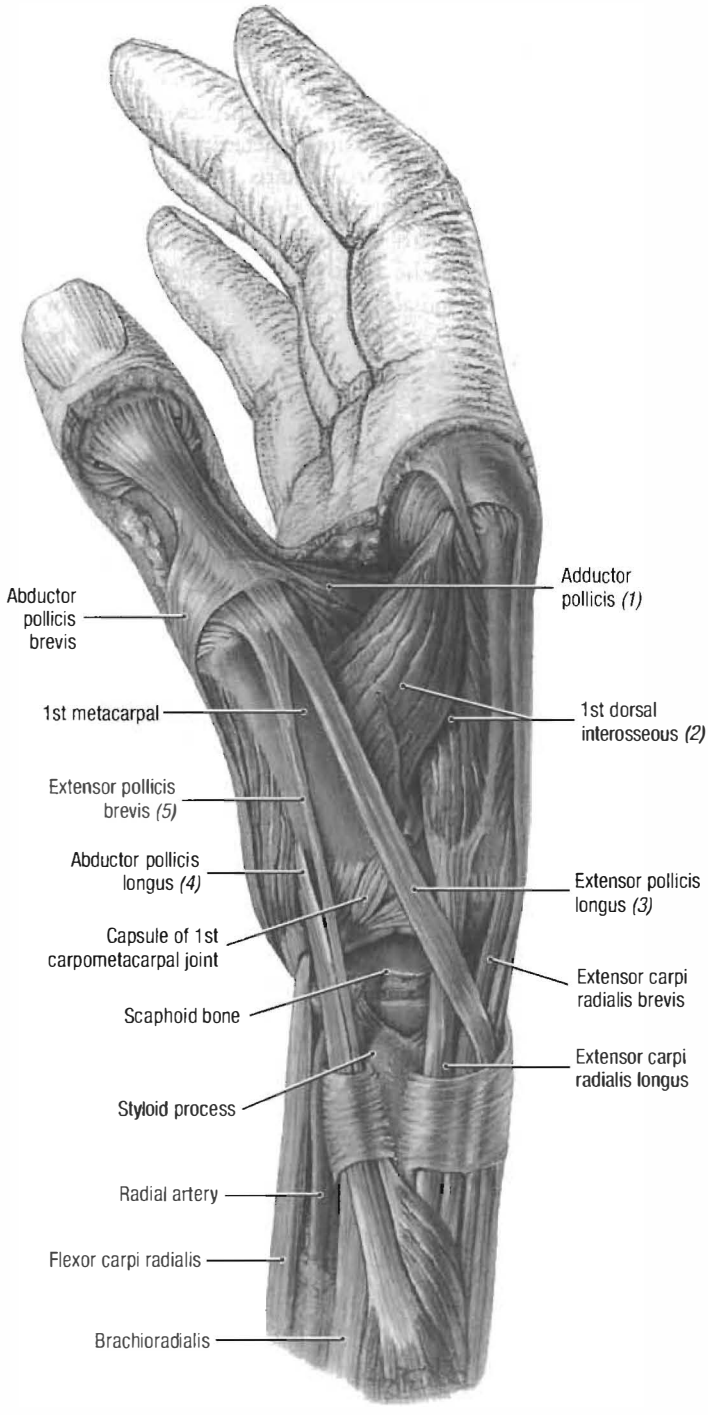

FIGURE 19.1.3. Wrist and hand, lateral view. Note the location of the snuffbox, between the extensor pollicis longus and the paired abductor pollicis longus and extensor pollicis brevis tendons, over the scaphoid bone. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

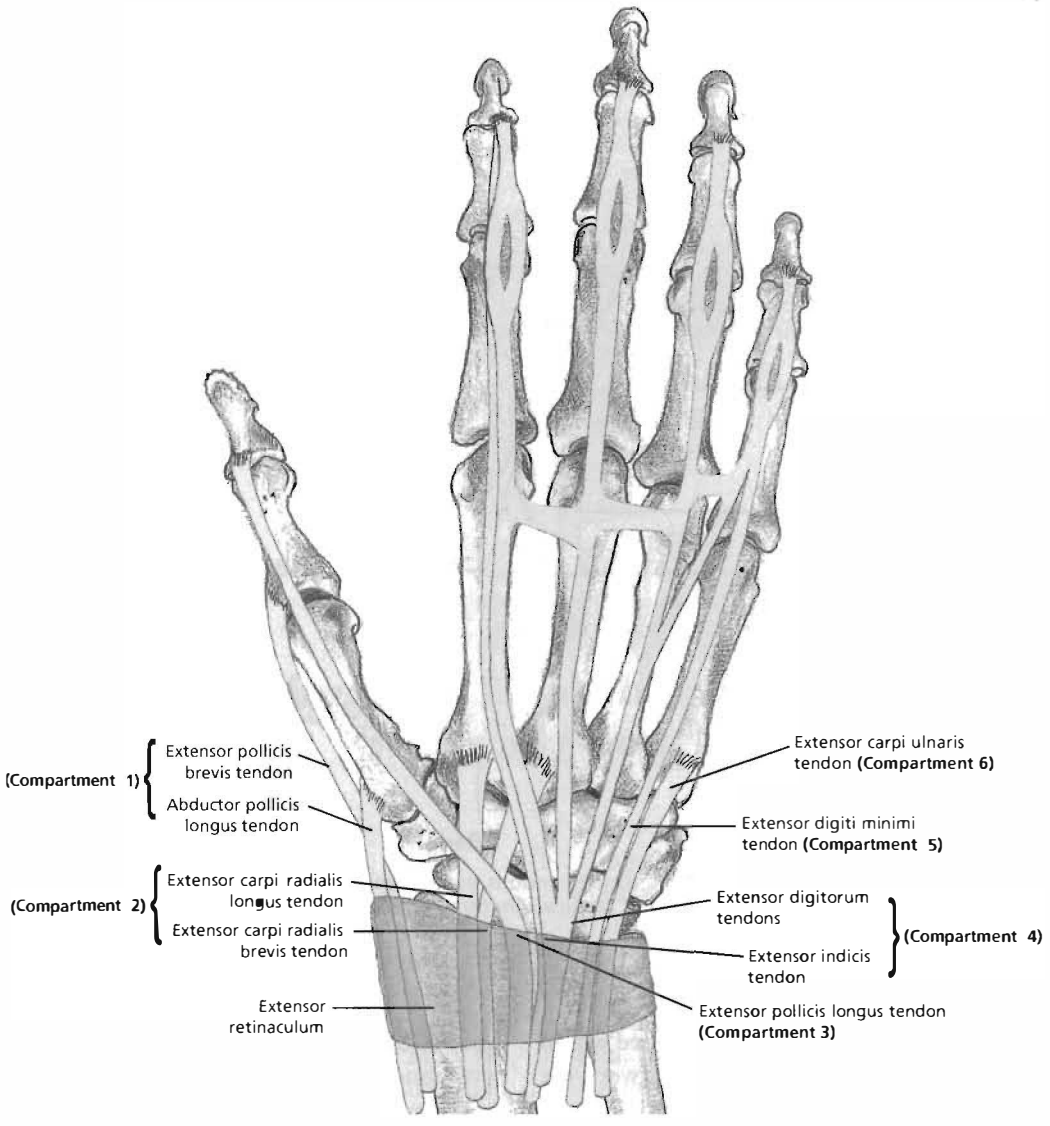

FIGURE 19.1.4. Extensor tendons of the hand and wrist, dorsal view. (From Stoller DW. *MRI, Arthroscopy, and Surgical Anatomy of the Joints.* Baltimore: Lippincott Williams & Wilkins, 1999.)

carpal tunnel (Fig. 19.1.5). The FDP lies deep, and the FDS more superficial, with the tendons to each digit encased in synovial sheaths that begin just proximal to the MCP joints, and run to the points of attachment. There is an interruption of the synovial sheath in the distal palm, and then it continues proximally through the carpal tunnel to the proximal borders of the flexor retinaculum. The sheath of the small finger is continuous with the more proximal common flexor sheath. The extrinsic finger extensors—the extensor digitorum communis (EDC), extensor indicis proprius, and extensor digit minimi—divide into the central and collateral slips or bands to form a complex extensor hood.

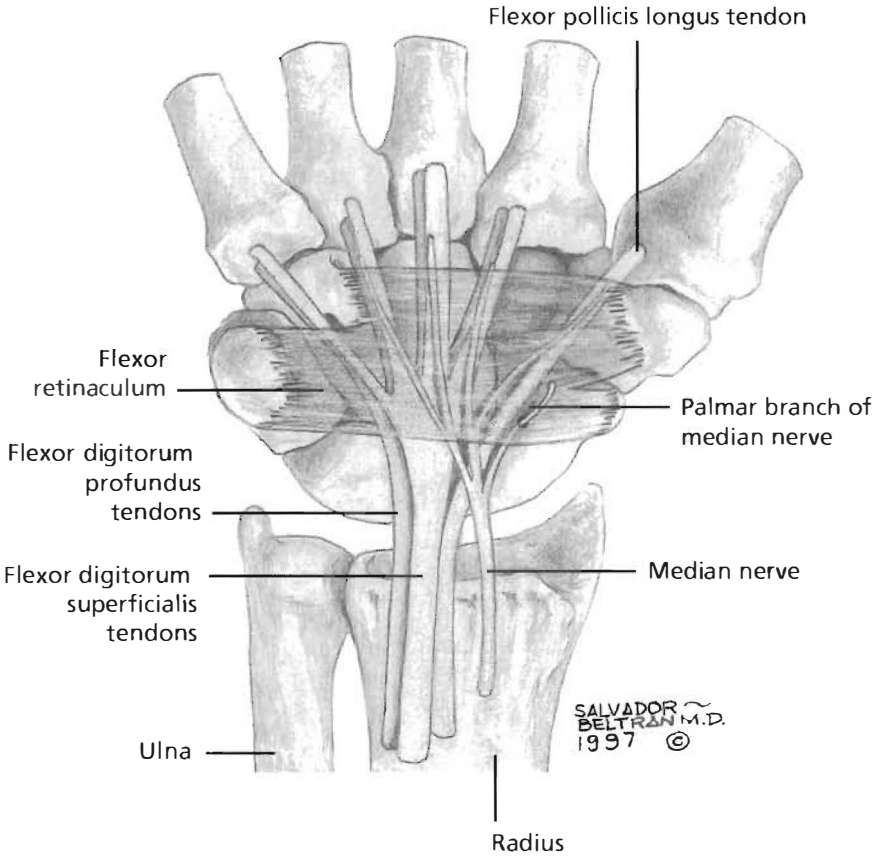

FIGURE 19.1.5. The flexor digitorum tendons in relation to the median nerve. (From Stoller DW. *MRI, Arthroscopy, and Surgical Anatomy of the Joints.* Baltimore: Lippincott Williams & Wilkins, 1999.)

The intrinsic hand muscles are housed within the confines of the hand, arranged between the metacarpal bones. The interossei muscles insert laterally and medially onto the proximal dorsal hood to perform finger adduction and abduction. The lumbrical muscles lie parallel to the interossei along the metacarpal bones. The proximal attachment of these muscles is along the FDP tendon; the distal attachment is just distal to the proximal interphalangeal (PIP) joints on the lateral border of the middle phalanx of digits 2 through 5. These muscles act to assist in full extension of the fingers at the PIP joints. Innervation is split between the first and second lumbricals by the median nerve (C8, T1) and the deep branch of the ulnar nerve (C8, T1). The deep branch of the ulnar nerve innervates the interossei muscles.

The wrist and hand are supplied by the radial and ulnar arteries. The two arteries have both deep and superficial branches (7). The wrist receives its blood supply from the anterior and posterior interosseous arteries, which are distal continuations of the brachial artery. The superficial palmar arch is primarily supplied by the ulnar artery (5,7). The deep palmar arch is formed by the deep palmar branch of the ulnar artery and the radial artery and is also located at the midpalmar aspect of the wrist and is distal to the carpal tunnel.

Nerves that supply the DRUJ are the anterior branch of the median nerve (C6-C7) and

the posterior interosseous branch of the deep radial nerve (C7-C8) (6).

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19.2

Physical Examination

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OBSERVATION

Evaluation of the wrist and hand starts when the examiner enters the room. It is important to note the manner in which the athlete is holding the hand and whether or not he or she can shake hands. Flexor tendon injuries are easily noted because the injured digit will not be maintained in its natural flexed position. Although rudimentary, it is always important to count all digits and note any previous amputations.

Palmar Surface

The palmar, or volar, aspect of the hand and wrist has multiple creases that are located where the underlying fascia attaches to the skin. The four important creases are as follows (Fig. 19.2.1):

1. The *distal palmar crease* marks the volar location of the metacarpophalangeal (MCP) joints.
2. The *proximal palmar crease* lies in the midmetacarpal area and is found just proximal to the distal palmar crease.
3. The *proximal interphalangeal crease* is located at the proximal interphalangeal (PIP) joint and marks the distal aspect of no man's land.
4. The *thenar crease* denotes the thenar eminence.

Note the "attitude" of the hand, that is, its natural position when relaxed (Fig. 19.2.2). The MCP and interphalangeal (IP) joints should have slight flexion at rest, and the fingers should line up parallel to each other and pointing toward the thenar and hypothenar eminences. Any significant discrepancy could mean a tendon injury or a contracture held in flexion with the fingers directed toward the thenar eminence (1,2). The normal appearance of each eminence has a prominence, which is important to note because atrophy of either eminence is representative of nerve damage to the muscle

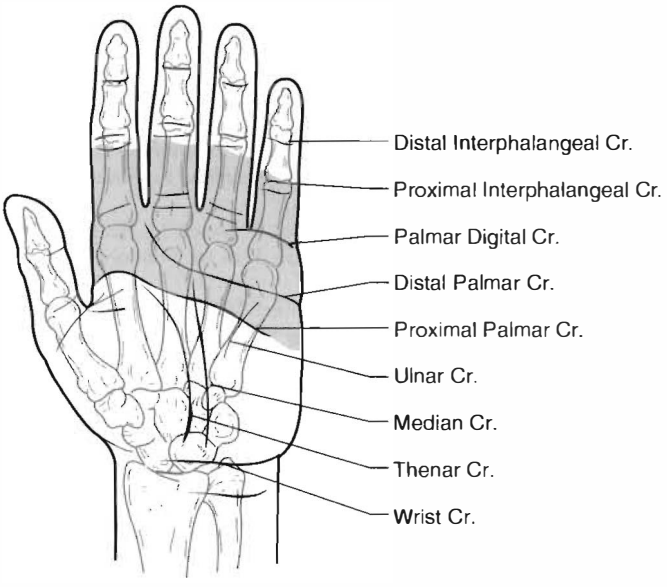

FIGURE 19.2.1. Palmar view of the hand with the four main creases: distal palmar, proximal palmar, proximal interphalangeal, and thenar crease.

bellies that control the thumb (median nerve) and little finger (ulnar nerve) (1,3).

The skin of the volar surface is usually thicker than the dorsal side because it is the working side of the hand. The volar skin is fixed with fascia, which binds it with the structures beneath it at the creases. This fixation is important because it allows for objects to be held securely by the hand.

The skin on the fingers is connected to bone with septa and small ligaments running from skin to bone along the lateral and medial sides of the fingers (Cleland's and Grayson's ligaments) (4). This arrangement results in little rotatory movement of the skin around the fingers.

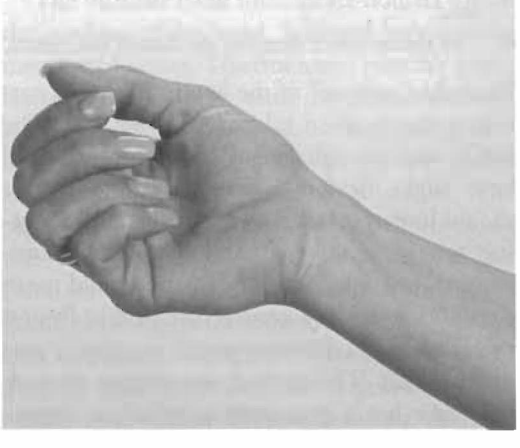

FIGURE 19.2.2. Attitude of the hand, noting the natural flexion at the metacarpophalangeal and proximal interphalangeal joints.

Flexor Zones (Fig. 19.2.3)

Zone I: The profundus tendon exists on its own and inserts at the proximal aspect of the distal phalanx.

Zone II: The profundus tendons become volar to the superficialis tendons. The profundus tendon will emerge through the superficialis tendons at the level of the MCP joint. The superficialis tendons bifurcate and then insert on the midaspect of the middle phalanx.

Zone III: The lumbricals originate from the profundus tendon in this zone.

Zone IV: The median nerve and the nine flexor tendons run through the carpal canal.

Zone V: The flexor tendons begin in the distal aspect of the forearm at the musculotendinous junction. The superficialis tendons lie volar to the profundus tendons.

Dorsal Surface

The skin on the dorsal aspect of the hand is more mobile, primarily to allow for MCP joint

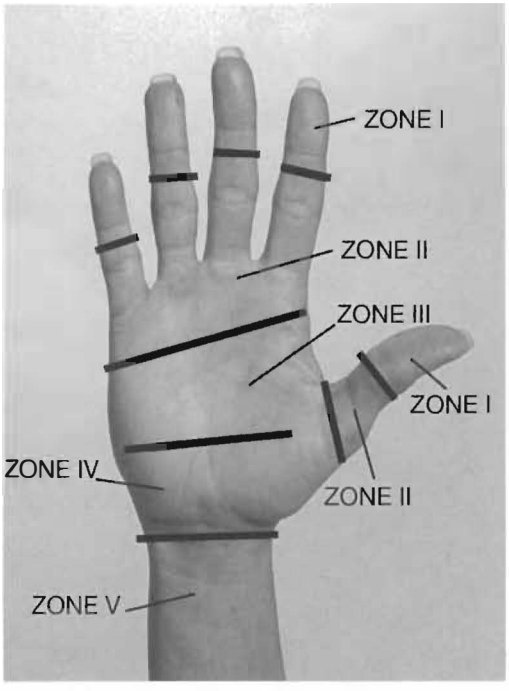

FIGURE 19.2.3. Palmar zones of the hand.

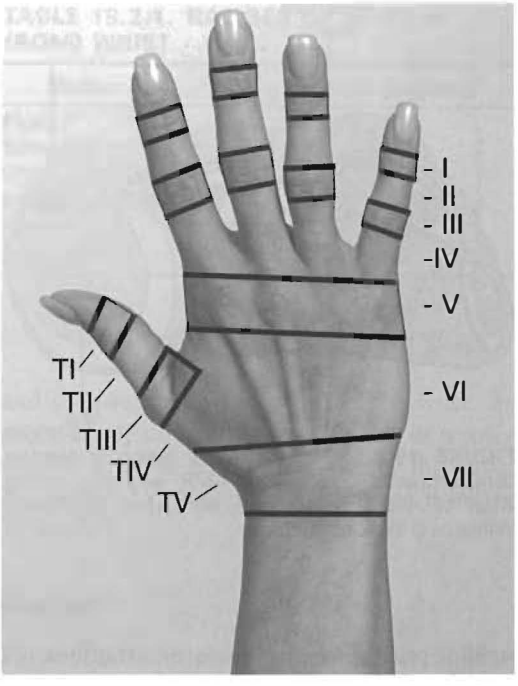

FIGURE 19.2.4. Dorsal aspect of the hand with extensor tendon zones.

flexion. The knuckles on the dorsal aspect of the hand are usually most prominent, the middle one more so. When an athlete clenches a fist, the fifth knuckle will become less prominent. The valleys between each knuckle should all be symmetrical. Any asymmetry, for example, as a result of swelling, is indicative of an injury (1,4).

Inspection of the nails is also important. The normal nail bed should have a white base (the lunula) with the remainder of the bed being pink. Observation of the nail is important because a nail bed that is lighter in color could be indicative of anemia. Nails that have ridges or are brittle could be a result of malnutrition. Clubbing of the fingertips and nails may indicate pulmonary disease.

Extensor Tendon Zones

As with the volar aspect of the wrist and hand, the extensor side is subdivided into zones and compartments for the purpose of describing extensor tendon injuries (Fig. 19.2.4). The six dorsal extensor tendon compartments are as follows (Fig. 19.2.5) (1,4,5):

Compartment	
1	Abductor pollicis longus, extensor pollicis brevis
2	Extensor carpi radialis longus and brevis
3	Extensor pollicis longus
4	Extensor carpi digitorum communis, extensor indicis proprius
5	Extensor digiti minimi
6	Extensor carpi ulnaris

PALPATION

Palpation of the wrist bones starts at the radius and ulna and proceeds distally. Note tenderness on the radial and ulnar styloids (Fig. 19.2.6). The indentation at the distal aspect of the radial

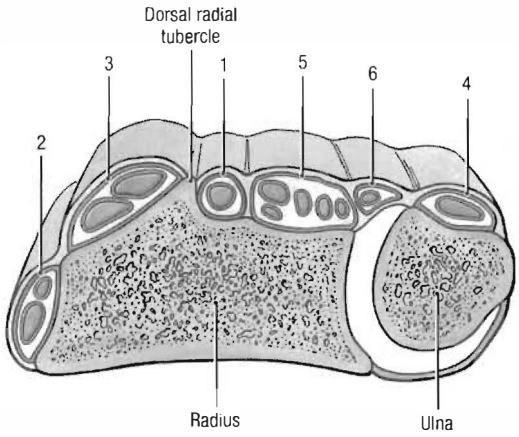

FIGURE 19.2.5. The six dorsal extensor tendon compartments. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

styloid process is the anatomic snuffbox. A branch of the radial artery can be palpated within the snuffbox, but any tenderness located in the area could represent a scaphoid fracture (1,4,6). The scaphoid is the most commonly fractured carpal bone, and depending on the location of the fracture, healing can be difficult due to recurrent blood flow. It is the most radial of the proximal carpal row. Palpation is facilitated by placing the wrist into ulnar deviation (Fig. 19.2.7).

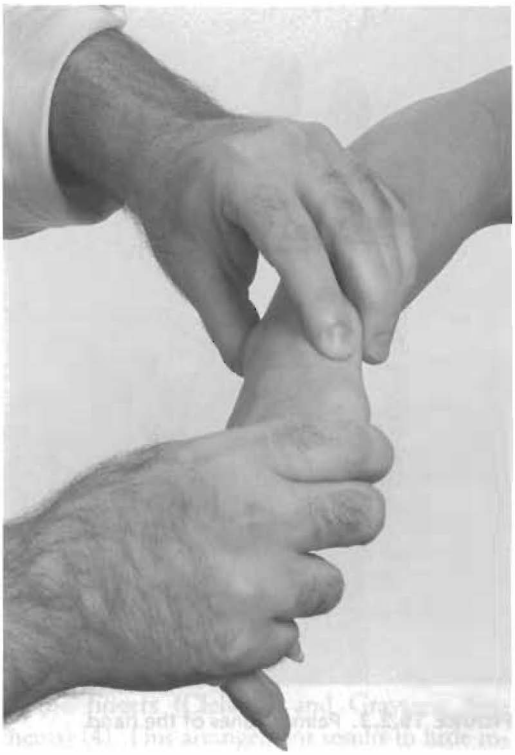

FIGURE 19.2.7. Snuffbox with ulnar deviation to palpate the scaphoid.

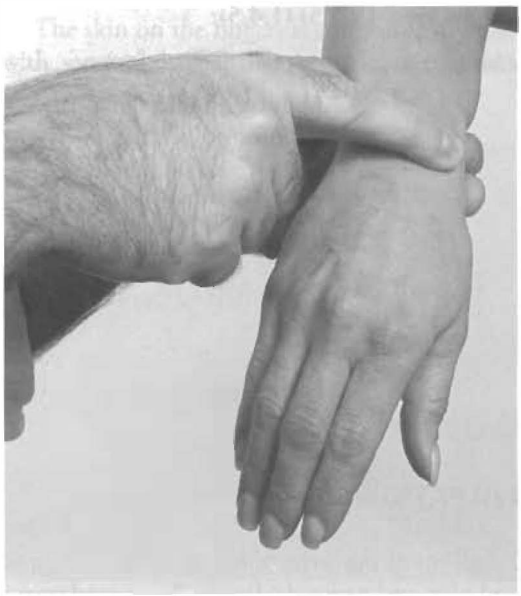

FIGURE 19.2.6. Palpation of the radial styloid.

Examine the proximal carpal row carefully (scaphoid, lunate, triquetrum, pisiform). The distal row includes the trapezium, which can be easily palpated by moving distal to the anatomic snuffbox and asking the athlete to flex and extend the thumb. The trapezoid, capitate, and hamate round out the row. The hamate hook can be easily palpated by placing the IP joint of the thumb on the pisiform and then rolling the thumb forward (Fig. 19.2.8). The hamate is important because it forms the ulnar border of Guyon's canal, which contains the ulnar nerve and artery (7).

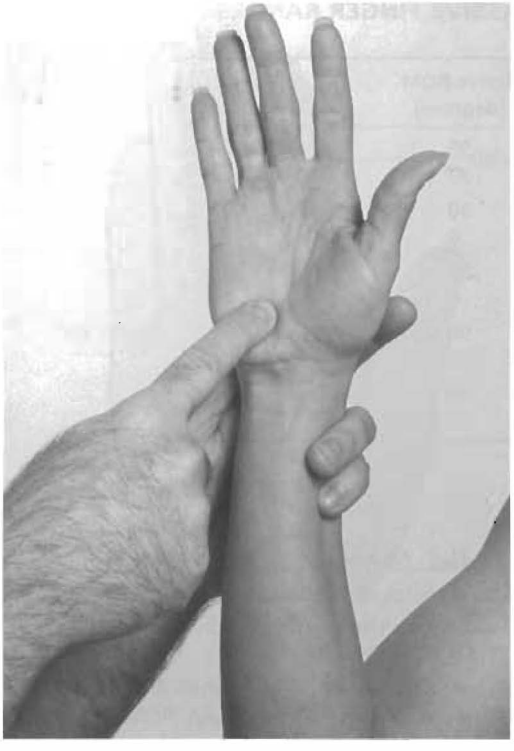

FIGURE 19.2.8. Hook of the hamate.

RANGE OF MOTION

Wrist

The wrist range of motion (ROM) should be evaluated by assessing both passive and active ranges before continuing the examination. Significant loss of motion from trauma may need radiographs to rule out unstable fractures. Bilateral comparison is essential to evaluate for any unilateral restrictions. Ranges of motion move in three planes as follows:

1. Flexion and extension.
2. Ulnar and radial deviation.
3. Supination and pronation (this motion actually emanates from the elbow, not the wrist).

To assess passive motion, the examiner isolates the wrist joint by holding the forearm with one hand and using the other hand to manipulate the wrist. At the same time, he or she puts the wrist into radial and ulnar deviation and then supinates and pronates the wrist. Be aware of any discrepancies in (ROM) or restrictions that could reflect an injury to the wrist. Normal passive ranges of motion are listed in Table 19.2.1.

TABLE 19.2.1. RANGES OF MOTION (ROM) WRIST

Motion	ROM (degrees)
Flexion	80
Extension	70
Ulnar deviation	30
Radial deviation	20
Supination	90
Pronation	90

Finger

Finger motion involves three joints per digit, so the examiner should look at all of them. To grossly assess active finger flexion and extension, ask the athlete to make a fist while observing the finger motion. Make sure the fingers are all moving together throughout the range of motion. When all joints of the fingers are completely flexed, the fingers should be at the level of the distal flexion crease.

The specific ranges of motion that need to be evaluated are as follows:

1. Finger and thumb flexion and extension at the MCP joint.
2. Finger and thumb flexion and extension at the IP joints.
3. Finger and thumb abduction and adduction at the MCP joint.
4. Finger and thumb opposition.

Normal ranges of motion for the finger are listed in Table 19.2.2.

To examine the passive motion of the fingers, the examiner grasps the hand at the distal metacarpal using his or her index finger and thumb to stabilize each joint. The mobilizing hand then moves the digit through its ROM. To test MCP flexion and extension, the examiner grasps the metacarpal with the stabilizing

TABLE 19.2.2. ACTIVE AND PASSIVE FINGER RANGES OF MOTION (ROM)

Joint	Motion	Active ROM (degrees)	Passive ROM (degrees)
MCP	Flexion	95	100
	Extension	30	35
PIP	Flexion	90	100
	Extension	0	10
DIP	Flexion	90	90
	Extension	0	20
Thump IP	Flexion	90	90
	Extension	20	20
Thumb MCP	Flexion	50	50
	Extension	10	10

hand and the proximal phalanx with the mobilizing hand. The MCP joint has some lateral movement when in extension, but no lateral movement when in flexion because the collateral ligaments are loose in extension and tight in flexion. This becomes an important factor when the hand is placed into a cast that extends beyond the MCP joint. The cast should maintain MCP joint flexion in order to prevent collateral ligament tightness.

To evaluate flexion and extension of the PIP joint and the distal interphalangeal (DIP) joint, the examiner again uses his or her stabilizing hand just proximal to the joint and then uses the other hand distal to the joint. Abduction of the fingers is a motion that occurs through the MCP joint.

Evaluate finger abduction and adduction by putting the fingers in extension, then stabilizing the target joint and moving it through its range of motion. Thumb abduction and adduction is a function of the carpometacarpal (CMC) joint, so the examiner stabilizes proximal to the joint and then moves the thumb away from the palm (abduction) and toward the palm (adduction).

NEUROVASCULAR EXAMINATION

Sensory Testing

The wrist and hand sensorium is supplied by the radial, median, and ulnar nerves.

Radial Nerve. The radial nerve innervates the dorsum of the hand and wrist on the radial side, from the thumb to the third metacarpal (1,8).

Median Nerve. The median nerve innervates the radial volar border of the wrist as well as the volar aspects of the thumb, index, and middle fingers. The most specific area of innervation by the median nerve is the volar aspect of the index finger (1,8).

Ulnar Nerve. The ulnar nerve innervates the volar ulnar aspect of the hand, specifically the ring finger and the little finger. The purest area of innervation for the ulnar nerve is the volar tip of the little finger (Fig. 19.2.9) (1,8).

Dermatomal Distribution

C6: Dorsal aspect of the thumb and volar aspect of the thumb and the thenar eminence.

C7: Dorsum of the first web space, the index and middle fingers, and from the ulnar border of the thumb to the index and middle fingers and the skin overlying their metacatpals.

C8: Dorsal and volar sides of the ring and little fingers and the ulnar aspect of the palm (9,10).

Motor Testing

For gross strength testing, the grip test is simple and telling. Note the force applied in the

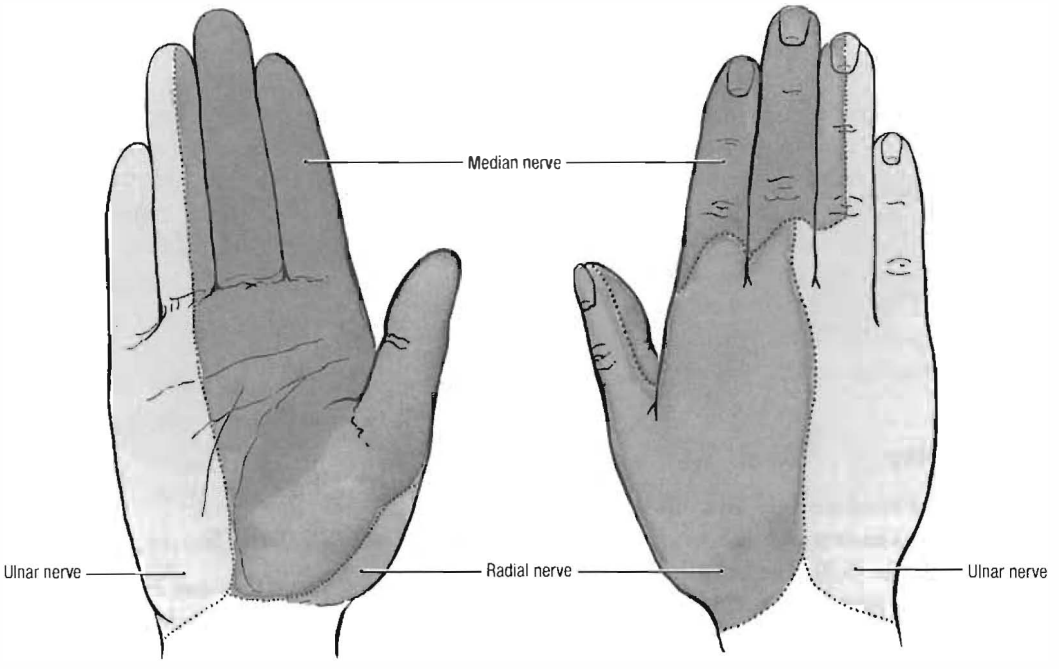

FIGURE 19.2.9. Sensorium of the hand. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

grip by each finger. Athletes with an ulnar nerve injury can still give a forceful grip, but the ring and little fingers may have limited strength.

The specific motions that should be tested in the wrist and hand are listed below.

1. *Wrist extension* (radial nerve). The radial nerve controls the extensor carpi radialis longus and brevis and the extensor carpi ulnaris. Wrist extension is a predominant function of the radial nerve.
2. *Wrist flexion* (median and ulnar nerves). The ulnar nerve controls the flexor carpi ulnaris, while the median nerve activates the stronger flexor carpi radialis.
3. *Finger extension* (radial nerve). Testing this motion involves the extensor digitorum communis, the extensor indicis, and the extensor digiti minimi.
4. *Thumb extension* (radial nerve). Thumb extension is controlled at the MCP joint by the extensor pollicis brevis, while the extensor pollicis longus extends the IP joint.
5. *Finger flexion* (median and ulnar nerves). Flexion is tested at three different levels:
   - *MCP joint* (median nerve): Motion is controlled by the lumbricals. The radial two lumbricals are innervated by the median nerve, while the ulnar two lumbricals are innervated by the ulnar nerve.
   - *PIP joint* (median nerve): Flexion of the PIP joint is controlled by the flexor digitorum superficialis.
   - *DIP joint* (ulnar nerve): DIP flexion is controlled by the flexor digitorum profundus.
6. *Thumb flexion* (median and ulnar nerves). MCP flexion is the domain of the flexor pollicis brevis, which has two innervations: the medial aspect by the ulnar nerve and the lateral aspect by the median nerve. Thumb flexion at the IP joint is controlled by the flexor pollicis longus and is innervated by the median nerve.
7. *Finger adduction* (ulnar nerve). Finger adduction is controlled by the volar interossei muscles.

8. *Thumb adduction* (ulnar nerve). Thumb adduction is controlled by the adductor pollicis.
9. *Finger abduction* (ulnar nerve). Motion at the MCP is controlled by the dorsal interossei and the abductor digiti minimi.
10. *Thumb abduction* (radial and median nerves). Thumb adduction is controlled by the abductor pollicis longus, which is innervated by the radial nerve, and the abductor pollicis brevis, which is innervated by the median nerve.

Vascularity

The wrist and hand are supplied by the radial and ulnar arteries. The two arteries have deep and superficial branches (6,9). The superficial branch of the radial artery joins the superficial branch of the ulnar artery in the midpalmar aspect of the hand and together form the superficial palmar arch. The superficial palmar arch is primarily supplied by the ulnar artery (9,10). Arising from the superficial palmar arch are the digital arteries. Each digit has its own radial and ulnar digital artery, which terminates in an anastomosis at the distal phalanx. The deep palmar arch is formed by the deep palmar branch of the ulnar artery and the radial artery and is also located at the midpalmar aspect of the wrist and is distal to the carpal tunnel. The radial artery is the primary contributor to the deep palmar arch (9).

Allen's Test. The athlete opens and closes the hand multiple times and then makes a fist. Next, the examiner holds down the radial and ulnar artery with the thumb and index finger. Let go of the artery being tested and the athlete's hand should pink up on the side of the artery that was released. Repeat the examination for the other artery to evaluate its function (1,4).

Positive test: The skin stays white, with no apparent return of blood flow after one artery is decompressed.

Indicates: Vascular compromise of either the radial or ulnar arteries; also used to ensure vascular patency.

PROVOCATIVE TESTS

Bunnel-Littler Test. This test evaluates tightness of the intrinsic hand muscles. It is also useful for distinguishing PIP joint contracture from intrinsic hand muscle tightness. The examiner places the stabilizing hand around the metacarpals while the mobilizing hand extends the MCP joints of each digit a few degrees of extension and moves each PIP joint through its ROM. If the PIP joints can be placed into flexion, then there is no intrinsic tightness (Fig. 19.2.10).

Positive test: Limited ROM of the PIP joint.

Indicates: Intrinsic muscle tightness or PIP joint capsule restriction.

FIGURE 19.2.10. Bunnel-Littler test.

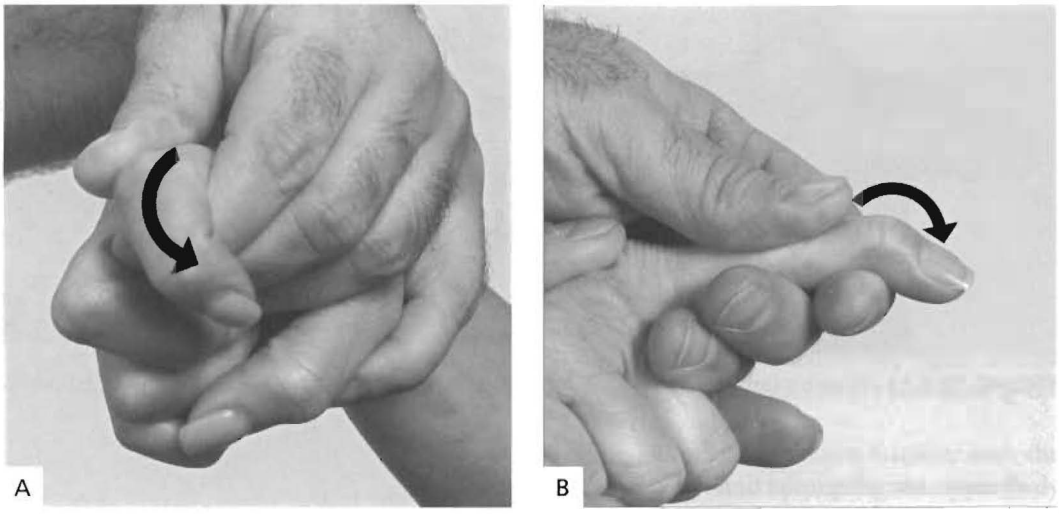

FIGURE 19.2.11. Finger flexor test. **A,** Testing the flexor digitorum superficialis. **B,** Testing the flexor digitorum profundus.

Note: To differentiate between hand intrinsic tightness and PIP joint tightness, the examiner flexes the MCP joint of the involved digit. This move should relax the hand intrinsics and allow the PIP joint to be put through its range of motion. If the PIP joint is now capable of flexion, then the hand intrinsics were tight. If there is still limited ROM, then the PIP joint is tight (1,4).

Finger Flexor Test. The flexor digitorum superficialis (FDS) evaluation can be performed by the examiner holding the MCP and having the athlete flex the PIP (Fig. 19.2.11A). For the flexor digitorum profundus (FDP), the examiner holds the PIP joint of the target finger so the distal phalanx will flex (Fig. 19.2.11B) (11).

Positive test: Loss of flexion at the target joint.
Indicates: FDS or FDP damage (depending on the isolated joint).

Finkelstein's Test. The athlete makes a fist with the thumb tucked in. The examiner deviates the wrist in the ulnar direction (Fig. 19.2.12).

Positive test: Pain in the area of the first dorsal compartment.

Indicates: First dorsal compartment stenosing tenosynovitis, or DeQuervain's disease (1,4,12).

Phalen's Test. The athlete places the dorsal aspect of one hand against the dorsal aspect of the other hand, thus causing flexion at the wrist. This is held for at least 30 seconds while the athlete reports changes in sensation or pain to the examiner (Fig. 19.2.13).

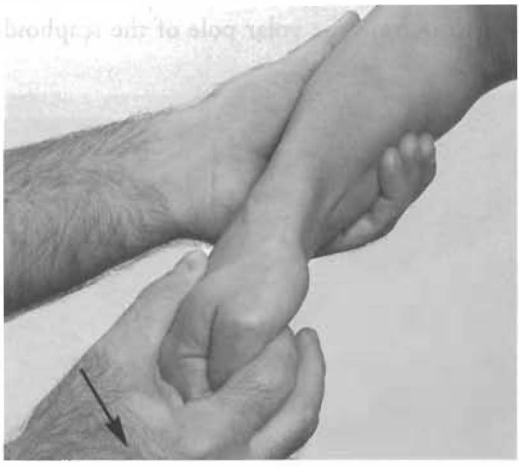

FIGURE 19.2.12. Finkelstein's test.

FIGURE 19.2.13. Phalen's test.

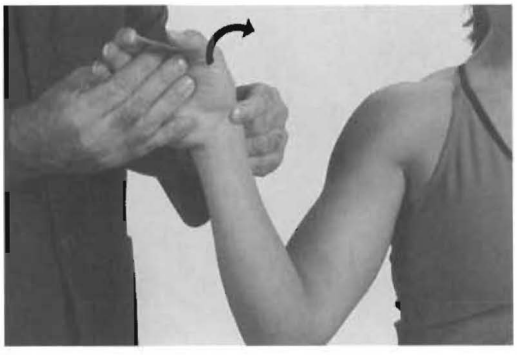

FIGURE 19.2.15. Watson's test, with radial deviation of the wrist.

Positive test: Reproduction of neurologic symptoms (numbness, tingling, pain).

Indicates: Carpal tunnel syndrome.

Tinel's Sign. The athlete is sitting and has both wrists facing up and lying on his or her legs. The examiner uses a reflex hammer to tap the transverse carpal ligament on the volar aspect of the wrist (Fig. 19.2.14).

Positive test: Paresthesias in the median nerve distribution with percussion (3,4).

Indicates: Carpal tunnel syndrome.

Note: This test can be used in other areas of the body where there is suspicion of a compressive neuropathy, such as the tarsal and cubital tunnels and Guyon's canal (13).

Watson's Test. The examiner places his or her thumb on the volar pole of the scaphoid while holding the wrist in ulnar deviation. The athlete then radially deviates the wrist (Fig. 19.2.15) (10).

Positive test: Pain with radial deviation of the wrist.

Indicates: Scapholunate instability and/or scapholunate ligament disruption.

Lunotriquetral Ballottement Test. This evaluates stability of the lunotriquetral joint. In this test, the examiner fixes the lunate between the index finger and thumb of the stabilizing hand. The mobilizing hand has its index finger and thumb on the triquetrum and displaces it in a volar and dorsal direction (Fig. 19.2.16) (13).

Positive test: Pain, crepitus, and excessive laxity.

Indicates: Lunotriquetral instability.

Triangular Fibrocartilage Complex (TFCC) Stability. The TFCC holds the radius and ulna together, so joint play best assesses the integrity of this structure. (See distal radioulnar joint anteroposterior glide [Fig. 19.2.20]).

Positive test: Increased play of the distal ulna against the distal radius compared with the opposite side.

Indicates: TFCC disruption.

FIGURE 19.2.14. Tinel's sign.

JOINT PLAY

The wrist and hand have tremendous adaptability and dexterity, more so than any other

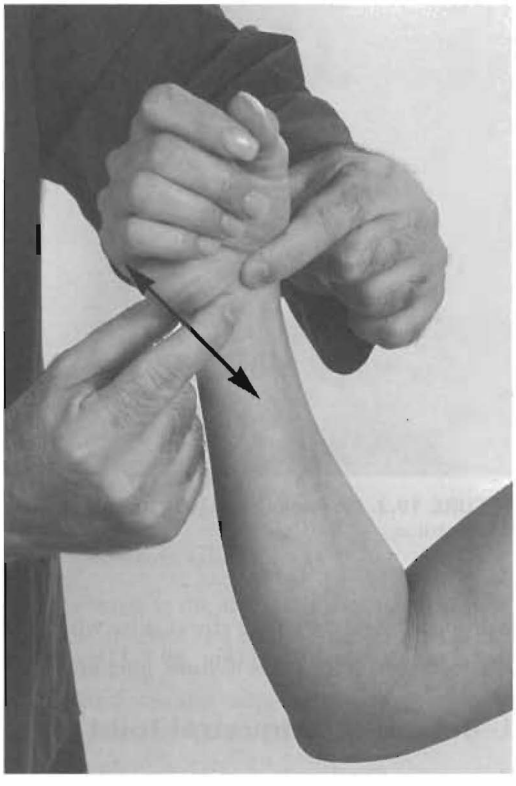

FIGURE 19.2.16. Lunotriquetral ballottement test.

region of the body. The functional demands are significant in sports, ranging from punching to gripping with force to delicately manipulating equipment. To accommodate this, the musculoskeletal structure has many joints with significant and variable amounts of play. Injuries to the wrist and hand should always incorporate joint play evaluation so that restrictions can be addressed that may otherwise be missed (14).

Evaluation of joint play in the wrist consists of examining the following joints:

1. Radiocarpal joint
2. Distal radioulnar joint and TFCC
3. Ulnomeniscotriquetral joints
4. Midcarpal joints

Evaluation of the hand consists of examining the following joints:

1. Metacarpal heads
2. MCP and IP joints
3. First metacarpal and trapezium joint

Radiocarpal Joint

Long-axis Extension

1. The examiner uses one hand to stabilize the distal anterior humerus of the athlete.
2. The mobilizing hand grasps the wrist, with the thumb just distal to the radial styloid process and the index finger just distal to the ulnar styloid process.
3. The examiner introduces traction with the mobilizing hand by rotating the upper body away (Fig. 19.2.17).

Posterior Scaphoid and Lunate Tilt

1. The examiner has the athlete's hand palm down.
2. The right thumb is placed over the dorsal side of the navicular.
3. The left thumb is placed over the lunare.
4. The index fingers curl under the wrist, pressing against the volar side of the navicular and lunate.
5. The examiner gently flexes the carpal bones (and the wrist) by lifting the forearm, then suddenly whips the forearm down toward the floor (Fig. 19.2.18).

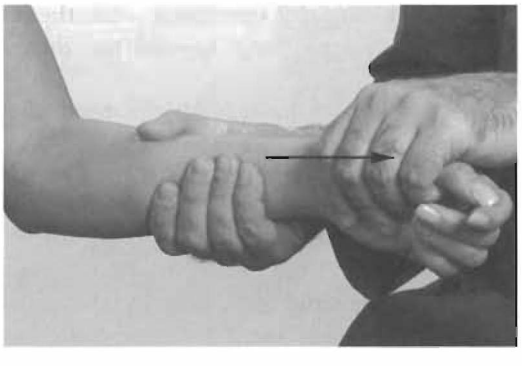

FIGURE 19.2.17. Joint play of the radiocarpal joint: long axis extension.

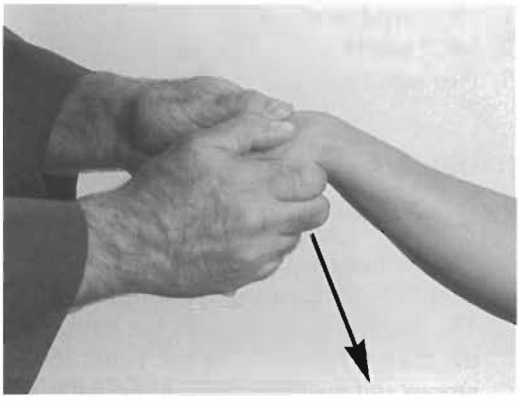

FIGURE 19.2.18. Radiocarpal joint: posterior scaphoid and lunate tilt.

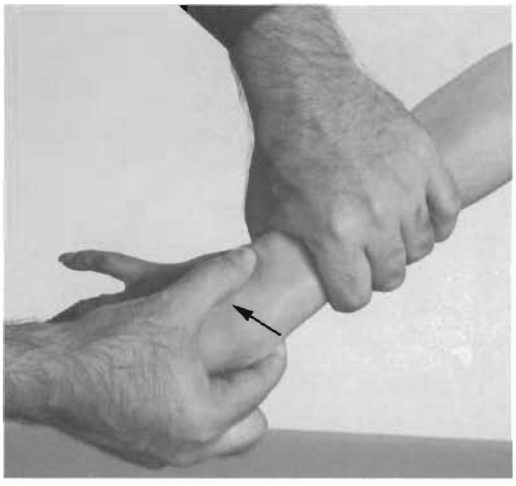

FIGURE 19.2.19. Radiocarpal joint: tilt of scaphoid from radius.

Tilt of Scaphoid from Radius

1. The examiner places the stabilizing hand proximal to the athlete's wrist with the thumb just proximal to the triquetrum.
2. The mobilizing hand grasps the proximal row of carpal bones, placing the thumb against the triquetrum and the index finger against the scaphoid.
3. The athlete's wrist is ulnar-deviated using the thumbs as a pivot point (Fig. 19.2.19).

Distal Radioulnar Joint

Anteroposterior Glide (Triangular Fibrocartilage Complex Test)

1. The examiner's stabilizing hand has its palmar side on the radial side of the athlete's wrist. The index finger lies in the first to second metacarpal interspace, while the remaining fingers wrap around the thenar eminence.
2. The mobilizing hand holds the distal ulna between the index finger and thumb.
3. The examiner moves the distal ulna anteriorly and posteriorly, assessing the amount of joint play in each direction (Fig. 19.2.20).

Rotation. The setup is the same as for the distal radioulnar joint anteroposterior glide, but the examiner wings his or her forearms out at a right angle to the athlete's forearm. The examiner keeps a stiff wrist and flexes the forearm to introduce rotation at the distal ulna while the distal radius is stabilized (Fig. 19.2.21).

Ulnomeniscotriquetral Joint

Long-axis Extension. Similar setup for radiocarpal long-axis extension (Fig, 19,2,22) but stabilizing hand is holding the distal humerus.

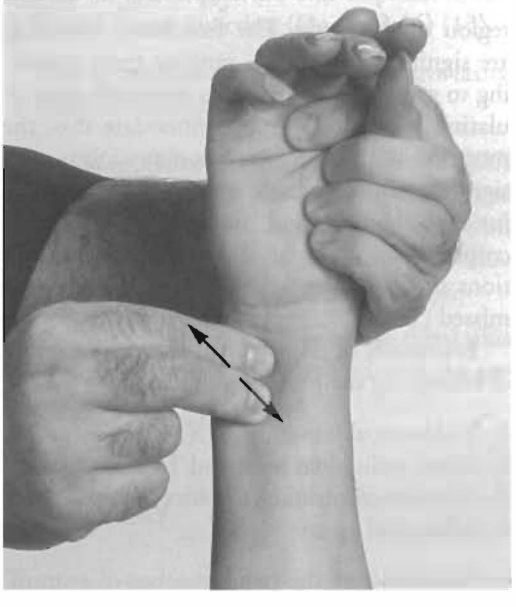

FIGURE 19.2.20. Joint play of the distal radioulnar joint: anteroposterior glide.

FIGURE 19.2.21. Distal radioulnar joint rotation.

Anteroposterior Glide

1. The setup is the same as for anteroposterior glide of the distal radioulnar joint, but the mobilizing thumb and index finger are on the pisiform and triquetrum.
2. The examiner introduces anterior and posterior motion (Fig. 19.2.23).

Side Tilt

1. Same as setup for scaphoid tilt on radius.
2. The examiner radially deviates the wrist, using the index fingers as a pivot (Fig. 19.2.24).

Midcarpal Joint

Long-axis Extension. Same as for radiocarpal and ulnomeniscotriquetral joints.

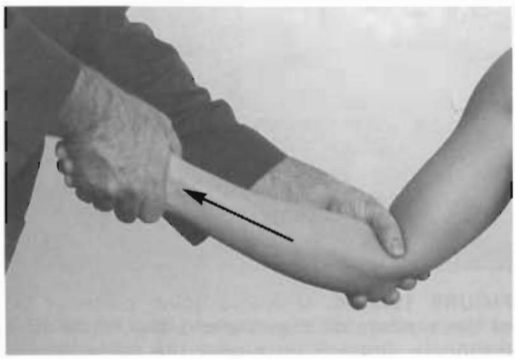

FIGURE 19.2.22. Ulnomeniscotriquetral joint: long-axis extension.

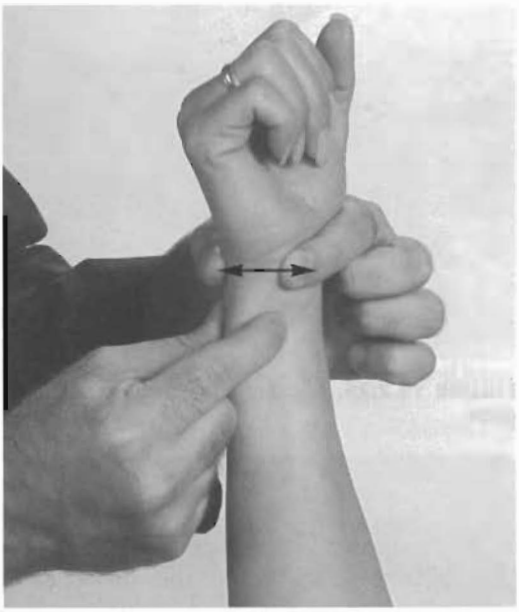

FIGURE 19.2.23. Ulnomeniscotriquetral joint: anteroposterior glide.

Anteroposterior Glide

1. The athlete has the hand palm down while the examiner places the stabilizing hand around the forearm just proximal to the proximal carpal row.
2. The mobilizing hand firmly grasps the proximal hand around the carpal region.

FIGURE 19.2.24. Ulnomeniscotriquetral joint: side tilt.

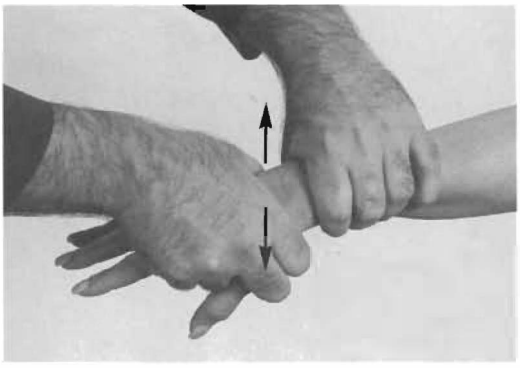

FIGURE 19.2.25. Midcarpal joint: anteroposterior glide.

3. The examiner holds the arm still with the stabilizing hand while the other hand translates the athlete's hand anteriorly and posteriorly, assessing for the amount of play (Fig. 19.2.25). The wrist should not be flexed or extended during this technique.

Posterior Tilt of Capitate on the Scaphoid and Lunate. *Note:* This technique depends on proper placement of the hands.

1. The examiner places one hand on the volar aspect of the athlete's wrist with the thenar eminence on the proximal row of carpal bones.
2. The opposite hand is against the dorsal side with the thenar eminence against the distal row of carpal bones.
3. The examiner dorsiflexes his or her wrists so that the forearms are at right angles to the carpal bones, then both hands squeeze together (Fig. 19.2.26). This tilts backward the distal row of carpal bones, which gaps the capitate-lunate articulation.
4. When this technique is done correctly, the athlete's fingers should spread apart as the examiner squeezes his or her own hands.

Metacarpal Heads

Anteroposterior Glide. The examiner stabilizes one head in place with a firm hold while the mobilizing hand gently holds the adjacent head being tested (Fig. 19.2.27). Mobilize the head anterior to posterior, and assess for amount of play.

Rotation. In the same position as for anteroposterior glide of the metacarpal head above, the examiner gently rotates the head of the metacarpal clockwise and counterclockwise, noting the amount of play (Fig. 19.2.28).

Metacarpophalangeal and Interphalangeal Joints

Long-axis extension (Fig. 19.2.29)
Anteroposterior tilt (Fig. 19.2.30)
Lateral tilt (Fig. 19.2.31)
Rotation (Fig. 19.2.32)

FIGURE 19.2.26. Midcarpal joint: posterior tilt of the capitate on the scaphoid and lunate. This technique depends on proper placement of the hands.

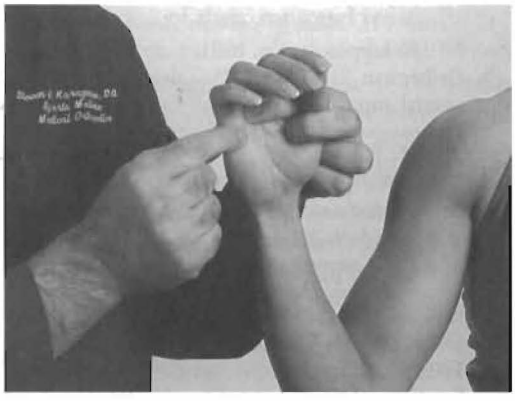

FIGURE 19.2.27. Metacarpal heads: anteroposterior glide.

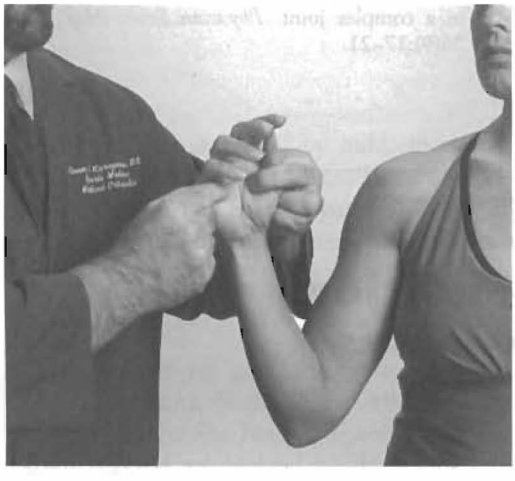

FIGURE 19.2.28. Metacarpal heads: rotation.

FIGURE 19.2.29. Joint play at the metacarpophalangeal and interphalangeal joints: long-axis extension.

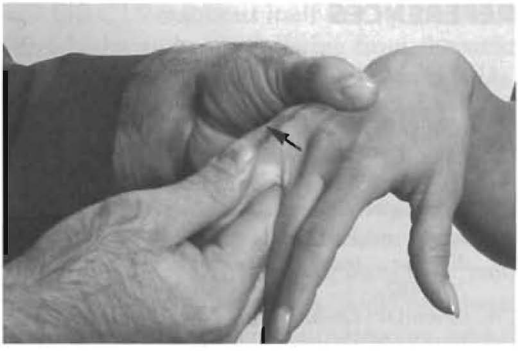

FIGURE 19.2.30. Metacarpophalangeal and interphalangeal joints: anteroposterior tilt.

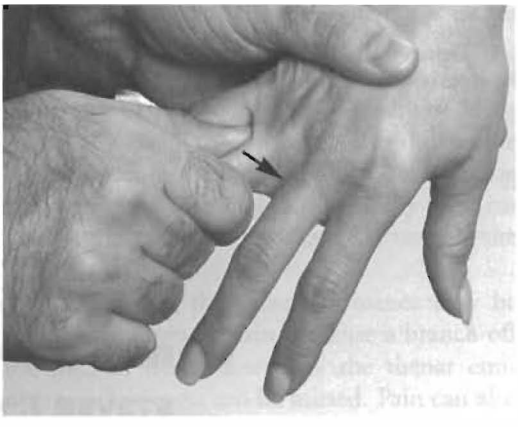

FIGURE 19.2.31. Metacarpophalangeal and interphalangeal joints: lateral tilt.

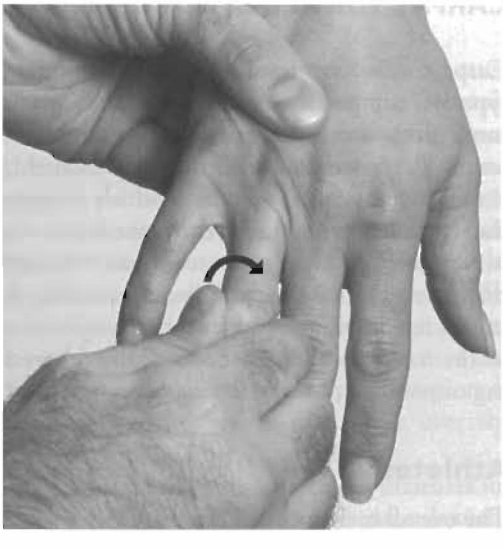

FIGURE 19.2.32. Metacarpophalangeal and interphalangeal joints: rotation.

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19.3

Common Conditions

STEVEN J. KARAGEANES

CARPAL TUNNEL SYNDROME

Carpal tunnel syndrome (CTS) is a common type of compressive neuropathy that occurs more often in the industrial setting than in athletics. The anatomic carpal tunnel is created by the transverse carpal ligament, which traverses the carpal bones and lies over the median nerve, nine flexor tendons, and occasionally the median artery. When the nerve becomes entrapped in this region, motor and sensory changes occur in the hand and fingers, occasionally progressing to permanent nerve damage in severe cases.

Athletes

The overall incidence of CTS in athletics is low. Athletes using racquets are at higher risk for developing CTS, such as tennis, racquetball, and squash. Field hockey, ice hockey players, and golfers also can develop CTS, particularly during multiple sessions of repetitive practice. In most any activity, athletic or not, overuse is the trigger for the syndrome. Trauma to the wrist can be as troublesome as overuse if there is bleeding or inflammation in the carpal tunnel. Fractures cause bleeding that leads to acute neurologic symptoms from compression, but the symptoms usually resolve as the bones heal, assuming the fracture is reduced.

The population at highest risk is the wheelchair athlete, who repetitively uses his or her arms and wrists for transportation and competition. Wheelchair athletes compete in races from 100 meters to a marathon, swimming, basketball, tennis, rugby, and football. In response to this, many companies now make specially designed wrist braces for athletes with frequent

wheelchair use of their arms and wrists. Part of their pre-participation physical should always include a thorough examination of the hands and wrist, as they will likely suffer overuse and abuse. Nevertheless, any athlete who sustains repetitive wrist trauma is susceptible to CTS, whether the compressive force comes from inflammation, chronic ligament scarring, structural changes, or biomechanical carpal shifting.

Pregnant athletes do not usually compete in contact sports, but they are at high risk for CTS, even without repetitive exercise. These cases spontaneously resolve after delivery, so few are surgically released. However, the athlete may have to live with 5 to 6 months of discomfort if manual medicine is not applied in some form.

Grading

Severity of CTS is graded by mild, moderate, and severe neuropathic changes. These changes are best identified on electromyography, the gold standard for CTS diagnosis.

Symptoms

Symptoms can be graded according to the Global Symptom Scale (Table 19.3.1). The scale assesses the five basic symptoms involved with CTS: pain, numbness, paresthesias, weakness or clumsiness, and nocturnal awakening. Each is rated from 1 to 10, and a composite of the five symptoms allows the range to run from 5 to 50. A higher score means more severe progression of disease.

TABLE 19.3.1. GLOBAL SYMPTOM SCALE FOR CARPAL TUNNEL SYNDROME

Pain
Numbness
Paresthesias
Weakness/clumsiness
Nocturnal awakening

From Chang MH, Chiang MH, Lu SS, et al. Oral drug of choice in carpal tunnel syndrome. *Neurology* 1998;51: 390–393.

Mild CTS manifests itself first by nocturnal paresthesias and pain radiating from the wrist through the hand and into the thumb, index, and middle fingers. As the disorder progresses, the paresthesias and pain last longer and occur more frequently during the day. The onset is more easily provoked and intensity increases, but the symptoms can resolve with restored circulation. Severe CTS involves intractable pain and muscle atrophy and a poor prognosis, since the median nerve is undergoing axonal degeneration. Do not let the athlete wait for muscle atrophy to occur; axonal loss can occur before atrophy is visible. If nerve decompression is not performed, permanent neural changes will occur.

The classic pattern of sensory changes is along the palmar side of the hand extending from the thenar eminence of the thumb to the lateral aspect of the fourth digit. Changes along the fourth and fifth digits are typically from injury to the ulnar nerve, which lies outside the carpal tunnel.

Occasionally, the thenar eminence may be the only location of pain. Because a branch off the median nerve innervates the thenar eminence, subtle cases can be missed. Pain can also radiate back through the forearm and medial epicondyle.

Pathophysiology

Overuse of the wrist is the most common cause of CTS. As the wrist is repetitively used, chronic inflammation and carpal bone shifting from muscular imbalance narrow the carpal tunnel space for the distal median nerve. Its entrapment as it courses through the osseofibrous carpal tunnel alters neural microperfusion and leads to ischemic changes, demyelination, and axonal degeneration. Static wrist hyperflexion compresses the median nerve between the finger flexors and the flexor retinaculum, while hyperextension of the wrist stretches the median nerve over the flexor tendons and the distal radius.

Congestion in the carpal tunnel increases intratunnel pressure and decreases neural nutrition. Chronic congestion starts to increase neural edema, which then increases the compressive

effect. Neural insult is manifested by symptoms, but at this point the process is still reversible with decompression. Only when the axons themselves break down do permanent changes occur.

Intratunnel pressures normally are in the neighborhood of 2.5 mm Hg. In CTS, the pressures can increase to 32 mm Hg in neutral position, up to 90 mm Hg with wrist flexion, and over 100 mm Hg with wrist extension. Prolonged exposure leads to nerve injury by impeding circulation (thereby contributing to the intraneural ischemia) when pressures exceed 30 mm Hg. In fact, hyperflexion or hyperextension of the wrist can increase intracanal pressures from 40 to 100 times, depending on the study cited.

Overuse of the wrist also leads to biomechanical changes. Excessive flexion and extension of the wrist impacts the median nerve and leads to an imbalance of flexor strength compared to the extensors. This causes a subtle volar shift in the carpal bone alignment, which is the floor of the carpal tunnel. The shift flattens the floor, decreasing tunnel space and increasing nerve compression.

Acute trauma can cause CTS as well. Trauma to the volar wrist—particularly with the wrist extended—can cause internal bleeding and nerve compression, even more so if the trauma causes a fracture. This is often seen in falls where the athlete puts his or her arm out to break the fall.

People at risk for CTS are those who work with computers, vibratory tools, painting brushes, scrapers, and upholstery. Activities that require forceful finger flexion (more than 4 kg) and repetition with repeat cycles less than 30 seconds contribute markedly to CTS pathophysiology and symptoms.

CTS is common in pregnant women partly due to hypervolemia. Hobbies demanding manual dexterity, such as musical instruments, knitting, crocheting, and gardening, also put people at higher risk. Predisposing medical conditions include pregnancy, hypothyroidism, diabetes mellitus, gout, and rheumatoid arthritis.

Structural changes to the carpal tunnel itself should be ruled out in the workup. This includes trauma, such as a Colles fracture, stenosis or fibrosis of the carpal tunnel or ligament, osteophytes, tenosynovitis, or a ganglion cyst.

Differential Diagnosis

Every carpal tunnel workup should include a cervical spine examination (see head and neck examination in Chapter 16.2) to rule out radiculopathy. Even if symptoms do not exhibit the classic "shooting pain down the arm," neuropathy can still emanate from the cervical spine. The differential diagnosis should include a more proximal median nerve injury, generalized neuropathy, and intrathoracic malignancies. Other conditions with potential to cause CTS include alcohol abuse, benign lipoma, malignant tumors (especially multiple myeloma), congestive heart failure, renal failure, hematoma formation, and obesity.

Shoulder pathology may not refer pain in a median nerve distribution to the fingers, but conditions such as rotator cuff impingement, instability, and bicipital tenosynovitis can affect shoulder biomechanics to the point at which they increase the risk of CTS. For example, a tennis player with shoulder impingement may not be able to swing the arm around hard enough, so he or she may compensate by flexing the wrist harder during groundstrokes, which increases stress at the wrist and sets off CTS.

Examination

On clinical examination, test sensation with two-point discrimination. Differentiate between paresthesias and loss of sensation, as athletes can have paresthesias but normal two-point discrimination.

Wasting of the abductor pollicis brevis, innervated by a branch of the median nerve, causes thenar atrophy. This is seen in more severe cases where axonal degeneration is occurring. Phalen's test is the most sensitive test for CTS (see Fig. 19.2.13). Rest the athlete's elbows on a table and allow the wrists to fall into complete volar flexion for 1 minute. This may reproduce the wrist paresthesias. Reverse Phalen's test has been documented as well, using extension

instead of flexion. A modification of this test is a provocative wrist flexion test, in which the examiner presses on the median nerve in the Phalen's test flexed position. Pain within 20 seconds is considered a positive test (1).

Electromyography (EMG) is the gold standard for diagnosing median nerve entrapment. Grading of severity is accomplished by looking for changes in distal sensory latency, distal motor latency, and nerve conduction velocity. Sensory latency changes are the most sensitive and earliest indicator of CTS, while distal motor latency prolongation is seen later in the course of CTS. Changes in signal amplitude indicate a progressing disorder, while severe motor changes signify significant neuropathy.

It should be noted that with clinicians who use manual medicine, EMG is more beneficial in diagnosing severity of nerve damage and localizing the source of the nerve entrapment (2). A thorough history and physical, with special attention paid to abnormal tissue restriction and congestion, will typically be enough to make the diagnosis.

Standard Treatment

Acute treatment of CTS is conservative use of nonsteroidal anti-inflammatory drugs (NSAIDs), ice, and relative rest. Any concomitant conditions affecting or causing CTS (such as diabetes and hypothyroidism) should be treated while treating the nerve and wrist, or the condition may not improve. Because night symptoms are common, neutral wrist splints (not extended) should be used at night to avoid excessive flexion and provocation. Splints may be used at work for activity-related symptoms.

Physical therapy is usually successful in controlling early CTS, but compliance with home programs needs to be stressed to avoid recurrence. Basic components of a CTS protocol include wrist stretches, grip exercises, wrist flexor and extensor strengthening, and use of modalities such as electrical stimulation and moist heat.

With any therapy session, the athlete should have his or her work and home environment evaluated for proper ergonomics, particularly home and work offices with computers. Pads in front of a mouse or keyboard can help. Look at the height of the monitor, keyboard, or chair in the office. The workplace should be evaluated for evidence of poor ergonomics, particularly if multiple employees suffer from similar musculoskeletal maladies.

Oral corticosteroids have temporary benefit at best (4). Corticosteroid injections have varying and temporal efficacy. The symptoms occasionally get worse due to the increased compression in the carpal tunnel from the volume of medicine injected. Vitamin B_6 and magnetic therapy have anecdotal support but little controlled research recommending their use. Both have minimal side effects, so many try them as a lower-tier option.

Manual Medicine

The goal of manual medicine in CTS is to decrease congestion in the carpal tunnel. Research demonstrates that manual medicine is effective in mild and moderate cases (2). This can be approached from several aspects:

1. Lengthening or loosening the transverse carpal ligament.
2. Increasing carpal tunnel diameter.
3. Improving lymphatic flow through the carpal tunnel.
4. Restoring function and mobility to the radiocarpal and ulnocarpal joints.
5. Restoring balance between the wrist flexors and extensors.

Lengthening

Extension Stretch of Wrist and Thumb

1. The athlete is sitting and places the elbow of the affected extremity against the medial aspect of the knee. The fingertips of that hand press against the medial aspect of the opposite knee. The palm is facing up.
2. The athlete squeezes the knees together, introducing more extension into the wrist, until the wrist is painful or the restrictive barrier is met.
3. The athlete grabs the opposite thumb and rolls it into abduction, extension, and external

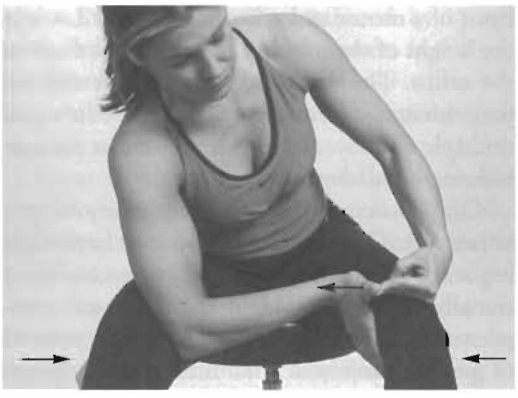

FIGURE 19.3.1. Extension stretch of wrist and thumb.

rotation until the pain or restrictive barrier is met (Fig. 19.3.1).

4. The maneuver is gradually performed over 5 to 10 seconds, and the final position is held for 15 to 30 seconds. The athlete repeats this two-phase process five to ten times daily.

Alternate Technique 1 (Sucher Self-stretch Technique). This is the same technique as above, but the athlete's hand leans against a door frame or wall, forcibly dorsiflexing the wrist and fingers.

Alternate Technique 2. The athlete takes one hand and gently pulls back the digits of the other hand, holding for 15 to 20 seconds.

Exercises

Carpal Pump

Rationale: This helps to work excess fluid out of the transverse carpal ligament while working the flexor and extensor muscle groups.

1. The athlete holds the arms outstretched in front.
2. The athlete flexes the wrist and fingers so that a fist is made. The contraction should be forceful enough to fire the whole flexor bundle. Hold for 2 to 4 seconds (Fig. 19.3.2A).
3. The athlete then extends the wrist and fingers forcefully, contracting with near maximal effort and holding for 2 to 4 seconds (Fig. 19.3.2B). Repeat back and forth for 1 to 2 minutes. This can be done hourly during exercise or work. (Carpal pump exercise from the American Academy of Orthopedic Surgeons Conference poster February 25, 1996.)

Wrist Flexion and Extension Curls

Rationale: To improve the strength of both flexor and extensor groups and reduce flexor strength dominance compared with the extensors (Fig. 19.3.3).

Manual Techniques

Myofascial Release

Rationale: To increase flexibility and release restrictions in the transverse carpal ligaments,

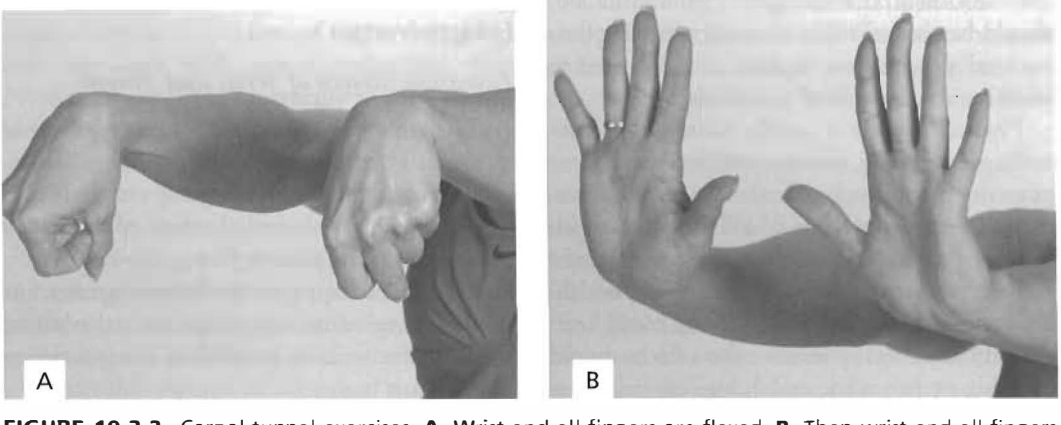

FIGURE 19.3.2. Carpal tunnel exercises. **A,** Wrist and all fingers are flexed. **B,** Then wrist and all fingers are extended.

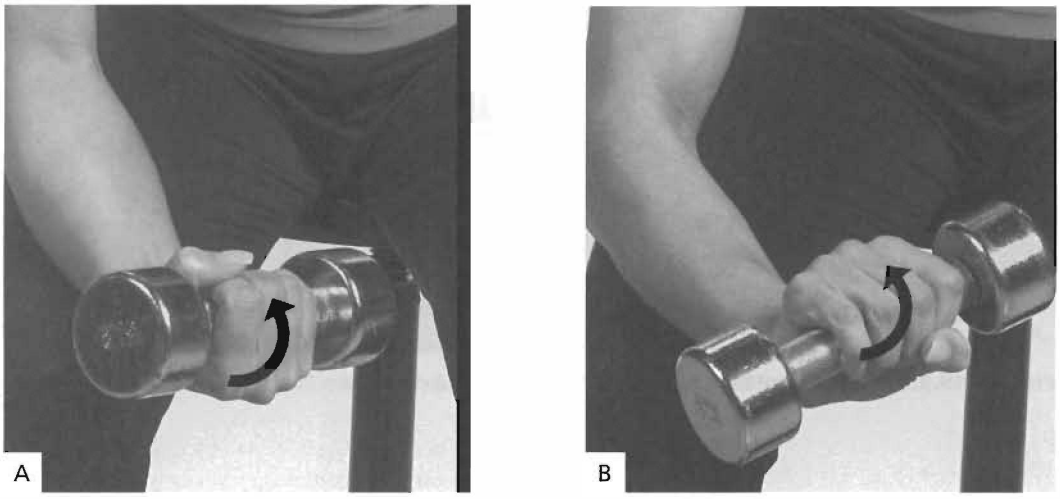

FIGURE 19.3.3. A, Wrist flexion curls. **B,** Wrist extension curls.

and radiocarpal and ulnocarpal ligaments, thereby decreasing pressure on the median nerve.

Technique 1: Direct Release (Left Wrist) (7)

1. The clinician stabilizes the athlete's hand by holding it with the left palm against the dorsum and the fingers reaching around to hold the thenar eminence.
2. The left hand holds the athlete's wrist with the fingers over the volar aspect (Fig. 19.3.4A).
3. The clinician rotates his or her hands opposite to each other, reaching the end point and holding (Fig. 19.3.4B).
4. Repeat in opposite directions and hold at the restrictive barrier.

Technique 2: Sucher Myofascial Release (Left Wrist) (5)

1. The clinician holds the athlete's hand with the fingers against the dorsum, the thumbs pressing on the medial and lateral attachments of the transverse carpal ligament, and the athlete's thumb held by the right hand (Fig. 19.3.5A).
2. The clinician's fingers press up on the cen-

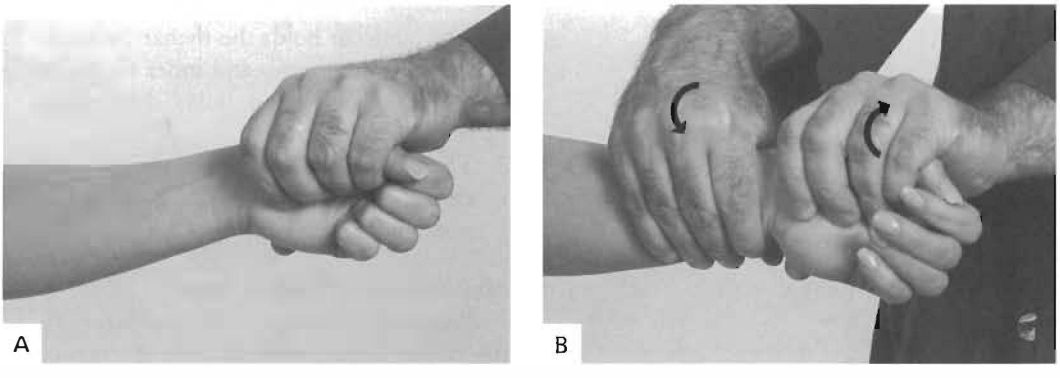

FIGURE 19.3.4. Direct myofascial release to the wrist and transverse carpal ligament. **A,** Starting position with the athlete's left wrist. **B,** Rotating the wrist and distal forearm in opposite directions.

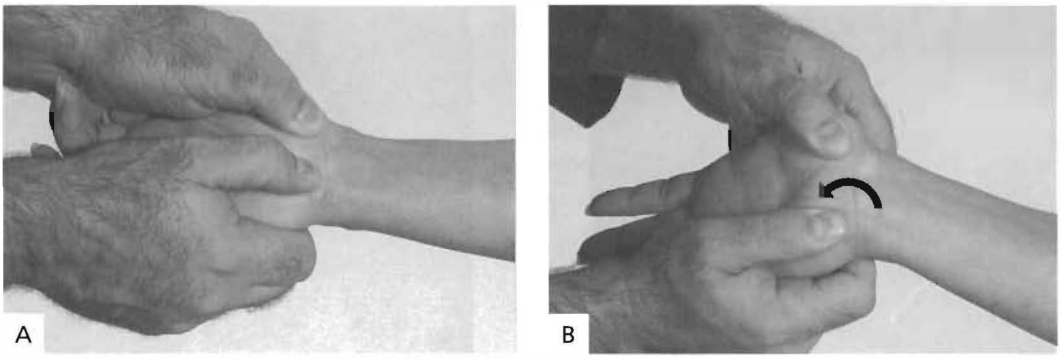

FIGURE 19.3.5. Sucher myofascial release. **A,** Starting. **B,** In extension.

tral dorsal wrist as the thumbs apply pressure on the ligament insertions and the athlete's thumb is pulled into radial abduction and extension (Fig. 19.3.5B).

Muscle Energy

Rationale: Increases flexibility of flexors and extensors, improves range of motion.

Technique for Flexors

1. The clinician stabilizes the athlete's wrist with the proximal hand holding the wrist proximal to the palmar crease.
2. The clinician's distal hand moves the athlete's hand into wrist extension by pushing against the athlete's metacarpophalangeal joints on the palmar side (Fig. 19.3.6).
3. The athlete pushes 3 to 5 seconds, holds 1 second, repeats three to five times, repositioning after each contraction.

FIGURE 19.3.6. Muscle energy technique to the wrist flexors.

For *extensors*, the athlete's wrist is moved into the flexion barrier.

For *ulnar deviation*, the athlete's wrist is moved into the radial deviation barrier.

For *radial deviation*, the athlete's wrist is moved into the ulnar deviation barrier.

For *opponens muscle*, move the athlete's thumb into extension.

Opponens Roll Maneuver (Right Wrist) (6)

Rationale: This is helpful in lifting the carpal ligament off the median nerve and its thenar branch using lateral axial rotation on the opponens muscle. It may also stretch the ligament and loosen adhesions.

1. The clinician holds the thenar eminence in between the thumb and index finger, while the stabilizing hand holds the ulnar side firmly (Fig. 19.3.7A).
2. The clinician moves the thumb and thenar eminence into lateral axial rotation, extension, and abduction (Fig. 19.3.7B).

Mobilization of Carpal Bones

Rationale: Treats anterior subluxations of the carpal bones, most notably the lunate, to increase carpal tunnel space and decrease compression.

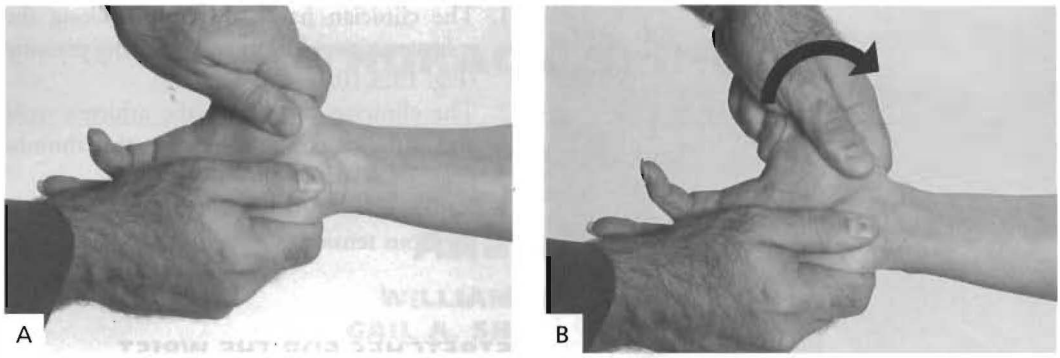

FIGURE 19.3.7. Opponens roll maneuver: **A,** Starting. **B,** After lateral axial rotation.

Technique for Restricted Extension

1. The clinician holds the athlete's hand with both of his or her hands, while the athlete rests the hand in pronation.
2. The clinician's thumbs rest on the dorsal scapholunate region, fingers clasped around the thenar and hypothenar eminences.
3. The clinician extends the athlete's wrist into the restrictive barrier (Fig. 19.3.8A).

Technique for Restricted Flexion

1. The clinician introduces an impulse by thrusting the wrist down toward the floor.
2. The clinician flexes the athlete's wrist into the restrictive barrier.
3. The clinician introduces an impulse by thrusting the wrist up toward the ceiling (Fig. 19.3.8B).

Carpal Tunnel Pump

Rationale: To move congestion out of the tunnel by a lymphatic mechanical pump action of the clinician's hands.

1. The clinician places one hand on the volar side with the thenar eminence against the proximal crease. The other hand is on the dorsal side one fingerbreadth distal to the volar hand (Fig. 19.3.9).
2. The clinician then rhythmically squeezes his or her hands together, alternating between the volar and dorsal hands, to generate a pumping motion.

Cross-friction Massage

Rationale: Decompresses the median nerve by slowly loosening the transverse carpal ligament.

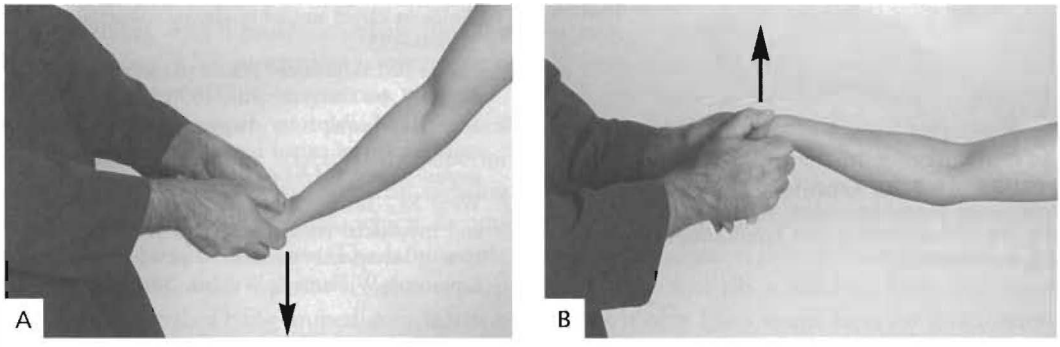

FIGURE 19.3.8. Lunate mobilization. **A,** With extension restriction. **B,** With flexion restriction.

FIGURE 19.3.9. Carpal tunnel pump.

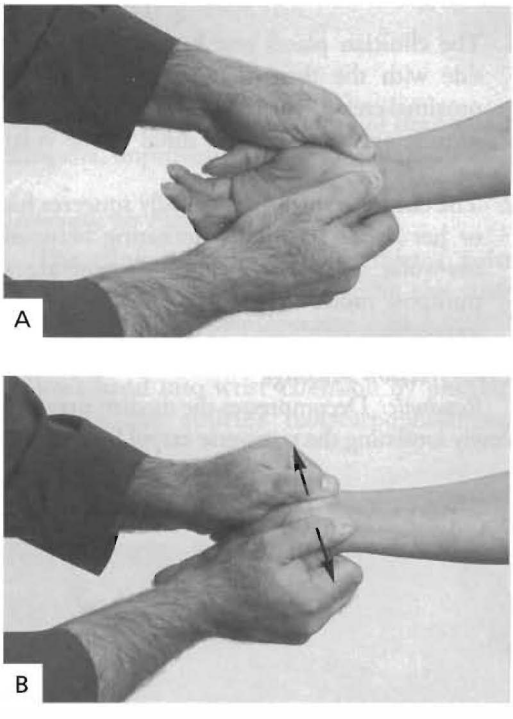

FIGURE 19.3.10. Cross-friction massage to the transverse carpal ligament. **A,** Setup. **B,** Dorsiflexing the athlete's wrist and simultaneously separating the thumbs.

1. The clinician has both thumbs along the transverse carpal ligament, applying pressure (Fig. 19.3.10A).
2. The clinician dorsiflexes the athlete's wrist and simultaneously separates the thumbs (Fig. 19.3.10B).
3. Repeat as needed for softening of any ligamentous tension.

STRETCHES FOR THE WRIST

- Stretch of *volar forearm muscles:* See stretches for the elbow in Chapter 18.3 (Fig. 18.3.10A).
- Stretch of *dorsal forearm muscles:* The athlete can use a tabletop or a wall variation, depending on the comfort level. See stretches for the elbow in Chapter 18.3 (Fig. 18.3.10B).
- Stretch of *extensor pollicis longus muscle, extensor pollicis brevis muscle, abductor pollicis longus muscle:* See stretches for the elbow in Chapter 18.3 (Fig. 18.3.10D).

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20

THORACIC SPINE

20.1

Anatomy

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GAIL A. SHAFER-CRANE

The thoracic spine, which consists of the 12 thoracic vertebrae, contains the thoracic and upper lumbar portions of the spinal cord and associated nerve roots whose branches supply the back and the anrerolateral thoracic and abdominal walls in a segmental fashion from the neck to the pelvis. This region of the vertebral column serves as an attachment for the ribs and transmits the weight of the body to the lumbosacral spine. The thoracic spine is concave anteriorly. Anatomy of the thoracic spine is presented in detail in major anatomic textbooks (1--6).

The distinguishing feature of all 12 thoracic vertebrae is that they articulate with at least one pair of ribs (Fig. 20.1.1). Typical thoracic vertebrae (T2-T9) articulate with two pairs of ribs; the head of the rib contacts a facet on the superior margin of one vertebral body and a similar facet on the inferior margin of the vertebral body above. In addition, there is a facet on the transverse processes of thoracic vertebrae T1-T10.

Bodies of thoracic vertebrae increase in size from T1 to T12 and display highly convex anterior surfaces, which protrude deeply into the thoracic cavity. This protrusion is exaggerated by the direction of the transverse processes, which project posteriorly and superiorly as well as laterally. The pedicles attach to the superior ends of the vertebral bodies thereby creating deep inferior vertebral notches, which accommodate the spinal nerve roots. The laminae are broad and those of the superior vertebra overlap those of the vertebra lying immediately inferior.

The long and slender spinous processes point inferiorly and overlap the vertebra lying inferiorly. The spinous processes of the lower thoracic vertebrae resemble those of lumbar vertebrae; they are shorter, stouter, and project more posteriorly. The superior and inferior articular processes are situated anterior to the transverse process and their facets are oriented in the coronal plane. The superior facet faces posteriorly and laterally, while the inferior facet faces anteriorly and medially (Fig. 20.1.2). There is an abrupt transition in the orientation of the articular processes at the level of T12 where the inferior articular process becomes oriented to match those of the lumbar vertebrae with an anterolaterally directed facet.

There are 12 pairs of ribs, which articulate with the thoracic vertebrae through their heads and short necks. The head of a typical rib has a superior and inferior facet divided by a crest for articulation with the bodies of two adjacent thoracic vertebrae. The superior border of the narrower neck also displays a crest for the attachment of ligaments. The neck terminates laterally in an articulating tubercle, which bears an oval, convex facet for articulation with the transverse process of the similarly numbered thoracic vertebrae. Lateral to the facet is a rough area for attachment of a ligament. The tubercle marks the junction of the neck and the body of the rib, which turns sharply forward at the angle and slants inferiorly and forward. The head of the first rib is different from the others in that it only has a single facet for articulation with the facet on the superior margin of the body of the T1 vertebra.

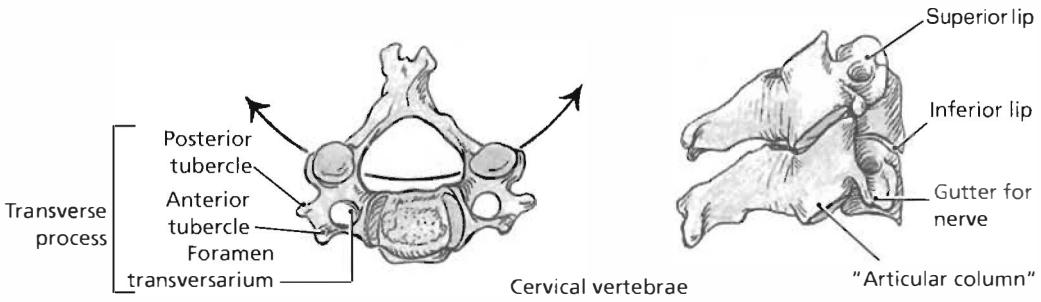

FIGURE 20.1.1. The thoracic vertebrae. From Olson T: A.D.A.M student Atlas of Anatomy. Philadelphia: Williams & Wilkins, 1996.

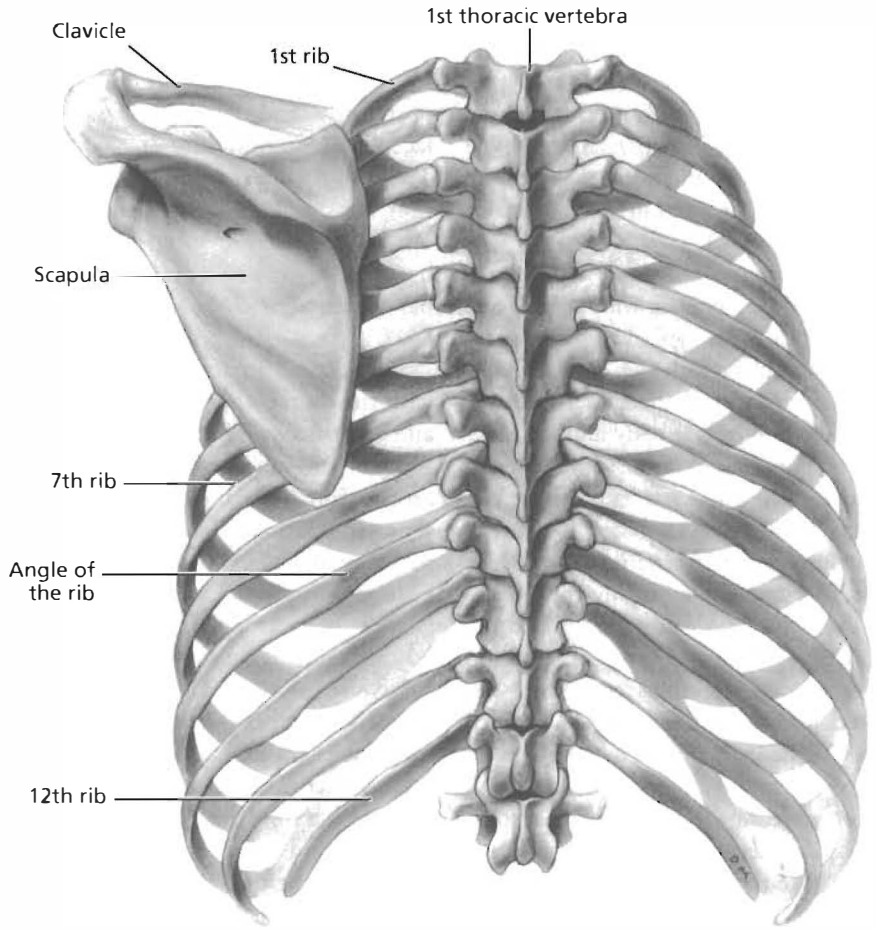

FIGURE 20.1.2. The bony thorax, posterior view. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

The thoracic spine is composed of two types of joints, which work together to provide mobility for the spine. These include facet joints (zygapophyseal joints) and intervertebral body joints (intervertebral discs). The facet joints are synovial joints between the articular processes of adjacent vertebrae. A thin, loose articular capsule surrounds each joint. The facet joints permit gliding movements between the vertebrae, and the shape of the articular surfaces limits the range of motion possible. Branches arising from the posterior primary rami of spinal nerves innervate these joints.

The intervertebral discs are fibrocartilaginous joints, designed for weight bearing and strength, situated between the bodies of adjacent vertebrae. Each intervertebral disc consists of an outer anulus fibrosus, composed of concentric lamellae of fibrocartilage, which surrounds a gelatinous nucleus pulposus. The anuli insert into the rounded rims on the articular surfaces of adjacent vertebral bodies. Branches arising from the anterior primary rami of spinal nerves innervate the intervertebral discs. The intervertebral discs are avascular structures, that receive their blood supply by diffusion from the vertebral bodies. Mobility of the thoracic spine, especially in its superior and middle regions is limited mainly due to the ribs and rib cage as well as the narrowness of the intervertebral discs. Rotation takes place primarily in the thoracic spine, with flexion, extension, and lateral bending being extremely limited.

Ligaments reinforce and stabilize the facet joints and the intervertebral discs. The anterior longitudinal ligament is a broad, thick band attaching to anterior and anterolateral surfaces of the vertebral bodies and intervertebral discs. It limits extension of the vertebral column. The posterior longitudinal ligament runs within the vertebral canal and attaches to the posterior surfaces of the vertebral bodies and the intervertebral discs. In the thoracic region it narrows over the surfaces of the vertebral bodies and expands over the intervertebral discs. The posterior longitudinal ligament checks flexion of the vertebral column. The strong, highly elastic, flattened ligamenta flava attach to the laminae of adjacent vertebrae. These ligaments help to preserve the normal curvature of the vertebral column and straighten the vertebral column after it has been flexed. Short interspinous ligaments and a strong cordlike supraspinous ligament join adjacent spinous processes. The intertransverse ligaments connect adjacent transverse processes and are insignificant in the thoracic spine.

The ribs articulate with the thoracic spine at two synovial joints: the costovertebral and costotransverse joints. At the costovertebral joint, the head of a rib articulates with two vertebral bodies (Fig. 20.1.3). A ligament attaching the crest of the head to the intervertebral disc divides the joint cavity. The articular capsule is thickened anteriorly by the radiate ligament, which anchors the head of the rib to the intervertebral disc and to the thoracic vertebral bodies superior and inferior to the disc. The costotransverse joint is between the articulating tubercle on the rib and the transverse process of the vertebra. The joint has a small cavity and is strengthened by three costotransverse ligaments. These extend between the crest on the rib neck and the transverse process superior to the rib (superior costotransverse ligament), between the posterior aspect of the rib neck and its own transverse process (costotransverse ligament), and between the tip of the transverse process and the rough part of the articulating tubercle (lateral costotransverse ligament). These joints are responsible for the pump-handle and bucket-handle movements of the ribs. Both joints receive their arterial supply from branches of the posterior intercostal arteries and are innervated by branches of the posterior primary rami of the thoracic spinal nerves.

The control and strength of movements in the thoracic spine, as well as in other regions of the spine, are dependent on muscles. Muscles help to stabilize the spine and control the effects of gravity. The extensor muscles are the one major functional group of the thoracic spine. This group is also capable of rotating and side bending the spine. Flexor muscles located on the anterior aspect of the spine in other regions are absent in the thoracic spine. The extensor muscles (deep back muscles) span the

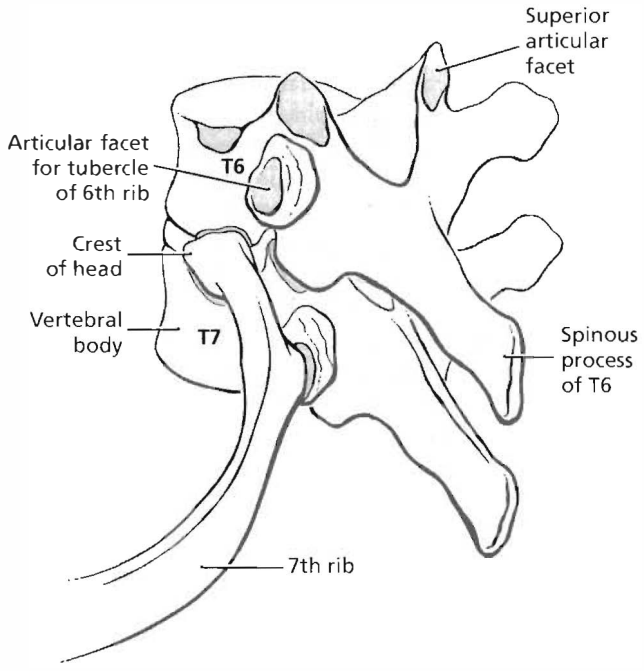

FIGURE 20.1.3. Costovertebral joint. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

entire back from the skull to the sacrum and in the thoracic region are situated posterior to the laminae and transverse processes between the spinous processes medially and the angle of the ribs laterally. They are innervated segmentally by the posterior primary rami of the thoracic spinal nerves. These muscles consist of two groups: erector spinae and transversospinalis. The muscles are enclosed in the thin thoracic portion of the thoracolumbar fascia, which attaches to the spinous processes of the thoracic vertebrae and the angles of the ribs and separates the muscles from overlying superficial muscles. The erector spinae muscles (Fig. 20.1.4) are responsible for restoring the spine to the erect posture after flexion and also act to control flexion of the spine against gravity. The transversospinalis muscles act primarily to rotate the spine.

The erector spinae and transversospinalis muscles are arranged in three layers: superficial, intermediate, and deep. In the thoracic spine, the superficial layer consists of three vertically running columns of muscles. The most lateral column is the iliocostalis, which runs along the angles of the ribs. Portions of all three subdivisions of the iliocostalis—lumborum, thoracis, and cervicis—can be found in the thoracic spine. The intermediate column is the longissimus, which is associated with the transverse processes. In the thoracic spine, the thoracis and part of the cervicis subdivisions of the thoracis muscle can be found. The medial column is the spinalis, which is associated with the spinous processes. The thoracis subdivision of the spinalis muscle is well developed and is always present. The intermediate layer is composed of two subgroups of the transversospinalis muscles. As the name of this muscle group implies, the transversospinalis muscles arise from transverse processes and insert into spinous processes.

In the thoracic spine the intermediate layer is composed of the thoracis and cervicis portions of the semispinalis muscle and deep to them, the multifidus muscles. The semispinalis muscles span up to six vertebrae, while the multifidus muscles span two to four vertebrae. The deep layer is composed of segmental muscles: interspinous, intertransverse, and rotator groups.

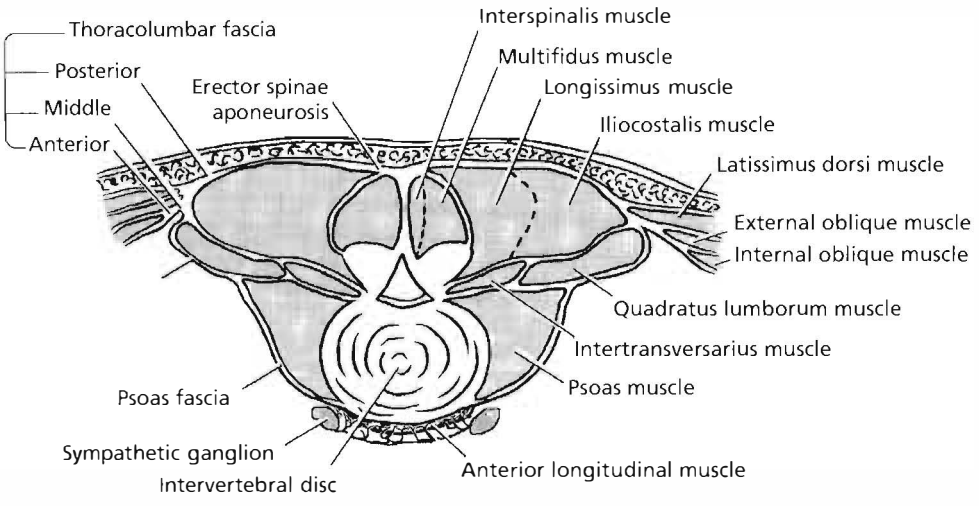

FIGURE 20.1.4. Erector spinae muscles of the thoracic region. (From Ward RC, ed. *Foundations of Osteopathic Medicine, 2nd ed.* Philadelphia: Lippincott Williams and Wilkins, 2003.)

All of these groups are better developed in the cervical and lumbar spine than they are in the thoracic spine. The rotators typically span one to two vertebrae. In the thoracic spine the deep back muscles and the overlying thoracolumbar fascia are covered by other muscles, which are not innervated by the posterior primary rami of the thoracic spinal nerves and which attach the upper limb to the vertebral column. These include the trapezius, latissimus dorsi, and deep to the trapezius, the rhomboids.

Blood supply to the thoracic spine, associated muscles, nerve roots, and spinal cord is derived from 12 pairs of posterior intercostal arteries arising from the costocervical trunk of the subclavian artery (posterior intercostals 1 and 2) and the thoracic aorta (posterior intercostals 3 to 12), which enter and course in the intercostal spaces on the anterolateral thoracic wall. Near its origin each posterior intercostal artery gives off a posterior branch that passes posteriotly with the posterior primary rami of the corresponding thoracic spinal nerve. This branch supplies the back muscles and overlying skin as well as the thoracic spine and contributes to supply the nerve roots, the spinal cord and its coverings, and the thoracic vertebrae through a spinal branch that enters through the intervertebral foramen.

The spinal cord and its coverings (meninges) are located in the vertebral canal of the thoracic spine. The thoracic spine contains the thoracic and upper lumbar spinal cord segments along with their anterior and posterior nerve roots. Thoracic spinal nerves (12 pairs) are formed just outside the intervertebral foramen by the union of the anterior and posterior roots. In the thoracic spine, the spinal nerves are formed below the vertebra they correspond to in number. Thus, the T1 spinal nerve is formed just outside the intervertebral foramen between the T1 and T2 vertebrae. After the spinal nerve is formed, it divides almost immediately into anterior and posterior primary rami. The posterior primary rami innervate segmentally skin and muscles of the back as well as the zygapophyseal joints of the thoracic spine. The anterior primary rami enter the intercostal spaces and become the intercostal nerves innervating segmentally skin and muscles of the anterolateral thoracic and abdominal walls. Situated on the antetior aspect of the necks of the ribs, on each side of the thoracic spine, are the thoracic sympathetic trunks consisting of 11 to 12 ganglia linked together. Each ganglion is connected to an intercostal nerve by two branches. The last ganglion may be connected to both the eleventh and twelfth intercostal nerves.

Arising from the ganglia of the thoracic sympathetic trunks are three splanchnic nerves (greater, lesser, and least), which descend toward the diaphragm on the anterior aspects of the thoracic vertebral bodies.

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20.2

Physical Examination

ROBERT N. PEDOWITZ

OBSERVATION

The examiner should be situated directly in front of the athlete, standing far enough away to view the entire body so that posture can be assessed. Assessment begins by noting the appearance of the spine and related structures. Observe the athlete's posture, both in a seated and standing position. Note any obvious deformities, such as asymmetry on either side of the spine or in an anterior-posterior view, keeping in mind the relationship the thorax has with the trunk and axial skeleton (Fig. 20.2.1).

The areas of the thoracic spine generate power for the lower extremities and stability for the upper extremities. These principles of observation are key for athletes who develop instability patterns, for the dysfunction and its cause may not always be in the same place.

Look at the chest wall and determine if there is right-to-left symmetry. Note any abnormalities such as pectus excavatum (funnel chest), pectus carinatum (pigeon chest), or barrel chest, which may be related to an underlying medical problem. From the side, observe the anterior-posterior diameter for an increase or decrease, and note the right versus the left. From the posterior aspect, note the symmetry from one side of the spine to the other, any curvatures to the spine, any "fullness" of the musculature, or any skin problems, such as rashes or moles. The athlete should first be examined in a standing position, to assess normal posture (Fig. 20.2.2), then in the seated position, to note any "slumping" or bending of the torso, indicating either poor posture or abnormalities of the musculature or spine itself. Correlate any findings with the rest of the evaluation in determining the correct diagnosis and associated treatment.

PALPATION

Begin with the athlete standing. Place the finger pads along the spine, first over the spinous processes, then the transverse processes, and finally over the paraspinal muscles. Feel for tissue

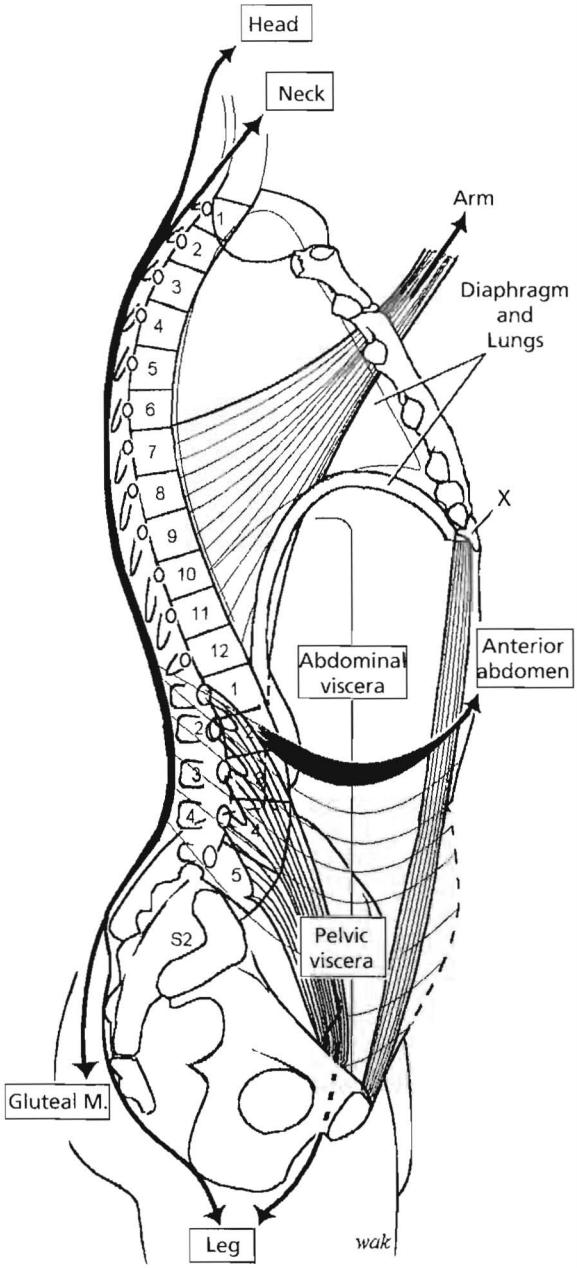


FIGURE 20.2.1. Thoracic region and its relationship to the axial skeleton. (From Ward RC, ed. *Foundations of Osteopathic Medicine,* 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2003.)

texture changes, *a*symmetry of landmarks, *r*estriction of motion, and *t*enderness to palpation (TART findings).

When evaluating the athlete, it is possible to find areas of tenderness while palpating along the spine, which are related to muscles that are in spasm. In considering the diagnosis of a tender point, it is helpful to know where tender points are most common. They may be located posteriorly or anteriorly. Posterior points are found while examining the athlete's back, and may be located either on the tip of the lateral surface of the spinous process or at the tip of the transverse process. Anterior points are found

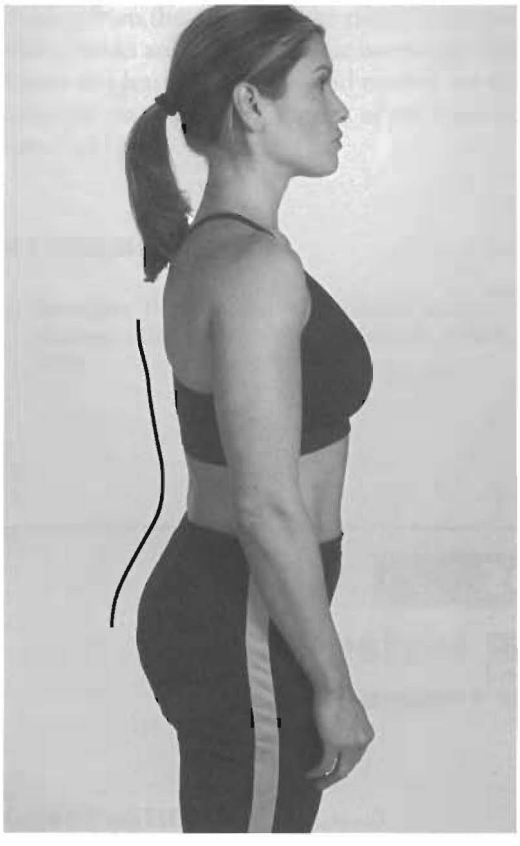


FIGURE 20.2.2. Lateral view of standing posture.

when examining the athlete's front, while assessing the rib cage and other pertinent areas related to the thoracic spine (2). Anterior tender points are listed in Table 20.2.1.

Often, a tender point along the costovertebral junction in the thoracic spine will lead to a tender point on the corresponding anterior sternochondral junction. For instance, an athlete with a tender left T5 dysfunction will likely have a tender point at the left fifth sternochondral junction anteriorly; a dysfunction at one end of the rib attachment leads to dysfunction at the other end.

MOTION TESTING

Gross Active and Passive Range of Motion

To test *passive motion*, the athlete sits in a "natural" seated posture. The examiner places one thumb on the athlete's uppermost thoracic transverse process on the side being evaluated and the forefinger of the same hand over the acromion, cupping the shoulder with the web of his or her hand, and inducing the appropriate motion (Fig. 20.2.3). The examiner's body can be placed against the athlete's back to isolate the thoracic spine and block out accessory movement created by the lumbar spine, cervical spine, or extremities.

Active range of motion can be assessed by asking the athlete to slump over (flexion), arch the back as far as the athlete can (extension), and side-bend with the arms to the side, either sitting or standing, as seen in Figures 20.2.4A to C. Rotating the upper torso would be enough to judge thoracic rotation (Fig. 20.2.4D). Normal

TABLE 20.2.1. ANTERIOR TENDER POINTS OF THE THORACIC CAGE

Tender Point	Location
AT 1	Suprasternal notch
AT 2	Midline on sternal manubrium, above angle of Louis
AT 3–5	Midline on body of sternum across from corresponding rib attachment
AT 6	Midline at xiphisternal junction
AT 7	Midline at tip of xiphoid process or inferior costal margin at same level as tip of xiphoid process
AT 8–9	Midline to a couple of inches lateral in abdominal wall. *Note:* AT 9 is 45 degrees superior and lateral to the umbilicus by 2 in.
AT 10–11	AT 10 is midline, a couple of inches lateral in abdominal wall, and 45 degrees lateral and inferior to the umbilicus by 2 in. AT 11 is at McBurney's point.
AT 12	Superomedial surface of iliac crest in midaxillary line

FIGURE 20.2.3. Passive range of motion of segments (side bending).

TABLE 20.2.2 ACTIVE RANGE OF MOTION (ROM) OF THE THORACIC SPINE

Motion	ROM (degrees)
Flexion	20–45
Extension	25–45
Side bending	20–40
Rotation	35–50

Adapted from Magee D. *Orthopedic Physical Assessment,* 2nd ed. Philadelphia: WB Saunders, 1992: 221–227, 236–237.

ranges of motion for the thoracic spine are listed in Table 20.2.2.

Segmental Motion Testing

While the athlete is seated, the examiner tests the spine for TART, as done for palpation. Next, examine flexion and extension. Place the fingers in the interspinal spaces and feel for changes, or gaps, as you flex or extend at that segment. Gaps widen in flexion and narrow in extension. Note any asymmetry among levels. For side bending and rotation motion, place the pads of your second and third fingers on either side of the specific spinous process, and note changes or gaps as motion is induced.

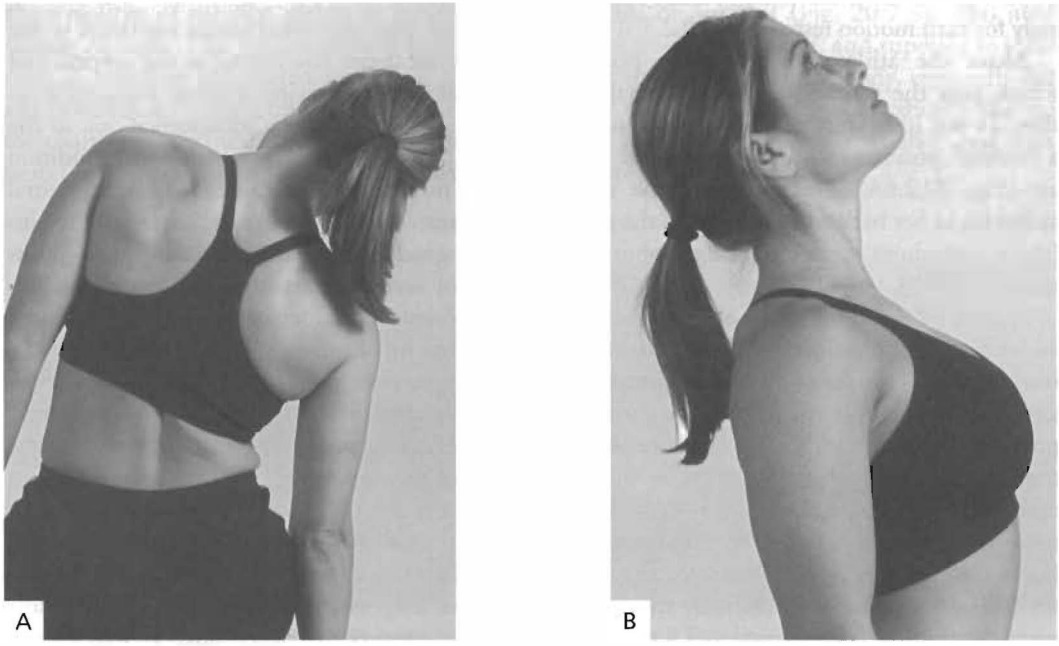
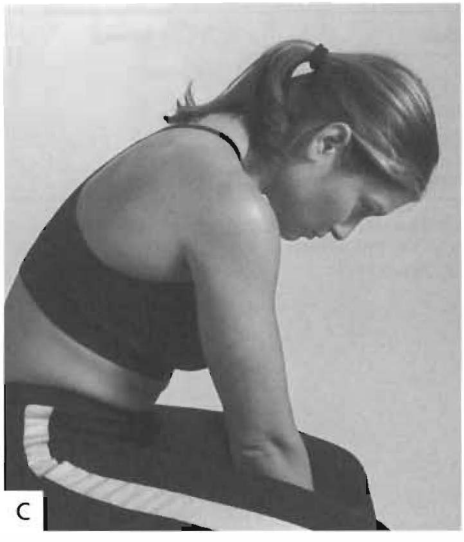


FIGURE 20.2.4. Active range of motion. **A,** Side bending. **B,** Extension (arch back).



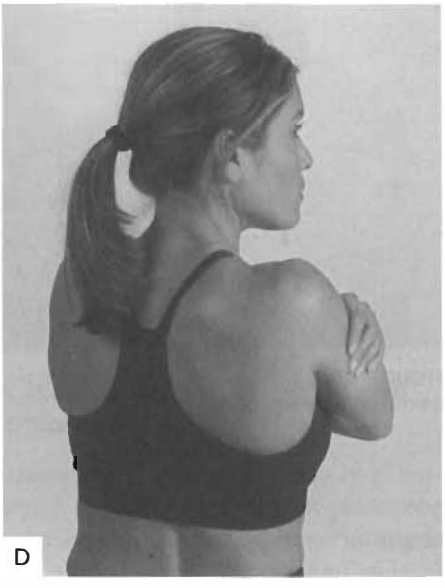


FIGURE 20.2.4. (*continued*) C, Flexion (slump over). **D,** Rotation.

To examine the upper spine (T1-T6), move the athlete's head in the direction you want the spine to move (Fig. 20.2.5A). To examine the lower spine (T5-T12), grasp the athlete's shoulder (from the front) opposite the segment you are palpating and move the athlete's body appropriately for each motion tested (Fig. 20.2.5B) (4).

Move the athlete to the prone position, which puts the thoracic spine in neutral position. To test in flexion, the athlete curls up into a "catlike" position, with the hips raised in the air (Fig. 20.2.6A). For extension, the athlete arches his or her back while leaning on the elbows (this is sometimes referred to as the "sphinx" position) (Fig. 20.2.6B) (7).

In each of the above positions, side bending and rotation are monitored by palpating the intersegmental spaces as mentioned previously, and by pressing on the transverse processes, posteriorly to anteriorly. If there is resistance, tenderness to palpation, or pain at the site, then there is a dysfunction at the segment. For example, if an athlete is in the sphinx position (extension) and the transverse process of T8 on the left is tender and difficult to push toward the right, it is diagnosed as T8 extension, side-bent left, rotated left (T8 E SL RL), per Fryette's second principle.

SCOLIOSIS

Scoliosis is an abnormal lateral curvature to the spine, but it can also extend to the cervical and lumbar areas in varying degrees. Scoliosis is diagnosed most often in adolescence due to the rapid growth spurt, which makes this condition more noticeable. The causes may be structural or nonstructural. Structural changes can involve genetic, idiopathic, or congenital problems of vertebral formation, or loss of flexibility. Nonstructural changes can involve poor posture, inflammation, nerve root irritation, leg-length discrepancy, or hip contracture. Scoliosis is classified by the curve pattern found, which is designated according to the level of the apex of the curve (Fig. 20.2.7). For example, a left thoracic curve would have a convexity toward the left, where the apex sits.

When palpating the spine, note any muscle spasms and changes in the bony and soft tissue contours on either side, as well as any

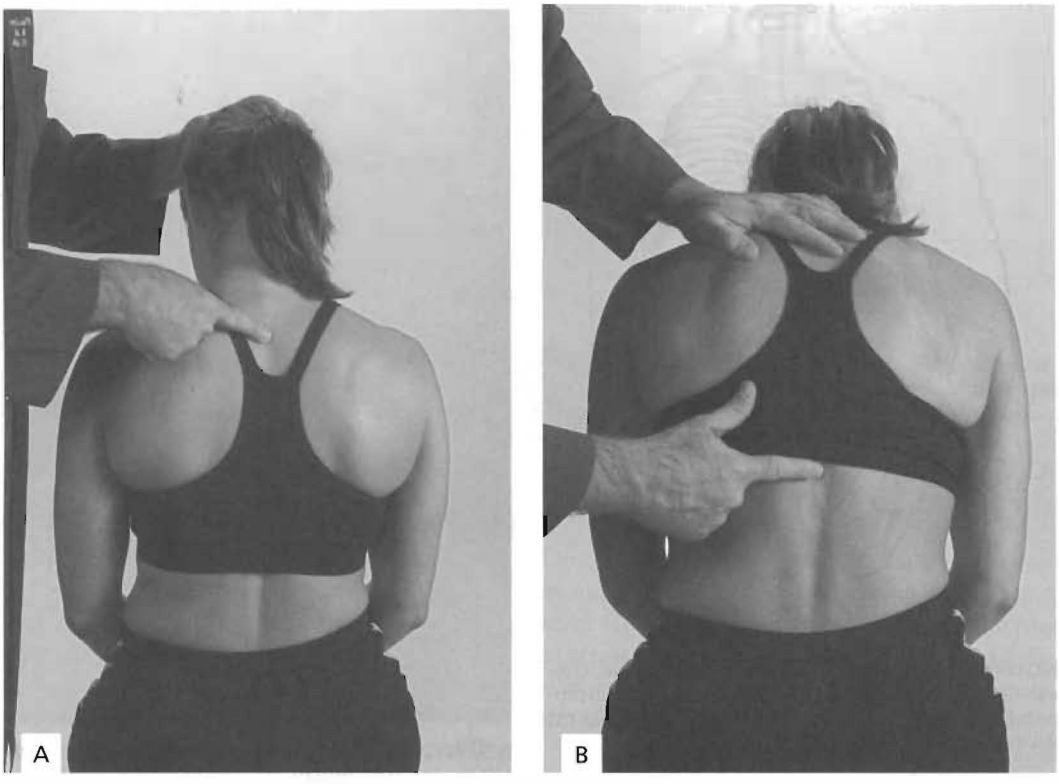


FIGURE 20.2.5. Segmental motion testing. **A,** Upper spinous process T3-T4 (head). **B,** Lower spinous process T9-T10.

abnormalities of the ribs, either in position or during motion. The athlete should be examined in standing and seated positions, noting any postural deficiencies, such as slouching in the chair or not standing straight. Look at the posterior spine as the athlete bends forward to touch his or her toes (Fig. 20.2.8). Also note changes from left to right, and superior to inferior, as you view the entire body, especially the tips of the ears, shoulders (specifically the tip of the acromion process), scapulae, and hips (iliac crest).

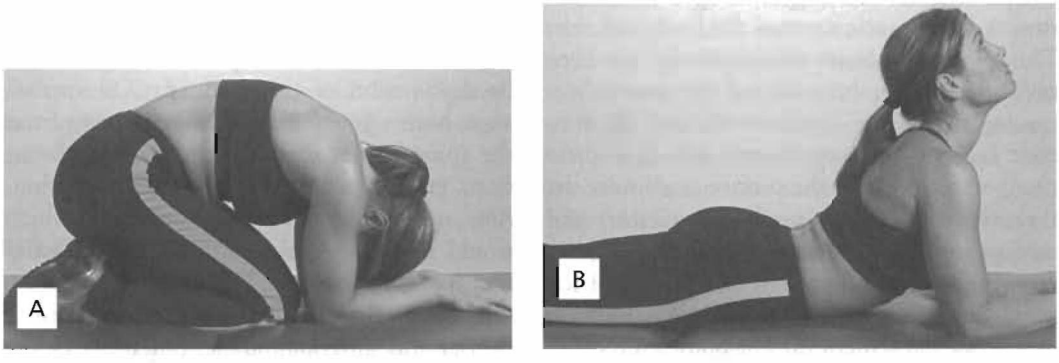


FIGURE 20.2.6. A, Prone thoracic flexion (catlike). **B,** Prone thoracic extension (sphinx).

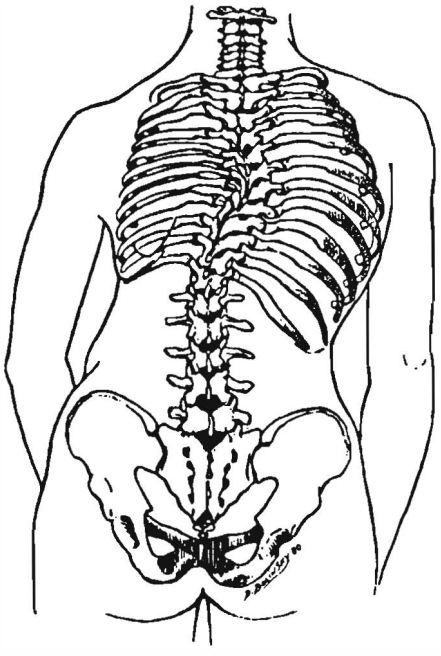


FIGURE 20.2.7. Scoliosis of the thoracic spine, convex right. (From Ward RC, ed. *Foundations of osteopathic medicine,* 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2003.)

FIGURE 20.2.8. Examining the thoracic spine with full flexion (toe touch).

The pathologic process that occurs with scoliosis may have long-lasting clinical impact on the athlete, so prompt recognition and treatment are important. Primary thoracic scoliosis carries the worst prognosis of any type because of its rapid progression and the severity of associated clinical deformities. When there is evidence of a structural scoliosis, the spine appears distorted from the vertebral bodies rotating to the convex side, and this pushes the ipsilateral ribs posteriorly---causing a "hump"—while narrowing the thoracic cage on the ipsilateral side. The spinous processes deviate toward the concave side, shifting the ipsilateral ribs anteriorly—causing a "hollow"—while widening the thoracic cage on the contralateral side (1). These changes can decrease the surface area under the thoracic cage. This can result in respiratory and cardiac problems, such as increased intrathoracic pressure and decreased compliance of the lungs. Severe cases can even cause compression of the vascular structures. The body always tries to compensate for the scoliosis in order to keep the eyes balanced and even with the horizon. This results in changes to the spine, hips, and legs, which may exacerbate pain and other medical conditions.

SCAPULAR DYSFUNCTION

The scapula is an important structure because of its relationship to the thoracic spine. With its muscular attachments to the spine and the rib cage, motion about the spine can be inhibited by dysfunction of either scapula. On appearance, both scapulae should be equidistant from the spine and at equal height, without deviation, including any abduction or adduction. Also, note if one side appears "winged," which would look like a bird with an injured wing that hangs to the side. This condition may be due to long thoracic nerve palsy or weakness of the serratus anterior muscle. Sprengel's deformity occurs when one scapula is congenitally

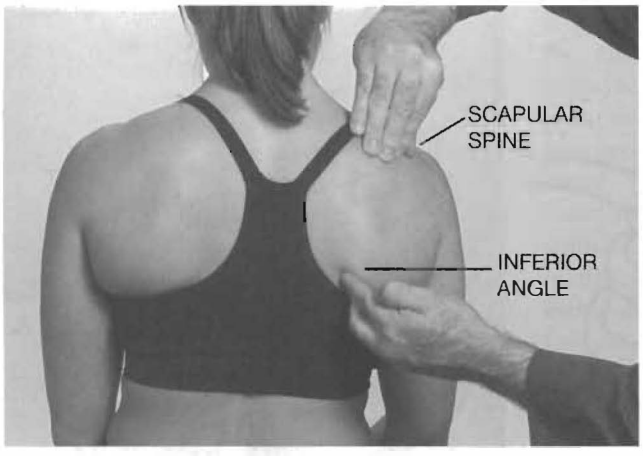


FIGURE 20.2.9. The right scapula, palpating the inferior medial angle with the left hand and the scapular spine with the right hand.

elevated and possibly atrophied, or smaller than the other.

When physically examining the scapula, palpate along the superior, medial, and lateral borders of the bone (5). The acromion and the scapular spine are superior and should be palpated in a lateral to medial direction. The scapular spine is felt just beneath the acromion, and sits at the level of the third thoracic spinous process. Medially, moving downward along the edge of the scapula, feel for any muscle changes (this is the levator scapulae area), such as tender points or tissue texture changes. The medial border should be parallel to the spine and sit about 5 cm lateral to the vertebrae. The inferior angle of the medial border sits at the level of the seventh thoracic spinous process (Fig. 20.2.9).

If the scapula is winged, you may appreciate the medial border coming off the rib cage instead of sitting flat against it, as expected in a normal scapula. The lateral border, which is harder to palpate than the medial due to multiple muscle attachments, should be palpated moving from the inferomedial to the superolateral edge.

RIB CAGE

The thoracic spine has articulations with the rib cage posteriorly, and any dysfunction of the ribs can affect the spine to various degrees. Often a rib dysfunction will cause or correspond with a thoracic dysfunction at the same level. Conditions such as scoliosis affect the ribs in terms of position and motion. Because of the ribs' association with the lungs, rib abnormalities can result in altered respiratory mechanics.

The ribs should be palpated individually and as a group, noting asymmetry, differences in space between the ribs on one side compared with the other, and tissue texture changes. Having the athlete inhale and exhale allows the examiner to assess the motion of the ribs. Keep in mind the rules of rib motion: Ribs 1 to 5 exhibit primarily "pump-handle" motion, whereas ribs 6 to 10 exhibit primarily "bucket-handle" motion. Ribs 11 and 12, which do not articulate with the thoracic cage, undergo caliper motion. Pump-handle rib motion is best palpated by placing the ulnar side of your hand on the athlete's rib cage anteriorly and laterally to the sternum. Bucket-handle motion is best palpated by placing your hands, with fingers pointed laterally, on the anterior rib cage, just below the level of the sternum. Caliper motion is appreciated by putting your hands on the anterior-inferiormost portion of the rib cage, again with fingers pointing outward, and monitoring the ribs as the athlete inhales and exhales. Figure 20.2.10 demonstrates contact points to monitor rib motion.

The first and second ribs are best monitored with finger pads in the first intercostal space.

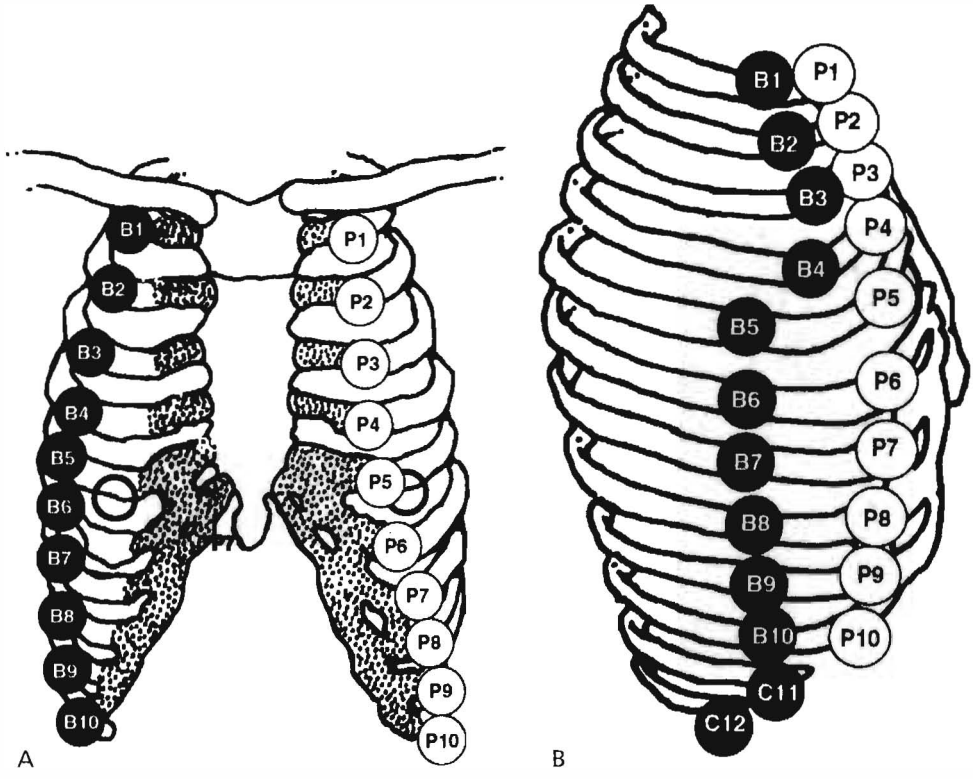


FIGURE 20.2.10. A. and **B.** Rib contact points for palpation and monitoring respiratory movement. view B, Bucket-handle motion; C, caliper motion; P, pump-handle motion.

First rib pump-handle contacts are against the inferior rib margin on either side of the manubrium, while the bucket-handle contact is lateral at a point where the rib passes under the clavicle. The other ribs have pump-handle contacts at the costochondral margins. The other bucket-handle contact points are along the rib shafts in the midaxillary line.

Rib Motion Testing

The athlete can be supine or sitting, although a sitting athlete allows the examiner to flex and extend the spine. The examiner places both hands on the chest wall, checking three areas:

1. Anterior upper bucket-handle motion (Fig. 20.2.11A).
2. Anterior middle pump-handle motion (Fig. 20.2.11B).
3. Posterior rib angles (Fig. 20.2.11C).

In each case, the fingers lightly touch the ribs and follow their motion through inspiration and expiration and compare for asymmetry.

Floating Ribs

The athlete is best examined prone, as the examiner places his or her hands on the lumbar paravertebral and flank region. Sliding the skin up, down, and in circles will unveil the twelfth rib shafts. Allow the ribs to move your hands and follow the hand movement; there is no need for the hands to change positions. Check for symmetry.

NEUROVASCULAR EXAMINATION

In the thoracic spine, there are no specific superficial or deep tendon reflexes to indicate

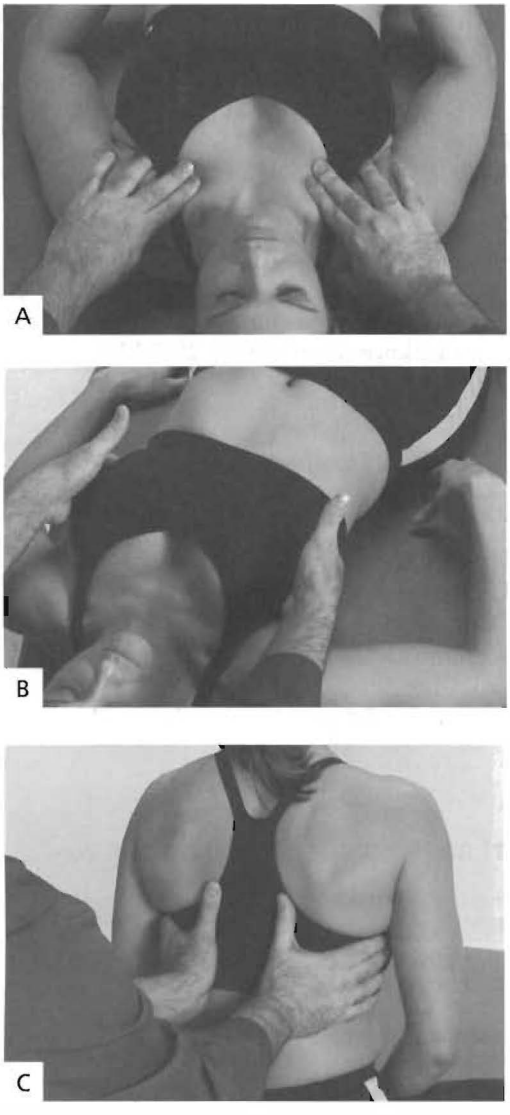


FIGURE 20.2.11. Rib motion testing. **A,** Anterior upper bucket-handle motion. **B,** Anterior middle pump-handle motion. **C,** Posterior rib angles.

nerve root problems. However, there is a great deal of association between sympathetic reflexes and dermatomes (or cutaneous reflexes) and thoracic spine dysfunction.

Dermatomes

The dermatomes that are supplied by the thoracic nerve roots follow the pattern of the ribs (Fig. 20.2.12). The anatomic arrangement of the neurovascular bundle is known by the acronym VAN (*V*ein *A*rtery *N*erve), and it runs along the inferior edge of the rib body. Dermatomes represent cutaneous branches of the dorsal and ventral rami, and each spinal nerve innervates a continuous strip of skin that extends from the posterior midline to the anterior midline (6). There is much overlapping of segments in the thoracic region, where one dermatome may be in the same area as the next.

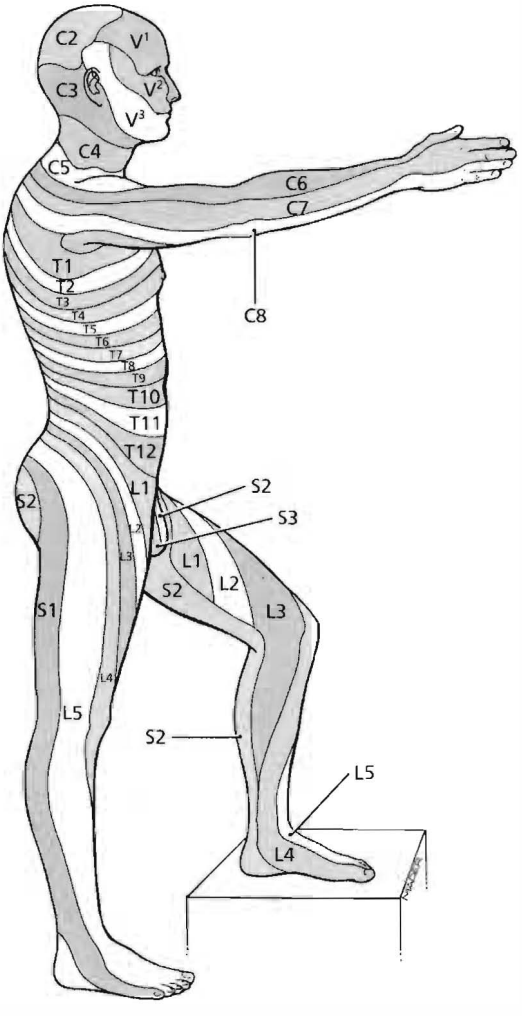


FIGURE 20.2.12. Dermatomes of the Thoracic Cage. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

Note any lesions or pathology that follows a dermatomal pattern. One such disorder of clinical significance is herpes zoster (shingles), which is a viral infection that is embedded in the spinal nerve ganglia. Herpes zoster causes sharp, burning pain in a dermatomal distribution of the nerve it infects, followed shortly after by an erythematous (red) vesicular rash. It usually sits in only one dermatome unilaterally without crossing over the midline.

Sympathetic Reflexes

A somatic dysfunction in the thoracic spine may sometimes correlate with a clinical finding or pathology from a visceral structure. Sympathethic innervations that arise from the thoracic rami supply various organs in the body, and when there is a problem with one of these organs, a viscerosomatic reflex may occur between an organ and the spine (via the nerve pathway). Table 20.2.3 lists visceral organs and/or structures and the corresponding thoracic spine level(s) that are affected by visceral dysfunction.

PROVOCATIVE TESTS AND MANEUVERS

Brudzinski's/Kernig's Test. With the athlete supine and the hands behind the head, he or she flexes the head onto the chest (Brudzinski) (Fig. 20.2.13A) and/or flexes the hip with an extended knee (Kernig) (Fig. 20.2.13B).

Positive test: Pain or stiffness of the head, neck, or low back.

Indicates: Meningeal irritation, nerve root problems, or dural irritation.

Sitting Dural Stretch Test (Slump Test). The seated athlete "slumps" forward with the shoulders sagging and the spine flexed. The examiner flexes the neck of the athlete, then passively extends one knee to 0 degrees (Fig. 20.2.14) (1).

TABLE 20.2.3. VISCERAL ORGANS OR STRUCTURES AND THEIR CORRESPONDING THORACIC SPINE LEVELS

Body Region	Visceral Organ/Structure	Thoracic Spine Level
Head and neck	Pupils, lacrimal and salivary glands, sinuses, carotid body and sinus, thyroid	T1-T4
Thoracic/Chest	Trachea/bronchi, mammary glands, esophagus (lower 2/3), aorta, heart, lungs, pleura	T1-T6
Upper extremity	Arms	T2-T8
Upper abdomen	Stomach, duodenum	T5-T9 L
	Liver	T5 R
	Gallbladder and ducts	T6 R
	Pancreas	T7 R
	Spleen	T7 L
Lower abdomen and pelvic	Right colon, ovary and testes, small intestines, upper ureter	T10-T11
	Kidney	T10-L2
	Adrenal gland	T11
	Appendix	T12 R
	Lower ureter	T12-L1
	Left colon, bladder, uterine body, prostate, genital cavernous tissues	T12-L2
Lower extremity	Legs	T11-L2

Adapted from Kuchera WA, Kuchera ML. *Osteopathic principles in practice,* 2nd ed, rev. Columbus, OH: Greyden Press, 1994:68–69.

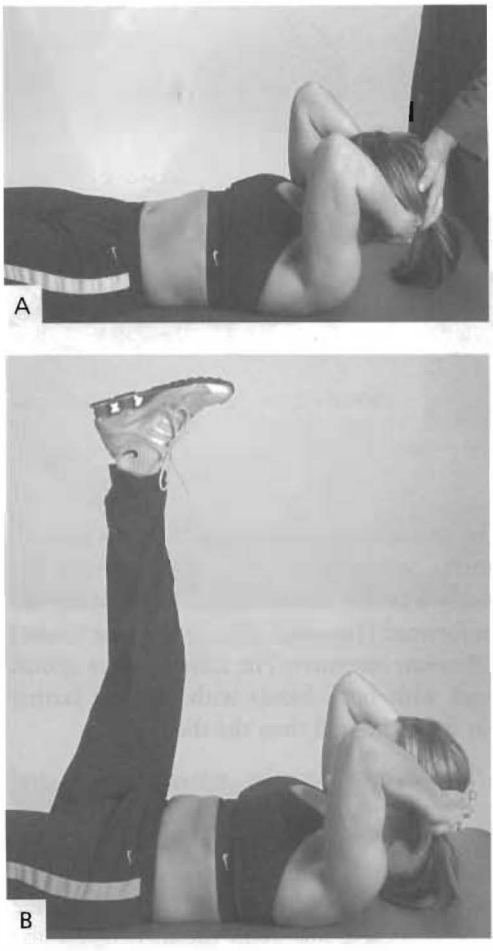


FIGURE 20.2.13. A, Brudzinski's test. **B,** Kernig's test.

FIGURE 20.2.14. Sitting dural stretch test (slump test).

Positive test: Pain on the ipsilateral side at the site of the dysfunction.

Indicates: Impingement of the dura and spinal cord or nerve roots.

First Thoracic Nerve Root Stretch. The seated athlete abducts the arm 90 degrees and flexes the pronated forearm 90 degrees, then the examiner puts the athlete's hand behind the neck (Fig. 20.2.15) (1).

Positive test: Pain in the scapular area or the arm.

Indicates: First thoracic nerve injury, ulnar nerve injury.

Passive Scapular Approximation. The examiner has the athlete lying prone on a table, then

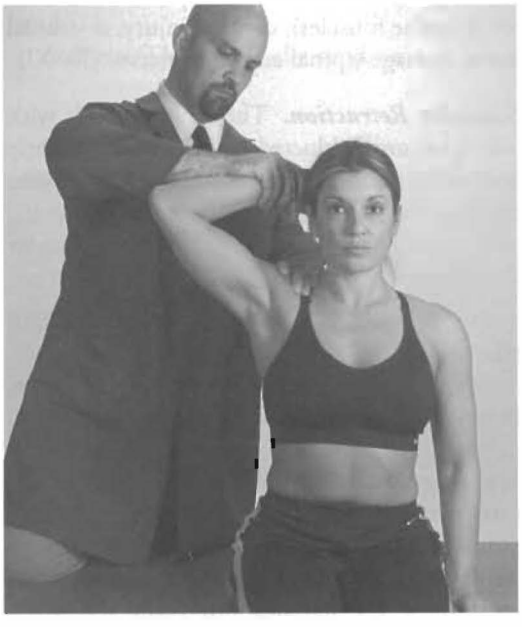


FIGURE 20.2.15. First thoracic nerve root stretch.

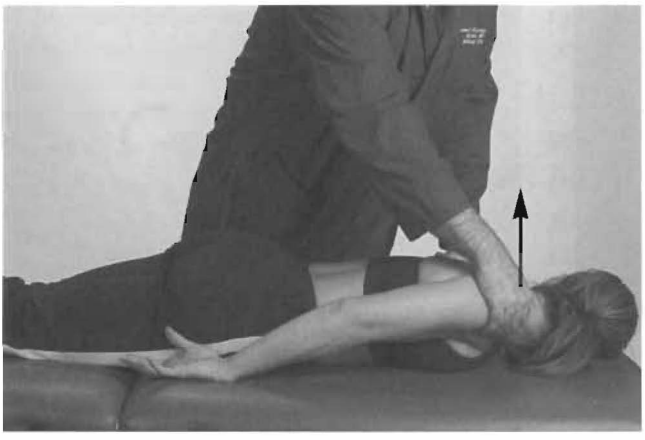


FIGURE 20.2.16. Passive scapular approximation.

passively lifts the athlete's shoulder (scapula) up and back (Fig. 20.2.16).

Positive test: Pain in the ipsilateral scapular area.
Indicates: T1 and T2 neural impingement.

Scapular Elevation. The examiner stands behind the standing athlete and places his or her hands on each acromion. With arms resting at the side, the athlete shrugs his or her shoulders against examiner resistance (Fig. 20.2.17).

Positive test: Weakness or pain against resistance.
Indicates: Tendinitis (of trapezius and levator scapulae muscles), cervical injury, or cranial nerve damage (spinal accessory nerve, CN XI).

Scapular Retraction. The athlete stands with his or her arm adducted and the elbow slightly bent. The examiner stands behind the athlete, cupping his or her hand on the ipsilateral elbow, and has the athlete resist as the examiner abducts to the scapula (Fig. 20.2.18).

Positive test: Painful or weak response during resistance
Indicates: Rhomboids and/or trapezius dysfunction, thoracic spinal dysfunction.

Scapular Protraction (Winging). The examiner stands behind the upright athlete, whose arm is flexed forward, and elbow bent, both at 90 degrees, and places one hand over the thoracic spine for stabilization, while the other hand grasps the proximal aspect of the athlete's forearm and attempts to pull the arm backward, while the athlete resists by moving the arm forward (Fig. 20.2.19).

Alternate maneuver: The athlete pushes against a wall with both hands with the feet farther away from the wall than the shoulders.

Positive test: Scapular winging, pain, and weakness during maneuvers.
Indicates: Serratus anterior muscle weakness. Lower trapezius dysfunction may result in the medial winging of the scapula, partly because the trapezius muscle maintains the medial scapular border close to the vertebral column (5).

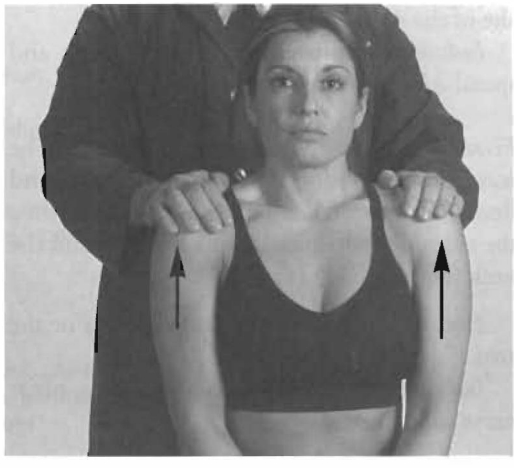


FIGURE 20.2.17. Scapular elevation.

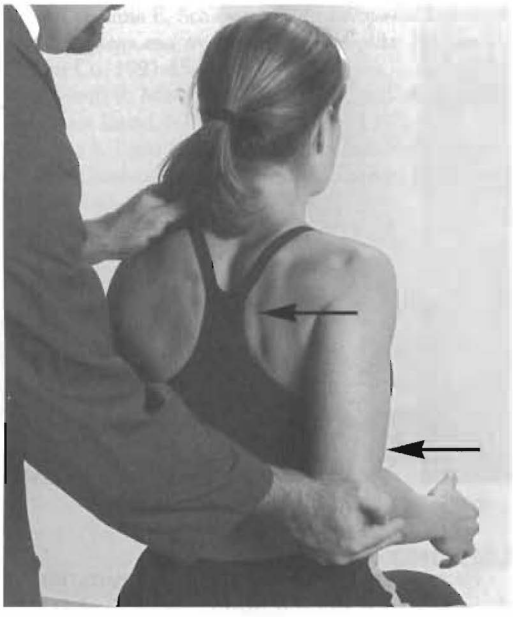


FIGURE 20.2.18. Scapular retraction.

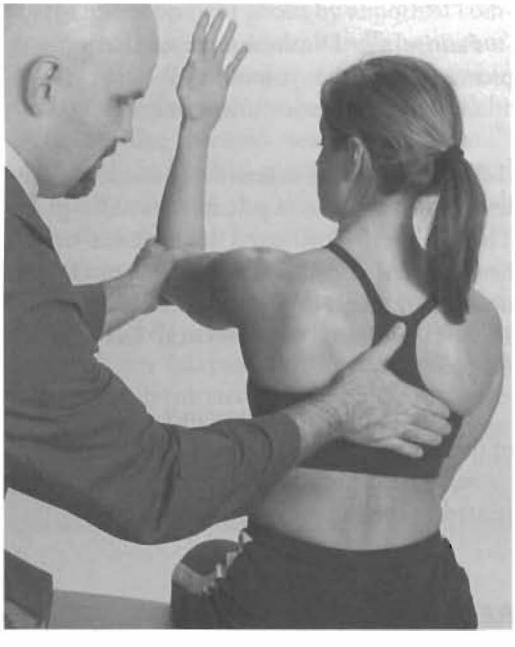


FIGURE 20.2.19. Scapular protraction (winging).

TABLE 20.2.4. PROVOCATIVE TESTS FOR THORACIC OUTLET SYNDROME

Test	Positive Test
Adson	Diminished radial pulse in abduction
Spurling	Pain radiating down the arm from cervical axial compression
Hyperabduction	Pulses diminish with 180 degrees of abduction
Costoclavicular	Pulses diminish with drawing the shoulders down and back (assuming a "military" posture)
Stress	Pulses diminish with hand opening and closing slowly for 3 minutes

THORACIC OUTLET SYNDROME PROVOCATIVE TESTS (Table 20.2.4)

Elevated Arm Stress Test (EAST), or "Hands-up" Test. The athlete externally rotates the shoulders with the elbows slightly behind the head. The athlete then opens and closes his or her hands slowly for 3 minutes (Fig. 20.2.20).

Positive test: Pain, heaviness, or profound arm weakness or numbness and tingling of the hand (1).

Indicates: Thoracic outlet syndrome.

FIGURE 20.2.20. Elevated arm stress test (EAST), or "hands-up" test.

FIGURE 20.2.21. Costoclavicular test.

Costoclavicular Test. The examiner monitors the radial pulse. The examiner draws the shoulder down and back as the athlete assumes a "military" posture (chest out) (Fig. 20.2.21).

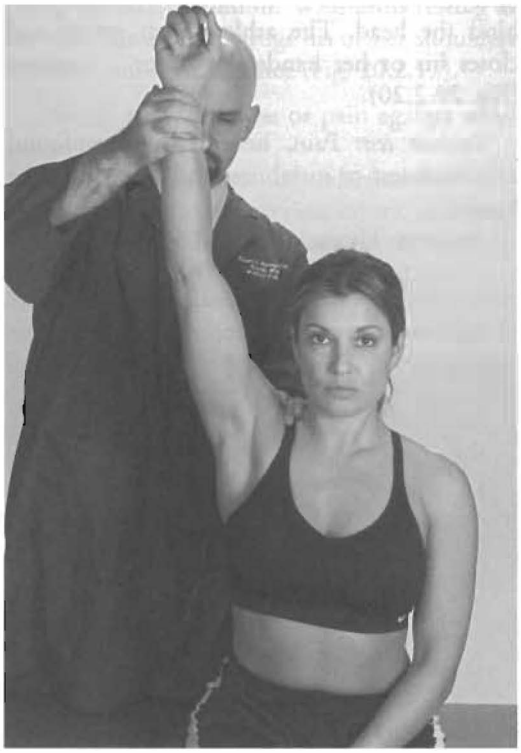


FIGURE 20.2.22. Hyperabduction test. The examiner monitors the radial pulse while elevating the athlete's arms 180 degrees.

Positive test: Disappearance or diminution of the pulse, or if the symptoms are elicited (1).

Indicates: Thoracic outlet syndrome.

Hyperabduction Test. The examiner monitors the radial pulse, then elevates the athlete's arm 180 degrees

Positive test: Diminishment of the pulse or provocation of the symptoms (3).

Indicates: Thoracic outlet syndrome.

Adson's Test. The examiner abducts the athlete's affected arm and palpates the radial pulse. The athlete is instructed to turn his or her head toward the affected side and extend the neck, then take a deep inhalation (see Chapter 16.2, Head and Neck Physical Examination Fig. 16.2.10).

Positive test: The disappearance or diminution of the pulse or provocation of symptoms (3).

Indicates: Thoracic outlet syndrome, usually related to the scalene musculature.

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20.3

Common Conditions

SHERMAN GORBIS

THORACIC OUTLET SYNDROME

Thoracic outlet syndrome (TOS) is a collection of syndromes brought about by abnormal compression of the neurovascular bundle by osseous, ligamentous, or muscular structures between the cervical spine and the lower border of the axilla (1). The involved neurovascular bundle consists of the brachial plexus plus the C8 and T1 nerve roots and the subclavian artery and, rarely, the subclavian vein (Fig. 20.3.1) (1).

Compression of the neurovascular structures can occur in athletes who repetitively hyperabduct their arms. This group can include tennis players (players with a heavy, drooped, and internally rotated arm shoulder may develop neck and arm pain owing to nerve compression [2]), baseball pitchers, swimmers, and volleyball players (1). Women between the ages of 20 and 50 years are commonly affected (3).

FACTORS

Intrinsic. The neurovascular structures must pass through several narrow passageways. When their size and shape are altered, compression occurs. Three regions are most notorious:

1. Anterior scalene, where tightness can occur affecting the triangle formed by the anterior and medial scalene muscles and the first rib, which provides a route for the subclavian artery and nerve plexus.
2. Pectoralis minor, where tightness can affect the border of the pectoralis minor muscle near its attachment to the coracoid process as the neurovascular bundle passes between it and the rib cage. Arm hyperabduction tightens the pectoralis minor and may compress the neurovascular bundle against the rib cage (2,3).
3. Costoclavicular approximation affects the neurovascular bundle as it passes between the clavicle and first rib.

Cervical ribs, which are far less frequent than the above factors, can obstruct the normal path of the neurovascular structures as the spinal nerves exit the neural foramina.

Extrinsic. The most prominent extrinsic factor is trauma, which can cause contusions, clavicular fractures, or whiplash injuries, all which can predispose to TOS (8). In general, anything that compresses the local neurovasculature, with or without trauma, can produce TOS complaints.

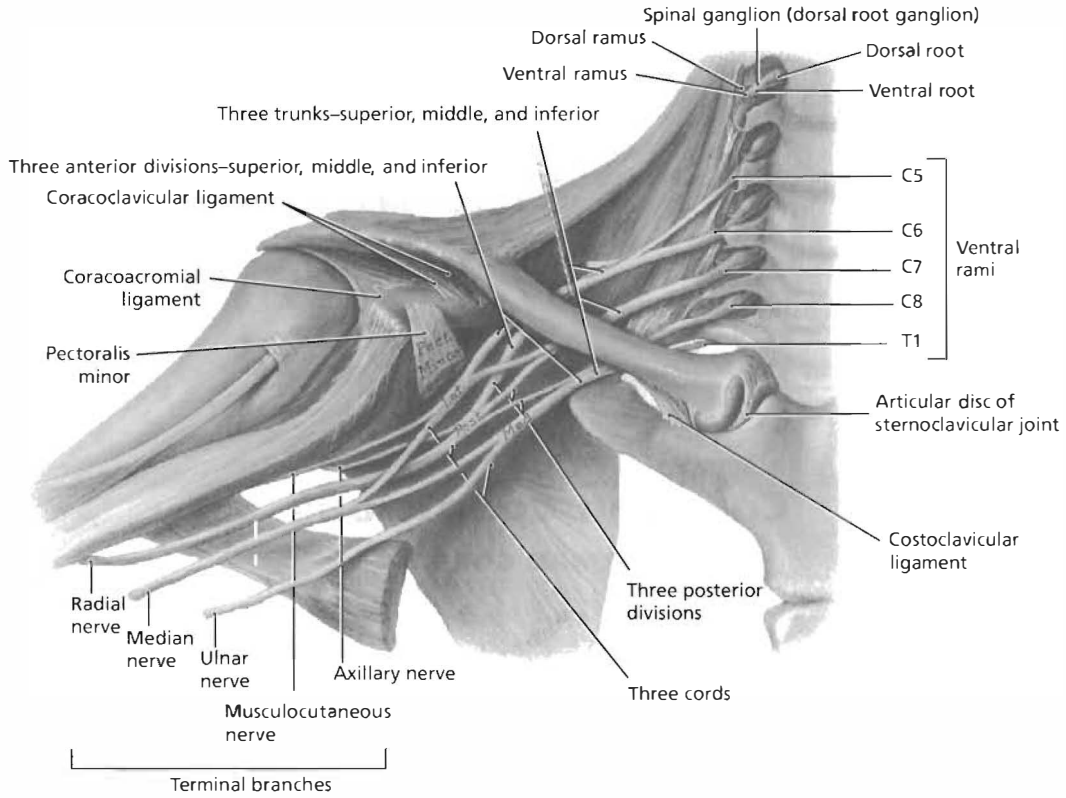

FIGURE 20.3.1. Anatomy of the thoracic outlet and neurovascular bundle. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

History and Physical Examination

A host of symptomatology can be related to TOS, occasionally making the diagnosis difficult to make. Symptoms are classified into two types of TOS: vascular and neurologic subgroups (Table 20.3.1). A third type is often called "disputed TOS," in which symptoms do not fit neatly into any category (7,8).

Vascular symptoms include swelling, puffiness, or heaviness in the arm or hand; bluish discoloration of the hand; pulsating lump over the clavicle; deep, boring toothache-like pain in the neck and shoulder region that increases at night; and superficial vein distension in the hand (1).

Neurologic symptoms can masquerade as many different types of neuropathies emanating from the neck, shoulder, elbow, or wrist. The symptoms common to TOS are paresthesias along the inside forearm and the palm (C8, T1 dermatome), muscle weakness and atrophy of the gripping muscles and small muscles of the hand (thenar and intrinsics), difficulty with fine motor tasks of the hand, cramps of the muscles of the inner forearm, pain in the arm and hand, and numbness and tingling in the neck, shoulder region, arm, and hand (2).

Examination usually reveals tight scalene and pectoralis muscles, which may have taut bands or trigger points that can influence symptomatology. Restricted respiratory motion may be observed on the affected side, particularly in ribs 1 through 5 (10). More advanced cases may show shoulder protraction, scapular restriction, and scapulothoracic dysfunction. Such significant shoulder involvement illustrates the need to restore balance to

TABLE 20.3.1. SYMPTOMATOLOGY OF THORACIC OUTLET SYNDROME

Vascular Symptoms
Swelling or puffiness in the arm or hand
Bluish discoloration of the hand
Feeling of heaviness in the arm or hand
Pulsating lump over the clavicle
Deep, boring toothache-like pain in the neck and shoulder region that seems to increase at night
Superficial vein distension in the hand
Neurologic symptoms
Paresthesia along the inside forearm and the palm (C8, T1 dermatome)
Muscle weakness and atrophy of the gripping muscles and small muscles of the hand (thenar and intrinsics)
Difficulty with fine-motor tasks of the hand
Cramps of the muscles of the inner forearm
Pain in the arm and hand
Tingling and numbness in the neck, shoulder region, arm, and hand

From Kulund DN. *The injured athlete*, 2nd ed. Philadelphia; JB Lippincott Co, 1988.

the agonist-antagonist muscle relationships in the shoulder girdle.

The examination should rule out other conditions that produce referred pain, numbness, or paresthesias into the arm and hand. This includes Pancoast's tumor of the lung, neurofibromas, cervical root compression, cervical spondylosis, cervical disc herniation, and carpal tunnel syndrome.

Provocative tests to localize the area of neurovascular compression include Adson's test, Spurling's test, the hyperabduction test, the EAST test, and the costoclavicular test (see Chapter 20.2).

STANDARD TREATMENT

Conservative treatment typically includes local heat and a program addressing postural retraining and upper extremity flexibility, particularly with restricted pectoralis minor and scalene muscles. Avoidance of provocative maneuvers is crucial in the early stages of treatment. Physical therapy should attempt to treat any cervicoscapular muscle imbalance and scapulothoracic dyskinesia. Modalities such as ultrasound have some benefit.

Corticosteroids can be used, either orally or injected into the affected regions. Scalene or pectoralis blocks can help narrow the diagnosis and possibly release the muscles. Long-term success depends on the athlete's compliance with a home exercise program and modification of activity (10).

MANUAL MEDICINE

Acute. Assess motion of the following structures from proximal to distal. Treat somatic dysfunctions with osteopathic manipulation therapy (OMT):

1. Cervical and upper thoracic vertebrae.
2. First rib superior subluxation with shortening of scalene muscles.
3. Clavicle motion restriction in abduction and flexion.

Subacute. After motion is restored to vertebrae, ribs, and clavicles, reevaluate the thoracic inlet for myofascial restrictions and treat with myofascial release. The athlete can then be placed on a home exercise program to continue stretching the muscles of the cervical and upper thoracic spine, pectorals, and shoulder girdle (3).

Precautions

The cervical spine must be protected at all times when doing techniques, more so if the athlete has cervical osteoarthritis or disc pathology. Any prolonged compression by the clinician on the brachial plexus will likely cause significant discomfort. The operator should avoid abducting the arm over the shoulder or in external rotation (10). Pain levels should be closely monitored, because certain myofascial or indirect techniques may cause too much discomfort for the athlete (10). This also includes stretches performed by either the athlete or the clinician. The athlete should also feel that his or her head is moved through a range of comfort.

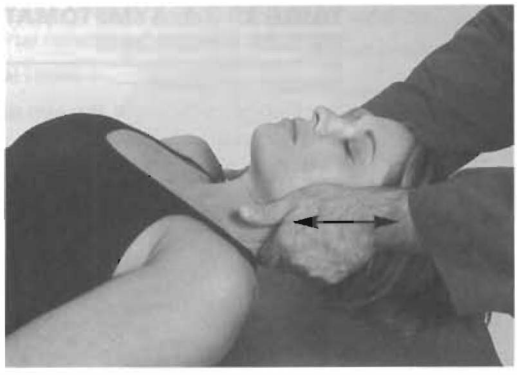

FIGURE 20.3.2. Bilateral-lateral stretch for the cervical soft tissue.

Techniques

Bilateral-lateral Stretch: Cervical Soft Tissue. This treats hypertonic paraspinal soft tissue of the cervical region.

Rationale: This stretch helps to relax the cervical paraspinal soft tissue that limits motion.

1. The athlete is supine, with the clinician standing or seated at the head of the table.
2. The clinician's finger pads bilaterally contact the medial side of the cervical paraspinal musculature, just lateral to the spinous processes.
3. The clinician places simultaneous bilateral-lateral stretch on both sides of the cervical paraspinal musculature. This can be from superior to inferior or inferior to superior. The clinician focuses on the side of greater tissue tension and muscle hypertonicity (Fig. 20.3.2).
4. The clinician may also apply long-axis extension superiorly, simultaneously with the bilateral-lateral stretch by leaning backward through the extended arms (4).

Muscle Energy: Typical Cervical Vertebrae (C2-T1). This treats a single-segment type II dysfunction that is flexed, rotated, and side-bent to the same side.

Rationale: To directly improve the range of motion through the restrictive barriers of extension, rotation, and side bending to the left (this can relieve tension at cervical nerve roots).

1. The clinician's left index finger contacts the articular pillar of C6, and the right hand supports the athlete's head.
2. The extension barrier is found by lifting C6 anteriorly with the left index finger until tension is felt.
3. The clinician's right hand is placed on the right side of the athlete's head to stabilize and to be used later to resist right side bending. Using the left finger contact, C6 is translated to the right until the left side-bending barrier is palpated (Fig. 20.3.3).
4. Using the right hand, the clinician rotates the athlete's head to the left until tension builds at the C5-C6 segment, signaling the presence of the left rotation barrier.
5. The athlete is asked to attempt to push his or her head gently to the right or to lift it off

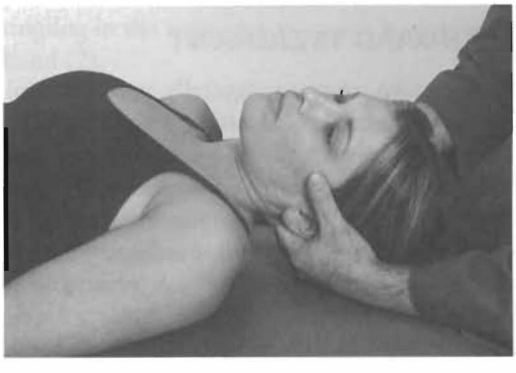

FIGURE 20.3.3. Muscle energy technique for flexed, rotated, and side-bent right (FRS R) at C5-C6.

the table. The effort is maintained for 3 to 5 seconds.
6. Relax, reposition, repeat, and reassess (5).

Muscle Energy: Typical Cervical Vertebrae (C2-T1). This treats a single-segment type II dysfunction that is extended, rotated, and sidebent right (ERS R).

Technique. Same as above, but the cervical spine is flexed into the barrier instead of extended.

Functional Technique. This can be applied in place of the muscle energy techniques on the cervical spine.

1. The clinician supports the athlete's head and upper cervical spine with the index and middle fingers over the dysfunctional segment (Fig. 20.3.4).
2. The clinician seeks the dynamic balance point (point of maximal ease) in the following planes: anterior-posterior translation, left-to-right/right-to-left translation, and rotation bilaterally.
3. Distraction/compression is attempted at the dysfunctional segment. Continue that which maintains the dynamic balance point.
4. Motion is initiated to the direction of ease and followed until tissue tension releases. Reassess (4).

Muscle Energy to the Sternoclavicular Joint

Rationale: These two techniques treat restrictions in abduction and horizontal flexion of the sternoclavicular (SC) joint. They increase the space between the clavicle and first rib through which the neurovascular bundle passes.

FIGURE 20.3.4. Functional technique for the cervical spine.

Restricted Abduction of the Sternoclavicular Joint

1. The athlete is supine on the table with the dysfunctional SC joint at the edge of the table.
2. The clinician stands on the side of the dysfunction, facing cephalad.
3. The clinician places one hand on the medial end of the dysfunctional clavicle while the other hand grasps the athlete's forearm just proximal to the wrist (Fig. 20.3.5).
4. The clinician internally rotates the dysfunctional uppet extremity into extension off the edge of the table to the resistant barrier while monitoring with the other hand at the SC joint (Fig. 20.3.5).
5. The athlete is asked to perform 3- to 5-second muscle contraction to extend the arm toward the ceiling against resistance.
6. Relax, reposition, and repeat (4).

Restricted Horizontal Flexion of the Sternoclavicular Joint

1. The athlete is supine on the table. The clinician stands on the side opposite the dysfunctional SC joint.
2. The clinician places the cephalic hand over the medial end of the dysfunctional clavicle. The caudad hand grasps the athlete's shoulder girdle over the posterior aspect of the scapula.
3. The athlete's hand grasps the back of the clinician's neck with an extended arm.
4. The clinician engages the horizontal flexion barrier by standing more erect and lifting the scapula.
5. The athlete pulls down on the clinician's neck with a 3- to 5-second muscle effort with three to five repetitions. During this effort, the clinician maintains posterior compression on the anterior aspect of the medial end of the clavicle (Fig. 20.3.6).
6. Retest (4).

Muscle Energy: Superior Subluxation, First Rib. This treats a superior subluxation of the first tib (left-side example).

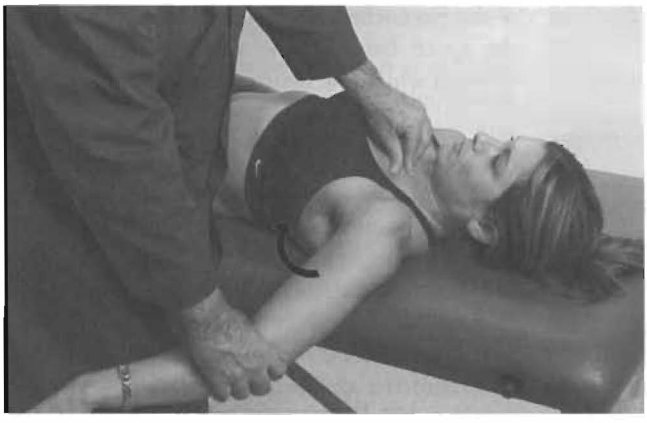

FIGURE 20.3.5. Muscle energy to the sternoclavicular joint.

Rationale: This treats a first rib dysfunction that can lead to difficulty turning the head, which is important in many sports such as football and baseball. First rib dysfunctions can lead to scalene spasm and shortening, which could irritate the neurovascular bundle.

1. The clinician stands behind the seated athlete.
2. The clinician's right foot is on the table; the athlete's right arm is draped over the clinician's right thigh.

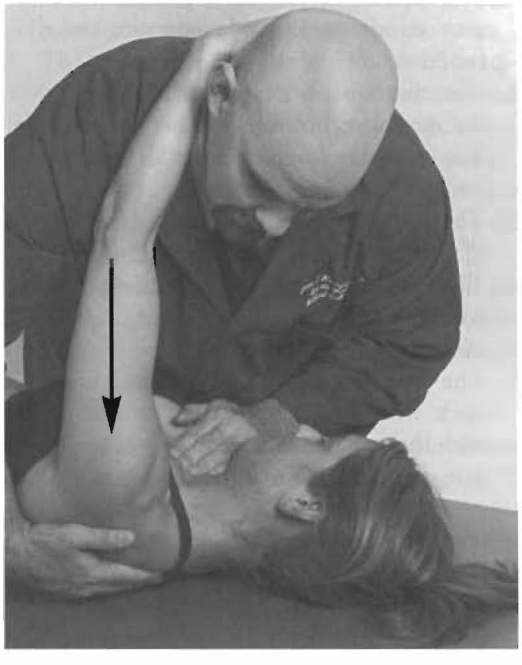

FIGURE 20.3.6. Restricted horizontal flexion of the sternoclavicular joint.

3. The clinician's left-hand fingers overlie the left upper trapezius muscle, which is pulled posteriorly. The clinician's right hand and forearm control the athlete's head and neck.
4. The clinician's left thumb contacts the posterior shaft of the left first rib through the trapezius muscle.
5. The clinician passively rotates and side-bends the athlete's head to the left to unload the left scalene muscles. Caudal pressure is placed on the shaft of the left first rib while a posterior force is maintained on the left trapezius muscle.
6. Using his or her left thumb, the clinician maintains an anterior force on the posterior aspect of the shaft of the first rib to slide it anteriorly in relation to the T1 left transverse process.
7. The athlete actively side-bends his or her head and neck against the clinician's right arm, contracting the right scalene muscles, resulting in reciprocal inhibition of the left scalene muscles. Maintain caudal force on the shaft of the left first rib (Fig. 20.3.7).
8. Relax, reposition, repeat, and reassess.

Muscle Energy: Elevated First Rib (Supine). This directly uses the scalenes to restore first rib mobility and relax the muscle itself.

Direct Myofascial Release to the Thoracic Inlet. Anterior boundaries: clavicles and first ribs; lateral: first ribs; posterior: cervicothoracic

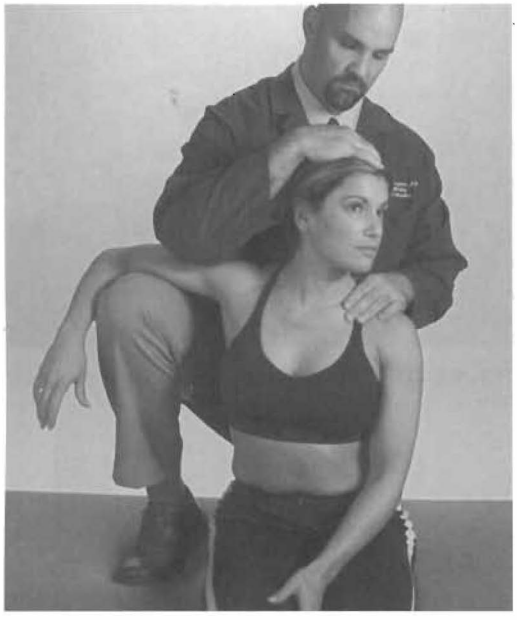

FIGURE 20.3.7. Muscle energy, elevated first rib left, sitting.

[CT] junction). This treats myofascial tension at the thoracic inlet.

Rationale: This allows the range of motion of the clavicles, first ribs, and CT junction to be as physiologic as possible. This also allows for optimal lymphatic drainage.

1. The athlete is seated with relaxed posture, the clinician standing behind.
2. The clinician's hands are placed across the shoulders with the thumbs facing each other at the CT junction.
3. Simultaneous bilateral, anterior, inferior, and circumferential twist and stress are induced across and around the CT junction (Fig. 23.3.8).
4. The clinician takes the tissues to the direction of bind.
5. Use deep inhalation, as a release enhancer, to make the bind tighter.
6. During exhalation, follow the soft tissues through a release.
7. Retest (6).

Myofascial Release, Pectoralis Minor (Right)

Rationale: Pectoralis minor restriction and tension compress the neurovascular bundle passing by the muscle under the coracoid process.

1. The athlete is supine with the clinician sitting at the side of the dysfunctional muscle.
2. The clinician drapes the athlete's right arm over his or her elbow to allow the arm to relax.
3. The right hand palpates the lateral aspect of the pectoralis minor and moves the fingers slowly across the fibers medially, applying pressure the whole time (Fig. 20.3.9).
4. Repeat until the muscle is released.

Myofascial Release: Scalene

Rationale: Scalene restriction compresses the neurovascular structures that pass between the anterior and middle scalenes.

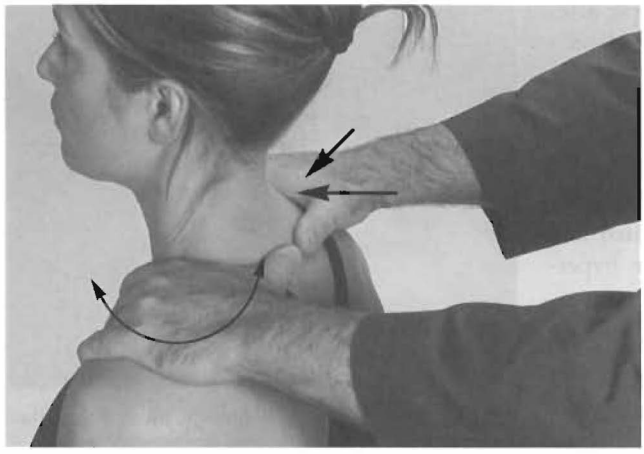

FIGURE 20.3.8. Direct myofascial release to the thoracic inlet (boundaries include anterior: clavicles and first ribs; lateral: first ribs; posterior: cervicothoracic [CT] junction). This treats myofascial tension at the thoracic inlet.

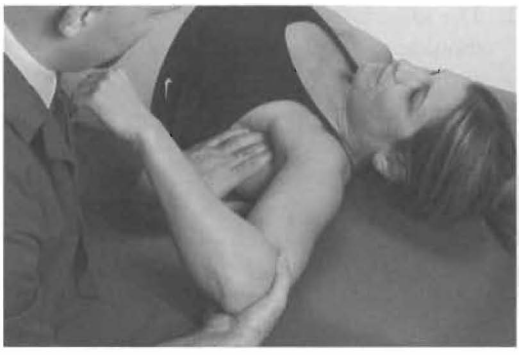

FIGURE 20.3.9. Myofascial release for the pectoralis minor (left).

FIGURE 20.3.10. Counterstrain, protracted shoulder.

Technique. Same as for the pectoralis minor, but the athlete's head is extended off the table and supported by the clinician's knees while the technique is applied to the scalenes.

Counterstrain: Protracted Shoulder

Rationale: A protracted shoulder usually manifests an imbalance with the scapular stabilizers and other shoulder girdle muscles. Without retraction, the glenohumeral joint is not as stable and more stress is shifted to the soft tissues, including the neurovascular bundle.

1. The athlete is prone with the clinician standing opposite the affected shoulder.
2. The clinician brings the athlete's hand of the affected side toward the contralateral knee and rests it on the posterior body.
3. The clinician gently distracts the arm longitudinally with the more distal arm, while the other arm stabilizes the scapula (Fig. 20.3.10).

Muscle Energy: Levator Scapulae Muscle

Rationale: This can contribute to multiple flexion restrictions (extended, rotated, and side-bent [ERS]) dysfunctions that are rotated and side-bent to the ipsilateral side of the hypertonic muscle) affecting C2-C4.

1. The athlete lies on the left shoulder.
2. The athlete's left arm is positioned so that the head can lie over the upper arm and side-bend to the left, with the cervical spine positioned into flexion and left rotation toward the table.
3. The athlete's right arm is placed with the hand resting on the right hip.
4. The clinician stabilizes the athlete's cervical spine by placing the right hand over the right articular pillars of C1-C4. The left hand is used to depress and posteriorly tilt the athlete's right scapula, aligning the left forearm and wrist with the fiber direction of the levator scapulae (Fig. 20.3.11).
5. The athlete is asked to lift the right shoulder against the clinician's resistance for 3 to 5 seconds.

FIGURE 20.3.11. Muscle energy, levator scapulae muscle.

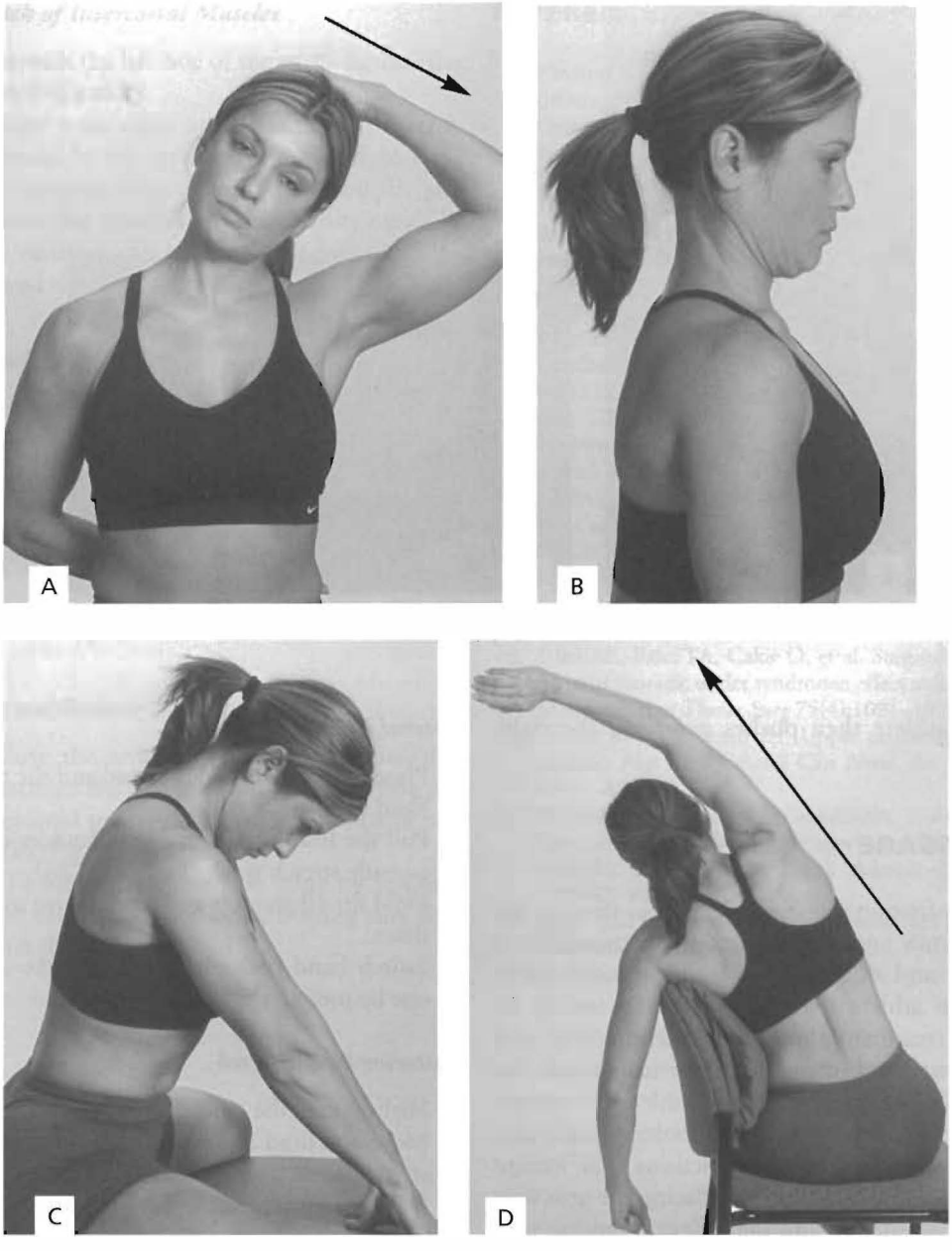

FIGURE 20.3.12. Stretches of the thoracic region muscles: **A,** Lateral neck; **B,** posterior neck; **C,** paraspinals of cervicothoracic region; **D,** intercostals.

6. After relaxation, the shoulder is further depressed inferiorly with an additional posterior tilt, while the head is further rotated to the left.
7. Relax, reposition, repeat, and reassess.

Modification. This technique can be done supine, so that to treat the right levator, the clinician's left arm cradles the athlete's head while the right hand depresses the scapula.

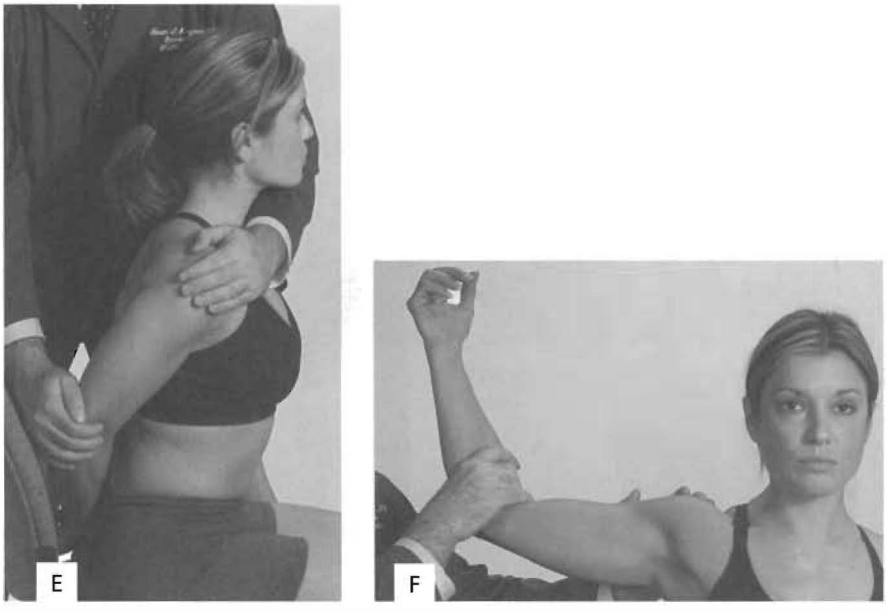

FIGURE 20.3.12. (*continued*) E, serratus anterior; **F,** external rotator.

The athlete then pushes gently to the right (12).

MASSAGE

Cross-friction massage is helpful in treating the pectoralis minor and scalene restrictions. Deep tissue and shiatzu may be too uncomfortable for an athlete if the spasm is too tender, so each treatment should be individualized and discussed with the athlete. Internally rotate the arm during massage if the athlete is uncomfortable. Massage to the shoulder girdle can help relax soft tissue restrictions that irritate the neurovascular bundle. Placing the arm into internal rotation and slight flexion reduces tension while massaging.

STRETCHES FOR THE CERVICOTHORACIC REGION

Postural retraining and the following stretching exercises can be helpful in the prevention of TOS.

Lateral Neck Stretch

1. Place the left hand on the head and the right hand behind the back.
2. Pull the head toward the left shoulder until a gentle stretch is felt (Fig. 20.3.12A).
3. Hold for 10 seconds and repeat three to five times.
4. Switch hand positions and repeat the exercise by pulling the head to the right.

Posterior Neck Stretch

1. Slightly tuck the chin.
2. Move the head straight back until a gentle stretch is felt (Fig. 20.3.12B).
3. Hold for 10 seconds and repeat three to five times.

Stretch of Paraspinals of Cervicothoracic Region

1. Tuck the chin in and flex or bend the head forward.
2. Stretch should be felt in the posterior cervical and possibly thoracic musculature (Fig. 20.3.12C).

Stretch of Intercostal Muscles

1. Sit with the left side of the trunk against the back of a chair.
2. Raise your right arm overhead. A stretch should be felt in the area of the right ribs/intercostals (Fig. 20.3.12D). Good for any sport that involves upper extremity use.
3. A variation can be done on a Swiss ball instead the side of a chair.

Stretch of Serratus Anterior Muscle

1. Sit with the right hand on the sacrum and the right elbow bent.
2. The clinician pushes the right shoulder blade/scapula upward and toward the spine (Fig. 20.3.12E).
3. A stretch may be felt in the right ribs/intercostals area. Good for any sport that involves upper extremity use.

External Rotator Stretch

1. Move the arm into external rotation (this stretches the pectoralis minor by moving the coracoid process away from the muscle origin) (Fig. 20.3.12F).
2. The same stretch can be done by pushing the shoulder into a table, making sure first that the table is soft.

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21

LUMBOSACRAL SPINE

21.1

Anatomy

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The lumbosacral spine, consisting of the five lumbar vertebrae, the sacrum, and the coccyx, contains the cauda equina (lumbar, sacral, and coccygeal nerve roots) whose branches supply the lower limb, gluteal region, and perineum. This region of the vertebral column also provides mobility for the back and support for the upper body, and transmits the weight of the upper body to the pelvis and the lower limb (Fig. 21.1.1). The lumbar spine is convex anteriorly, while the sacrum has an anterior concavity. Anatomy of the lumbosacral spine is presented in detail in major anatomic textbooks (1–6).

The lumbar vertebrae (L1-L5) are larger and heavier than those in other regions of the vertebral column. The vertebral body is considered massive and displays a kidney shape when viewed superiorly. The body, together with the strong pedicles and laminae that form the neural arch, surrounds the triangle-shaped vertebral foramen. This foramen is larger than in the thoracic spine and smaller than in the cervical region. The laterally projecting transverse processes are long and slender and are situated anterior to the articular processes. The superior articular process has a facet directed posteromedially and a mamillary process on its posterior surface. An anterolaterally directed facet characterizes the inferior articular process. Approximately halfway between the superior and inferior articular process is the pars interarticularis. A bony defect at this location is referred to as a spondylolysis. The spinous processes are short and sturdy and are often referred to as looking "hatchet-shaped." The interspace between the L4 and L5 spinous processes serves as an excellent reference point from which to identify the other lumbar vertebrae and the sacrum. This interface is situated at the same level as the most superior aspects of the iliac crests.

The large wedge-shaped sacrum is composed of the five fused sacral vertebrae. The sacrum provides stability and strength to the pelvis and transmits the weight of the body to the pelvic girdle and the lower limbs through the sacroiliac joints. The superior surface of the S1 vertebra forms the base of the sacrum. It has superior articular processes whose facets articulate with the facets of the inferior articular processes of the L5 vertebra. The anterior edge of the body of the S1 vertebra is called the sacral promontory. On the pelvic and posterior surfaces of the sacrum are four sacral foramina for the exit of the anterior and posterior primary rami of the first four sacral spinal nerves and accompanying vessels. The pelvic surface of the sacrum is smooth and concave with four transverse ridges indicating the points of fusion of the sacral vertebrae. The posterior surface of the sacrum is rough and convex and has a median sacral crest formed by the fusion of the first three sacral spinous processes. Inferior to the median crest is the sacral hiatus, which results from the absence of the laminae and spinous processes of the S4 and S5 sacral vertebrae. The hiatus leads into the inferior end of the vertebral canal. The sacral cornua (horns) project inferiorly on each side of the sacral hiatus. The lateral surface of the sacrum

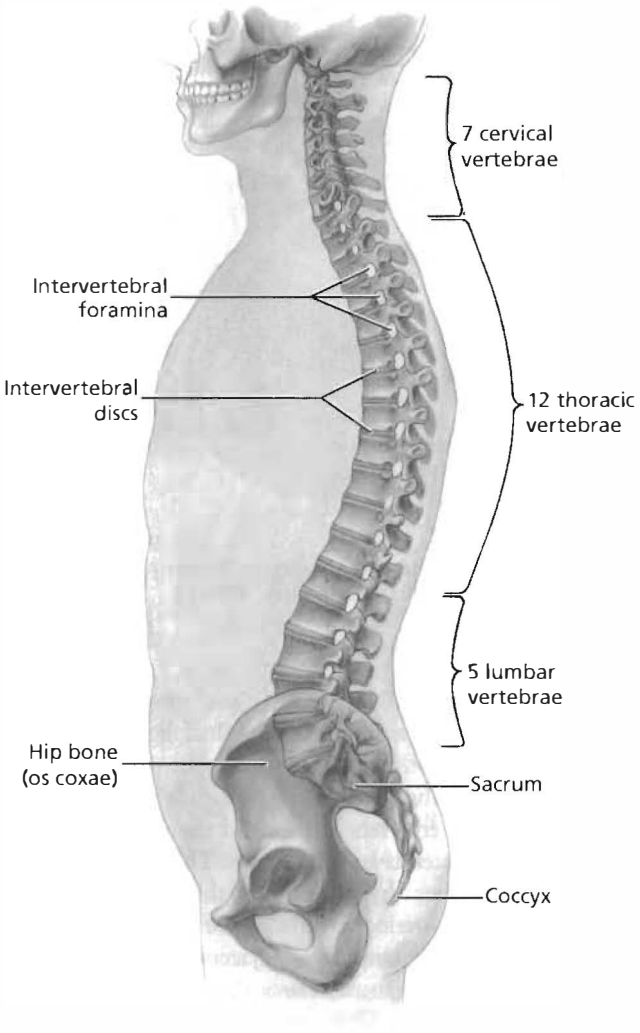

FIGURE 21.1.1. Lumbosacral spine. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy*, 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

presents an articular surface that participates in the formation of the sacroiliac joint. At the inferior end of the lateral surface, at the point where the sacrum tapers toward its apex at the coccyx, is the inferior lateral angle. The coccyx is a remnant of three fused coccygeal vertebrae.

The lumbosacral spine is composed of two types of joints, which work together to provide mobility. These include the facet joints (zygapophyseal joints) and the intervertebral body joints (intervertebral discs). The vertebral bodies and intervertebral discs (anterior segment) are designed for support, weight bearing, and shock absorption. The arch and facet joints (posterior segment) are designed to protect the spinal cord and provide for movement. Branches arising from the posterior primary rami of the spinal nerves innervate these joints.

The intervertebral discs are fibrocartilaginous joints, designed for weight bearing and strength, situated between the bodies of adjacent vertebrae (Fig. 21.1.2). Each intervertebral disc consists of an outer anulus fibrosus, composed of concentric lamellae of fibrocartilage, which surrounds a gelatinous nucleus pulposus. The anuli insert into the rounded rims on the articular

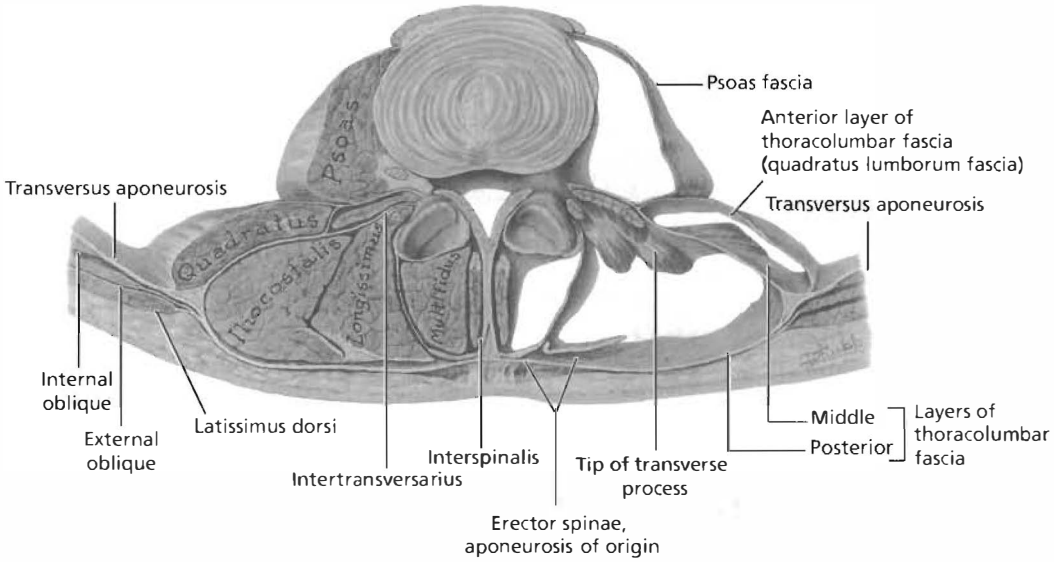

FIGURE 21.1.2. Lumbar musculature and intervertebral disc, transverse section. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy*, 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

surfaces of adjacent vertebral bodies. The most inferior functional intervertebral disc is between L5 and the sacrum. In the lumbar region the discs are the thickest, particularly the anterior portions, when compared with those in other regions of the vertebral column. Branches arising from the anterior primary rami of the spinal nerves innervate the intervertebral discs. The intervertebral discs are avascular structures that receive their blood supply by diffusion from the vertebral bodies (Fig. 21.1.3).

Movements in the lumbar spine consist of lateral bending, flexion, and extension. Extension is most marked in the lumbar region, when compared with other regions of the vertebral column, and is usually more pronounced than flexion in this region of the vertebral column.

Ligaments stabilize the facet joints and intervertebral body joints and support the lumbosacral spine. The anterior longitudinal ligament is a strong, broad fibrous band that covers and connects the anterior aspects of the vertebral bodies and the intervertebral discs. It extends onto the pelvic surface of the sacrum. This ligament maintains stability of the intervertebral body joints and helps to prevent hyperextension of the vertebral column. The posterior longitudinal ligament is narrower and somewhat weaker than the anterior longitudinal ligament. It extends along the posterior aspects of the vertebral bodies inside the vertebral canal. It is attached to the intervertebral discs and the vertebral bodies and extends to the sacrum. This ligament helps to prevent hyperflexion of the vertebral column and posterior protrusion of the intervertebral discs. The laminae of adjacent vertebrae are connected by broad elastic bands called the ligamenta flava. These ligaments help to preserve the normal curvature of the vertebral column and straighten the vertebral column after it has been flexed. Short and strong interspinous ligaments and a strong cordlike supraspinous ligament join adjacent spinous processes. The supraspinous ligament is at its broadest in the lumbar region and can be palpated. The intertransverse ligaments connect adjacent transverse processes and in the lumbar spine they are membranous and more substantial when compared with those in other regions of the vertebral column.

Because most of the body weight is situated anterior to the vertebral column, many strong muscles are attached to the transverse and spinous processes to support the vertebral column. This is particularly true in the lumbosacral

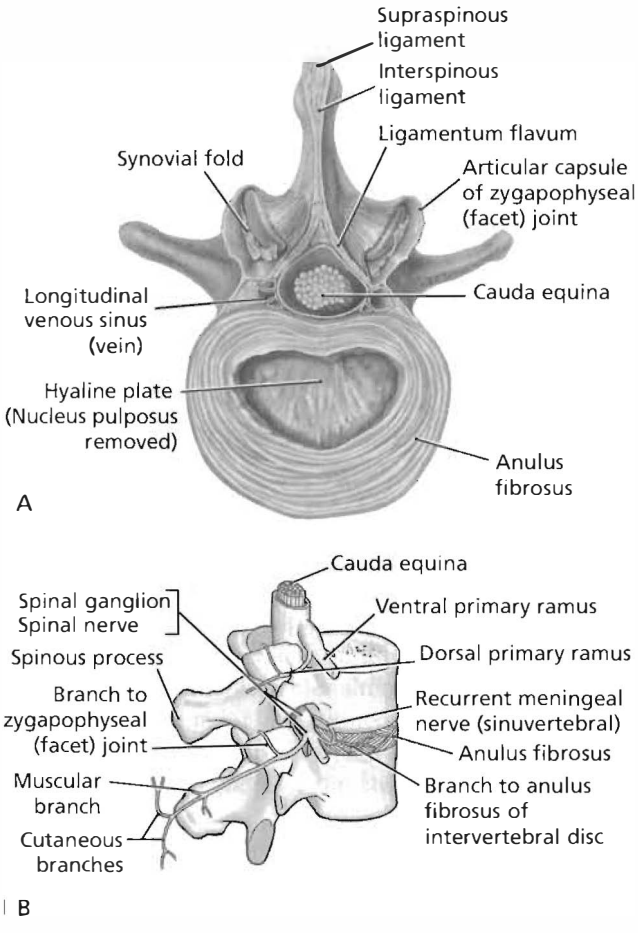

FIGURE 21.1.3. Intervertebral lumbar segment. **A,** Superior view. **B,** Posterolateral view with spinal nerve and ganglion visible. From Moore KL, Agus AMR, Essential Clinical Anatomy. Baltimore: Williams & Wilkins, 1995.

spine. Three groups of muscles are situated in the back: superficial, intermediate, and deep. The superficial and intermediate groups are extrinsic back muscles that control limb movements and respiration, respectively. The most prominent of these extrinsic muscles in the lumbosacral region is the latissimus dorsi, which connects the trunk and pelvis to the upper limb. The deep group of back muscles comprises the intrinsic back muscles that act on the vertebral column to produce movements and control posture. These muscles are enclosed in fascia (thoracolumbar), which in the lumbosacral region is strong and thick and attaches to the tips of the spinous processes, supraspinous ligament, median sacral crest, and transverse processes.

The intrinsic back muscles are divided into three layers: superficial, intermediate, and deep. Muscles in the intermediate and deep layers attach to the lumbosacral spine. The intermediate layer consists of the massive erector spinae, which lies in the trough on each side of the spinous processes and forms a prominent bulge on each side of the midline. The erector spinae is the chief extensor of the vertebral column. Deep to the erector spinae is the deep layer consisting of the transversospinal muscles running obliquely between the transverse and spinous processes. In the lumbosacral spine these muscles include the well-developed multifidus and the rotatores. The multifidus is involved in extension, lateral bending, and rotation while the rotatores act in rotation of the lumbar spine. Branches of the posterior primary rami of the lumbosacral spinal nerves innervate the intrinsic back muscles segmentally.

There are three paired muscles on the posterior abdominal wall that attach to and act on the lumbosacral spine: psoas, iliacus, and quadratus lumborum. The psoas muscle laterally flexes the lumbar spine, is used to balance the trunk when sitting, and when acting with the iliacus, flexes the lumbar spine. The quadratus lumborum extends and is the chief lateral flexor of the lumbar spine. Branches of the anterior primary rami of the L1-L4 spinal nerves innervate these muscles, for the most part. The anterior abdominal wall muscles are key to normal lumbar spine support. Weakness of these muscles results in an abnormal increase in lumbar lordosis and low back pain. These muscles receive segmental innervation from branches of the anterior primary rami of the spinal nerves.

Blood supply to the lumbosacral spine, associated muscles, and nerve roots is derived from four pairs of lumbar arteries and the median sacral artery arising from the posterior aspect of the abdominal aorta. Sometimes the median sacral artery gives off a fifth pair of lumbar arteries. Each lumbar artery gives off a posterior branch that turns posteriorly to supply the musculature of the back and overlying skin and, in turn, gives off a spinal branch to the vertebral column, nerve roots, and meningeal coverings. An anterior branch runs deep to the psoas and quadratus lumborum muscles and supplies the anterolateral abdominal wall.

The spinal cord and its coverings (meninges) are located in the vertebral canal. In the adult lumbar spine the spinal cord, containing L5, sacral (5) and coccygeal (1) segments, usually terminates at the level of the intervertebral disc between the L1 and L2 vertebrae. From this point to the S2 segment of the sacrum, the vertebral canal of the lumbosacral spine contains the dural sac, underlying arachnoid mater, and

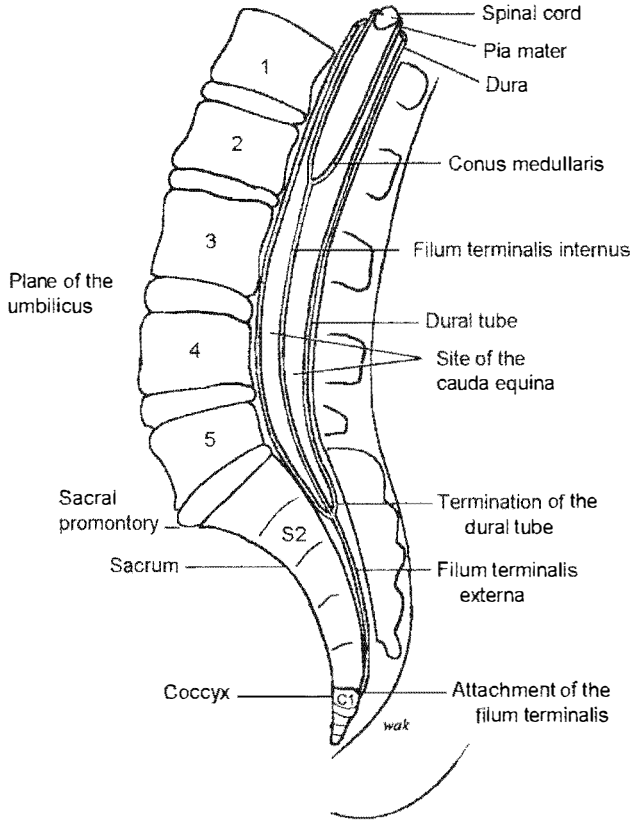

FIGURE 21.1.4. Relationships among the spinal cord, lumbar vertebral column, filum terminalis, and cauda equina, lateral view. (From Ward RC, ed. *Foundations of Osteopathic Medicine,* 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2003.)

the cauda equina bathed in the cerebrospinal fluid of the lumbar cistern (Fig. 21.1.4). The cauda equina are dorsal and ventral roots arising from lumbar, sacral, and coccygeal spinal cord segments which are descending to exit the vertebral column through the appropriate intervertebral foramina and form spinal nerves. The dura mater extends into the intervertebral foramina as dural root (meningeal) sleeves covering the dorsal and ventral roots and ends by blending with the epineurium of the spinal nerve. The inferior end of the spinal cord is called the conus medullaris. From the conus medullaris, a slender strand of pia mater, the filum terminalis, descends among the nerve roots of the cauda equina. At the level of S2 the filum acquires a layer of dura mater as it leaves the inferior end of the dural sac and becomes the coccygeal ligament. This ligament passes through the sacral hiatus and attaches to the coccyx. Each spinal nerve is formed immediately outside the intervertebral foramen and divides almost immediately into an anterior and posterior primary ramus. The posterior primary rami segmentally supply skin and muscles of the back and skin over the gluteal region. Anterior primary rami (L2-S3) form the lumbosacral plexus, which innervates the lower limb, gluteal region, and the perineum. The lumbar portion of the lumbosacral plexus (L2-L4) is formed within the psoas muscle. Emerging from this muscle laterally is the femoral nerve, which descends deep to the inguinal ligament to enter the anterior thigh. Emerging from the psoas muscle medially is the obturator nerve, which courses forward on the lateral pelvic wall and enters the medial thigh through the obturator foramen. A portion of the L4 anterior primary ramus along with the L5 anterior primary ramus forms the lumbosacral trunk, which descends into the pelvis and joins the sacral portion of the lumbosacral plexus. The sacral portion of the lumbosacral plexus (L4-S3) arises from the anterior primary rami of the S1-S3 spinal nerves, which emerge from the pelvic sacral foramina and join the lumbosacral trunk on the posterior wall of the pelvis anterior to the piriformis muscle. The large sciatic nerve leaves the lumbosacral plexus through the greater sciatic foramen, inferior to the piriformis muscle, enters the gluteal region, and descends into the posterior thigh. In the gluteal region the sciatic nerve is located halfway between the greater trochanter of the femur and the ischial tuberosity. In addition to the three major branches, there are several small lumbar and sacral branches given off by the lumbosacral plexus; they supply skin and muscle in the lower limb, gluteal region, and the perineum.

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21.2 Physical Examination

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Lumbosacral pain can have many sources and etiology (1,2). The physical examination works with the history to narrow down the differential diagnoses, so both must be done comprehensively. The history and physical should be thorough to avoid missing a grave diagnosis.

OBSERVATION

The athlete should be standing as the examiner first evaluates posture. This should be examined from the anterior, posterior, and lateral positions to observe any asymmetry or gross deformity. An imaginary line is placed through the midline of the body. From the anterior view, the line should run from the nose, through the sternum, umbilicus, and genitalia. Posteriorly, the spine should serve as the imaginary line. Laterally, the imaginary line extends from the external auditory canal over the greater trochanter to the lateral malleolus. Note any flattening or exaggeration of lumbar lordosis as well as scoliotic curvatures. Excessive stooping or leaning to a particular side could signal an acute process. Uneven innominate heights could suggest a possible leg-length discrepancy or a contralateral lumbar dysfunction. Always look at the knees and feet for the presence of misalignments such as pes planus or cavus, and genu varum or valgum.

Measure symmetry by placing the fingertips on the acromia, anterior superior iliac spines (ASIS), posterior superior iliac spines (PSIS), gluteal folds, patella, and lateral malleoli, and note which ones are higher (Fig. 21.2.1). This is incorporated in the overall musculoskeletal examination, but it is pertinent for the lumbar spine.

GAIT

Gait observation can yield a wealth of information. The analysis should be conducted with a barefoot athlete walking on a flat surface. The examiner should be looking for alterations such as a footdrop, lack of a push-off, restricted hip extension, or overpronation. Observe the motion in the lumbosacral region as the athlete walks. Note any restriction in motion, in particular, the sacroiliac joints and the lumbosacral junction. More information can be found in Chapter 25 on gait analysis.

RANGE OF MOTION

Gross range of motion has nearly as much variability as there are personalities. Despite these vast differences in athletes, knowing the normal ranges of motion aids in identifying the source of the problem and later aids the clinician in checking the response to treatment (3).

Assessment of gross motion of the lumbosacral spine is performed with the athlete standing. Make sure the flexion is coming from the lumbar spine and not in the pelvis, knees, or thoracic spine. The range of active motion for the lumbar spine is 40 to 60 degrees for forward flexion, 20 to 35 degrees for extension, 15 to 20 degrees for side bending, and 3 to 18 degrees for rotation (4).

Lumbar flexion can be restricted but appear to be normal due to compensatory mechanisms. The examiner should see the normal lordotic curve of the lumbar spine disappear as the athlete flexes. He or she can further confirm true lumbar flexion by placing one thumb on

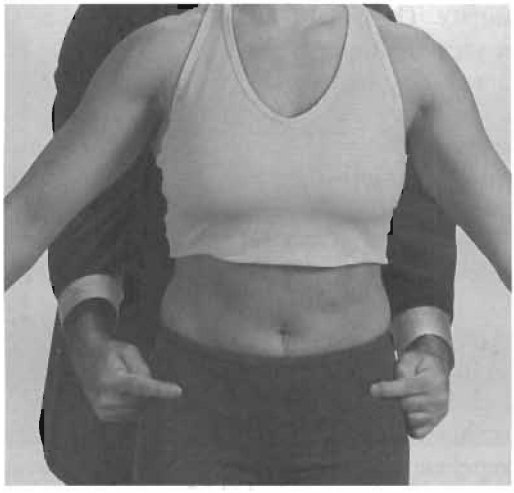

FIGURE 21.2.1. Assessing for unleveling of the anterior superior iliac spine.

the spinous process of L1 and the other on the base of the sacrum, and then see if the thumbs separate (Fig. 21.2.2). The same technique applies to the other planes of motion.

Hamstring flexibility should always be assessed during every athletic lumbosacral examination. With their origin on the pelvis, the hamstrings are a direct link between the lower extremity and pelvis as well as a primary propulsive force in the gait cycle. Hamstrings with reduced flexibility create an abnormal vector force on the ipsilateral innominate bone, resulting in a tendency toward posterior rotation. This has a direct effect on the sacroiliac joint and the lumbar musculature connected to the pelvis (quadratus lumborum, erector spinae).

The popliteal angle is an objective way to measure hamstring flexibility (4). With the athlete supine, the examiner flexes the hip to 90 degrees. The examiner then passively extends the leg to the physiologic barrier. The angle formed at the knee is the popliteal angle (Fig. 21.2.3). The normal range is 120 to 180 degrees, depending on the athlete's age, sex, fitness, and genetics.

The symmetry of motion is just as important as its range. If an athlete is able to sidebend the lumbosacral spine 20 degrees to the right and only 10 degrees to the left, this may indicate focal dysfunction and restriction, which can lead to breakdown along the kinetic chain. The examiner can more closely assess symmetry by comparing the range of a vertebral segment on both the involved and uninvolved sides.

PALPATION

Using the TART system (*t*issue texture changes; *a*symmetry of landmarks; *r*estriction of motion; *t*enderness to palpation), the examiner should

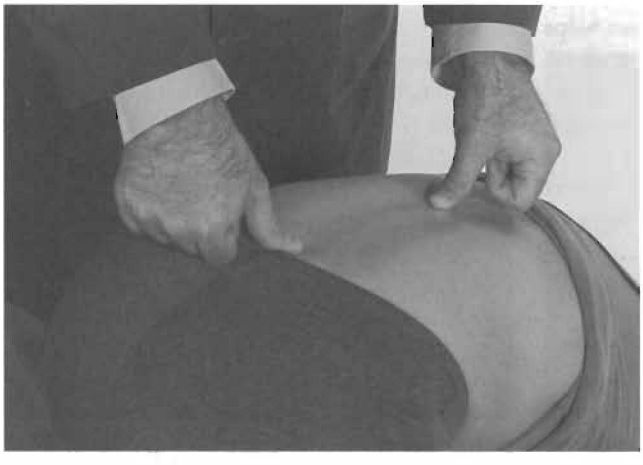

FIGURE 21.2.2. Confirmation of lumbar flexion.

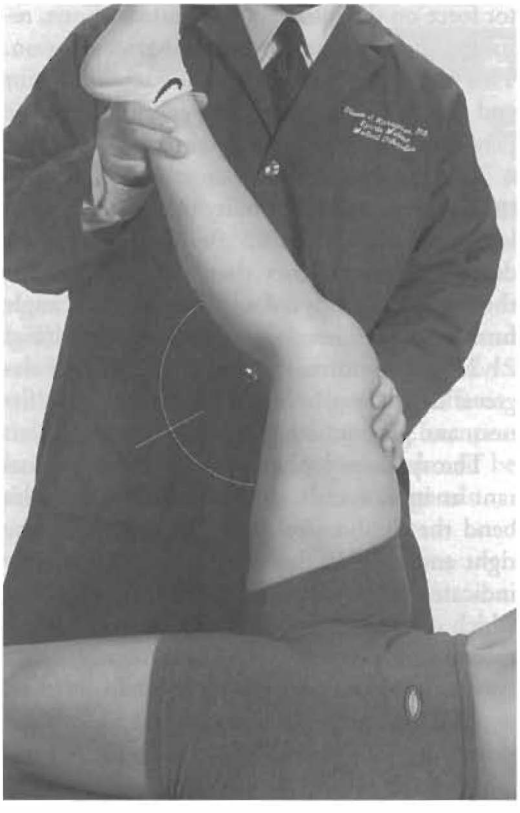

FIGURE 21.2.3. Popliteal angle.

begin by excluding nonmusculoskeletal ailments (6,7,8). Palpating the costoverterbral angles and suprapubic region for tenderness can help exclude infections of the genitourinary tract. Percussion of the spinous processes helps to exclude infections of the vertebrae or discs, such as discitis or osteomyelitis. Palpate the abdomen to look for an aortic aneurysm, pancreatitis, or cholecystitis.

Palpating the tissue gives the examiner a feel for the turgor and tone of the tissue. Regions with increased turgor and tone often have tender points. By palpating these tender points, the examiner can assess the severity of strain patterns and dysfunctions. The fascia can thicken or scar over time, restricting its motion. To palpate the fascia, the examiner puts just enough pressure on the skin so that the fingers can glide the subcutaneous tissue over the smooth fascial surface. The glide should be smooth and nontender; any findings to the contrary should be noted and investigated.

Palpatory findings typically confirm the acute or chronic processes elicited in the history. Acute processes generally have a warm, boggy feel with focal tender points. The tender points in acute processes are usually sharp and severe. In contrast, chronic tender points are firmer, cooler, and more diffuse (6,7).

After assessing the superficial tissue, the examination proceeds beneath the muscle and fascia to the lumbar vertebrae, sacrum, and ligaments. The examiner presses the transverse vertebrae and assesses the ease of motion with right and left rotation. A dysfunction is named for the anterior body of the vertebra in relation to the vertebra inferior and in the direction of ease to which a vertebra moves (7). For example, if L4 rotates to the right more easily (pressing down on the left transverse vertebra) than to the left, it is described as being rotated right.

By palpating the spinous processes of the individual vertebrae, the examiner can identify such conditions as spina bifida occulta or spondylolisthesis, in which the involved spinous process lies more anterior than the remaining vertebrae and spinous processes. Tenderness on the interspinous ligaments may indicate sprain or significant dysfunction. A tender spinous process requires radiographic evaluation, which can demonstrate spina bifida and spondylolisthesis.

The sacrum is the junction between the lumbar spine and pelvis, and its position makes it a common source of dysfunction. Palpate the sacral sulci formed bilaterally with the junction with L5 and note how shallow or deep they are (Fig. 21.2.4). Label the sulcus based on whatever side is dysfunctional. For example, if the seated flexion test is positive on the right and the left sulcus is shallower than the right, one would say "deep right sulcus" rather than "shallow left sulcus." Palpate the inferior lateral angles (ILAs) and note which is more posterior and/or inferior (Fig. 21.2.5). Examine the sacroiliac joints, both superior and inferior poles, and note any tenderness or loss of joint play or spring.

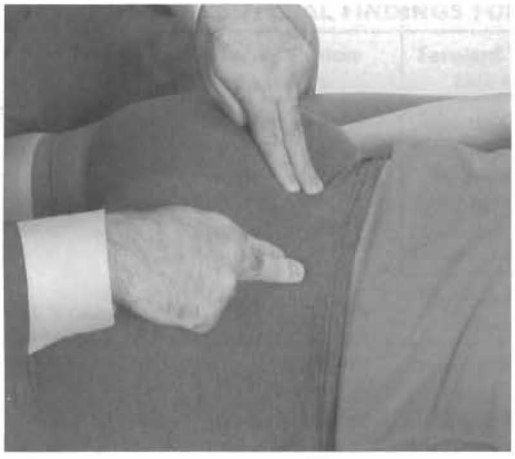

FIGURE 21.2.4. Palpation of the sacral sulcus and the posterior superior iliac spine.

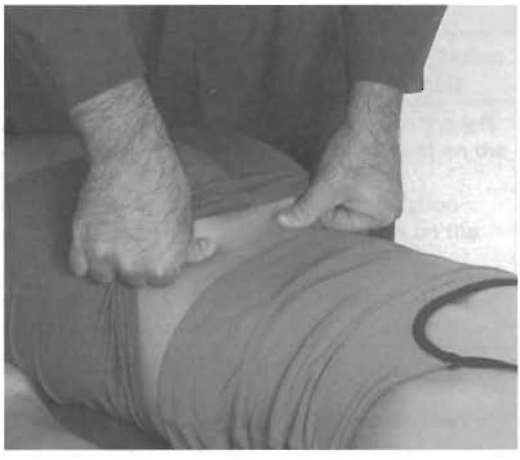

FIGURE 21.2.6. Palpation of the iliolumbar ligament.

Always palpate the posterior superior iliac spines (PSIS) when checking the sacral sulci. Often, pain from this articulation between the iliac crest and sacrum mimics lumbar radiculopathy and can be treated quickly.

Dysfunctions of the sacrum can also lead to dysfunctions of the ligaments and muscles directly connected to the sacrum. The iliolumbar ligament is an extension of the quadratus lumborum and runs from the transverse processes of L4-L5
iac joint. It can be palpated just superior to the PSIS and is deeper than the fascia and muscles (Fig. 21.2.6).

The sacrotuberous ligament runs from the inferior border of the sacrum to the ischial tuberosity. Palpate inferiorly and laterally from the sacral ILAs (Fig. 21.2.7).

Palpating the sciatic notch can give some information about the piriformis, particularly an acute piriformis syndrome, but this maneuver is not necessarily sensitive for piriformis dysfunction because the sciatic nerve may be irritated independently and tender to palpation.

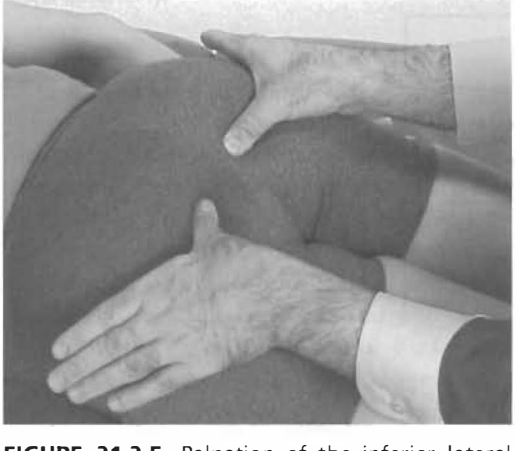

FIGURE 21.2.5. Palpation of the inferior lateral angles of the sacrum.

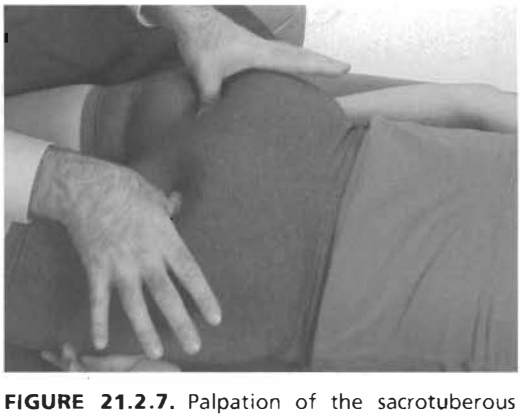

FIGURE 21.2.7. Palpation of the sacrotuberous ligament.

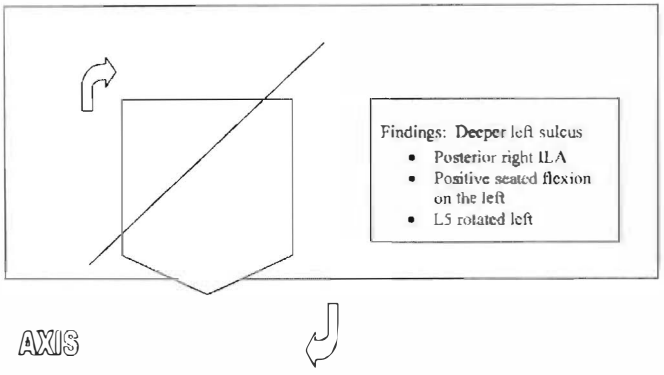

FIGURE 21.2.8. Forward sacral torsion with right axis. ILA, inferior lateral angle.

The relationship between L5 and the sacrum can be assessed. Sacral torsion describes a sacrum that is rotated on an oblique axis and in the opposite direction of L5 (Fig. 21.2.8). This is the opposite motion of L5 relative to the sacrum and causes the torsion (6).

Generally, an L5 dysfunction leads to a sacral dysfunction, so the examiner should look closely at this segment. Three common rules apply (5):

1. The sacral axis is the same as the side bending of L5.
2. L5 and the sacrum rotate in opposite directions.
3. There is decreased motion of the sacroiliac joint at the side *opposite* the axis.

Another common sacral dysfunction is the sacral shear. A sacral shear occurs when the sacrum rotates anteriorly or posteriorly within the sacroiliac joint on a transverse axis (6). Shears are the result of two opposing forces at the sacroiliac joint (7), and are described as being in flexion or extension. Sacral shears can be the result of stepping unexpectedly off a curb or hole, falling on the sacral area, or a short leg (7). Often unilateral flexion or extension of the sacrum is demonstrated (Fig. 21.2.9).

A proficient way to distinguish shears and torsions is to look at the side of the positive seated flexion test. In shears as well as torsions, the sacral base on the dysfunctional side is shallow (shallow sulcus) or deep; the difference between the two is the palpatory findings of the inferior lateral angles (ILAs) of the sacrum. In torsions, one can palpate an ILA that is more anterior or posterior *opposite* the side of the

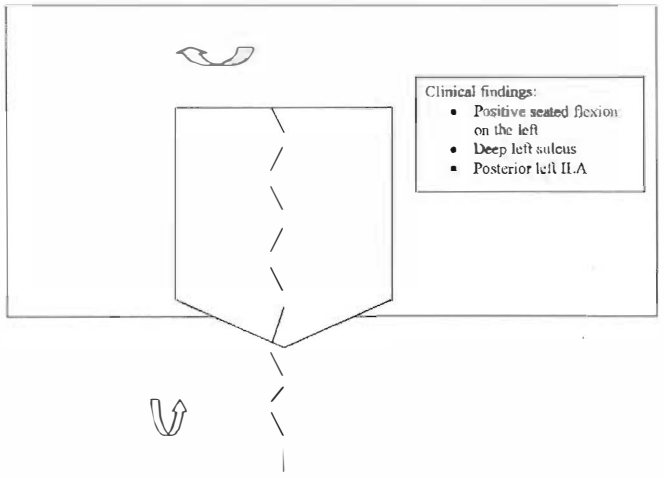

FIGURE 21.2.9. Unilateral flexion or extension of the sacrum. ILA, inferior lateral angle.

TABLE 21.2.1. POSITIONAL FINDINGS FOR TORSIONS AND SHEARS

	Forward Torsion Right Axis	Forward Torsion Left Axis	Posterior Torsion Right Axis	Posterior Torsion Left Axis
Sacral sulcus	Deep on the left	Deep on the right	Deep on the right	Deep on the left
Inferior lateral angle	Posterior on the right	Prominent on the left	Prominent on the left	Prominent on the right
L5 rotation	Left rotation	Right rotation	Right rotation	Left rotation
Seated flexion	Positive on the left	Positive on the right	Positive on the left	Positive on the right
	Left Unilateral Flexion	**Right Unilateral Flexion**	**Left Unilateral Extension**	**Right Unilateral Extension**
Sacral sulcus	Deep on the left	Deep on the right	Shallow on the left	Shallow on the right
Inferior lateral angle	Prominent on the left	Prominent on the right	Deep on the left	Deep on the right
Seated flexion	Positive on the left	Positive on the right	Positive on the left	Positive on the right

positive seated flexion test. With a sacral shear, the examiner places his or her thumbs on the ILAs and notices an abnormality on the *ipsilateral* side. Table 21.2.1 lists the various findings for sacral torsions and sacral shears.

After assessing the relationship between L5 and the sacrum, the evaluation needs to exclude any rotational component of the pelvis. Palpating the PSIS and ASIS and noting their level and relation to the midline can aid in assessing for any rotational component of the innominates. For example, an inferior right ASIS, superior PSIS, and pain over the SI joint could simply be the result of the right innominate rotated anteriorly.

LEG-LENGTH DISCREPANCY

Although the research does not completely support the correlation between a short leg and lumbar pain (3), most practitioners agree that a short leg can have an effect in such problems as gait and running mechanics and economy, standing posture, postural sway, as well as increased incidence of scoliosis, low back pain, osteoarthritis of the hip and spine, aseptic loosening of a hip prosthesis, and lower extremity stress fractures (10). The lumbosacral examination should include a leg-length evaluation.

To check for leg-length discrepancy, do the following:

1. The athlete lies supine on the table with the knees and hips flexed while the clinician holds the ankles.
2. The athlete lifts the hips off the table (Fig. 21.2.10).
3. The athlete sets the hips down while the clinician pulls down on the legs to take up any slack.

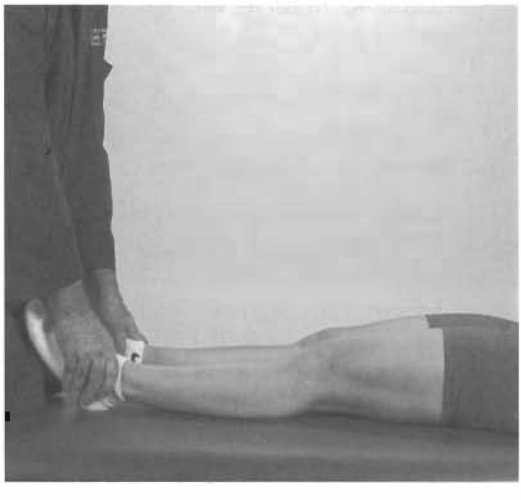

FIGURE 21.2.10. Evaluating leg lengths with the athlete supine.

4. The clinician places the thumbs under the medial malleoli and compares for evenness.

The difficulty with the leg-length discrepancy test is the intrusion of factors that can skew results, such as innominate shears and rotations, sacral torsions, and asymmetrical pes planus, particularly in cases of plantar fascia or posterior tibialis insufficiency. A more precise but time-consuming method is as follows:

1. Manipulation
   a. Pubic symphysis release.
   b. Piriformis stretches and muscle energy (if needed).
   c. Treatment of L5.
   d. Treatment of sacral torsion.
   e. Treatment of innominates, if still dysfunctional after steps (a) to (d).
   f. Treatment of any other significant dysfunctions.
   g. Check medial malleolar heights with the athlete supine.
2. Standing anteroposterior pelvis radiograph (set so sacral promontories are easily visible)
   a. Use a plumb line to hang to gravity for use as a perpendicular to aid in measuring. A small-bead chain will suffice.
   b. Draw a straight line through each promontory and extend it off the film.
   c. Draw a straight line perpendicular to the plumb line running through at least one promontory.
   d. Measure the angle at the intersecting promontory between both lines. This measures the amount of sacral declination the athlete experiences when standing.

This method takes into account the compensatory mechanisms the body uses to cope with a structural inequity. It is important to recheck the athlete after any inserted heel lift to see the body's response. In particular, there should be symmetry between the PSIS and ASIS landmarks, and the standing and seated flexion tests should be improved, if not negative, when the proper heel lift is finally inserted. Follow-up radiographs with the heel lift in place are not necessary, but may be helpful. The best candidate for heel lift correction is an athlete with a leg-length inequity who has lumbosacral dysfunction and pain that resolves with realignment.

INTERSEGMENTAL VERTEBRAL MOTION

One of the crucial elements in making the correct diagnosis in lumbosacral dysfunction is the assessment of the motion of the individual segments relative to each other. The history typically specifies the dysfunction to the lumbosacral spine, but it is the examiner's challenge to find which specific dysfunctions have led the athlete into the office.

To evaluate intersegmental motion, the examiner places the pads of the thumb on the pillars just lateral to the spinous process and notes which one is more posterior. The athlete flexes and extends the lumbar spine while the examiner monitors for change. The side that protrudes more posteriorly is the dysfunctional facet. If the facet is provoked in flexion, the lesion is restricted in that direction and positionally stuck in the opposite side, and it will get worse in the direction opposite the facet position.

Type I group dysfunctions have no flexion or extension component; rotation and side bending are opposite each other, which contrasts with type II segmental dysfunctions. The apex of the curve should be identified and targeted in treatment.

NEUROVASCULAR EXAMINATION

Sensory testing should focus on the L4-S1 nerve roots where most disc herniations occur (8,11). The reflexes for these locations are patellar (L4) and Achilles (S1). There is no reliable reflex for the L5 nerve root (3). A summary of the dermatomes, reflexes, and myotomes is found in Table 21.2.2.

Strength testing is quick, but gives invaluable information. Leg extension evaluates L4, while dorsiflexion of the extensor hallucis longus

TABLE 21.2.2. DERMATOMES, MYOTOMES, AND REFLEXES FOR THE LUMBAR NERVE ROOTS

Nerve Root	Dermatome	Myotome	Reflex
L1	Skin overlying inguinal canal	Hip flexion	Cremasteric
L2	Medial proximal thigh	Hip flexion	Cremasteric
L3	Medial distal thigh	Hip flexion	
L4	Medial foot Medial leg	Leg extension	Patellar
L5	Dorsum of foot	Extensor hallucis longus extension	None
	Lateral leg	Leg flexion	
S1	Lateral foot	Foot plantarflexion Toe flexion Leg flexion Foot eversion	Achilles

and plantarflexion of the foot evaluate L5 and S1, respectively. Light touch of the medial (L4), dorsal (L5), and lateral (S1) aspects of the foot assesses the dermatomes (4,5). Any asymmetry or abnormality in these regions should be noted and fully evaluated.

Babinski's Sign. The examiner takes a sharp object such as a key or the handle of a reflex hammer and rubs it along the lateral aspect of the athlete's foot, crossing over to the skin overlying the metatarsophalangeal joints (Fig. 21.2.11).

Positive test: The extensor hallucis longus reflexively dorsiflexes instead of plantarflexes.

Indicates: Upper motor neuron lesion, potentially by herniation.

Oppenheimer's Sign. The examiner takes a blunt object and slides it down the medial aspect of the athlete's lower leg (Fig. 21.2.12).

Positive test: The extensor hallucis longus reflexively dorsiflexes instead of plantarflexes.

Indicates: Upper motor neuron lesion, potentially by herniation.

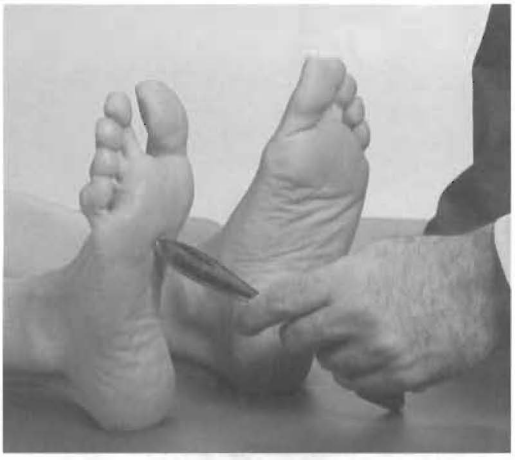

FIGURE 21.2.11. Babinski's sign.

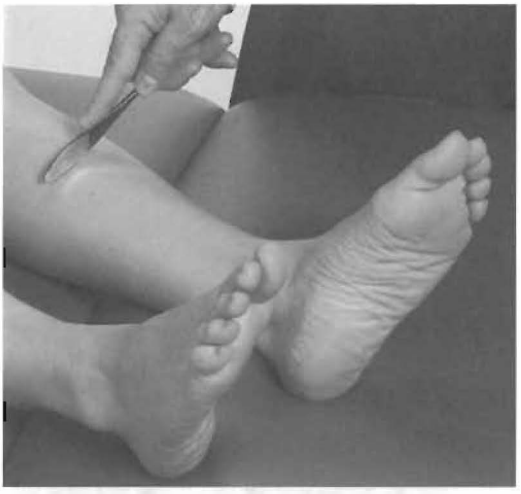

FIGURE 21.2.12. Oppenheimer's sign.

PROVOCATIVE TESTS AND MANEUVERS

Squatting Test. This is a gross function test that can be done at the beginning of the examination to localize the source of pain. The athlete stands facing the examiner and performs a full squat down to maximal knee and hip flexion (12).

Positive test: Pain produced by squatting localized in the lumbosacral spine, hips, or knee.

Indicates: Pathology in the spine, hip, or knee, depending on the location of the pain.

Stork Sign. This provocative test assesses the posterior spine. The athlete stands in front of the examiner, and then lifts one foot off the ground. The athlete then extends the back and reports on any provoked pain (Fig. 21.2.13A, B) (4,5).

Positive test: Pain produced by lumbar extension.

Indicates: Spondylolysis and/or spondylolisthesis of the lumbar pars interarticularis.

The knee lift typically provokes pain in such pathology. Unilateral injuries are usually located on the ipsilateral side of the flexed hip.

Standing Flexion Test. With the athlete standing, the examiner places his or her thumbs over the PSIS joints and asks the athlete to bend forward, which flexes the lumbar spine (Fig. 21.2.14A).

Positive test: Asymmetrical rise of one thumb above the other (6,7).

Indicates: Gross restriction at the sacroiliac joint, either intrinsically in the joint or from extrinsic factors, such as hamstring restriction.

Seated Flexion Test. With the athlete sitting and the feet flat on the floor or stool, the examiner places his or her thumbs over the PSIS joints and asks the athlete to bend forward, which flexes the lumbar spine (Fig. 21.2.14B).

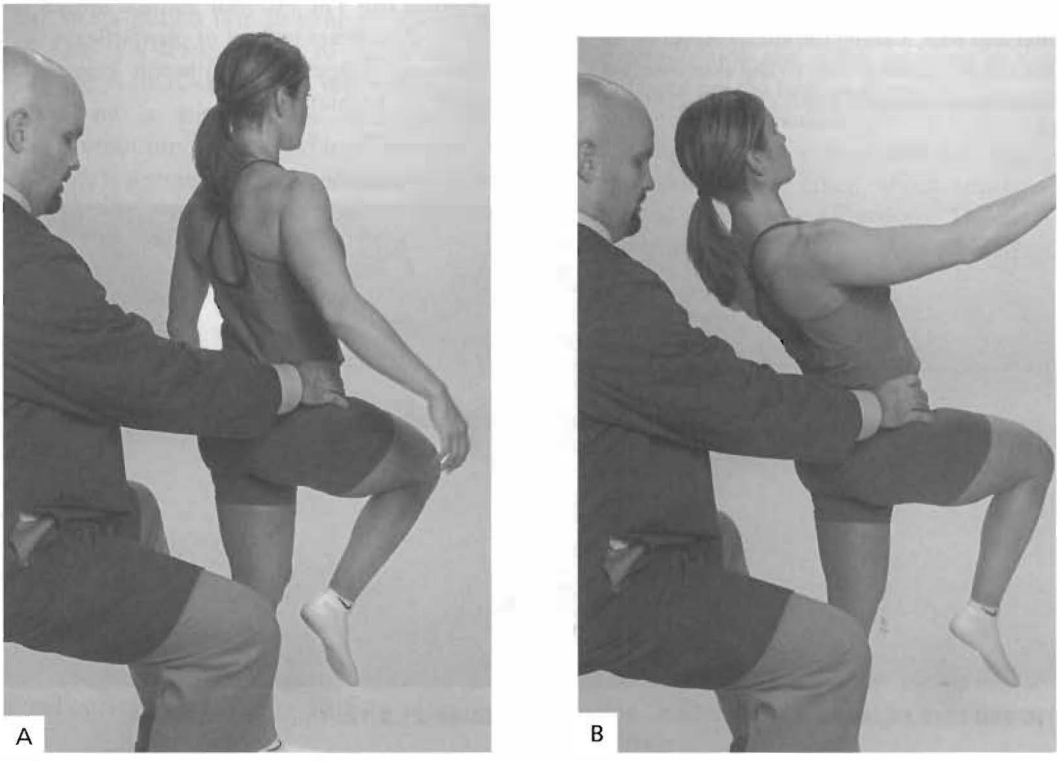

FIGURE 21.2.13. A and **B,** Stork test.

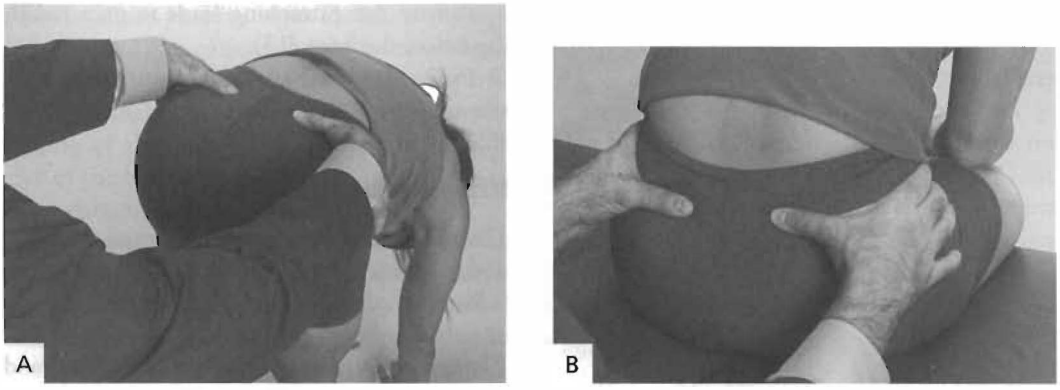

FIGURE 21.2.14. A, Standing flexion test. **B,** Seated flexion test, sacral torsion.

Positive test: Asymmetrical rise of one thumb above the other.

Indicates: Restricted sacroiliac joint on the positive side. The seated flexion test isolates the sacroiliac joint by anchoring the ischial tuberosities to the table and taking out any hip flexion. This also removes the hamstrings from the equation, so that tightness does not affect the test result, as opposed to the standing flexion test, which looks at gross hip restriction.

Straight Leg Raising Test. With the athlete supine and seated, the examiner raises the extended leg off the table, which stretches the sciatic nerve in normal athletes without herniation (Fig. 21.2.15A).

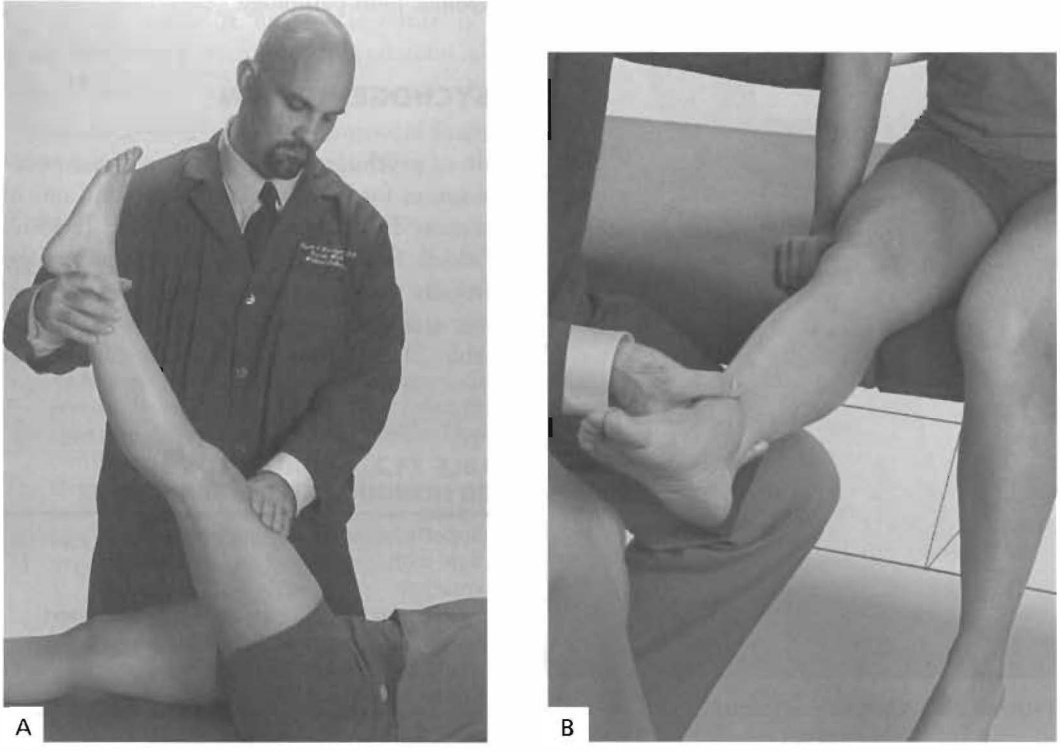

FIGURE 21.2.15. A, Supine straight leg raise. **B,** Seated straight leg raise (bench test).

Positive test: Pain starting at the lumbar region that radiates below the knee on the ipsilateral side.

Indicates: Disc herniation, which impinges on the nerve root. The pain elicited by nerve root impingement is between 30 and 60 degrees, and flexion of the knee relieves the pain (1,8).

Variation 1. Crossed leg raising test.

Positive test: Pain elicited in the contralateral extremity.

Variation 2. Bench (seated straight leg) test: The athlete is sitting and the examiner extends the leg up to tension (Fig. 21.2.15B).

Positive test: Pain elicited in the ipsilateral hip and leg.

Lasègue's Sign. The examiner lifts the athlete's extended leg off the table to the point of pain. The examiner then lowers the leg just below the level of pain and adds dorsiflexion of the ankle, which stretches the sciatic nerve (Fig. 21.2.16).

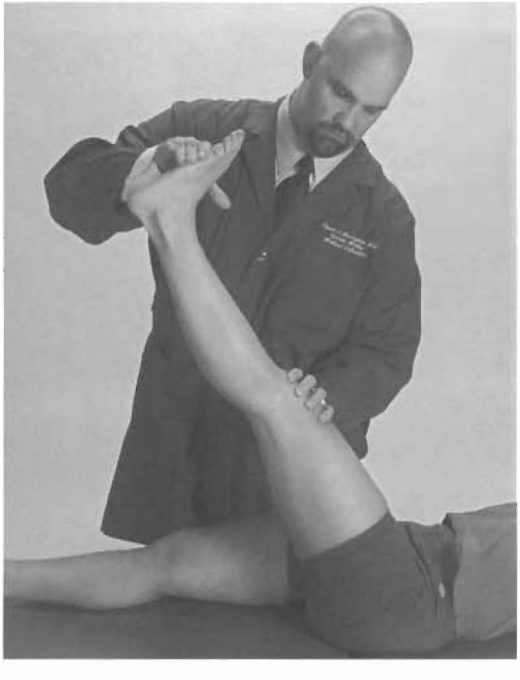

FIGURE 21.2.16. Lasègue's sign.

Positive test: Stretching leads to pain radiating below the knee (13).

Indicates: Disc herniation, neural impingement

Hip Provocative Tests

This aids in narrowing the differential diagnoses down by attempting to provoke the symptoms from the hip.

Thomas's Test. See Chapter 22.2, Hip and Pelvis Examination (Fig. 22.2.4).

Indicates: Dysfunction of the iliopsoas, rectus femoris, teasor fascia lata (TFL), or iliotibial band.

Patrick's (FABER [Flexion, Abduction, External Rotation]) Test. See Chapter 22.2, Hip and Pelvis Examination (Fig. 22.2.7).

Positive test: Pain elicited posteriorly or anteriorly in the hip (1,3).

Indicates: Anterior groin pain suggests hip joint pathology, while posterior pain suggests sacroiliac joint pathology.

PSYCHOGENIC PAIN

Pain of psychogenic origin is one of the possible causes for low back complaints and one of the most difficult causes to eliminate. In 1980, Waddell found five nonorganic signs to be clinically useful in differentiating between organic and nonorganic causes of low back pain (Table 21.2.3) (14). Waddell concluded that

TABLE 21.2.3. WADDELL'S SIGNS FOR NONORGANIC LUMBAR PAIN

1. Superficial, nonanatomic tenderness
2. Pain with stimulation, i.e., axial loading, rotation
3. Distraction tests, i.e., straight leg raising test
4. Nonorganic regional disturbances—not explained by neuroanatomy
5. Overreaction

three or more positive signs are clinically significant, and the patient should be considered for further psychological testing (14). Superficial, nonanatomic tenderness describes the finding of the skin being tender over a wide area of the lumbar region and often extends to other parts of the back.

The distraction tests most commonly used in an athlete are both the seated and supine straight leg raising test. In athletes with true lumbar disc herniation, pain radiates below the knee in both positions. Several other tests to gauge malingering or poor effort exist.

Axial Loading Test. The examiner forces a vertical load onto the athlete's skull. Pain should not be elicited in the low back.

Positive test: Athlete reports pain in the lower back.

Indicates: Exaggeration of symptoms, malingering.

Hoover's Test. With the athlete lying supine, the examiner places a hand under each heel. The athlete is then asked to raise the affected leg off the table. A normal response is to push downward with the contralateral side (Fig. 21.2.17).

Positive test: Feel a lack of downward force.

Indicates: Lack of effort from athlete.

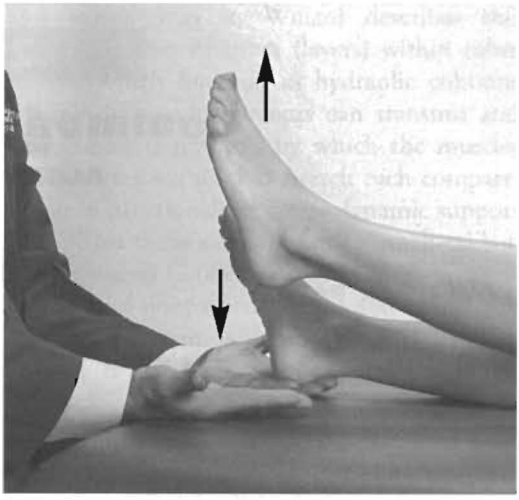

FIGURE 21.2.17. Hoover's test.

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21.3 Common Conditions

ALBERT KOZAR

LOW BACK PAIN

Epidemiology

Incidence

Low back pain is a common acute and recurrent dysfunction in athletes. It is one of the most disabling athletic injuries, affecting 5% to 15% of athletes and accounting for 10% to 15% of sports injuries as a whole (1). In comparison, 85% of the general population has low back pain at some point and roughly 2% to 5% of the general population reports low back pain annually. Reports have stated the prevalence is as high as 11% in gymnasts and 50% in football linemen (2). The vast majority of cases in the athletic population are self-limited, with acute episodes usually resulting from repetitive microtrauma (overuse) and at times single-episode macrotrauma. Those with repetitive microtrauma as the cause will usually present without a specific mechanism of injury.

Prevalence

Low back pain has been found to affect all levels of sport from adolescent to professional (3–5). A variety of sports appear to be associated with low back pain as a common cause of limited practice and playing time. Some of those specifically cited in the literature include cross-country skiing (6), dancing (7), football (8), golf (9), gymnastics (10), racquet sports (11), rowing (12), swimming (10), tennis (13), triathlon (14), and weightlifting (15).

Natural History

Athletes

The natural history of low back pain in athletes from clinical experience appears to be more self-limited, with less episodes of recurrence than the general population. Nachemson confirmed this when he reported that two thirds of injured athletes are relatively symptom-free and able to function in sport within 2 weeks and that 88% are doing well by 6 weeks (16). Although the literature examining the natural history of low back pain in athletes is limited, recent studies have found that the recurrence of low back pain in adolescent and college athletes seems to parallel that seen in the general adult population. Recurrence rates in youths in Finland have been postulated to be as high as 26% in males and 33% in females (17). Greene et al. surveyed all 679 varsity athletes at Yale in 1999 and reported that 18% (124) reported a low back injury within the past 5 years and that 6.8% (46) sustained a low back injury in the follow-up season (18).

There were no differences in rates between men and women or between athletes involved in contact versus noncontact sports. These data, though limited due to sample size, suggest that there is a three- to sixfold greater risk of low back injury in those with a prior history of injury and a 37% (46 of 124) overall incidence of recurrent low back pain. In 2003, O'Kane surveyed 1,829 former intercollegiate rowing athletes to determine whether preexisting back pain was a significant factor for back pain in intercollegiate rowers (19). He reported that 57% of these athletes with preexisting back pain developed further back pain during their collegiate rowing career compared with 36% who developed low back pain for the first time. The intensity level of back pain and missed practice time appeared to be relatively equal in both groups. In summary, the clinician's mindset should view athletic low back pain as a common injury with a potentially episodic na-

ture that can affect the athlete's ability to function in both sports and personal life. Treatment must be focused on complete functional recovery and prevention, not just elimination of acute pain.

Functional Anatomy

The "Fascial Sling"

From an anatomic perspective, the anatomy of the lumbosacral-pelvic region is best viewed as a continuous, ligamentous stocking based on the interconnections of the various regional ligaments and fascial structures outlined in the literature (20). This myofascial stocking acts as the primary support of the osseous elements throughout the lumbosacral-pelvic region, and is anchored through the thoracolumbar fascia of the back and the hamstring-sacrotuberous ligament complex of the pelvis (Fig. 21.3.1).

Throughout this myofascial sling is a large sheet of fascia that is the attachment site for multiple major muscle groups of the spine, abdomen, and upper and lower extremities. This sling is a "sculptured mountain of fascia" of the body with muscles, nerves, and bones inserted and woven into it. Willard describes this sling as a series of tubes (layers) within tubes (layers) which function as hydraulic columns whereby the compartments can transmit and share tensile stress and by which the muscles can contract within and stretch each compartment, to functionally increase dynamic support (20). This fascia contains both a small-caliber, unmyelinated C-fiber system (typical of nociceptors and sympathetic axons) and a large-caliber fiber system with encapsulated endings (typical of mechanoreception and proprioreception). Force transfers through this region may be under the proprioceptive control of neural elements within this tissue. There are at least three separate sources of these small-caliber primary afferent fibers throughout the region (21).

The continuous nature of this myofascial stocking gives the entire lumbosacral-pelvic region a unitary function. Injury to any component of this system, no matter how small, will have widespread effects throughout this region if healing does not rapidly ensue. The interactive role of these neural elements is essential to the normal trophic activity of this tissue. It is their breakdown and degeneration that can lead to chronic pain syndromes.

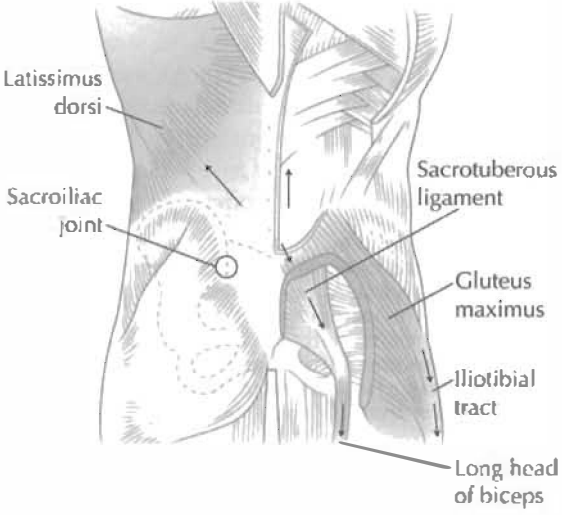

FIGURE 21.3.1. Musculoligamentous sling of the lumbosacral-pelvic region. The right side shows a part of the longitudinal muscle-tendon-fascia sling. Below is the continuation between the biceps femoris tendon and the sacrotuberous ligament, above a continuation of the biceps femoris tendon to the aponeurosis of the erector spinae. To show the right erector spinae, a part of the thoracolumbar fascia has been removed. The left side shows the sacroiliac joint (*circle*) and the cranial part of the oblique dorsal muscle-tendon-fascia sling: the latissimus dorsi muscle and thoracolumbar fascia. In this drawing, the left part of the thoracolumbar fascia is tensed by the left latissimus dorsi and the right gluteus maximus. (From Brolinson PG, Kozar AJ, Cibor G. Sacroiliac joint dysfunction in athletes. *Curr Sports Med Rep* 2003;2:47–56, with permission.)

Lumbosacral Gait Mechanics

In the normal gait cycle, there are combined activities that occur simultaneously in the right and left innominate bones and function in connection with the sacrum and spine (22,23). As one steps forward with the right foot, at heel strike, the right innominate rotates posteriorly, and the left innominate rotates anteriorly. During this motion, the anterior surface of the sacrum is rotated to the left and the superior surface is level, while the spine is straight but rotated to the left. Toward midstance, the right leg is straight and the innominate is rotated anteriorly. The sacrum is rotated right and side-bent left, while the lumbar spine is side-bent right and rotated left. At left heel strike, the opposite sequence occurs and the cycle is repeated (Fig. 21.3.2).

Throughout this cycle there is a rotatory motion at the pubic symphysis, which is essential to allow normal motion through the sacroiliac (SI) joint. According to Greenman

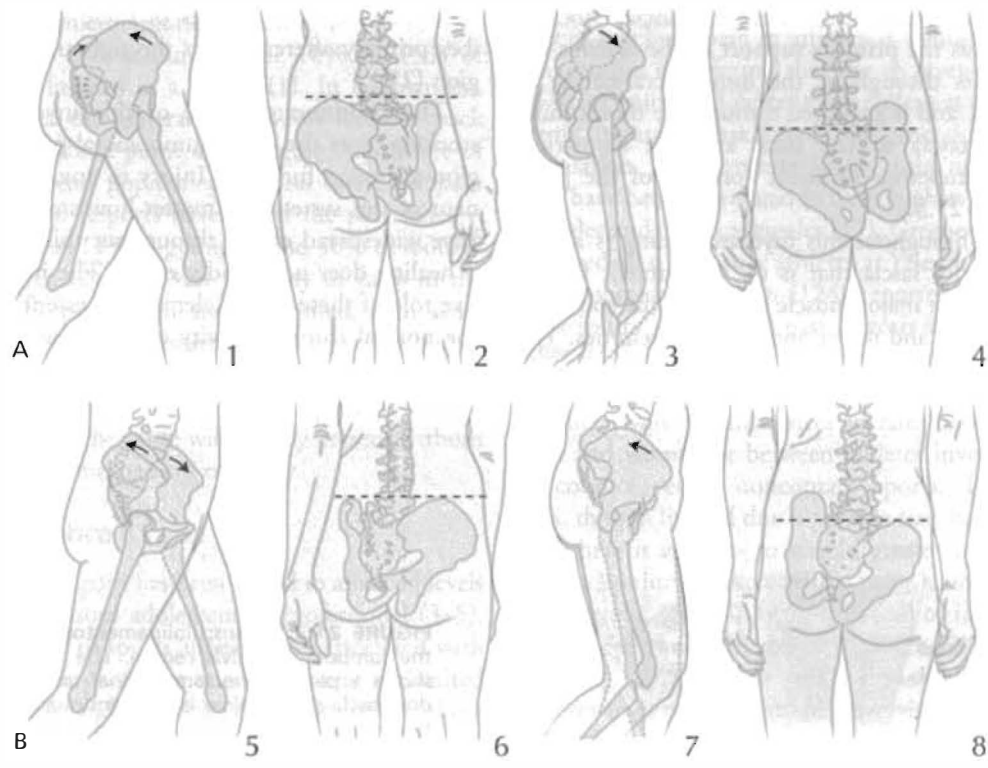

FIGURE 21.3.2. Sacroiliac joint motion during walking. **A,** (*1* and *2*): At right heel strike. *1,* The right innominate has rotated in a posterior direction and the left innominate has rotated in an anterior direction. *2,* The anterior surface of the sacrum is rotated to the left and the superior surface is level, while the spine is straight but rotated to the left. (*3* and *4*): At right midstance. *3,* The right leg is straight and the innominate is rotating anteriorly. *4,* The sacrum has rotated right and side-bent left, while the lumbar spine has side-bent right and rotated left. **B,** (*5* and *6*): At left heel strike. *5,* The left innominate begins anterior rotation; after toe-off, the right innominate begins posterior rotation. *6,* The sacrum is level but with its anterior surface rotated to the right. The spine, although straight, is also rotated to the right, as is the lower trunk. (*7* and *8*): At left leg stance. *7,* The left innominate is high and the left leg is straight. *8,* The sacrum has rotated to the left and side-bent right, while the lumbar spine has side-bent left and rotated right.

(22,23), pubic symphysis dysfunction in walking is one of the essential or leading causes of the development of SI joint dysfunction (SIJD). In static stance, when one bends forward and the lumbar spine regionally extends, the sacrum regionally flexes, with the base moving forward and the apex moving posteriorly. During this motion, both innominates go into a motion of external rotation and outflaring. This combination of motion during forward bending is called *nutation of the pelvis.* The opposite occurs in extension, which is called *counternutation.* As the sacrum goes into extension with the base moving posteriorly and the apex anteriorly, the innominate components internally rotate and in-flare. This motion is clearly demonstrated and illustrated by Kapandji (24).

Normal Lumbosacral Motion

Coupled Motion in the Lumbar Spine

The vertebral unit is composed of two adjacent vertebrae with their associated disc, arthroidial, ligamentous, muscular, vascular, lymphatic, and neural elements (25). Motion defined, for example, about L3, describes motion of L3 and its associated soft tissue structures moving on L4 and its associated soft tissue structures. Vertebral motion is guided based on the tripod arrangement of the disc and facet joints. In the lumbar spine, the facet joints lie in the sagittal plane allowing for significant flexion and extension movement, with side bending and rotation to a lesser degree. During normal lumbar flexion or extension, there should not be any associated rotation or side bending. However, when one initiates either side bending or rotation, the other will always occur. This reciprocal movement is called *coupled motion* and occurs in opposite directions in normal lumbar spine movement (type I motion). That is, one motion can never occur without the other. When this coupled motion occurs in the same direction (non-neutral, or type II motion), it is dysfunctional. If this coupled motion occurs in the opposite direction (neutral, or type I motion) upon initiation of flexion or extension, it is also considered dysfunctional.

Coupled Motion at the Lumbosacral Junction

At the lumbosacral joint, sacral motion primarily occurs in the opposite direction of L5. When they occur in the same direction, it is considered dysfunctional. This description of motion is such because gross, forward bending of the body is compared to regional sacral motion. For example, during forward flexion, the sacrum moves into posterior nutation, in which the sacral base moves posteriorly, and the apex moves anteriorly. However, when describing this motion using purely regional motion terminology, they occur in the same direction, that is, regional lumbar extension (forward bending) occurs with regional sacral extension (posterior nutation). It is important to the terminology one is working with so that incorrect comparisons are not made.

Normally functioning lumbar vertebrae and the normal adaptive response of a functional lumbar spine is that of side bending and rotating to opposite sides as a group in response to side bending of the sacral base in a neutral plane. When the lumbar spine is forward-bent with non-neutral mechanics with side bending and rotation to the same side, the sacrum participates with a backward or posterior torsional movement about an oblique axis in which the sacrum side-bends and rotates to opposite sides. For example, when bending forward and to the right, L5 flexes, side-bends, and rotates to the right, while the sacrum goes into a backward left-on-right torsion with side bending to the right, rotation to the left, and the left base going into posterior nutation. Therefore, the normal adaptive response of L5 in relation to the sacrum is *opposite* the base of the sacrum, and vice versa. Sacral side bending and rotation always occur to opposite sides. Whenever side bending or rotation of L5 in relation to the sacrum occurs in the same direction, dysfunction is present. Posterior torsional movements can be described as non-

neutral sacroiliac mechanics and are not part of the normal walking cycle.

Role of Sacroiliac Joint Coupling

Biomechanics between the Spine and Pelvis

In 2001, Vleeming et al (26). described their integrated model of joint dysfunction. This functional description comes from extensive study of the lumbosacral-pelvic region over the past 10 to 15 years, and is the most studied and supported biomechanical model for this region. It integrates structure (form and anatomy), function (forces and motor control), and the mind (emotions and awareness) in human performance. Integral to the biomechanics of lumbosacral-pelvic stability is the concept of a self-locking mechanism of the SI joint. It is the only joint in the body that has a flat joint surface that lies almost parallel to the plane of maximal load. Its ability to self-lock occurs through two types of closure: form and force. *Form closure* describes how specifically shaped, closely fit contacts provide inherent stability independent of external load. *Force closure* describes how external compression forces add additional stability.

Only the ligaments in this region were thought to provide additional support. Evidence now shows that the fascia and muscles within this region provide significant self-bracing or self-locking to the SI joint and its ligaments through their crosslike anatomic configuration (Fig. 21.3.3). Ventrally, this is formed by the external abdominal obliques, linea alba, internal abdominal obliques, and transverse abdominals, whereas dorsally, the latissimus dorsi, thoracolumbar fascia, gluteus maximus, and iliotibial tract contribute significantly. Additionally, there appears to be an arthrokinetic reflex mechanism by which the nervous system actively controls this added support system.

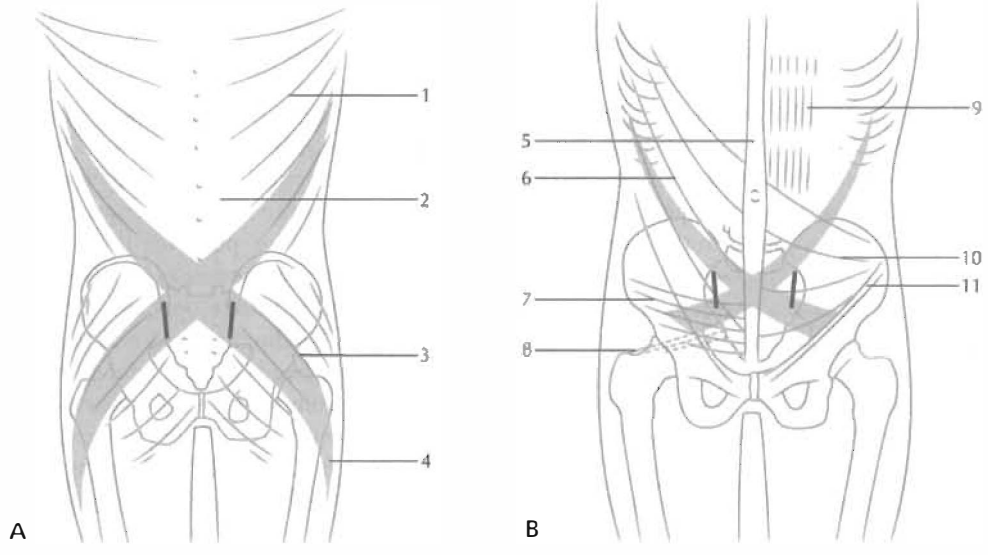

FIGURE 21.3.3. The crosslike configuration demonstrating the force closure of the sacroiliac joint. The SI joint becomes stable on the basis of dynamic force closure via the trunk, arm, and leg muscles that can compress it, as well as its structural orientation. The crosslike configuration indicates treatment and prevention of low back pain with strengthening and coordination of trunk, arm, and leg muscles in torsion and extension rather than flexion. The crossing musculature is noted. **A,** Latissimus dorsi (*1*); *2*, thoracolumbar fascia; *3*, gluteus maximus; *4*, iliotibial tract. **B,** Linea alba (*5*); *6*, external abdominal obliques; *7*, transverse abdominals; *8*, piriformis; *9*, rectus abdominis; *10*, internal abdominal obliques; *11*, ilioinguinal ligament. (From Moore KL, Agur AMR, Essential Clinical Anatomy. Baltimore: Williams & Wilkins, 1995.)

These supports are critical in asymmetrical loading, when the SI joint is most prone to subluxation. The important concept to gain from this understanding of integrated function with regard to treatment and prevention of low back pain is that a lumbosacral strain is a "neuromyofascial musculoligamentous" injury (27).

Coupled Motion of the Contralateral Latissimus Dorsi and Gluteus Maximus

Vleeming et al. (28) defined the posterior layer of the thoracolumbar fascia as a mechanism of load transfer from the ipsilateral latissimus dorsi and the contralateral gluteus maximus (Fig. 21.3.4). This load transfer is critical during rotation of the trunk, helping to stabilize the lower lumbar spine and pelvis. This was demonstrated through cadaveric and electromyographic (EMG) studies (29). The stretched tissue of the posterior thoracolumbar fascia assists the muscles by generating an extensor influence, and by storing elastic energy during lifting to improve muscular efficiency.

Lumbopelvic Rhythm and the Hamstrings

Recent studies show there is both a functional and anatomic connection between the biceps femoris muscle and the sacrotuberous ligament (30). This relationship allows the hamstring to

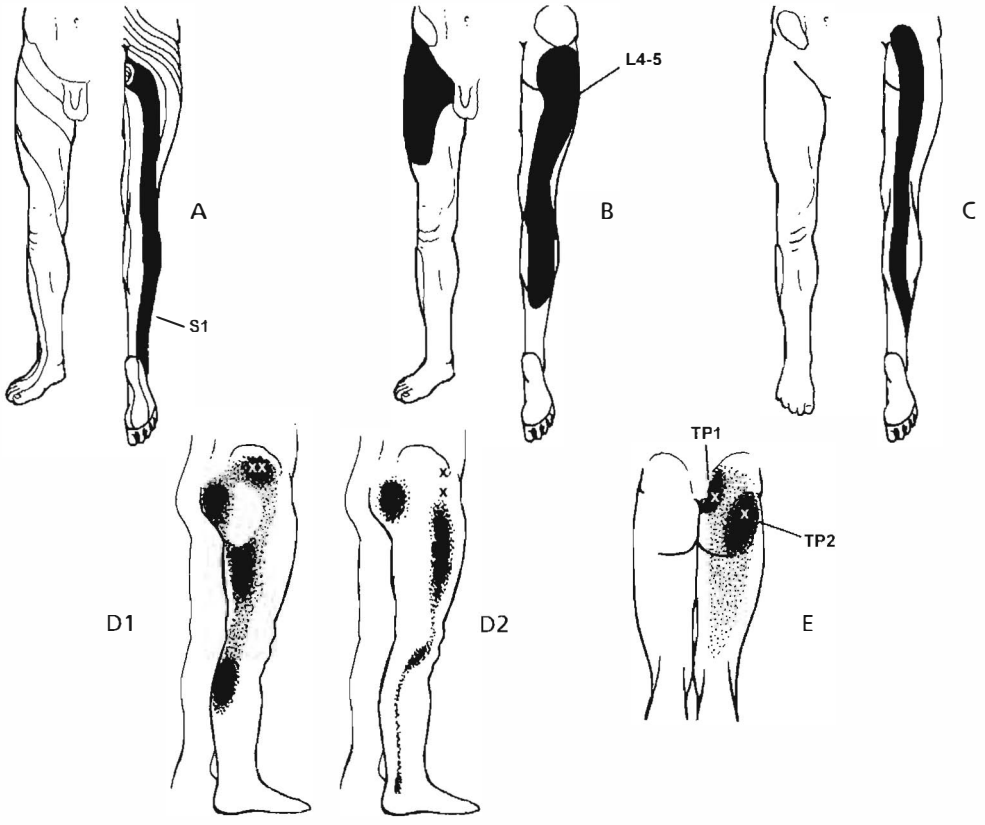

FIGURE 21.3.4. Differential diagnosis for referred pain to the posterior buttock, thigh, calf, and ankle. **A,** Dermatomal referred pain from irritation of the S1 nerve root. **B,** Sclerotomal referred pain from irritation of the L4-L5 facet joint and/or capsule. **C,** Sclerotomal referred pain from the sacroiliac joint and/or sacroiliac ligaments. **D,** Myotomal referred pain from the gluteus minimus muscle (*1*) posteriorly and (*2*) anteriorly. **E,** Myotomal referred pain from the piriformis muscle in full-blown piriformis syndrome.

play an integral role in the intrinsic stability of the SI joint. It appears that the biceps femoris, often found to be short on the pathologic side in low back pain, may actually be a compensatory mechanism via the previously described arthrokinetic reflexes to help stabilize the SI joint. In healthy individuals, a normal lumbopelvic rhythm exists, during which the first 65 degrees of forward bending is via the lumbar spine, followed by the next 30 degrees via the hip joints. Increased hamstring tension prevents the pelvis from tilting forward, thereby diminishing the forward-bent position of the spine, which results in reducing spinal load (31). Normalization of the lumbopelvic rhythm is an essential component to treatment of low back pain and sacroiliac joint dysfunction.

Lumbosacral-pelvic "Core" Strength: The Essential Role of the Transversus Abdominis

The transversus abdominis has been shown to be the key muscle to functional retraining of trunk mechanics (commonly referred to as the "core"), due to its observed patterns of firing before and independent of the other abdominal muscles (32). The first muscle to fire in the body with the initiation of any movement of the body is the transversus abdominis. It stabilizes the lumbosacral-pelvic region in order to generate stability for that movement. Exercise techniques that promote independent contraction of the transversus abdominis have been shown to reduce recurrence rates after an acute low back pain episode (33), and decrease pain and disability in chronic low back pain (34). Most recently, a study by Richardson et al (35) appears to show that these clinical benefits focusing on the transversus abdominis occur due to significantly reduced laxity in the sacroiliac joint.

Differential Function of the Iliacus from Psoas Muscle

In recent years, intramuscular electromyographic studies of the hip flexor muscles during human locomotion have revealed a separate role of the psoas and iliacus muscles for stability and movement of the lumbar spine, pelvis, and hip (36,37). In 1995, Anderson et al. presented evidence that the iliacus muscle was selectively recruited in the standing position with extension of the contralateral leg and, in standing, maximal, ipsilateral abduction, significantly higher levels of activation in the iliacus muscle when compared with the psoas muscle were found (36). This suggested preferential action of involved single-joint muscles when possible to achieve local pelvic control. In 1997, Anderson et al. studied walking and running and found that the iliacus muscle was the main "switch muscle" during low-speed walking (37). That is, the iliacus muscle is key to reversing lower extremity motion from extension to flexion. In a later study in 1997, Anderson et al. reported that the iliacus and sartorius muscles performed a static function needed to prevent a backward tilting of the pelvis during trunk flexion sit-ups (38). With static leg lifts, there was progressively more activation of these muscles with increasing elevation of the extremity. They also observed that a change in pelvic tilt influenced activation of the iliacus and sartorius muscles. A backward pelvic tilt combined with a hyperlordotic back decreased activation of these muscles, whereas forward pelvic tilt combined with a hyperlordotic back increased activation of these muscles. This suggests an important and separate role of the iliacus from the psoas in function and dysfunction of the low back.

Tensegrity: The Tension–Integrity Approach to the Lumbosacral-pelvic Region

Although still in development, a new biomechanical model of body design is *tensegrity*, which refers to a self-stabilizing system in which tension is continuously transmitted across all elements (39,40). Therefore, the stability of a tensegrity structure lies not in the strength of individual members but in the ability of all the members to distribute and balance mechanical forces. Current biomechanical models of spinal

mechanics view the spine as a column or pillar. The sacrum as the base locks into the pelvis as a wedge or other gravity-dependent closure. For human postures, this model may not explain the functionality present. According to Levin (40), "the hallmark of a pillar is stability, but the hallmark of a spine is flexibility and movement." In the tensegrity model, triangulated structures (a truss) form the basis for inherently stable structures. When viewed three-dimensionally, they become the tetrahedron, octahedron, and icosahedron. Further, the sacrum, as opposed to being viewed as a wedge or base, is suspended as a compression element within the ligamentous tension system of muscles, ligaments, and fascia. To date, cellular structure and mechanotransduction are established by this system (41).

FACTORS INVOLVED IN LUMBOSACRAL PAIN

Basic Mechanisms Involved with Low Back Injury in Sports

The pain-sensitive structures of the lumbosacral spine are listed in Table 21.3.1. Though low back injury during sport is most commonly thought to be due to microtrauma, it can occur by any of the following mechanisms:

- Chronic stressful position, e.g., field hockey or jogging
- Repetitive loading, e.g., rowing
- Hard repetitive contact, e.g., football and hockey
- Lifting of human bodies, e.g., ballet and figure skating
- Hyperextended lumbar spine, e.g., gymnastics and football
- Stressing an immature spine, e.g., weight training and gymnastics
- Sudden violent muscle contraction, e.g., throwing sports and sprinting

TABLE 21.3.1. PAIN-SENSITIVE STRUCTURES OF THE LUMBOSACRAL SPINE

Facet joint capsules (rich in nociceptors)
Ligaments and fascia (rich in nociceptors)
Intervertebral disc
Vertebrae (periosteal nociceptors)
Nerves
Muscle

In the differential diagnosis of low back pain in adult athletes, though mechanical and discogenic are the most common causes, one must also include the possibility of tumor, infection, vascular, metabolic, inflammatory, congenital, degenerative (including spinal stenosis, disc disease, and facet syndrome), trauma (including single-episode injuries to the apophysis, muscle tears, ligament sprains, fractures), and repetitive injury (including postural, stress fracture, and spondylolisthesis). Additionally, remember congenital ot developmental disorders such as scoliosis and dysplastic spondylolisthesis. Visceral pathology such as upper gastrointestinal disease and retroperitoneal disorders, which can end up being a neoplasm, must also be in the differential.

In the adolescent athlete, mechanical, spondylolysis, and apophysitis are the most common causes. The differential in this population should still include tumor, infection, vascular, metabolic, inflammatory, congenital, trauma (including single-episode injuries to the apophysis, muscles tears, ligament sprains, fractures), and repetitive injury (including postural, stress fracture, and spondylolisthesis). Though somewhat uncommon, the differential in this population must also include Scheuermann's disease, atypical Scheuermann's, and apophyseal ring fractures.

Athletes versus Nonathletes

When evaluating an athlete for low back pain it is important to recognize certain distinctions between the athletic and nonathletic populations (1). The forces that the athlete's back is subjected to are often much greater than those seen by nonathletes. Further, these forces are usually sustained for longer periods of time. Specific movements and body positions are required in different sports that will predispose

the athlete to specific injuries unlike those found in the general adult population. For example, the increased incidence of spondylolysis is well documented in gymnasts, who are constantly hyperextending their low back.

The repetitive nature in athletics, though not unique, is often more prevalent, especially with increasing levels of play. The mechanism of low back pain in athletes is usually overuse. In nonathletic groups, there is a much higher incidence of degenerative and arthritic conditions compared with athletes, most likely reflected in the fact that the athletic group comprises a much younget population. Finally, the nonathletic population may have motivations to quickly report even minor injuries because of potential compensation, while many athletes are often reluctant to report back problems for fear of losing playing time, or, in the case of professional athletes, monetary compensation. This reluctance may also lead to an underestimation of the incidence and types of various back ailments in the general athletic population. For these reasons, epidemiologic data gathered in relation to the general adult population cannot be directly applied to athletic populations.

Adolescent versus Adult Athletes

Michele and Wood have reported on significant differences in the etiologies of low back pain between these groups, in particular a 47% incidence of spondylolysis in young athletes compared with only a 5% incidence in the general adult population (42). They also cited an 11% incidence of disc-related back pain in adolescent athletes compared with a 48% incidence of discogenic back pain among an athletic adult population.

The uniqueness of injuries to the young athlete's spine most often reflects certain growth-related risk factors. Factors specific to the adolescent athlete include the consideration of the adolescent growth spurt, which can affect thoracolumbar fascia and hamstring flexibility, and the consideration of growth cartilage in the apophyseal joints in the spine.

Growth cartilage and immature ossification centers of the adolescent spine are susceptible to compression, distraction, and torsional injury. During growth, these areas often are the weakest link of force transfer (43). Specifically, the traction apophysis is a common area of overuse injury. Ossification of the growth plate begins between 7 and 8 years of age. By ages 12 to 15, these foci form the arrangement seen on radiographs, with the final ring apophyseal closure occurring sometime around ages 21 to 25. The ring apophysis does not extend around the entire circumference of the vertebral body but only around it centrally. This apophyseal ring contributes little to longitudinal growth of the vertebral body, but is responsible for the growth of vertebral body breadth.

The posterior column of the immature lumbar spine forms from an ossification center on each side of the neural arch at the pedicles. Ossification progresses posteriorly and may be congenitally incomplete on the superior portion of the pars interarticularis on the lower lumbar vertebra, most common at L5, in 4% to 6% of the general population. The posterior arch also contains the growth cartilage of the facet joint and spinous process apophysis and is subject to traction of the thoracolumbar fascia and lordotic impingement.

The adolescent growth spurt itself can play a significant role in athletic injury. Kinetic force abnormalities can develop from intrinsic musculoskeletal inflexibility and from extrinsic collision and ground reactive forces. Intrinsic biomechanical defects related to the adolescent growth spurt include iliopsoas inflexibility, femoral anteversion, thoracolumbar fascial tightness, abdominal weakness, and thoracic kyphosis. Increased lumbar lordosis will put additional compression on the posterior elements, whereas excess lumbar flexion will increase anterior column compression. Iliopsoas inflexibility increases lumbar lordosis and shear forces to the disc. Other factors that increase lumbar lordosis include tight thoracolumbar fascia, genu recurvatum, thoracic kyphosis, and weakened abdominal musculature. Femoral anteversion limits hip turnout and is often compensated for in the immature dancer, for example, by increasing lumbar lordosis, which releases the ilioinguinal ligament. The adolescent growth

spurt may also predispose to an acute apophysitis or apophyseal avulsion at various thoracolumbar attachment sites including the iliac crest, and spinous and transverse processes.

Intrinsic Factors

A number of significant predisposing conditions must be considered when evaluating an athlete for low back pain and should include leg-length inequality, genu recurvatum, iliacus and/or psoas tightness, femoral anteversion, weak core strength, neuromuscular imbalance (lower crossed syndrome), abnormal flexion to extension strength (normal 1:1.3), poor fitness, underlying somatic dysfunction, previous lower extremity joint injury without complete rehabilitation, and nutrition.

While most athletes are in good general physical condition, one cannot assume that they spend enough time strengthening and stretching their low back and abdomen. In fact, studies have not demonstrated that athletes have stronger back muscles than nonathletes. Injuries are often due to poor core conditioning of the spine, asymmetrical biomechanics of the lower extremity and pelvis, and/or repetitive stress. This repetitive stress may be due to the physical and contact nature of one's specific sport or to the excess and asymmetrical range of motions placed on the torso during competition. As one moves up in the level of competition, there is an increasing demand for core strength and balance in order to transfer kinetic energy to produce power in a particular extremity. A weak core can lead to lumbosacral sprain and/or extremity injury.

Leg-length discrepancies allow for an unequal transmission of forces across the spine during weight-bearing activities. These forces can be amplified in sport due to the rapid acceleration of body mass or repetitive stress transmitted. Although traditional orthopedic teaching is that a minimum of 1 to 2 cm difference is essential to cause dysfunction, many in the sports medicine community have studied the effects of a short leg on athletes specifically and consider differences of as little as 4 mm to be significant (44).

Poor underlying fitness in any athlete, but especially in the weekend warrior, is a risk factor for injury. Poor aerobic capacity has not been shown to be a direct risk factor for back injury but its effect through muscular endurance, strength, flexibility, and body composition is felt by many to be a significant risk for injury. Weakness of the erector spinae, especially the multifidus, leads to an abnormal ratio of trunk extension strength to trunk flexion strength and has been shown to be significantly reduced in those athletes with low back pain (45). Inflexibility of the hamstrings increases lumbar lordosis. Pelvic mobility is essential in bending and lifting activities, and tightness of the hip flexor muscles may limit pelvic movement and cause excessive strain on the lumbar spine (45). Tightness of the hip extensor muscles may reduce lumbar lordotic curve, making the spine less resilient to axial loading (45). Finally, poor nutrition may result in irreversible osteopenia and stress fractures with possible recurrence such as spondylolysis.

One must always have a high index of suspicion for acute disc herniations in those young athletes not responding to conservative treatment. Sports involving flexion, axial loading, and rotation such as weightlifting, collision sports, and bowling most commonly are involved with nucleus pulposus herniation. Though they occur less frequently in young athletes than in the general adult population, the adolescent athlete with acute disc herniation may present with only back spasms, neurogenic scoliosis, hamstring tightness, and often may not have the hard signs such as sciatica, reflex loss, or muscle weakness.

Goldstein et al. (10) demonstrated an 11% incidence of degenerative disc disease in the preteen athlete, a 43% incidence in the elite athlete, and a 63% incidence in Olympic gymnasts. They also demonstrated that when the volume of training exceeded 15 hours a week, the risk of injury increased from 13% to 57%. Spinal changes of lumbar Scheuermann's kyphosis with apophyseal ring avulsion and Schmorl's nodes have been associated with flexion and axial loading, as seen in gymnasts, football players, and divers with a flattened lordosis.

Hyperextension in dancing, figure skating, and interior football linemen has been associated with spondylolysis.

Extrinsic Factors

Extrinsically one must always consider training errors, improper technique, poor equipment, and sport-specific forces as potential causative issues for low back pain in athletes. Collision sports such as rugby, football, and ice hockey can lead to acute fractures of the thoracic and lumbar spine, which most often involve the transverse or spinous processes. Though rare, compression fractures are possible when loading occurs in a flexible vertical posture.

Training errors are one of the most common risk factors for athletic injury. Any abrupt change in training that increases more than one of the following factors can lead to the production of an overuse syndrome: intensity, duration, equipment, and terrain. The end result is a marked increase in the total load to the body precipitating injury. Proper technique is fundamental and an important component for any athlete. The mechanics of throwers, hitters, and body movement in contact sports must be scrutinized for failure to maintain a fluid transfer of energy through the kinetic chain. For example, excessive lordosis in a football lineman while maintaining an erect posture or performing excessive repetitive lordotic movements by a dancer can precipitate injury. Failure to properly time the transfer of kinetic energy from the lower to upper extremity, such as in a pitcher, may result in low back injury. Poor equipment, or improper equipment fit, for example, with a cyclist whose seat height is too high, may lead to excessive pelvic rocking. This can lead to increased muscular activity and fatigue resulting in low back pain. In cycling, excessive distance between the seat and handlebars can lead to a hyperlordotic position which increases the repetitive strain on the low back.

Muscle Balance and Imbalance

Janda described how muscles respond to dysfunction in a predictable, characteristic pattern (46–48). This pattern is not random and occurs irrespective of the clinical diagnosis or specific regional injury. Postural (tonic) muscles will always become facilitated, hypertonic, and tight to some degree, while phasic (dynamic) muscles will become inhibited, hypotonic, and "weak," (pseudoparesis) to some degree.

Initially the pseudoparesis seen is a central nervous system inhibition, not a true weakness. Over a prolonged time period of inhibition the muscles actually may become weak. Attempts at strengthening the "weak" muscles only increases inhibition. Physiologically, a decrease in recruitment is seen with added resistance. These muscles actually may not appear grossly weak on initial testing, but they are seen to fatigue quickly, demonstrating poor endurance. It is important to remember that treatment must be directed at the cause of inhibition, the neural reflex, first.

This muscle dysfunction is characterized by a change in the sequence of muscle activation patterns and was termed by Janda as muscle imbalance. He described both an upper crossed syndrome and a lower (pelvic) crossed syndrome that when combined produce a layered syndrome that can be appreciated throughout the body. Superficial and deep electromyelographic analysis reveals that there are delays in the activation of phasic muscles, a decrease in amplitude and recruitment of phasic muscles, and that normal facilitatory input (resistance training) can have an inhibitory effect.

Triggers of muscle imbalance patterns include muscle disuse, repetitive movements, development of inflexibility, and pain. Of these, *pain seems to be the single dominant factor in the maintenance of these patterns.* Janda has documented muscle imbalance patterns starting as early as 6 to 7 years of age (48).

Muscle imbalance should be suspected any time there are abnormal firing sequences on range of motion testing, poor balance, recurrent somatic dysfunction, "weak" or easily fatigable phasic muscles on clinical examination, history of recurrent injury or other overuse injury in the same region, chronic pain, and postural imbalance. Muscle imbalances can

become a dominant factor in the cause of musculoskeletal pain and/or a major factor in the continuance of the pain. Failure to rehabilitate these patterns is sure to be a significant factor in recurrent injury.

REFERRED PAIN: DERMATOMES, MYOTOMES, AND SCLEROTOMES

Depending on the particular muscles, tendons, and/or ligaments injured, there can be referred pain into the gluteal muscles, hips, buttocks, posterior/lateral thighs, or up into the lower thoracic or thoracolumbar junction. Therefore, it is essential to have a full understanding of dermatomes, myotomes, and sclerotomes, which describe different types or sources of referred pain.

Lumbar dermatomes are located on the posterior lumbar paraspinal region and the anterior part of the thigh, leg, and foot (49). Pain or paresthesias in these areas of skin provide clues to the level of nerve root involvement and nerve dysfunction and/or irritation. Remember that these divisions vary slightly from person to person due to anatomic variation.

Myotomal pain is associated with cramps, weakness, and myofascial trigger points in the muscles that share innervation from the same irritated nerve roots (50). Sclerotomal pain describes vague, deep, toothache-like pain (51). This referred pain arises from ligaments, bones, and/or joints that share innervation from the same irritated nerve root. Most leg pain experienced by athletes in relation to low back pain, especially when it is variable in presentation, is sclerotomally referred.

Knee pain, for example, can be the result of irritation of the L3 vertebra, ligaments in the L3 region, the pubic symphysis, the hip, or the knee. All of these sites have the same L3 sclerotomal origin. Figure 21.3.4 demonstrates the five most common referred causes for what most physicians call "sciatica," and consider to be S1 nerve root irritation. However, only one of these patterns is dermatomal. *Remember, all that radiates is not radicular!*

Ligamentous laxity and tendinosis may produce sclerotomal referred symtoms (52). One or more of the following symptoms can arise from these types of degenerative structures:

1. Significant tenderness when pressure is applied.
2. An inability to maintain one position for prolonged periods of time or maintain repeated movement that often relieves the pain. The initial movements are painful. This is in contrast to pain of nerve or muscular origin, in which the pain is reduced with rest and worsened with movement. Degenerative tendons and ligament laxity produce more pain on initial movements after a period of rest, but the discomfort usually improves the more they are used.
3. Numbness and/or pins-and-needles sensation along the same specific sclerotomal pain distribution that may closely mimic patterns of neurologic origin. A key point in differentiating the source can be made by stroking the affected area. Sclerotomal referred numbness is comfortable (normal response) in contrast to the true numbness of neurologic origin, in which stroking produces hyperesthesia (abnormally sensitive response) or dysesthesia (painful response).
4. Local and referred pain. Referred pain is pain from a ligament or joint that is felt at some distance away from the injured site. This may mimic so closely pain referred from a neurologic origin that only a careful history will tell the difference.
5. Recurrent muscle tightness and spasms are very common. The abnormal joint movement associated with ligamentous laxity and tendinosis creates many protective actions by adjacent tissues. Muscles will contract in an attempt to pull the joint back to the correct location or stabilize it to protect it from further damage. There is a tendency for physicians to treat the muscle spasms as the primary cause of the problem. Many medical treatments may be directed toward the muscular spasms, and not the primary cause—the ligamentous or tendinous injury.

COMMON LUMBOSACRAL SOMATIC DYSFUNCTIONS

In athletes with acute low back pain, it is common to find significant somatic dysfunction. Common areas of somatic dysfunction in athletic injury include

- Psoas spasm (psoas syndrome)
- Iliolumbar ligament syndrome
- Flexed non-neutral upper lumbar dysfunction
- Extended non-neutral low lumbar dysfunction
- Sacral dysfunction, especially sacral shear (a unilaterally flexed or extended sacrum) and sacral torsions
- Innominate dysfunction, especially inominate shears and pubic shears
- Piriformis syndrome
- Iliacus dysfunction

In athletes with persistent low back pain who have previously not improved with additional therapies, it is important to consider the "dirty half-dozen," a term Greenman coined to describe those somatic dysfunctions that, in his experience, often underlie complaints of persistent pain in athletes with failed low back syndrome (23). These include

- Non-neutral lumbar somatic dysfunction
- Pubic symphysis dysfunction (primarily pubic shear)
- Restriction of the anterior movement of the sacral base
- Innominate shear dysfunction–upslip innominate
- Leg-length inequality and pelvic tilt syndrome
- Muscle imbalance of the trunk or lower extremities

Kuchera describes a high incidence of unilateral sacral flexion (sacral shear) with persistent pain (53). He notes that both sacral and innominate shears may be asymptomatic at their site, but eventually result in secondary subjective symptoms in the athlete's lumbar, upper rib, cervical, suboccipital, or cranial regions. These shears are nonphysiologic and cannot be removed without specific external treatment. Therefore, it is highly important to always screen athletes on initial examination for innominate and sacral shears.

Psoas Spasm (Psoas Syndrome)

During the evaluation of gait, if the athlete walks in a forward-bent position, he or she may have psoas spasm. Psoas syndrome is usually initiated when an athlete assumes any number of positions that shorten the origin and insertion of the psoas muscle for a significant length of time, and then arise rapidly, suddenly lengthening this origin and insertion. Specifically, any athlete who has to rapidly and forcefully flex the hip freely or against resistance can precipitate acute psoas spasm; two examples include a sprinter repeatedly practicing hurdles in reaching harder for that last one after a long day of practice, or the soccer player who forcibly kicks for a long goal or kicks directly into another player's body during a block. Psoas spasm can also be precipitated by overuse, such as during the performance of sit-ups with the lower extremities fully extended or during repeated hip flexion by such an athlete as a hurdles runner.

The following specific findings on physical examination may indicate the presence of psoas spasm (Fig. 21.3.5):

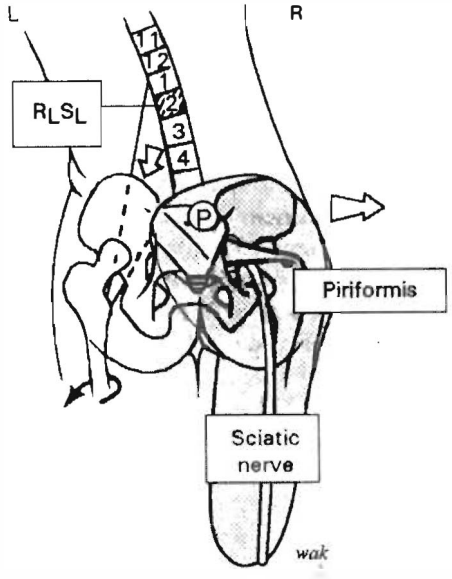

FIGURE 21.3.5. Acute left psoas syndrome. The athlete is forward-bent, leans to the left, and everts the left foot. (From Kuchera M. Lumbar region. In: Ward R, ed. *Foundations for Osteopathic Medicine*, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2003.)

- A beltline that is low on one side may indicate the presence of psoas spasm or short leg
- A flexed non-neutral somatic dysfunction in the upper lumbar spine at L1 or L2
- Pelvic side shift occurring toward the side of the longer leg or opposite to the side of the tighter psoas muscle
- Sacral somatic dysfunction on an oblique axis, usually to the side of lumbar side bending
- Hypertonicity of the piriformis muscle opposite the side of greatest psoas spasm

Treatment of psoas syndrome should always focus initially on the flexed lumbar component, followed by stretching the tight psoas muscle. If the psoas muscle remains persistently hypertonic and painful to stretch, counterstrain is a very effective method both on the field and in the office. Removal of all other somatic dysfunction is always important to prevent recurrence.

The physician should also be aware that there can be organic causes for psoas hypertonicity and spasm including femoral bursitis, hip osteoarthritis, prostatitis, salpingitis or other acute pelvic conditions, diverticulosis, ureteral calculus, and cancer of the descending sigmoid colon, to name a few. These should be ruled out by history and physical examination and appropriate testing.

Iliolumbar Ligament Syndrome

In those athletes presenting with vague groin pain, after appropriately ruling out organic causes, one should always consider referred pain from an irritated iliolumbar ligament (Fig. 21.3.6). The athlete may not even complain of back pain until a palpatory examination reveals ligament tenderness. The iliolumbar ligament is usually the first ligament stressed during postural decompensation, and, therefore, iliolumbar ligament palpatory tenderness should stimulate clinical evaluation for sacral base unleveling, scoliosis, and leg-length inequality. Direct injury and irritation to the iliolumbar ligament will often produce a tender point on the iliac crest located on the ipsilateral side of the complaint approximately 2.5 cm superior and lateral to the posterior superior iliac spine (PSIS); this ligament however, does not always refer pain to the groin.

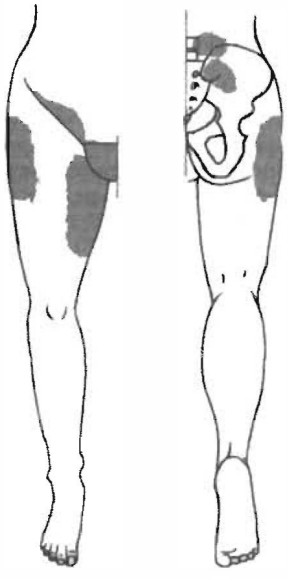

FIGURE 21.3.6. Referral pain patterns of a patient with an irritated right iliolumbar ligament syndrome. (From Kuchera, M. Lumbar region. In: Ward R. *Foundations for osteopathic medicine*, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2003.)

Sacroiliac-Iliosacral Dysfunction

Sacroiliac dysfunction is also commonly present in athletes. The type of sacroiliac dysfunction is variable. The sacrum that freely extends is typically more painful and difficult to treat. This may be related to flexed lumbar dysfunctions or anterior-posterior (A-P) curve issues. Bilaterally flexed sacrums are seen with an increased lumbar lordosis. Treatment of the iliopsoas hypertonicity typically helps sacroiliac dysfunction. If significant sacral dysfunction is still present after treating the psoas, muscle energy, myofascial, or indirect techniques for the sacrum are useful. Dysfunction of the innominates or pubes may also maintain sacroiliac dysfunction.

Piriformis Syndrome

A hypertonic piriformis muscle is a common finding in low back pain. Even though an athlete may have an acute piriformis syndrome, a hyper-

tonic piriformis muscle should be viewed as a symptom and the underlying sacroiliac joint dysfunction, pelvic obliquity, and/or weakness of the gluteus medius on the ipsilateral side should be sought after. In an acute piriformis syndrome, there is often significant pain in the middle of the buttock with radiation down the posterior thigh, usually not below the knee, with occasional lateral radiation. Examination findings include sciatic notch tenderness, positive straight leg raise test that may mimic lumbar radiculopathy, and pain reproduced with passive internal rotation and active external rotation.

The underlying sacroiliac and iliosacral dysfunctions should first be treated and then a technique to quiet the piriformis is often helpful to end the treatment session. This author has found that a significantly hypertonic piriformis muscle often does not respond well to direct stretch as either an office or home treatment modality, and counterstrain seems to be the most effective method. This can be taught to the athlete and continued at home on a daily or symptomatic basis.

Iliacus Dysfunction

The "iliacus complex" has been defined by Eland to represent all the significant *single-joint* factors that influence resistance to hip extension in motion (54). This complex includes the iliacus muscle and the iliofemoral ligament and their associated fascia, which appear to play the major role; and the pectineal muscle and the ischiofemoral ligament and their respective fascia, which likely play a lesser role. Eland also defined, studied, and introduced the "iliacus test," which was designed to assess the stretch capacity of the iliacus complex and has been found to be significantly different from Thomas's test, which assesses the psoas muscle (55).

Gravitational Strain

The model of suboptimal posture, though incomplete, has shown to be effective when used as a model to guide treatment (56). Posture can be defined as the size, shape, and attitude of the musculoskeletal system with respect to gravitational force (57). Subtle departure from ideal posture has been implicated as an important biomechanical factor in athletes with regard to injury because it results in increased mechanical stress throughout the body. Postural imbalance also appears to have a close relationship to the neuromuscular imbalance described by Janda (46). Posture must always be evaluated as part of the biomechanical evaluation. The size, shape, and attitude of the three cardinal bases of support should always be included: standing surface, the feet, and the base of the sacrum.

Recurrent somatic dysfunction and altered postural alignment should clue the physician in to the diagnosis of gravitational strain or postural imbalance. Gravitational strain is a systemic neuromuscular response of postural alignment and muscle firing patterns to chronic gravitational stress (59). Gravitational stress, an obligatory consequence of bipedal posture, is a constant and a greatly underestimated systemic stressor (58). It is most important that one understand that postural imbalance is a systemic neuromuscular dysfunction.

Initial treatment in these chronic pain states must focus on the reeducation of the neuromuscular system. This is accomplished by seeking optimization of posture through the use of one or more of the following modes of physical manipulation (57):

1. Contoured orthotics worn in the shoes to optimize foot and lower extremity biomechanics.
2. A flat orthotic of sufficient thickness to level the sacral base.
3. Manual medicine, with or without mobilization, directed to restore resilience to soft tissues and motion of restricted joint segment.
4. Daily practice of a therapeutic posture for 20 minutes to counter the bias of soft tissues reflective of the initial posture.
5. Use of pelvic belts (sacral belts) or a device for sacropelvic support during the postural retraining process.

During the implementation of the above, a principle-centered, functional rehabilitation program that focuses first on the stretching of

tight, hypertonic postural muscles, strengthening of weak phasic muscles, and proprioceptive retraining must be carried out (59,60). Muscle imbalances must be eliminated, and coordinated movement patterns returned to normal before strengthening of the core can begin effectively.

Hypomobility versus Hypermobility

Chronic stress can cause connective tissue structures to degenerate and lose their ability to compensate. Eventually ligamentous laxity and hypermobility can occur. Failure to identify the enthesopathy or tendinopathy that develops in the iliolumbar, sacroiliac, and sacrotuberous ligaments, and the attachments of the gluteals and erector spinae, can lead to incomplete resolution of symptoms or failure of treatment.

When choosing a treatment approach for hypermobile segments, one must try to differentiate between functional and structural hypermobility, because treatment can be quite different (58). Structural hypermobility implies a fixed defect, whereas functional hypermobility implies a compensatory change that can be altered through manual procedures and/or exercise. Not all hypermobile regions are structural.

Hypomobile somatic dysfunction can produce functional or compensatory hypermobility in the early stages. If the somatic dysfunction is left untreated, over time the dysfunctional segments can develop structural hypermobility. Initial treatment of functional hypermobility should include a multimodality treatment approach, including manual medicine treatment, neuromuscular and/or postural retraining, and functional core strengthening. A comprehensive manual medicine treatment can restore joint movement and resiliency to the soft tissue structures of the hypomobile segments while reducing segmental neural facilitation, thereby decreasing pain (61). Comprehensive neuromuscular and postural rehabilitation to supplement the function of weakened ligaments should be accomplished.

Clues to structural hypermobility are the bony exostoses at attachments of postural muscles and ligaments on radiography, which develop as a joint attempts to stabilize itself and/or recurrent somatic dysfunction. This recurrent somatic dysfunction often improves only in the short term with the combined treatment of osteopathic manipulative treatment OTM and active physical therapy focusing on neuromuscular retraining and core stabilization. Regenerative injection therapy (RIT), or prolotherapy as it was previously known, can then be used as an adjunctive treatment to eliminate the remaining residual structural hypermobility (62). Combining RIT with OTM and an active, functional, multimodal physical therapy program can resolve many chronic pain complaints (58).

LUMBOSACRAL SPRAIN

Pathology

In general, a lumbosacral strain can be differentiated and classified as primarily a result of joint dysfunction, true muscle strain, ligament strain, core weakness, neuromuscular imbalance, ligament laxity (microinstability), tendinitis, apophysitis, tendinosis, postural abnormality, and combinations of these. Pain, alone or absent, is not a reliable symptom to indicate whether or not dysfunction is present (63). It is not uncommon to find postural imbalance such as leg-length inequality, somatic dysfunction, and/or neuromuscular imbalance in athletes without pain. All dysfunctions that develop in the body further limit the body's ability to adapt to the environment. The resulting situation is one in which less stress to the system is needed in order to induce overuse injury. The higher the competing level of the athlete, the less ability he or she has to tolerate even the most minor or seemingly insignificant biomechanical, musculoskeletal, and neuromuscular imbalances.

The history should also find out information, such as

What makes the pain better or worse
Any exacerbating problems
Effect of coughing or sneezing on the pain
Associated neurologic symptoms
Previously known spinal curvatures

Situation at work and home (pending litigation, familial instability, abuse charges)

Medications used

Standard Treatment

Acute. The immediate consequence of an athletic back injury is a decrease in functional mobility with a resultant deconditioning of the athlete. Therefore, any treatment program should encourage as much activity as possible. Conventional medicine recommends *r*est, *i*ce, *c*ompression, and *e*levation (RICE) therapy. Sports physicians typically recommend keeping the athlete moving as much as he or she can tolerate, often preferring MEAT therapy (*m*ovement, *e*xercise, *a*nalgesics, and *t*reatments) that promotes increased blood flow and immune cell migration. Therapies that increase blood flow include therapeutic exercise, manual medicine, ultrasound, myofascial release, and electrical stimulation. Sports-minded physicians typically recommend a quick change from passive modalities to active, function-based therapeutic exercise (59).

Subacute. Treatment of subacute low back pain focuses on the specific identifiable biomechanical abnormalities, joint restrictions, postural imbalances, and muscle imbalances. Therapies should focus on movement using function-based therapeutic exercise to address muscle and balance and core strength.

MANUAL MEDICINE TECHNIQUES

Manual medicine can provide an indispensable benefit to the athlete. The clinician can use his or her hands immediately at the time of injury or just following the injury, providing local tissue support that can decrease the amount of pain the athlete will experience and accelerate healing. For example, in sports such as football, basketball, or hockey, the athlete can be immediately evaluated off the playing surface (once medical stability is determined) and placed through appropriate ranges of motion, thereby significantly reducing the immediate inflammatory response by providing an environment for the better exchange of fluids and less initial restriction of motion.

The specific method and application of force chosen should be determined on an individual basis, based on the specific athlete's level of hypertonicity, tissue texture feel, specific somatic dysfunction, and clinician's skill. The specific techniques are not usually chosen based on a specific underlying clinical condition (e.g., lumbosacral sprain, spondylolysis, and so on). Sequencing of treatment will vary between practitioners, but usually follows a regional sequence as discussed by Greenman (23), or sequential treatment of the overall area of greatest restriction (AGR). The following general techniques and sequence can be used in most sideline and office settings.

Soft Tissue Lumbar: Prone Pressure Technique

Rationale: Relaxes lumbar paravertebral muscles suffering from spasm or hypertonicity.

1. The athlete is prone on the table while the clinician stands at either side of the table.
2. The clinician contacts the lumbar paravertebral musculature on the opposite side of the spine with the heel of his or her cephalad hand.
3. The clinician grasps the anterior superior iliac spine (ASIS) with the caudad hand, pulling upward toward the ceiling while applying a simultaneous anterolateral force, stretching the lumbar paravertebral tissue like a bowstring (Fig. 21.3.7).
4. The clinician can use either a kneading motion or deep inhibitory pressure and repeat as many times as necessary.

Variation: Prone "Scissors" Technique

1. Instead of grasping the athlete's ASIS, the clinician grasps the athlete's opposite leg just above the knee with the caudad hand, lifting the leg far enough to cross it behind the nearer leg.
2. The clinician then contacts the lumbar paravertebral muscles on the far side with the heel of the cephalad hand. Pressure is applied anterolaterally with the cephalad hand, while

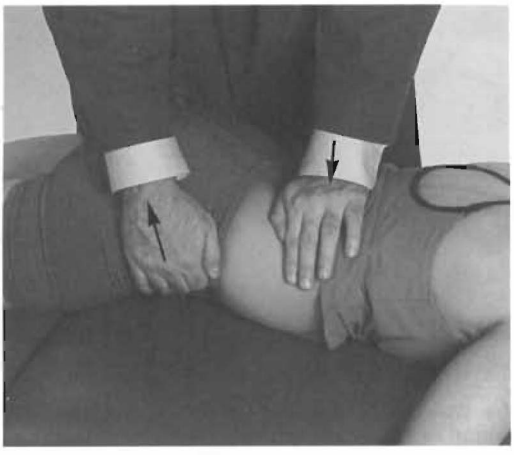

FIGURE 21.3.7. Soft tissue lumbar: prone pressure technique.

simultaneously increasing the amount of scissoring with the legs.
3. The clinician may use an intermittent kneading motion or sustained inhibitory pressure and repeat as many times as necessary.

Supine Lumbar Articulatory Technique (Left Side)

1. The athlete is supine while the clinician stands at the side opposite the side to be treated.
2. The athlete places his or her hands behind the head and locks the fingers.
3. The clinician slides his or her arm within the athlete's left arm and rests the dorsal hand on the athlete's sternum while the opposite hand puts pressure on the ipsilateral ASIS (Fig. 21.3.8).
4. The athlete takes a deep breath in and out, relaxing the body, while the clinician rotates the body toward the right ASIS, using his or her arm as a fulcrum for the athlete.
5. At end range of motion, the clinician introduces a small impulse posteriorly on the ASIS.

Direct Muscle Energy Technique ("Lumbar Walk")

Rationale: Increases lumbar side bending, reduces acute spasm, preparatory technique prior to high-velocity, low-amplitude (HVLA) thrust.

1. The athlete is laterally recumbent while the clinician stands facing the athlete.
2. The athlete's leg closest to the table is extended until motion is felt at the lumbosacral junction.
3. The athlete's back is rotated toward the table by pulling the arm next to the table forward until motion is palpated down to the lumbosacral junction.
4. The clinician grabs the upper leg at the ankle and flexes the knee to 90 degrees (Fig. 21.3.9).
5. The athlete's leg is then used as a long lever and a combination of elevation of the leg (which is abduction in this position) and internal rotation of the hip (which induces lumbar rotation opposite the side bending) is carried out until the restrictive barrier is felt at the palpating finger.
6. The athlete is then instructed to push the ankle directly down toward the floor at approximately one third his or her strength. An equal and opposite force maintains the leg position at the barrier for 3 to 5 seconds.
7. Relax, reposition, repeat, and retest.
8. The athlete's hip is then primarily flexed with the addition of some slight abduction and internal rotation until the restrictive barrier at the next lumbar segment moving cephalad is felt (Fig. 21.3.9B).

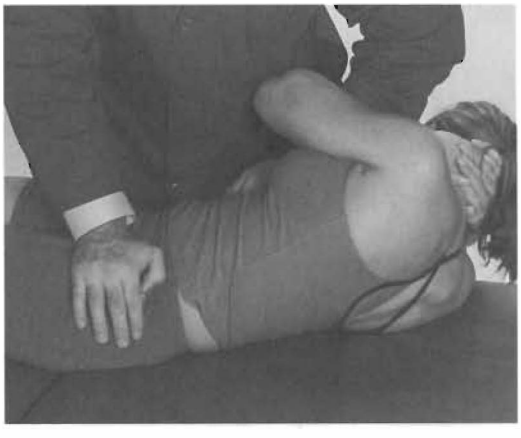

FIGURE 21.3.8. Supine lumbar articulatory technique (left side).

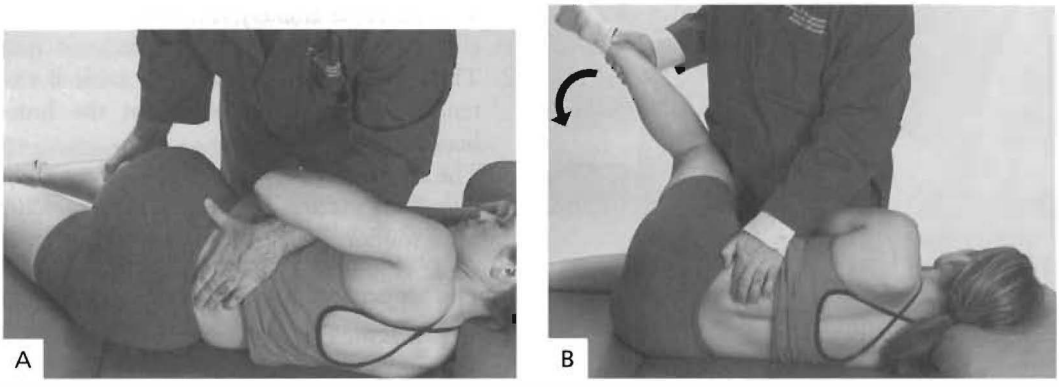

FIGURE 21.3.9. Direct muscle energy technique ("lumbar walk"). **A,** The down leg starts in extension, and the top leg is lifted. After the first contraction, the down hip can be flexed at the hip, and the top leg can be brought into more abduction and internal rotation in order to isolate the next vertebral segment up **(B)**.

Clinical Pearl: This is a useful technique for the athlete with acute low back pain. It can be used on the sideline or in the office to reduce acute spasm or as a preparatory technique prior to performing a specific HVLA technique for a non-neutral lumbar or sacral segment. It is usually well tolerated even in the most acute athlete.

Variation. Single-leg muscle energy technique for non-neutral flexed and extended lesions of the lumbar spine.

1. The posterior transverse process should be placed down on the table. Side bending and rotation will be to opposite sides as the athlete's hip is abducted and internally rotated. Therefore, when used for non-neutral dysfunctions, the technique focuses on the usual major restrictive motions in the lumbar spine of the flexed or extended component plus side bending, but is inherently not multiplanar.
2. Though the lateral recumbent position is often the most comfortable position for the acute athlete, this technique can be performed supine if the athlete prefers this position. The opposite leg can be flexed or flat on the table.

Indirect Ligamentous Articular Release of Thoracolumbar/lumbosacral Dysfunction

Rationale: This provides indirect release of ligamentous and myofascial restricted structures and somatic dysfunction from the sacrum to thoracolumbar junction that limit motion and function.

1. The athlete is supine while the clinician is seated on the side of the athlete that is the same as the dominant hand, just below the level of the sacrum, facing cephalad.
2. The clinician's dominant hand cups the sacrum and his or her palm while the other hand transverses longitudinally the axis of the spine at the thoracolumbar junction. The finger pads and thenar eminence should be on the paravertebral muscles bilaterally (Fig. 21.3.10).
3. The clinician brings the sacrum to a balance point indirectly by moving it superiorly, and inferiorly, sideways, and/or in rotation to the point of ease.
4. The clinician carries the affected vertebra of the thoracolumbar junction anteriorly and superiorly cephalad, which is almost always the position of ease.

Clinical Pearl: When there are several dysfunctional segments, start at the lowest area and work cephalad.

Muscle Energy: Lumbar Spine

Rationale: Direct technique to increase the restricted motion of L5 in extension, right rotation, and right side bending by treating non-neutral (type II) flexed lumbar dysfunctions

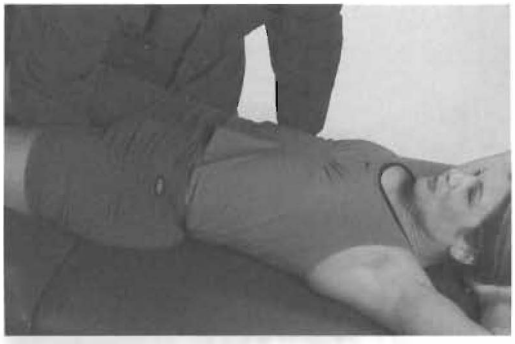

FIGURE 21.3.10. Indirect ligamentous articular release of thoracolumbar/lumbosacral dysfunction.

(i.e., flexed, rotated, and side-bent to the right) [L5 FRS R]).

1. The athlete is laterally recumbent with the posterior transverse process down (the rotation component closest to the table) while the clinician is standing and facing the athlete.
2. The clinician flexes the athlete's hips 45 degrees and the knees at 90 degrees.
3. The clinician places the middle of three fingers at the interspinous space between L5 and S1 with the cephalad hand. The fingers above and below go at the interspinous space above and below this level respectively.
4. The clinician extends the athlete's lumbar spine up to L5-S1.
5. The athlete's upper leg is flexed slightly further at the hip and dropped off the side of the table cephalad to the lower leg, and the pelvis is rotated anteriorly until the initial resistance reaches the segment to be treated.
6. The clinician palpates the L5-S1 interspinous space with the cephalad hand while the athlete's upper shoulder is carried posteriorly again until the initial resistance reaches the segment to be treated.
7. The athlete is instructed to pull gently forward with his or her shoulder for 3 to 5 seconds. (Fig. 21.3.11)
8. The athlete is then instructed to pull his or her hips gently backward. This contraction is held for 2 to 5 seconds. After 2 seconds of relaxation, the hip is carried forward until a new restrictive barrier is met.

Clinical Pearl: Lumbar muscle energy technique in a lateral recumbent position can be used to treat specific lumbat restrictions or performed bilaterally as a preparatory technique for HVLA treatments. This positioning is often comfortable and gentle enough for hurting athletes in various settings, particularly the sideline. It improves lumbar side bending and rotation while stretching the multifidus and small rotators and intertransverse muscles to decrease muscle tone and irritability.

Variation. The athlete is sitting with the arms folded across the chest, while the clinician stands behind. Side bending is caused by the

FIGURE 21.3.11. Muscle energy technique for the lumbar spine.

clinician leaning on the contralateral shoulder. Flexion and extension are created by deep respiration; to extend the spine, the clinician holds the athlete at end-inspiration (the spine extends during inspiration), then following the slumping of the spine during exhalation he or she brings the lumbar spine into flexion. Rotation is from holding the contralateral shoulder and walking around, bringing the lumbar spine into the restrictive barrier.

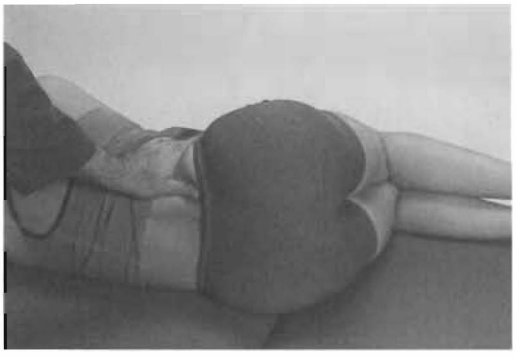

FIGURE 21.3.12. Direct ligamentous articular release of the iliolumbar ligaments and erector spinae muscles.

Direct Ligamentous Articular Release of the Iliolumbar Ligaments and Erector Spinae Muscles

Rationale: Direct release of the iliolumbar ligament and erector spinae muscle restriction to improve motion and function.

1. The athlete lies laterally recumbent with the restricted side up and the knees and hips flexed, while the operator faces the athlete's feet, standing posterior to the athlete at the level of the midthoracic spine.
2. The clinician places the pad of the thumb on the iliolumbar ligament superomedial to the PSIS between the ilium and the L4-L5 vertebra and tests the iliolumbar ligament by pressing anteriorly (Fig. 21.3.12).
3. The clinician continues pressing anteriorly and inferiorly with the thumb, maintaining balanced pressure at this barrier until the release occurs.

Clinical Pearl: Treating the strained iliolumbar ligaments is essential to achieving a full release of the sacrum and lumbar spine. After releasing the iliolumbar ligament, consider continuing to move anteriorly and superiorly to release the latissimus dorsi.

Direct Myofascial Release of the Iliopsoas Muscle

Rationale: Release of the hypertonic iliopsoas can be an essential treatment required to achieve full release of the sacrum and lumbar spine as well as reversal of neuromuscular imbalance.

1. The athlete lies supine while the clinician stands opposite the side to be treated facing the table at the level of the pelvis.
2. The clinician contacts the iliopsoas muscle just inferior and medial to the ASIS using the distal pad of the thumb of the dominant hand, placing the other hand's thumb on top to reinforce the pressure.
3. The clinician engages the muscle initially by directing a force posteriorly straight down toward the table on the medial aspect of the muscle and holding a steady, balanced force laterally once the muscle is engaged, until a releases is felt and the muscle relaxes (Fig. 21.3.13).

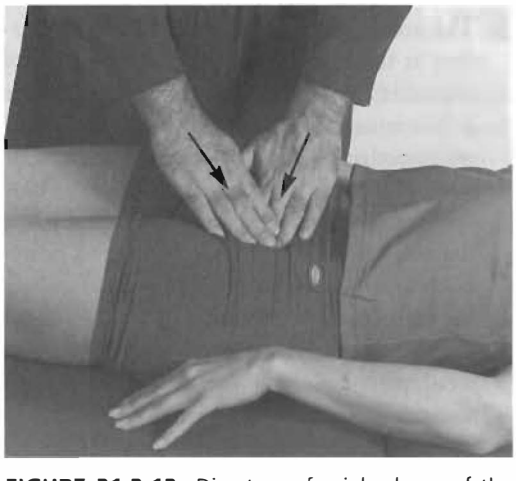

FIGURE 21.3.13. Direct myofascial release of the iliopsoas muscle.

Counterstrain for the Iliacus Muscle. Treats iliacus tender points: anterior and deep in the iliac fossa.

Rationale: Since the iliacus assists the iliopsoas in flexion, its release can be essential to achieving full release of the sacrum and lumbar spine and reversing neuromuscular imbalance.

1. The athlete lies supine with the clinician standing on the side to be treated, facing the table at the level of the pelvis. A pillow can be placed under the buttocks to raise the pelvis, increasing flexion.
2. The clinician places his caudad foot at the base of the athlete's buttocks with the knee flexed at 90 degrees.
3. The clinician places the athlete's ankles on top of his or her thigh with the ankles crossed, with the ankle initially closest to the clinician on the bottom.
4. The clinician then places the athlete's hips in an extreme bilaterally flexed and externally rotated position until the tender point is abolished (Fig. 21.3.14).
5. The clinician slowly places the athlete's legs back on the table and the tender point is rechecked.

FIGURE 21.3.14. Counterstrain for the iliacus muscle.

Lumbar High-Velocity, Low-Amplitude Technique

Rationale: HVLA is directed at the lumbar spine to increase lumbar joint motion. The classic "lumbar roll" is a convenient and comfortable position to use for most athletes.

1. The athlete is placed in the lateral recumbent position with the posterior transverse process of the desired treatment level up.
2. The athlete's back is rotated toward the table by using the arm of the shoulder next to the table until motion is felt at the segment to be treated.
3. The leg closest to the table is extended until motion is felt at the segment to be treated. The ankle of the upper leg is placed in the popliteal space of the lower leg so that the knee and hip are bent at roughly 45 degrees.
4. The clinician places his forearm over the upper hip and lumbar region, and the athlete's left forearm is placed on the athlete's upper chest. The rotation is carried out by both arms until all slack is taken up
5. The athlete takes a deep breath, and during expiration, further slack is taken up and the segment to be treated is localized.
6. The clinician introduces a short sharp thrust with the forearm over the hip and lumbar region while the other forearm maintains its position (Fig. 21.3.15).

FIGURE 21.3.15. Lumbar high-velocity, low-amplitude technique.

Variation for the Sacroiliac Joint. The dysfunctional sacroiliac joint is placed up in the lateral recumbent position. The same sequence is carried out as for the lumbar region, except the upper leg is draped off the table and the clinician uses his or her leg to lock the athlete's leg down onto the table and any flexed position. As the thrust is carried out, as opposed to a horizontally directed thrust as would be carried out to address the lumbar spine, the thrust is carried out in roughly a 45-degree angle horizontally, down, and across into the leg being held by the clinician.

PREVENTION

Self-mobilization and functional strengthening through active therapeutic exercise are the keys to prevention of back pain. A daily home program consisting of a principle-centered, functional rehabilitation program that focuses on the stretching of tight, hypertonic postural muscles, strengthening of weak phasic muscles, and proprioceptive retraining must be carried out. It is critical to remember that muscle imbalances must be eliminated and coordinated movement patterns returned to normal before strengthening of the core can begin effectively. Progressive advance of a functional and sport-specific–based core strengthening program can then be instituted.

Therapeutic Exercise

As flexibility improves, the athlete is started into movement patterns to restore firing sequences that have changed due to neuromuscular imbalance. It is only after the resolution of neuromuscular imbalance patterns that actual strengthening can ensue; otherwise, inhibitory stimuli will continue to facilitate neuromuscular imbalance patterns. Strengthening should be focused on both functional and sport-specific movement including stabilization of the core using muscular control while performing activities of daily living and sport while maintaining a neutral pelvis.

Principle-centered functional rehabilitation traditionally started by teaching the athlete how to find and maintain the neutral spine position. The athlete should be able to control pelvic motion in both a flexed and extended posture. Only when the athlete understands how to actually control pelvic movement can he or she truly appreciate and understand maintenance or holding of the neutral spine position.

LUMBAR STRETCHES

Comprehensive flexibility is always a critical starting point, focusing on the muscles that typically become hypertonic, and should include both upper and lower extremity muscles. In the lower body, these include the hamstrings, gastrocnemius and soleus, internal rotators of the hip, and hip flexors. Work by Eland, as presented earlier, has demonstrated an inherent functional difference between the psoas and iliacus muscles. When stretching, it is essential to isolate and stretch each of these separately. Eland proposes that a tight iliacus is a common event leading to low back pain (54). A restricted and tight anterior hip capsule is also often found and should be mobilized. This can be differentiated by testing internal rotation of the hip in the prone position. If a gradual end point is felt, typically any restriction present is primarily myofascial. If the end range has a sharp, sudden end point, hip capsule restriction is present, necessitating the need for hip capsule mobilization.

Hamstring Muscle (including semitendinosus, biceps femoris, semimembranosus). Lie on your back with a beach towel or belt attached on your right foot, and pull your right leg up toward your face. You should feel stretching in the back of your right thigh. Upon release of the stretch, the discomfort should cease (Fig. 21.3.16A). Good stretch for sports like track and field, basketball, football, softball/baseball, soccer, and hockey.

Quadriceps Femoris Muscle. Stand with the left hand supported on a stationary object to maintain your balance. The right hand holds the ankle. Pull the right leg toward the buttocks so the ankle approximates the buttocks.

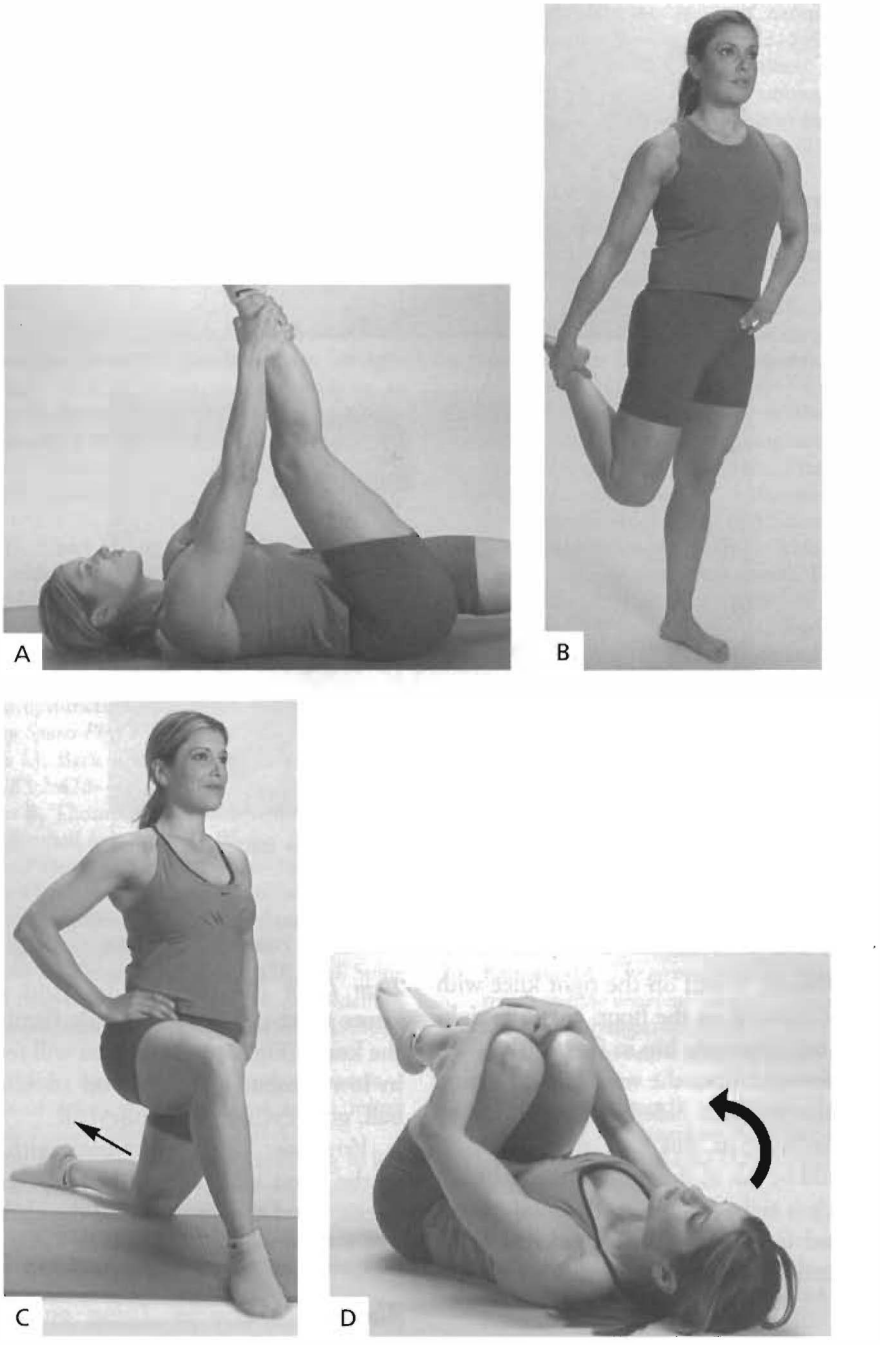

FIGURE 21.3.16. Stretches for the lumbar spine: **A,** Hamstring; **B,** quadriceps femoris; **C,** iliopsoas; **D,** lumbar erector spinae.

Then tighten/squeeze your buttocks muscles to produce a posterior tilt of the pelvis (Fig. 21.3.16B). You should feel further stretching in the front of your right thigh. Good stretch for track and field, basketball, soccer, volleyball, baseball/softball, and wrestling.

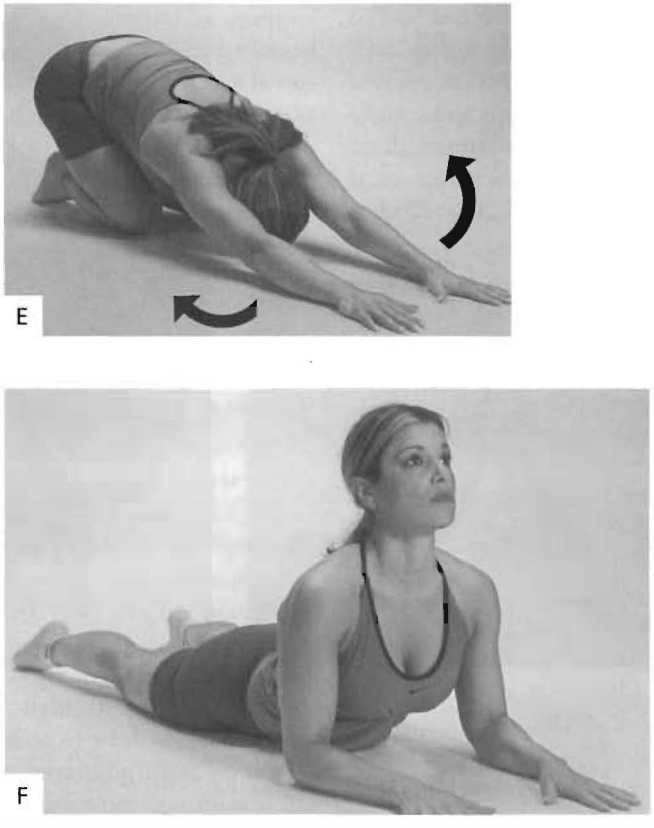

FIGURE 21.3.16. (*continued*) E, general low back; **F,** abdominal wall.

Iliopsoas Muscle. Kneel on the right knee with the left foot forward on the floor. Turn the right foot out to turn the right hip in (Fig. 21.3.16C). Then lean forward from the waist while keeping the back erect/straight. Side-bend the trunk to the opposite side, in this case to the left. A stretch should be felt in the front of the right hip if this muscle is tight or shortened. Good stretch for track and field, basketball, baseball/softball, hockey, soccer, hand/racquetball, dancers, and gymnasts.

Lumbar Erector Spinae Muscles, Stretching Left and Right. Kneel on the floor, flexed at the waist, laying your arms on the floor. Reach forward, then slide hands and body to the left, then right (Fig. 21.3.16D).

Low Back Muscles. Lie on your back with knees to the chest and hands firmly securing the knees (Fig. 21.3.16E). You will feel a stretch in low lumbar region. Good stretch for football, golf, cycling, and volleyball.

Variation: Lie on your back with the knees to the chest and the hand firmly securing the knees. Bend the head toward the chest.

Abdominal Muscles. Lying on stomach or prone, keep the hips securely against the table. Using the hands, push up so the elbows are extended/straight. Your lower back should be extended (Fig. 21.3.16F). You should feel a stretch in the abdominal area if these muscles are tight.

Variation: Lie on the back over a Swiss ball. You should feel a stretch in the abdominal area if these muscles are tight.

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22

HIP AND PELVIS

22.1

Anatomy

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The pelvic girdle is composed of three joints that work together to provide mobility for the lower limb and stability for the body. These include the acetabular-femoral (hip) joint, the sacroiliac joint, and the pubic symphysis. The sacroiliac joint and pubic symphysis are almost totally immovable joints, while the hip joint is very mobile. Anatomy of the hip and pelvis is presented in detail in major anatomic textbooks (1–6).

Several bony structures can be palpated as one examines the pelvic girdle. Beginning anteriorly, the subcutaneous anterior superior iliac spine can be easily palpated at the anterior end of the iliac crest (Fig. 22.1.1A). The iliac crest extends posteriorly from the anterior superior iliac spine to terminate at the posterior superior iliac spine, which lies directly deep to the dimples just superior to the buttocks. The iliac crest is subcutaneous and serves as the point of attachment for several muscles. Along the lateral lip of the iliac crest and approximately 7.5 cm from its apex lies the iliac tubercle, which marks the widest point on the crest. An imaginary transverse line connecting the tops of the iliac crests on each side crosses between the spinous processes of the L4 and L5 vertebrae.

The posterior edge of the greater trochanter of the femur can be easily palpated along the superior lateral aspect of the thigh (Fig. 22.1.2). The anterior and lateral portions of the greater trochanter are covered by the tensor fasciae latae and gluteus medius muscles and are less available for palpation. Following down along the inguinal crease medially and obliquely from the anterior superior iliac spine one can palpate the pubic tubercle deep to the pubic hair and mons pubis. It should be noted that the pubic tubercle and the superior edge of the greater trochanter lie on the same transverse plane.

Posteriorly, the ischial tuberosity lies in the middle of the buttocks at the level of the gluteal fold (Fig. 22.1.3). It is covered by the gluteus maximus muscle and fat and can only be palpated when the muscle moves superiorly during hip flexion. The ischial tuberosity lies on the same transverse plane as the lesser trochanter, which is not palpable and arises from the posterior medial surface of the proximal femur.

The sacroiliac joint is not palpable and the center of the joint is located at the level of the S2 segment of the sacrum. It is crossed by an imaginary transverse line connecting the posterior superior iliac spines on each side. Other important bony landmarks that are not palpable include the greater and lesser sciatic notches, the ischial spine and the acetabulum of the hip bone, and the head and neck of the femur.

The hip joint is a ball-and-socket synovial joint. At this joint the head of the femur articulates with the acetabulum. The depth of the acetabulum is increased by the fibrocartilaginous acetabular labrum, which attaches to the bony rim of the acetabulum and the transverse acetabular ligament. A strong fibrous capsule surrounds the joint, which attaches proximally to the rim of the acetabulum and the transverse acetabular ligament and distally to the intertrochanteric line of the femur anteriorly and the neck of the femur

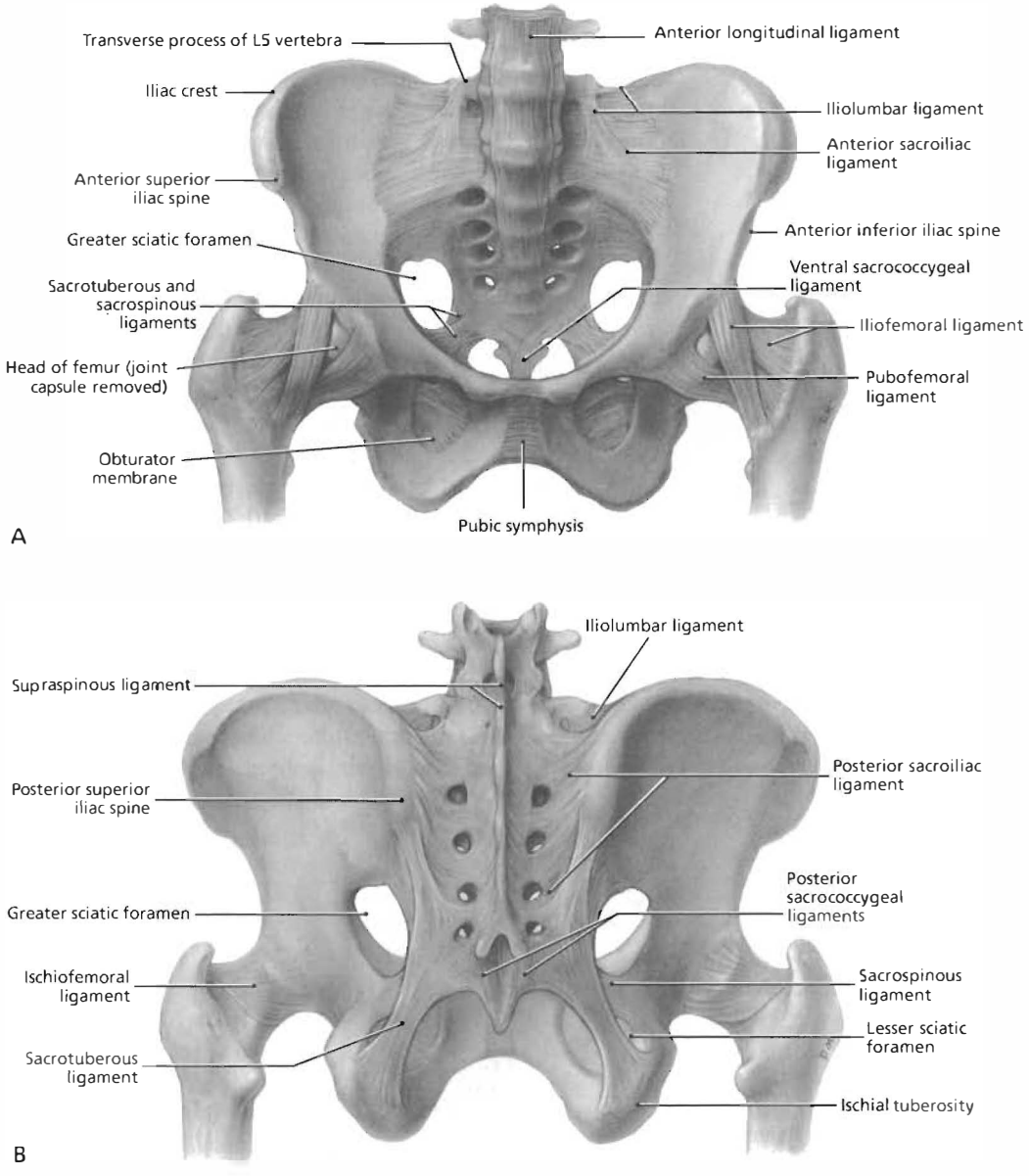

FIGURE 22.1.1. A, Anterior bony pelvis with important ligaments. **B,** Posterior bony pelvis. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy*, 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

posteriorly. Most of the fibers of the fibrous capsule take a spiral course from the hip bone to the femur. The synovial membrane, which lines the fibrous capsule, also covers the neck of the femur, which is intracapsular, and the nonarticular portion of the acetabulum as well as the ligament of the femoral head. The strong Y-shaped iliofemoral ligament reinforces the fibrous capsule anteriorly and is extremely important in preventing overextension of the hip during standing by screwing the head of the femur into the acetabulum.

The fibrous capsule is reinforced anteriorly and inferiorly by the pubofemoral ligament,

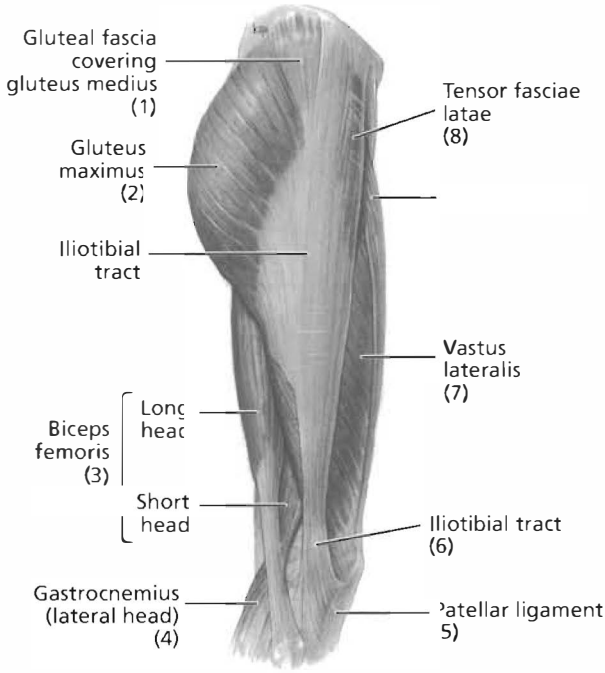

FIGURE 22.1.2. Lateral hip and thigh. Highlighted is the full extent of the iliotibial tract, from the iliac crest into the tibial plateau and fibular head. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

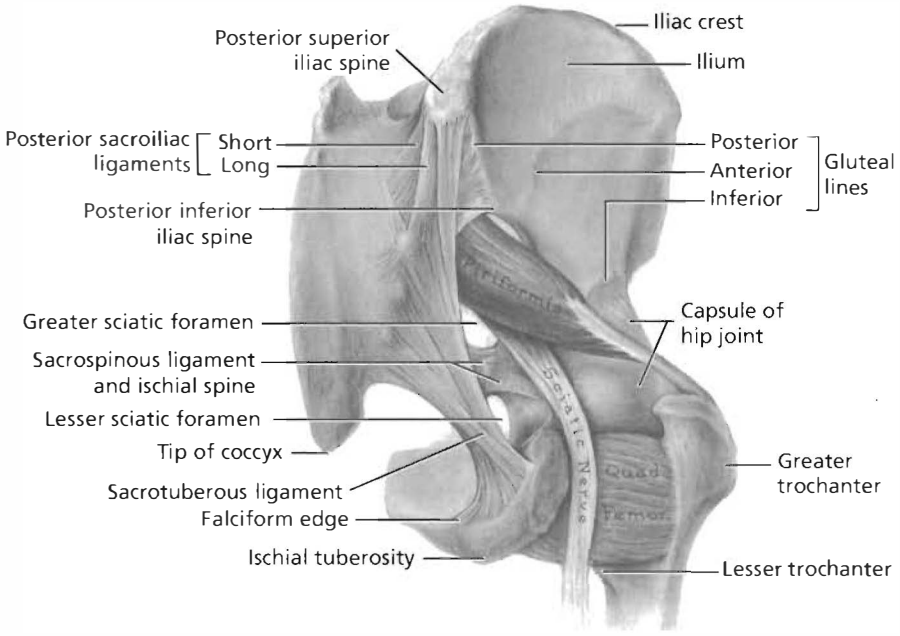

FIGURE 22.1.3. Posterior view of the hip. Note the relationship among the sciatic nerve, the piriformis muscle, the sacrotuberous ligament, and the hip capsule. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

which tightens during extension and abduction of the hip, thereby preventing overabduction at the joint. The ischiofemoral ligament reinforces the hip joint posteriorly where it screws the head of the femur medially into the acetabulum during extension, thereby preventing hyperextension of the joint. The intracapsular ligament of the femoral head, which connects the acetabulum to the femoral head, is weak and usually contains a small artery to the head of the femur. The hip joint movements are flexion-extension, abduction-adduction, medial-lateral rotation, and circumduction.

The joint receives its blood supply from branches of the medial and lateral femoral circumflex arteries, the artery of the femoral head, and a branch of the obturator artery. Branches of the femoral and sciatic nerves, the obturator nerve, and the superior gluteal nerve innervate the hip joint.

The sacroiliac joint is an extremely strong weight-bearing synovial joint between the articular surfaces of the ilium and the sacrum. These surfaces display irregular elevations and depressions, which serve to interlock the bones. Interosseous and anterior and posterior sacroiliac ligaments support the joint. The sacroiliac joint allows for very little mobility because of its role in transmitting the weight of the body to the hip bones. Movement at the sacroiliac joint is limited to gliding and rotatory movements when the joint is subject to considerable force. The sacrospinous and sacrotuberous ligaments allow only limited movement to the inferior end of the sacrum, thereby giving resilience to this region when the vertebral column sustains sudden weight increases.

The relatively immobile pubic symphysis is a fibrocartilaginous joint between the bodies of the pubic bones on the anterior midline. The fibrocartilaginous interpubic disc connects the two bones. The joint is reinforced superiorly by the superior pubic ligament and inferiorly by the arcuate pubic ligament.

Five anatomic regions around the hip and pelvic regions must be emphasized: (1) femoral triangle, (2) greater trochanter, (3) sciatic nerve, (4) iliac crest, and (5) hip and pelvic muscles.

The femoral triangle is situated in the superomedial third of the anterior thigh and is bounded by the inguinal ligament, deep to the inguinal crease, superiorly, the adductor longus muscle medially and the sartorius muscle laterally. The inguinal ligament, the base of the triangle, extends from the anterior superior iliac spine to the pubic tubercle. The triangle appears as a depression inferior to the inguinal ligament when the hip is flexed, abducted, and laterally rotated. The floor of the triangle is formed by portions of the adductor longus, pectineus, and iliopsoas muscles. The hip joint lies deep to the floor of the femoral triangle. The roof is formed by fascia lata and cribriform fascia. The apex of the triangle is the point where the medial border of the sartorius crosses the medial border of the adductor longus.

From lateral to medial, the main contents of the femoral triangle are the femoral nerve (L2-L4) and its branches, the femoral artery and its branches, the femoral vein and its tributaries, including the great saphenous vein, and the femoral sheath. The femoral head lies deep to the femoral artery. The femoral sheath is a funnel-shaped fascial tube that encloses the proximal portions of the femoral vessels and the femoral canal. It does not enclose the femoral nerve. The femoral sheath terminates about 4 cm inferior to the inguinal ligament by becoming continuous with the connective tissue covering the femoral vessels. The great saphenous vein and lymphatic vessels pierce the medial wall of the femoral sheath. This sheath allows the femoral vessels to glide deep to the inguinal ligament during hip flexion.

The femoral sheath is subdivided into three vertical compartments by connective tissue septa. These include a lateral compartment for the femoral artery, an intermediate compartment for the femoral vein, and a medial compartment space called the femoral canal. This latter space allows the femoral vein to expand when there is increased venous return from the lower limb. The femoral canal contains fat, lymphatic vessels, and some deep inguinal lymph nodes. The femoral canal opens into the abdomen at its superior end, the femoral ring. Superficial inguinal

lymph nodes can be palpated in the most medial portion of the triangle.

In the region of the greater trochanter is the trochanteric bursa, which protects the posterior and lateral portions of the greater trochanter. This bursa separates the overlying gluteus maximus muscle from the lateral side of the greater trochanter. The gluteus medius muscle attaches to the superolateral portion of the trochanter.

Posteriorly, in the region of the sciatic nerve, the nerve (L4-L5, S1-S3) can be located midway between the greater trochanter and the ischial tuberosity as it emerges through the inferior portion of the greater sciatic foramen, inferior to the piriformis muscle, into the gluteal region (see Fig. 22.1.7). When the hip is extended the sciatic nerve is covered by the gluteus maximus muscle, which moves out of the way when the hip is flexed, making the nerve accessible to palpation. The ischial bursa separates the gluteus maximus from the ischial tuberosity.

The region of the iliac crest is important because the sartorius and gluteal muscles attach just below it and the cluneal nerves cross over it. These nerves (L1-L3) supply the skin over the iliac crest between the posterior superior iliac spine and the iliac tubercle.

Muscles of the hip and pelvis lie in quadrants based on their position and functions. The anterior quadrant contains the flexor muscles. The most important muscles include iliopsoas, sartorius, and rectus femoris. The iliopsoas muscle is a prime stabilizer of the spinal column and the primary hip flexor. It is the conjoint muscle and tendon of the iliacus and psoas major. This muscle is crucial in several functions and roles:

1. Crosses L5-S1 and distributes forces from the large range of motion above through to the relatively limited movement of the sacrum.
2. Distributes forces from below the pelvis cephalad.
3. Originates at the inferior border of the transverse process of L1-L5 and the anterolateral surfaces of the vertebral bodies of T12-L5 and the intervertebral discs between them.
4. Anchors the crura of the diaphragm and is involved in respiratory function.
5. Moves the sacrum in an anterior-posterior fashion.
6. Acts as a prime mover of the lumbosacral junction, influencing sacral mechanics.
7. Affects the lumbar curve. Relaxation of the psoas muscle allows the normally present lumbar lordosis to flatten. Stretching of the iliopsoas muscle may alleviate low back discomfort. A home stretching program directed at the iliopsoas may alleviate pain in the low back.
8. Affects mechanics of gait, respiration, and the sequence of engagement, flexion, descent, and internal rotation of the fetus.

The sartorius flexes, abducts, and medially rotates the hip as well as flexing the knee. The iliopsoas crosses both the hip joint and the knee joint, acting as a flexor of the hip and an extensor of the knee. The iliopsoas is innervated by the anterior primary rami of the L1-L3 spinal nerves. The femoral nerve innervates the sartorius (L2-L3) and rectus femoris (L2-L4) muscles. The abdominal wall consists of the rectus abdominis, external and internal obliquus, and transversalis abdominis muscles (Fig. 22.1.4). Their function is crucial in the support of the lumbar spine and core stabilization. The abdominal wall inserts into the anterior aspect of the pelvis, and the transversalis aponeurosis blends into the inguinal canal. The conjoint tendon along the rim of the pelvic outlet is a popular location for the injuries often called "sports hernias." Inguinal hernias are also common sports injuries that can occur in more than one place (Fig. 22.1.5).

The medial quadrant contains the adductor muscles of the hip (Fig. 22.1.6). The most important muscles in this quadrant include the gracilis, pectineus, adductor longus, adductor brevis, and adductor magnus. The femoral nerve (L2-L3) innervates the pectineus, while the obturator nerve innervates the adductor longus (L2-L4), adductor brevis (L2-L4), adductor magnus (L2-L4), and gracilis (L2-L3).

The lateral quadrant contains the abductor muscles of the hip. The most important muscles in this quadrant include the gluteus

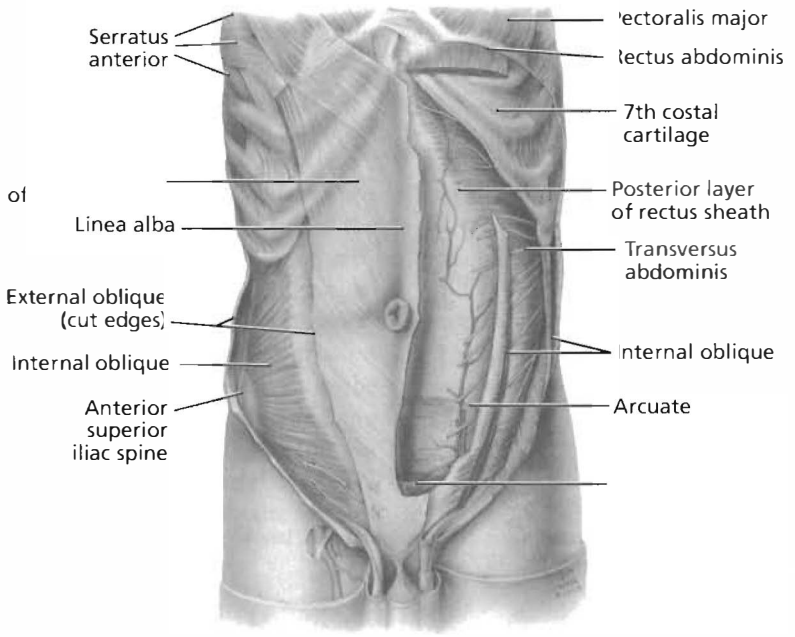

FIGURE 22.1.4. Abdominal wall musculature, deep dissection. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

medius and minimus (Fig. 22.1.7). The gluteus medius is the main hip abductor. The superior gluteal nerve (L5, S1) innervates both muscles.

The posterior quadrant contains the extensor muscles of the hip. The most important muscles in this quadrant include the gluteus maximus and hamstring (biceps femoris long

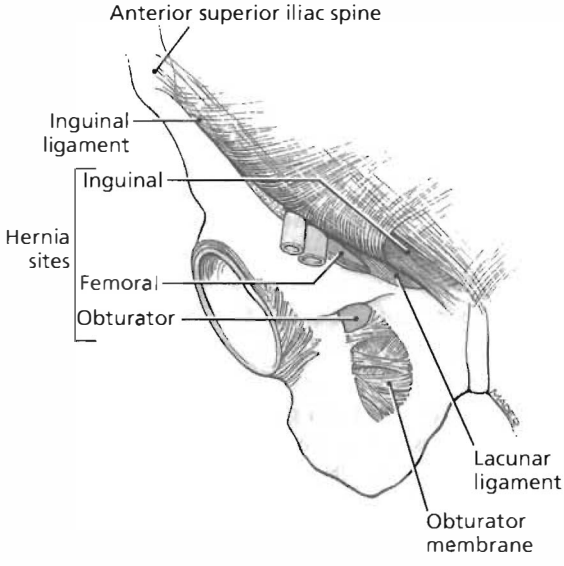

FIGURE 22.1.5. Anteroinferior view of the three hernia sites in the inguinal region. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

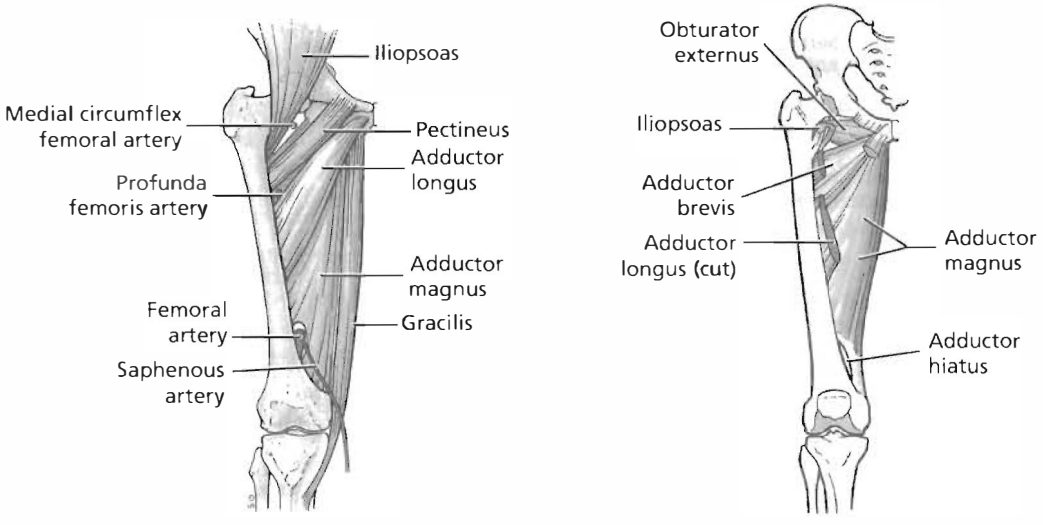

FIGURE 22.1.6. Medial thigh musculature. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

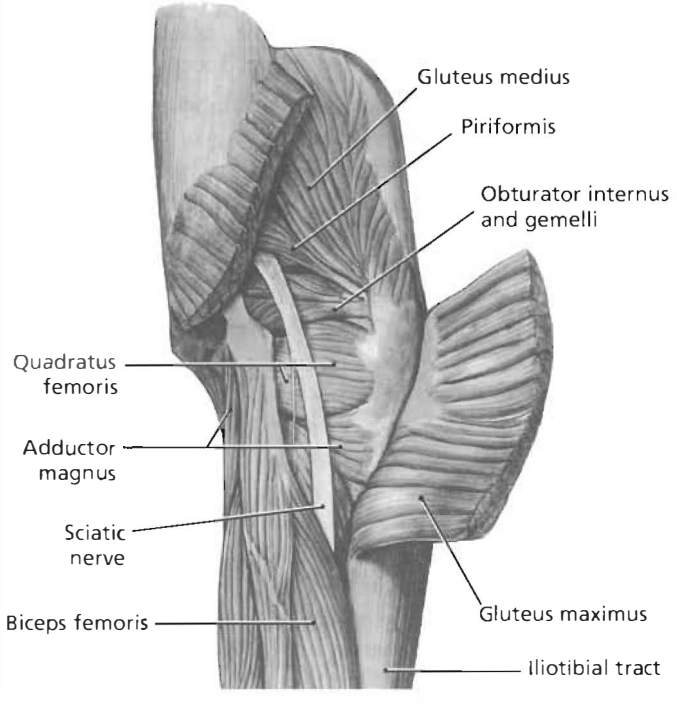

FIGURE 22.1.7. Muscles of the gluteal region. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

and short heads, semitendinosus, and semimembranosus) muscles. The gluteus maximus is the primary hip extensor and can be located between two imaginary lines drawn between the posterior superior iliac spine and rhe greater trochanter superiorly and the coccyx and ischial tuberosity inferiorly. The hamstring muscles, except for the short head of the biceps femoris, all attach proximally to the ischial tuberosity deep to the gluteus maximus. The inferior gluteal nerve (L5, S1-S2) innervates the gluteus maximus. The tibial division of the sciatic nerve (L5, S1-S2) innervates rhe semitendinosus, semimembranosus, and long head of the biceps femoris, while rhe common fibular portion of the sciatic nerve (L5, S1-S2) innervares the short head of the biceps femoris.

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22.2

Physical Examination

THOMAS R. PERKINS

OBSERVATION

The evaluation begins with careful observation and a generalized inspection of the athlete's stance, gait, skin, bony landmarks, lower spine, muscle atrophy, signs of injury, or prior surgery. The athlete's posture is examined and evidence of pelvic obliquity should be noted by placing the fingers just above the iliac crests, at the dimples, which overlie the posterior superior iliac spine (Fig. 22.2.1). Lumbar scoliosis can lead to apparenr obliquity and leg-length discrepancy; however, attempts at leveling the pelvis will fail unlike patients with true leg-length inequality. Abnormal spinal curves, irregular hair distribution and bony deformities represent underlying spinal disease and should be noted (1–3).

Abnormal thigh circumference, and atrophy of the trunk and buttock should also be recorded. Decreased lordosis may represent lumbar muscle spasm, whereas increases may be caused by weak abdominal muscles or fixed flexion deformity of the hip in an attempt to compensate for true hip extension (4). During inspection of the posterior aspect of the hip, the examiner should observe evidence of gluteal folds (both size and depth), which may represent leg-length differences, pelvic obliquity, and even congenitally dislocated hips in children (1,3,5).

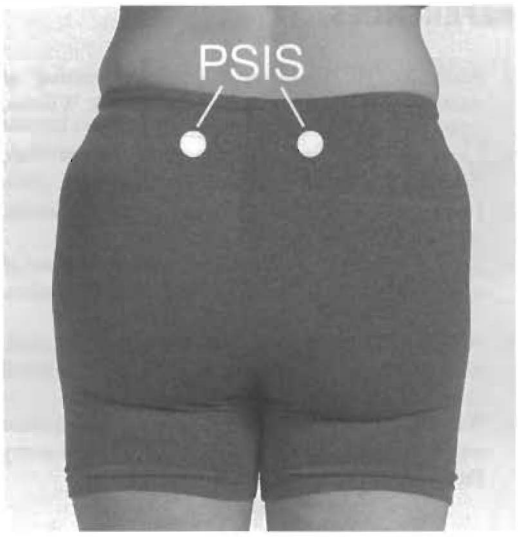


FIGURE 22.2.1. Posterior superior iliac spine.

PALPATION

The athlete is first asked to stand and the examination is carried out in a stepwise fashion. It is helpful to have the athlete lie on his or her side for palpation of deep posterior and lateral structures (Fig. 22.2.2). As one palpates these structures, the examiner should note tenderness and skin temperature. Palpate both sides for comparison (1,2).

Anterior

a. Anterior superior iliac spine (ASIS). Palpate the superior-anterior portion of the iliac crest.
b. Iliac crest. Assess pelvic obliquity.
c. Iliac tubercle. Located posteriorly on the lateral lip of the iliac crest, it marks the widest portion of the pelvis.
d. Pubic tubercle. Follow the inguinal ligament to the front of the pubis, which is at the same level as the top of the greater trochanter (5).
e. Femoral triangle (inguinal ligament, femoral artery, and inguinal lymph nodes). Place the athlete's leg in a figure-four position: The femoral artery is in the midpoint of the triangle, and the hip capsule is approximately 2 cm laterally and proximally. From medial to lateral, the neurovascular structures are the femoral vein, femoral artery, and femoral nerve (VAN) (1,3).
f. Adductor muscle group is palpable with the athlete's leg in an abducted position.
g. Abductor (gluteus medius).
h. Flexors (iliopsoas and sartorius).

Posterior

a. Posterior superior iliac spine. It lies under the dimples above the buttocks.
b. Ischial tuberosity (lateral side lying). Flex the athlete's hip for easy palpation; it is located at the level of the gluteal fold.
c. Extensors (gluteus maximus and hamstrings).
d. Sacroiliac (SI) joint. It is not palpable with the overhang of the iliac crest. The top of the SI joint is even with the top of the L4-L5 vertebrae.

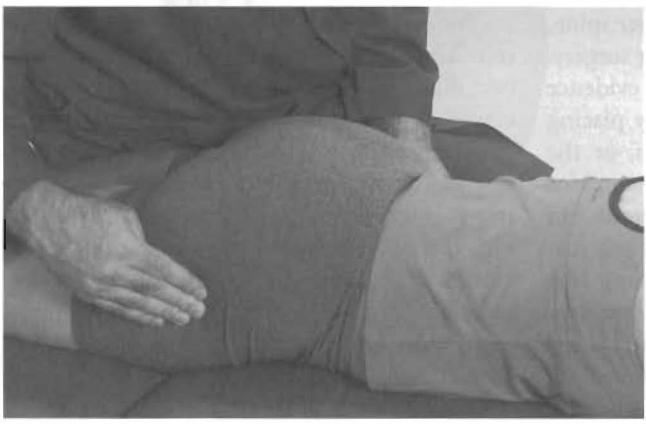
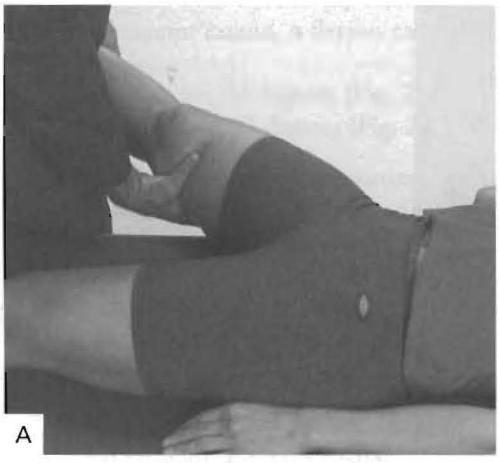
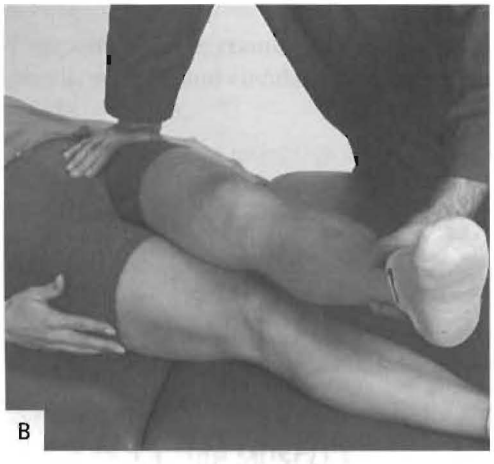


FIGURE 22.2.2. Lateral palpation of the greater trochanters.





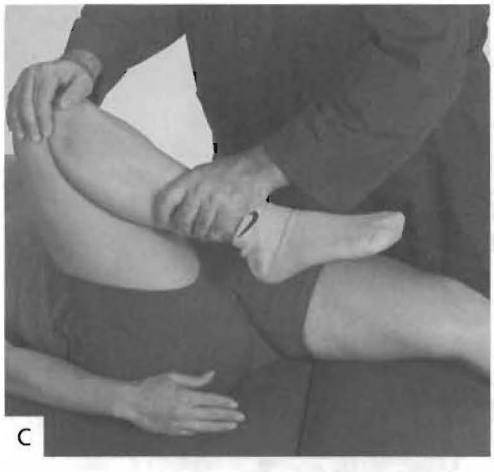


FIGURE 22.2.3. Range of motion. **A,** Abduction: 45 degrees. **B,** Adduction: 20 to 30 degrees. **C,** Flexion: 120 to 135 degrees.

e. Greater trochanter (lateral side lying). The posterior aspect of the greater trochanter is relatively uncovered in this position and easily palpable.
f. Greater trochanteric bursa (sciatic nerve—midway between the greater trochanter and the ischial tuberosity).

RANGE OF MOTION

1. *Abduction: 45 degrees* (Fig. 22.2.3A). With true hip pathology or degenerative arthritis, abduction is much more limited than adduction.
2. *Adduction: 20 to 30 degrees* (Fig. 22.2.3B).
3. *Flexion: 120 to 135 degrees* (Fig. 22.2.3C).

Thomas's Test. This test assesses iliopsoas restriction, which plays a significant role in lumbosacral instability. A tight iliopsoas can create an undue force on the pelvis due to its origin from the ilium.

a. The athlete lies supine on the examination table.
b. The examiner stabilizes the pelvis by placing a hand under the lumbar spine.
c. The examiner then flexes both hips up until the lumbar spine flattens.
d. The athlete can use his or her hands to hold the legs near the chest. From this point on, only hip flexion allows the leg to move, and the athlete should be able to touch the abdomen (120 to 135 degrees).
e. The athlete then extends one leg and lets it rest on the table. The leg will lie flat in a normal hip. (Fig. 22.2.4)

Positive test: The athlete cannot achieve (a) 90 degrees of knee flexion (rectus femoris), (b) a neutral angle of the hip (iliopsoas), or (c) less than 15 degrees of hip abduction relative to the pelvis (tensor fasciae latae or iliotibial band) (6,7).

Indicates: Dysfunction of the iliopsoas, rectus femoris, tensor fasciae latae, or iliotibial band.

4. *Extension:* 30 degrees (Fig. 22.2.5). This is best tested in the prone position with the

FIGURE 22.2.4. Thomas's test.

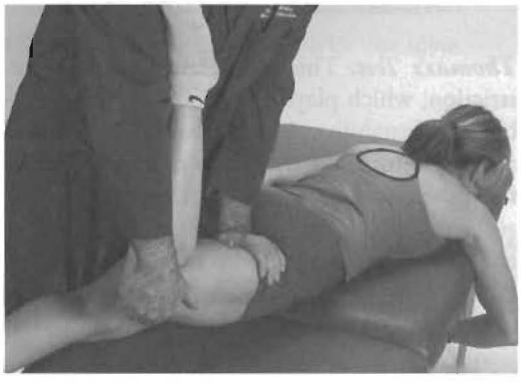


FIGURE 22.2.5. Extension.

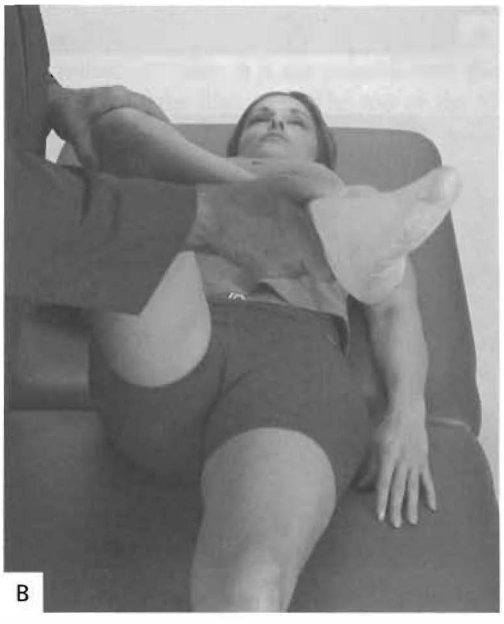


FIGURE 22.2.6. A, Internal rotation. **B,** External rotation.

knee slightly bent to relax the hamstrings. If the hip cannot extend, a flexion contracture is possible.
5. *Internal rotation:* 35 degrees (Fig. 22.2.6A).
6. *External rotation:* 45 degrees (Fig. 22.2.6B).

An alternative method for measuring internal rotation (IR) and external rotation (ER) is to place the athlete prone, flex the knees, and then internally and externally rotate the hips. Arthritis most commonly affects IR. Excessive IR or ER may be caused by femoral anteversion (IR) or retroversion (ER).

NEUROVASCULAR EXAMINATION

This portion of the examination should include muscle, sensory, and circulatory testing (8).

Muscle Testing. Test groupings of muscles (0–5 standard scale). See Table 22.2.1.

a. Flexors
b. Extensors
c. Abductors
d. Adductors
e. Internal rotators
f. External rotators

Sensory Testing. *Sensation* is evaluated by light touch in each dermatome of the hip and pelvis (Table 22.2.2).

Reflexes (patellar, Achilles) and *pulses* (femoral, posterior tibial, and dorsalis pedis) should be examined to complete the examination. The various innervations to the hip are found in Table 22.2.1.

PROVOCATIVE TESTS

Patrick/FABER test (Flexion ABduction External Rotation). This test grossly assesses the hip and sacroiliac (SI) joints. The athlete sits or lies supine and crosses one leg over the other, placing the lateral side of the foot on the opposite knee. The examiner can then push the knee toward the table to further stress the hip and SI anatomy (Fig. 22.2.7).

Positive test: Pain elicited posteriorly or anteriorly (1,6,10).

Indicates: Anterior groin pain suggests hip joint pathology, whereas posterior pain suggests SI joint pathology.

Trendelenburg's Test. This test is designed to evaluate the strength of the gluteus medius

TABLE 22.2.1. HIP MUSCLES

Action	Muscle	Origin	Insertion	Innervation	Nerve Root
Hip Flexion	Psoas	Iliac wing and lumbar spine	Lesser trochanter	L1-L3	L1-L3
	Iliacus	Iliac wing and lumbar spine	Lesser trochanter	Femoral	L2-L3
	Rectus femoris	Anterior inferior iliac spine and acetabular rim	Tibial tubercle	Femoral	L2-L3
	Sartorius	Anterior superior iliac spine	Pes anserinus	Femoral	L2-L3
	Adductor longus	Pubic ramus	Linea aspera of femur	Obturator	L2-L4
	Adductor brevis	Pubic ramus	Linea aspera of femur	Obturator	L2-3, L5
Hip Extension	Gluteus maximus	Iliac wing	Iliotibial band and femur	Inferior gluteal	L5, S1-S2
	Gluteus medius	Ilium	Greater trochanter	Superior gluteal	L5, S1

(*continued*)

TABLE 22.2.1. (*continued*)

Action	Muscle	Origin	Insertion	Innervation	Nerve Root
	Biceps femoris	Long head–ischial tuberosity Short head–linea aspera	Fibular head, lateral collateral ligament, lateral tibial condyle	Sciatic	L5, S1-2
	Semitendinosis	Ischial tuberosity	Medial tibial shaft	Sciatic	L5, S1-S2
	Semimembranosus	Ischial tuberosity	Medial tibial shaft	Sciatic	L5, S1-S2
Abduction	Tensor fascia lata	Iliac wing	Iliotibial band, Gerdy's tubercle	Superior gluteal	L4-L5
	Gluteus maximus	Iliac wing	Iliotibial band and femur	Inferior gluteal	L5, S1-2
	Gluteus medius & minimus	Ilium	Greater trochanter	Superior gluteal	L4-L5
Adduction	Adductor longus	Pubic ramus	Linea aspera of femur	Obturator	L2-L4
	Adductor brevis	Pubic ramus	Linea aspera of femur	Obturator	L2-L4
	Adductor magnus	Pubic ramus and ischial tuberosity	Linea aspera and adductor tubercle	Obturator	L2-L4
Internal Rotation	Adductor longus	Pubic ramus	Linea aspera of femur	Obturator	L2-L4
	Adductor brevis	Pubic ramus	Linea aspera of femur	Obturator	L2-
	Gluteus medius	Ilium	Greater trochanter	Superior gluteal	L5, S1
	Gluteus minimus	Ilium	Greater trochanter	Superior gluteal	L5, S1
	Tensor fascia lata	Iliac wing	Iliotibial band, Gerdy's tubercle	Superior gluteal	L4-L5
External Rotation	Gluteus maximus	Iliac wing	Iliotibial band and femur	Inferior gluteal	L5, S1-S2
	Obturator internus	Obturator foramen and membrane	Medial aspect of greater trochanter	N. to obturator	L5, S1
	Obturator externus	Obturator foramen and membrane	Medial aspect of greater trochanter	Obturator	L3-L4
	Quadratus femoris	Obturator ring	Medial aspect of greater trochanter	N. to quadratus	L5, S1
	Piriformis	Inferiolateral sacrum and greater sciatic notch	Superior aspect of greater trochanter	L5, S1-S2	L5, S1-2
	Gemellus superior	Ischial spine	Medial aspect of greater trochanter	N. to obturator	L5, S1
	Gemellus inferior	Ischial tuberosity and obturator ring	Medial aspect of greater trochanter	N. to quadratus	L5, S1
	Gluteus medius	Ilium	Greater trochanter	Superior gluteal	L5, S1

(Adapted in part from Anderson et al (16) and Agur AMR, Lee ML. *Grant's Atlas of Anatomy,* 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

TABLE 22.2.2. DERMATOMES OF THE HIP AND PELVIS REGION

Anterior
L1—groin and suprapubic region
L2—anterior thigh
L3—lower anterior thigh and knee
L4, L5—lower leg

Posterior
More curvilinear, especially around perianal area (S2-S4)

muscle. Ask the athlete to stand on one leg. If the gluteus medius is functioning properly, then the pelvis will remain horizontal. If it is not functioning, the side that has been lifted will drop; therefore, the leg the athlete is standing on has a weak gluteus medius (Fig. 22.2.8). This is demonstrated by a classic gait pattern (*Trendelenberg lurch*) in which the patient lurches to the unaffected side to counteract the imbalance from the muscle weakness. The patient attempts to center his or her body over the affected side (5,11).

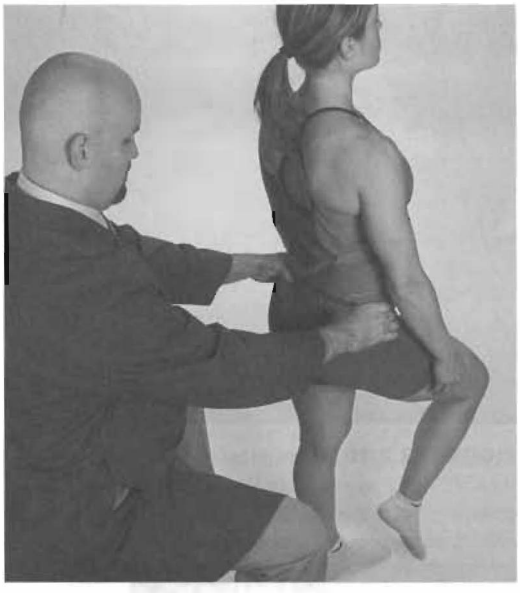


FIGURE 22.2.8. Trendelenburg's test.

Pelvic Rock Test. The examiner places both hands over the ASISs, then applies an alternating posterior vector force (Fig. 22.2.9). The normal finding is a soft springing return of the pelvis.

Positive test: Firm palpatory finding on the rock.

Indicates: Restriction and possible pathology of the SI joint and pelvis.

Leg-length Discrepancy. See Chapter 21.2, Lumbosacral Spine: Physical Examination (Fig. 21.2.10).

Ober's Test. The athlete lies on his or her side with the involved leg upward. The examiner abducts the leg and flexes it to 90 degrees, then releases the leg.

Positive test: The leg will stay abducted.
Indicates: Iliotibial band contracture (3,5).

FIGURE 22.2.7. FABER test (Flexion ABduction External Rotation).

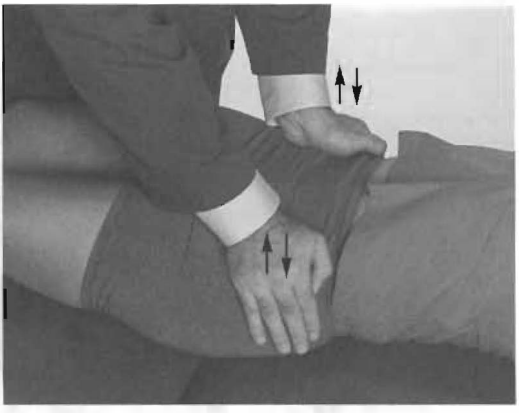


FIGURE 22.2.9. Pelvic rock test.

FIGURE 22.2.10. Ober's test.

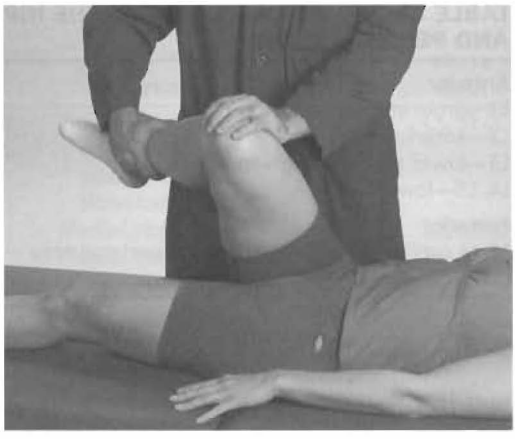


FIGURE 22.2.12. Apprehension test.

FIGURE 22.2.11. Ely's test.

Ely's Test. The athlete is placed prone and the knee is flexed (3,5).

Positive test: The hip on the ipsilateral side will spontaneously flex.

Indicates: Rectus femoris muscle contracture.

Apprehension Test. The athlete is supine and the hip is flexed, adducted, and internally rotated (Fig. 22.2.12).

Alternative method: The athlete slides down the table so that the leg is off the table and the examiner hyperextends and externally rotates the hip (Fig. 22.2.13).

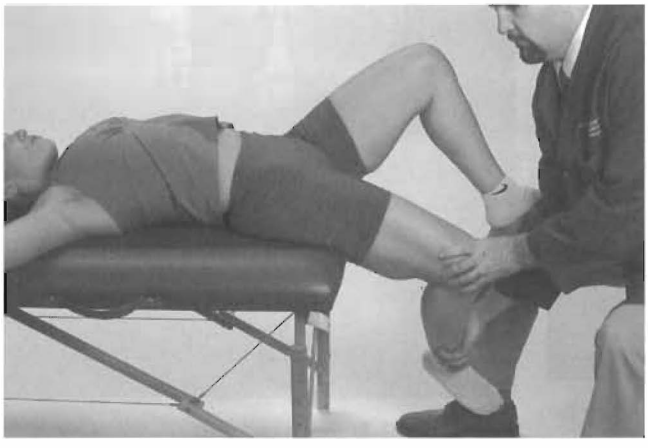


FIGURE 22.2.13. Alternate apprehension test.

Positive test: Anterior subluxation of the hip and anterior pain and apprehension.

Indicates: Anterior labral pathology.

Additional Apprehension Test. To elicit labral "catching," have the athlete perform a deep knee bend/squat. This potentiates the "catching" of labral tears and will result in pain and may be associated with audible crepitance (3,4,9).

Posterior Pelvis Pain Test. The athlete lies supine with the hip of the affected side flexed to 90 degrees while the examiner applies a force at the knee perpendicular to the table.

Positive test: Pain is elicited by this maneuver.

Indicates: Posterior pelvis pain (12).

GAIT ANALYSIS

This is a very complex science; however, we can simply break down the cycle into two phases: stance (60%) and swing (40%). Most problems are picked up during the stance phase because of weight bearing and pain.

Stance Phase. Most problems are noticed during this phase because of pain resulting in an antalgic (coxalgic) gait. Notice if there is a shortened time during the stance phase on the affected side: this is secondary to pain and the athlete will stand with the hip in a slightly flexed position (5,13–16). Muscle weakness of the anterior tibialis results in a "drop-foot" gait. Weakness of the gluteus medius results in a Trendelenburg lurch. Gastrocnemius weakness results in a flat-foot gait with no forceful push-off. Quadriceps weakness will force the patient to "lock out" the knee in hyperextension.

Swing Phase. Athletes with weak dorsiflexors develop a "steppage gait" in which they lift the foot high to clear the ground. Quadriceps weakness results in difficulty with the acceleration phase of gait, whereas hamstring weakness causes difficulties with deceleration before heel strike (5,13–16).

Instability. A widening of the base for gait may occur from neurologic disease or mechanical instability due to ligament or cartilaginous injury. The latter causes a sense of instability and the athlete walks with a wider-based gait (5,13–16). More discussion on gait analysis is found in Chapter 25.

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22.3

Common Conditions

VINCENT N. DISABELLA
CHRISTOPHER DAVIS

The hips and pelvis are the base of the athletic body, the core of the core. Without a strong base, the upper body and lower body cannot work in concert. Almost all athletic skills require the two to work together for maximal efficiency. For sprinting, the upper body generates thrust and momentum that aids speed. To field a ground ball, the player needs to run, stop, bend, twist, and throw in a matter of 3 seconds. To serve a tennis ball, the hips use the legs to propel the body forward, while the upper body uncoils to time the swing with the location of the ball already in the air.

This realization speaks to those who now emphasize core stability as the linchpin of an athlete's training. This idea supports the concept of proximal stability for distal mobility, which is in line with the kinetic chain principles.

Although the sacrum articulates with the ilia to make up the pelvis, its exam and common pathology will be discussed with the lumbar spine in a separate section. Because of the overlap of manual medicine techniques used to treat common conditions of the hips and pelvis, they are grouped together in one section at the end of this chapter.

GREATER TROCHANTERIC BURSITIS

Greater trochanteric bursitis is one of the most common causes of hip pain seen in sports medicine offices. This bursa lies just lateral and slightly posterior to the greater trochanter of the femur. There are actually two bursae in this area that can be involved. One is superficial, just under the iliotibial band, which is commonly affected in external snapping hip syndrome. The trochanteric bursa lies deeper between the gluteus medius and the tensor fasciae latae.

Inflammation and irritation of the trochanteric bursa can occur with repetitive hip flexion and extension such as in running. The repetitive rubbing of the musculature over the bony prominence of the greater trochanter can cause bursal irritation. This injury can also be caused traumatically by falls onto the hip or direct contusions over the lateral hip. Athletes who overpronate their feet can also have trochanteric bursal inflammation due to the resultant internal rotation of the hip (1).

Athletes

Runners are often affected by trochanteric bursitis due to the repetitive motion in this area. Women with broad hips, overpronators, and people with tibial torsions are more likely to have this condition (2). In road runners, the outside leg is more prone to bursitis in relation to the road surface. The camber in the road surface causes a greater adduction force over the hip, which then causes the abductors to contract more forcefully. Also, runners who adduct beyond the midline are considered to be at risk, as well as recreational runners who do not pay close attention to proper stretching and flexibility.

Ballet dancers and figure skaters can also suffer trochanteric bursitis as a result of prolonged activity balancing on one leg. Athletes who spend time balancing on one leg can also have the condition due to tensing of the gluteus muscles and the iliotibial band to maintain balance.

Traumatic bursitis can occur in any sport where there is a chance of collision, such as soccer, rugby, and basketball. The knee or greater trochanter can hit the playing surface, or another knee to inflame the bursae. Football and ice hockey players rarely suffer this injury due to protective padding.

Intrinsic causes of trochanteric bursitis include the following:

1. Wide pelvis
2. Prominent trochanter, particularly in women
3. Poor flexibility around the hip joint. Often athletes with trochanteric bursitis are found to have tight internal rotators (gluteus medius and minimus, and tensor fasciae latae). A tight iliotibial band can also cause increased friction over the bursa.
4. Athletes with poor foot structure or mechanics that result in overpronation that produces a tibial internal rotation that in turn produces an internal rotation of the femur. Leg-length discrepancies can cause more hip adduction on the longer side and increase tension on the trochanteric bursa on the shorter side for the same reasons road runners suffer this injury.

Runners can often develop trochanteric bursitis as a result of running on worn shoes. Once the resilience of shoes expires, overpronation can occur. Runners who train extensively on a track can suffer trochanteric bursitis in their outside leg while running in the turns because of increased forces across the hip.

History and Physical Examination

Athletes with trochanteric bursitis often present with deep burning pain in their lateral hip of insidious onset. They often report a recent increase in their training load or intensity. Many athletes report recently adding plyometrics training or dance aerobic activities, which include extreme repetitive hip flexion and extension in the conditioning programs. In traumatic bursitis, athletes report a fall or contusion to the lateral hip, but often they can walk with pain (3).

On examination, the athlete has pain over the lateral hip either directly over or slightly posterior to the trochanter. Pain is exacerbated by hip flexion-extension and forced adduction of the hip. The pain can radiate down the lateral thigh following the iliotibial band.

Standard Treatment

Acute care consists of relative rest, ice, and nonsteroidal anti-inflammatory drugs (NSAIDs). To maintain aerobic fitness, alternate aerobic activities should be used that do not aggravate the bursitis. Gentle stretching of the hip and pelvic area should be started as soon as tolerated. Emphasis should be placed on stretching the iliotibial band and gluteal muscles, which cross directly over the bursa. If conservative care fails, a corticosteroid injection into the bursa can be considered (1,4,5).

GROIN STRAIN

Adductor strain is the most common athletic injury occurring around the hip and groin area. The majority of groin strains are injuries to the musculotendinous junction of the adductor longus muscle. The gracilis is the second most commonly strained muscle (4). Less commonly, the injury occurs at the tendinous insertions into the pubic bone. Groin strains are usually minor to moderate strains, with grade 3 strains (severe) rarely occurring.

The injury usually occurs during cutting or kicking motions. Often the athlete is changing direction at full speed and overextends the step or loses his or her footing. In kicking sports the athlete can encounter the ground or another player, causing an eccentric contraction. Often the athlete has the knee fully extended with the hip flexed, abducted, and externally rotated (7). Besides the adductor longus and gracilis, any of the other adductor muscles (adductor magnus, adductor brevis, pectineus, and adductor minimus) can be affected. Often the athlete remembers the mechanism of injury, but it can also present with insidious onset. These athletes have a mild injury, which does not limit play but also never completely heals.

This injury is common in sports with play that involves a great deal of change of direction, such as football, ice hockey, basketball, and lacrosse. Soccer poses a double threat because of the change of direction and the kicking of the ball. Cheerleaders and gymnasts are also affected along with hurdlers and sprinters.

Renstrom and Peterson reported that 5% of all soccer injuries were groin strains, and 62% of the adductor strains seen in their study were injuries to the adductor longus (8). It is estimated

TABLE 22.3.1. INTRINSIC FACTORS CAUSING GROIN STRAINS

Poor flexibility
Muscle imbalance in hip and pelvis
Overuse
Piriformis dysfunction

that 25% of all soccer players will suffer a groin strain at some point in their career. Studies on ice hockey players report that anywhere from 9% to 57% will suffer a groin strain at some point in their career.

Intrinsic factors include poor flexibility and muscle imbalance around the hips and pelvis (Table 22.3.1). Overuse syndromes from repetitive kicking can cause weakness, which can result in adductor strain. Soccer-style kicking with predominantly one leg can cause the adductors of the dominant leg to overpower the nondominant leg, causing injuries to the weaker leg. This same activity can lead to overuse and fatigue of the dominant leg. Piriformis spasm can also cause external rotation of the hip, placing undue stretch on the adductors, especially with the hip extended. Many have postulated that foot overpronation and leg-length discrepancies are causes, but current research does not support this claim.

History and Physical Examination

Wet or slippery playing surfaces can cause acute overstretch of the adductors as the athlete falls with the hip flexed, abducted, and externally rotated. This can also occur when soccer players make a slide tackle. Kicking an inanimate object can interrupt the kicking motion and cause an adductor strain from severe eccentric loading (9).

The athlete will likely not recall the mechanism of injury. Often the athlete feels a tearing or popping sensation during an eccentric contraction, such as from cutting, lateral movement, or during kicking. Pain is reported in the medial upper thigh into the groin that worsens with activity. In chronic cases, the athlete may remember a "tweak" which was not enough to stop playing but has gotten progressively worse with playing.

On physical examination the athlete reports pain with passive abduction and active adduction over the medial thigh into the groin. The range of motion during Patrick's/FABER test may be limited and painful. Medial thigh ecchymosis and tenderness are appreciated over the adductor muscles; most often the adductor longus is involved. One should consider testing for osteitis pubis with a pubic spring test and for abdominal strain with a resisted abdominal crunch.

Athletes have tenderness over the adductor muscles at the musculotendinous junction. In grade 2 strains a defect is often palpable, and in the rare grade 3 tears the athlete presents with a painful groin mass and a distal muscle defect. Medial thigh ecchymosis with distal tracking is often seen.

Standard Treatment

Acute treatment consists of rest, ice, and gentle range of motion. Physical therapy modalities to limit swelling and pain should be instituted immediately. NSAIDs are used in this acute period for analgesia. The athlete should be restricted from all aggravating activities.

Subacute treatment begins once the athlete has full range of motion and is pain-free. All muscles that contribute to hip motion should be strengthened, with flexibility also being addressed. Once the athlete has returned to 70% of the strength of the unaffected leg, sport-specific activities can be added (10). Return to play can occur when the athlete's strength has returned to 90% and proprioception is adequate so he or she can protect the area from further injury (5).

Prevention

The most important exercises for preventing groin strains are a well-balanced hip and lower extremity strengthening program. The athlete should be training not only the quadriceps and hamstrings, but also the muscles that extend, flex, adduct, and abduct, the hips. An emphasis should be made to include adequate warm-up and use proper technique with each exercise.

The modifications of activity should limit the motions that aggravate the injury. Lateral movement or motions that cause leg abduction are usually the most painful. Limiting these

motions until the athlete is pain-free and has regained 90% of his or her strength usually allows for a safe return to play.

HIP POINTER

Hip pointers are contusions to the pelvis which cause bruising and muscle tightness. Most sports medicine physicians talk about contusions to the ASIS when they use the term hip pointer, but some broaden the definition to include any contusion to the iliac crest (14,15). These contusions can lead to bleeding, hematoma formation, and avulsion fractures in the adolescent athlete. This injury most often occurs when the athlete collides with another athlete or an inanimate object.

Often hip pointers cause severe pain and muscle spasm. It is important to recognize the attachments of the various muscles along the iliac crest. These muscles include the gluteus maximus and medius, iliacus, quadratus lumborum, latissimus dorsi, iliocostalis, transverse abdominis, and external abdominal oblique (16). The origin of the sartorius is at the ASIS, a common contusion site.

Any athlete who plays a physical contact sport and could suffer a collision is susceptible to a hip pointer. The injury is so common in certain collision sports that special padding is worn to prevent this injury, such as football and ice hockey. Rugby, soccer, and Australian Rules football players are more likely to suffer hip contusions because of not wearing protective equipment. Hip pointers can also be caused by repetitive contact with equipment such as a gymnast hitting the horizontal bars or Palma horse.

The factors predisposing an athlete to this injury are more environmental than anatomic with regard to the sports in which he or she participate. Participation in contact sports increases the likelihood of suffering a hip pointer. Harder playing surfaces may predispose the athlete to injury with a fall.

History and Examination

The athlete can usually recall the exact contact that caused the contusion. The pain is often so severe that an athlete may not be able to continue in competition. On physical examination, the athlete is tender over the contusion site at the ASIS. Often swelling and ecchymosis occur at the point of impact. Muscle spasm can cause abnormal posturing to protect the injured muscles. In younger athletes, radiographs should always be taken to rule out avulsion fractures (17).

Standard Treatment

Acute care consists of relative rest, ice, and NSAIDs. Alternate aerobic activities should be used that do not aggravate the muscles originating from the contused area. Gentle stretching of the hip and pelvic area should be started as soon as tolerated.

Subacute care works on establishing the basics of range of motion, strength, and function. The athlete also should begin running progressions. Once the athlete has progressed beyond straight running to pain-free cutting and once lateral movements are tolerated, sport-pecific activities can be added.

Once the athlete is pain-free, strengthening of the lower body can begin. The athlete often suffers minor weakness from prolonged rest. In some instances, muscle atrophy can occur from disuse and reflex inhibition of the injured muscles.

Manual Medicine

Counterstrain: Anterior Pelvis. Because a pelvic contusion causes pain and swelling, the surrounding tissues will likely become irritated and restricted, particularly if the athlete is trying to play through the contusion. The iliacus, iliopsoas, and lower abdominal wall would all benefit from indirect treatment to reduce tissue tension but not engage the restrictive barrier, particularly if the tissue is recently traumatized. Indirect techniques such as counterstrain and functional are effective choices for hip pointers.

OSTEITIS PUBIS

Osteitis pubis is an inflammation of the pubic symphysis and surrounding muscle insertions. Although the exact etiology is unknown, it is

most likely caused by repetitive microtrauma or shearing force to the pubic symphysis.

Beer described inflammation of the pubic symphysis as a result of urologic surgery in 1924. He called it an "orthopedic disease sponsored by urologic surgery" (18). In 1932, Spinelli wrote about this syndrome in athletes (19). Since that time there have been multiple reports of this syndrome in many sports. As shown by Alderink, sacroiliac motion has a very large impact on the motion about the pubic symphysis. Batt et al. postulated that osteitis pubis was a result of muscle injury to the adductors or abdominal musculature resulting in muscle spasm, which in turn causes increased shearing forces across the pubic symphysis (20).

Symptoms

The presenting symptoms for osteitis pubis can be almost any complaint about the groin or lower abdomen. The athlete complains about pain in the groin, hip, perineum, or testicle. Often the athlete presents with adductor pain or lower abdominal pain that then localizes to the pubic area. Usually the pain is unilateral and has been present for a few days to weeks. A study involving Canadian and Australian athletes by Fricker et al. showed that 8% of the males had scrotum or testicular pain (21). Often the pain increases with running, kicking, or pushing off to change direction.

Examination

Findings can vary greatly with osteitis pubis. One must always consider the athlete's sport and the chronicity of the pain. Early in the disease, the athlete may complain of groin or testicular pain. The pain is often aggravated by adduction of the leg or running. Symptoms are more often unilateral. Athletes may also complain of lower abdominal pain and be tender to palpation over the superior pubic ramus. When sacral innominate dysfunction is a cause, the athlete can have pain over one or both sacroiliac joints. This pain can often be accompanied by piriformis spasm and resultant sciatic-type pain. An athlete with a leg-length discrepancy might complain of hip pain on the longer limb. This can also be seen in runners who run in the same direction and functionally have one leg shorter secondary to the caber of the running surface. The most specific test is tenderness over the pubic symphysis with a direct pressure spring test.

Athletes

Osteitis pubis seems to be more prevalent in sports that involve running, kicking, or rapid lateral movements. Soccer has a great deal of running and rapid change of direction. This can lead to strains of the adductor muscles, which change the forces directed on the pelvis during recovery. The other inciting motion in soccer is the act of kicking. Many times the athlete is not well balanced when planting the foot to kick and puts a great deal of strain on the muscles stabilizing him or her to perform this kick. This is turn translates to abnormal forces across the pubic symphysis.

Sprinting can manifest symptoms of osteitis pubis due to repetitive microtrauma to the pelvis from the repetition of rapid acceleration. Muscle pulls are also common in track for this reason. This repetitive stress transmitted through the pubic symphysis accumulates and leads to tissue breakdown.

Ice hockey has multiple risk factors, which include the skating motion itself along with the contact with other players and the dasher boards. Many times the athlete may suffer a minor adductor strain, but the continued play and resultant changes in flexibility lead to abnormal forces across the symphysis. This can often be aggravated by a rapid change of direction that is often required in ice hockey.

American football also has a high rate of injury for several reasons. The first factor is due to the amount of sprinting involved in competition. The second factor is the excessive violent contact, which can often lead to minor injuries that "play through." Certain positions, such as defensive backfield, also demand a great deal of back-pedaling with a rapid abduction of one hip to turn and run with a receiver. This motion can lead to hamstring or adductor strains,

which in turn change the muscle balance and forces across the pubic symphysis. These are some of the sports where osteitis pubis is more often seen. This list can be expanded to any sport that includes running, jumping, contact, or rapid change in direction.

Standard Treatment

Osteitis pubis is a self-limiting disease with inflammation at one or both pubic margins. It resolves in about 9 months with conservative care (22). Conservative care usually consists of stretching, NSAIDs, and low-impact aerobic workouts. The principal author has seen much faster return to full activity with early corticosteroid injections, as recommended by Holt et al. (19), and aggressive stretching. Activity is limited to stretching for the first 7 to 10 days after injection. Manual medicine techniques are used to treat any contributing somatic dysfunctions, including muscle energy to the hip. Any dysfunction of the hips or pelvis can cause abnormal forces to be transmitted across the pubic symphysis.

SNAPPING HIP SYNDROME

Snapping hip syndrome is really a constellation of different entities that all cause a snapping sensation about the hip joint. Less than one third of individuals who experience snapping hip have any pain accompanying the snapping sensation (23). Snapping hip is usually separated into internal/anterior snapping hip and lateral/external snapping hip syndrome.

Internal snapping hip is most often caused by the iliopsoas tendon rubbing over the iliopectineal eminence of the pelvic brim or more rarely, over the lesser trochanter (24). The athlete feels a deep anterior snapping sensation with hip flexion. This is commonly seen in athletes who do a great deal of running, such as soccer players, or in dancers such as ballerinas. The snapping can often be aggravated during conditioning if the athlete is doing box jumps or running stairs. The condition can become painful if the iliopsoas bursa becomes inflamed.

A less common cause of internal snapping hip syndrome is the "suction phenomenon" of the hip joint itself. This is often seen in athletes with extreme range of motion such as gymnasts, dancers, and cheerleaders. Acetabular labrum tears, hip subluxations, and loose bodies in the joint are also rare causes of the internal snapping sensation.

External snapping hip is a much more common entity. It is most often caused by the iliotibial band snapping over the greater trochanter of the femur. The gluteus maximus tendon rubbing over the greater trochanter can also cause it. This can occur in any sport that involves repetitive flexion and extension or the hip, such as running or jumping. If the snapping is accompanied by trochanteric bursitis, the athlete will have pain over or just posterior to the greater trochanter. If the iliotibial band is severely tight, the athlete may also feel lateral knee pain.

Intrinsic Factors

Intrinsically snapping hip is often caused by muscle tightness or imbalance. Psoas tightness is the most common factor leading to internal snapping hip. If the snapping sensation is caused by subluxations, the intrinsic factors are usually an extreme range of motion and stretching of the joint capsule.

Tight tensor fasciae latae, gluteus medius, or gluteus maximus are usually the causes of external snapping hip syndrome. Reid also sites narrow bi-iliac width as a possible cause (25). In extreme cases of gluteus medius weakness, a gluteal lurch is present that can cause the iliotibial band to rub over the trochanter. Hyperpronation of the feet can also cause snapping hip due to the compensatory internal rotation of the hip (26).

Extrinsic Factors

The most common extrinsic cause of snapping hip is the running surface. Runners who always run on the same side of the road can often get snapping hip of their outside leg from stepping down with the camber of the pavement. Athletes who run stairs for conditioning or do step-ups can develop snapping hip symptoms if the

step or platform is too high, causing extreme hip flexion.

An athlete may not remember the exact time the snapping sensation in the hip began. When either trochanteric or iliopsoas bursitis is causing the snapping to become painful, a certain incident may be identified, such as one intense training session, or it may occur after a few weeks of more intense training. In collision sports, an athlete can suffer a hip pointer (contusion to the lateral hip), which causes swelling, bursitis, and snapping hip type symptoms. Athletes with internal snapping hip complain of pain or a clicking sensation when ascending stairs because of the firing of the iliopsoas muscle to initiate the step-up. External snapping hip is also aggravated by climbing stairs, but at a different phase of propulsion. As the athlete finishes going up a step, he or she fully extends the knee and hip, which causes the iliotibial band to snap over the greater trochanter.

Having the athlete lay supine and lift the affected leg off the table with the knee fully extended can elicit psoas tightness. During this maneuver, the psoas muscle is the only hip flexor activated (24). Palpation of the lesser trochanter and psoas muscle can also be achieved by flexing the hip and knee to 40 degrees and externally rotating the hip. Supporting the knee on a pillow allows enough relaxation for these deep structures to be palpated (26). Athletes with hip subluxations often report symptoms with passive extreme ranges of motion. Loose bodies and labral tears can cause symptoms with internal and external rotation with the hip flexed to 90 degrees.

External snapping hip can be elicited and often seen by having the athlete step up onto a box or platform. Ober's test is often positive, signifying a tight iliotibial band (27,28). Acute care consists of relative rest, modalities for pain and inflammation, and NSAIDs. Often there is no need for any limitation of activity if the snapping is painless. Once any pain is relieved, the athlete can begin to work on flexibility and muscle imbalances. Stretching of all the hip and pelvic muscles is the mainstay of treatment. Any muscle weaknesses should also be corrected. Mechanical causes such as foot overpronation should also be addressed. On the rate occasion when the snapping hip is painful and does not respond to conservative care, a corticosteroid injection can be considered.

MANUAL MEDICINE TECHNIQUES (7,29,30)

Muscle Energy for Superior Pubic Shear

Most Applicable Conditions: Osteitis pubis, groin strain.

1. The athlete lies supine on the table with the clinician standing on the side of the dysfunction facing cephalad.
2. The athlete's leg on the affected side is positioned off the table in a slightly extended position. With one hand the clinician facilitates extension of the leg, while the other hand monitors the contralateral ASIS for movement (Fig. 22.3.1A).
3. Once the motion barrier is engaged, the athlete is instructed to flex the extended leg with about 10 lb of pressure while the clini-

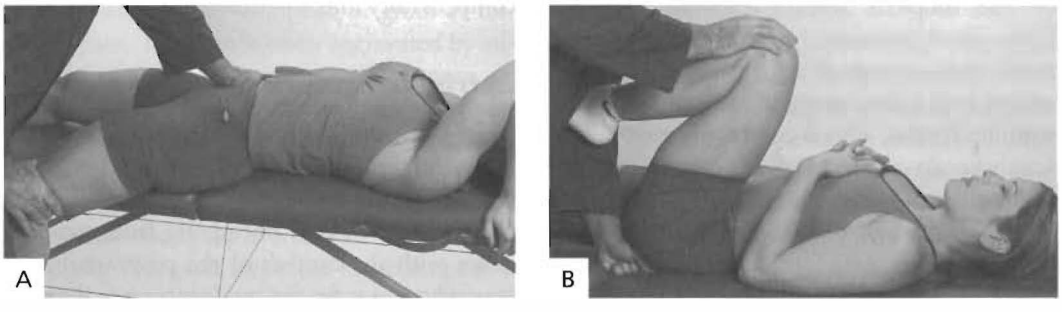

FIGURE 22.3.1. Muscle energy for superior pubic shear **(A)** and inferior pubic shear **(B).**

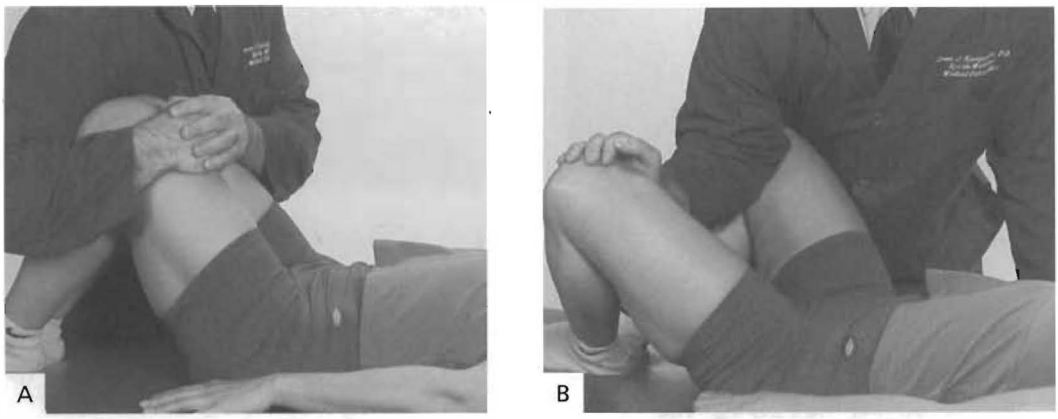

FIGURE 22.3.2. Muscle energy for gapped pubic symphysis **(A)** and compressed pubic symphysis **(B).**

cian resists. The isometric contraction is maintained for 3 to 5 seconds.
4. Relax, reposition, repeat, and reassess.

Muscle Energy for Inferior Pubic Shear
Most Applicable Conditions: Osteitis pubis, groin strain.

1. The athlete lies supine on the table with the clinician standing at the side of the dysfunction facing cephalad.
2. The athlete's knee on the side of dysfunction is flexed.
3. The clinician then places a fist on the table with cephalad pressure on the ischial tuberosity, while the other hand monitors the ASIS (Fig. 22.3.1B).
4. The clinician flexes the affected side until movement is felt with the monitoring hand.
5. Resting the athlete's flexed knee and shin on the clinician's shoulder, the athlete is instructed to straighten the knee while the clinician resists.
6. Relax, reposition, repeat, and reassess.

Muscle Energy for a Gapped Pubic Symphysis
Most Applicable Conditions: Osteitis pubis, groin strain.

1. The athlete is placed supine on the table with the knees and hips flexed to 90 degrees.
2. The clinician wraps both hands around the athlete's distal thighs and instructs the athlete to spread the knees apart with maximum intensity (Fig. 22.3.2A).
3. Relax, reposition, repeat, and reassess.

Muscle Energy for Compressed Pubic Symphysis
Most Applicable Conditions: Osteitis pubis, groin strain.

1. The athlete is placed supine on the table with the knees and hips flexed 90 degrees.
2. The clinician's forearm is placed between the athlete's knees with one hand on the medial knee and the elbow on the opposite medial knee (Fig. 22.3.2B).
3. The athlete is then instructed to bring the knees together against the force of the clinician's arm. This isometric contraction is held for 3 to 5 seconds and the athlete is instructed to relax.
4. Relax, teposition, repeat, and reassess.

Muscle Energy for Restricted Hip Abduction
Most Applicable Conditions: Osteitis pubis, greater trochanteric bursitis, groin strain, hip pointer, snapping hip syndrome.

1. The athlete is supine on the table with the restricted hip near the edge of the table.
2. The clinician stands between the athlete's legs.
3. The leg is lifted slightly off the table and taken to the barrier of abduction.

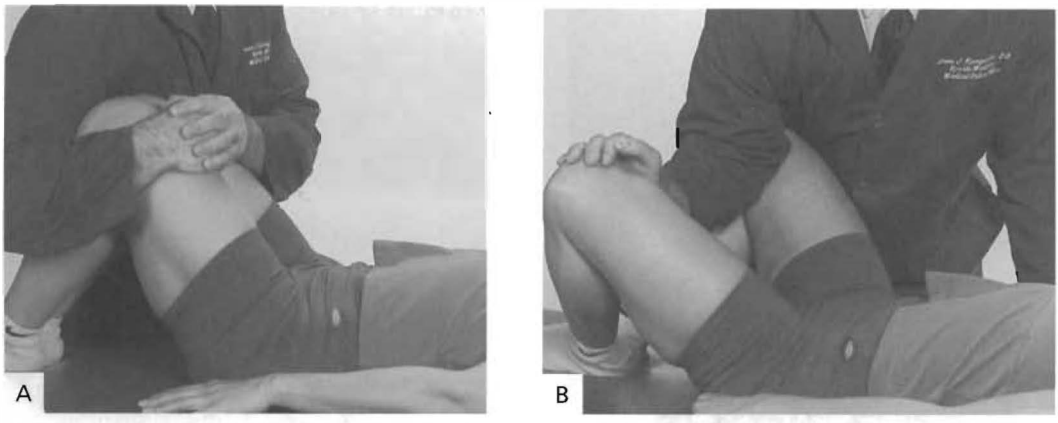

FIGURE 22.3.2. Muscle energy for gapped pubic symphysis **(A)** and compressed pubic symphysis **(B).**

cian resists. The isometric contraction is maintained for 3 to 5 seconds.
4. Relax, reposition, repeat, and reassess.

Muscle Energy for Inferior Pubic Shear
Most Applicable Conditions: Osteitis pubis, groin strain.

1. The athlete lies supine on the table with the clinician standing at the side of the dysfunction facing cephalad.
2. The athlete's knee on the side of dysfunction is flexed.
3. The clinician then places a fist on the table with cephalad pressure on the ischial tuberosity, while the other hand monitors the ASIS (Fig. 22.3.1B).
4. The clinician flexes the affected side until movement is felt with the monitoring hand.
5. Resting the athlete's flexed knee and shin on the clinician's shoulder, the athlete is instructed to straighten the knee while the clinician resists.
6. Relax, reposition, repeat, and reassess.

Muscle Energy for a Gapped Pubic Symphysis
Most Applicable Conditions: Osteitis pubis, groin strain.

1. The athlete is placed supine on the table with the knees and hips flexed to 90 degrees.
2. The clinician wraps both hands around the athlete's distal thighs and instructs the athlete to spread the knees apart with maximum intensity (Fig. 22.3.2A).
3. Relax, reposition, repeat, and reassess.

Muscle Energy for Compressed Pubic Symphysis
Most Applicable Conditions: Osteitis pubis, groin strain.

1. The athlete is placed supine on the table with the knees and hips flexed 90 degrees.
2. The clinician's forearm is placed between the athlete's knees with one hand on the medial knee and the elbow on the opposite medial knee (Fig. 22.3.2B).
3. The athlete is then instructed to bring the knees together against the force of the clinician's arm. This isometric contraction is held for 3 to 5 seconds and the athlete is instructed to relax.
4. Relax, reposition, repeat, and reassess.

Muscle Energy for Restricted Hip Abduction
Most Applicable Conditions: Osteitis pubis, greater trochanteric bursitis, groin strain, hip pointer, snapping hip syndrome.

1. The athlete is supine on the table with the restricted hip near the edge of the table.
2. The clinician stands between the athlete's legs.
3. The leg is lifted slightly off the table and taken to the barrier of abduction.

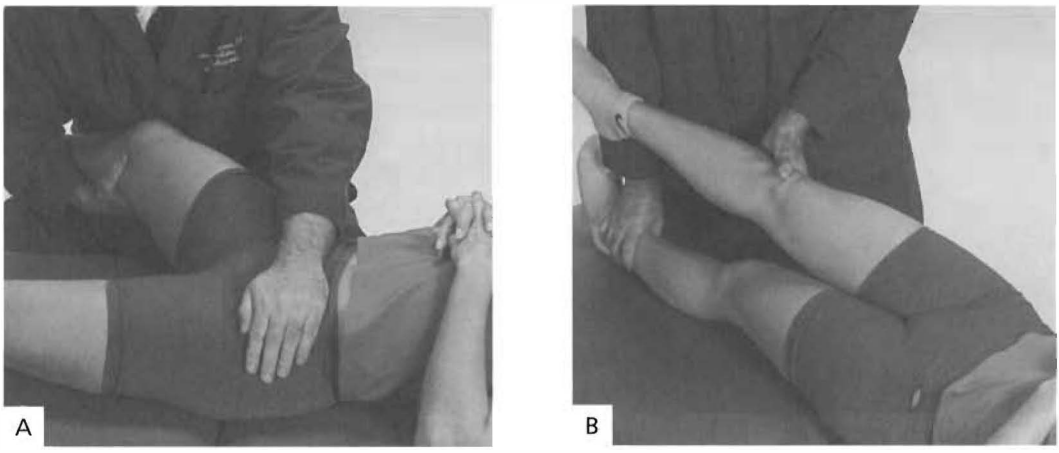

FIGURE 22.3.3. Muscle energy for restricted hip abduction **(A)** and restricted hip adduction **(B).**

4. The athlete adducts the leg against the clinician's thigh for 3 to 5 seconds (Fig. 22.3.3A).
5. Relax, reposition, repeat, and reassess.

Muscle Energy for Restricted Hip Adduction

Most Applicable Conditions: Osteitis pubis, greater trochanteric bursitis, hip pointer, snapping hip syndrome.

1. The athlete is supine with the feet at the end of the table.
2. The clinician lifts the involved leg and adducts it to the barrier.
3. The athlete abducts the leg against static manual resistance (Fig. 22.3.3B).
4. The barrier is engaged for 3 to 5 seconds and then the athlete and clinician simultaneously relax.
5. Relax, reposition, repeat, and reassess.

Muscle Energy for Resisted Hip Extension Secondary to Iliopsoas Restriction

Most Applicable Conditions: Osteitis pubis, snapping hip syndrome.

1. The athlete is supine with the clinician at the ipsilateral side to the restriction.
2. The athlete flexes both hips by bringing both knees to the chest.
3. As the athlete holds the contralateral knee to the chest, he or she extends the affected hip.
4. The clinician places his or her hand on the athlete's thigh just proximal to the knee to provide resistance as the athlete gently flexes it toward the ceiling (Fig. 22.3.4A). The barrier is engaged for 3 to 5 seconds and then the athlete and clinician simultaneously relax.
5. Relax, reposition, repeat, and reassess.

Muscle Energy for Restriction in Hip Flexion Secondary to Tight Hamstring Muscles

Most Applicable Conditions: Osteitis pubis, snapping hip syndrome.

1. The athlete is supine with the operator at the ipsilateral side of the table to the restriction.
2. The clinician cups the athlete's heel with his or her hand and gently lifts the leg off the table with a slight bend at the knee until the barrier is reached.
3. The athlete is instructed to bring the heel toward the table as the clinician applies an equal opposing force to the athlete's heel (Fig. 22.3.4B).
4. Relax, reposition, repeat, and reassess.

Alternative Method. The athlete rests the posterior thigh on the clinician's shoulder and attempt's to extend the hip against resistance.

Muscle Energy for Restriction in Hip External Rotation with Hip Flexed at 90 Degrees

Most Applicable Conditions: Osteitis pubis, hip pointer, snapping hip syndrome.

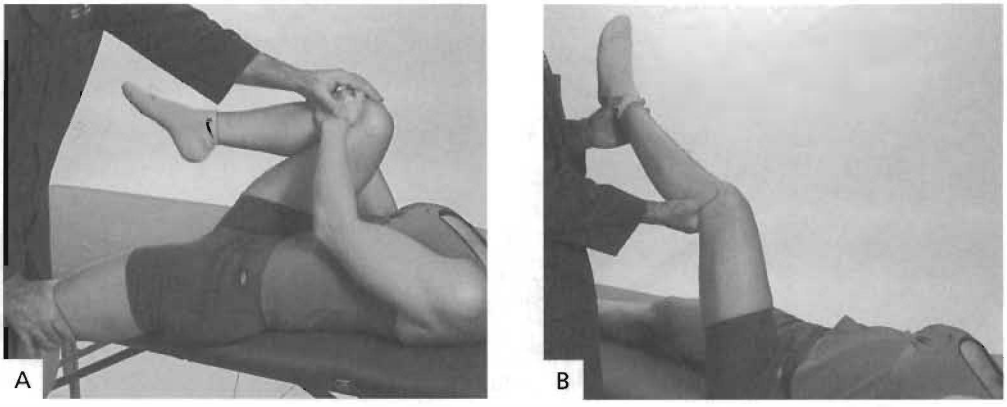

FIGURE 22.3.4. Muscle energy for resisted hip extension secondary to iliopsoas restriction **(A)** and restriction in hip flexion secondary to tight hamstring muscles **(B).**

1. The athlete is supine with the affected hip flexed to 90 degrees and the knee flexed to 90 degrees.
2. The clinician places his or her cephalad hand on the athlete's flexed knee for stabilization and uses the caudad hand to apply pressure to the ankle.
3. The athlete is told to externally rotate the hip against the clinician's equal resistance (Fig. 22.3.5).
4. Relax, reposition, repeat, and reassess.

Muscle Energy for Anterior Iliac Dysfunction

Most Applicable Conditions: Osteitis pubis, greater rrochanteric bursitis, hip poinrer, snapping hip syndrome.

1. The athlete is prone with the dysfunctional side at the edge of the table. The knee and hip are flexed and hanging off the table.
2. The clinician stands at the side of the table with the athlete's foot resting on his or her thigh.
3. The clinician uses his or her hand closest to the athlete to stabilize the athlete's sacrum and holds the flexed knee with the other hand.
4. The clinician passively flexes the athlete's hip and knee until the resrriction is felt (Fig. 22.3.6A).
5. The athlete is then instructed to extend the leg against the clinician's leg wirh mild force.
6. Relax, reposition, repeat, and reassess.

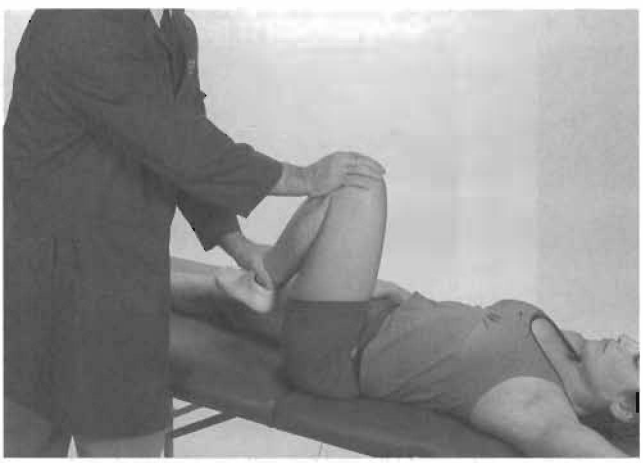

FIGURE 22.3.5. Muscle energy for restriction in hip external rotation with the hip flexed at 90 degrees.

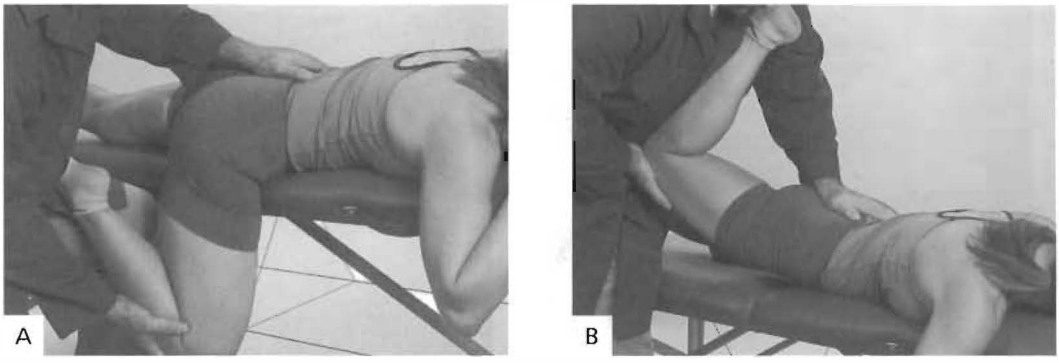

FIGURE 22.3.6. Muscle energy for anterior iliac dysfunction **(A)** and posterior iliac dysfunction **(B).**

Muscle Energy for Posterior Iliac Dysfunction
Most Applicable Conditions: Osteitis pubis, hip pointer, snapping hip syndrome.

1. The athlete is placed in the prone position with the clinician standing at the side of the table opposite the dysfunction.
2. The clinician uses his or her cephalad hand to stabilize the sacrum while using the caudad hand to lift the affected thigh into hip extension (Fig. 22.3.6B).
3. Once the barrier is engaged, the athlete is told to gently bring the leg down toward the table.
4. Relax, reposition, repeat, and reassess.

Muscle Energy for Superior Innominate Shear
Most Applicable Conditions: Osteitis pubis.

1. The athlete is supine with the clinician standing at the foot of the table.
2. The clinician holds the athlete's affected lower leg in both hands. The clinician extends and abducts the leg until the closed packed position is achieved.
3. The leg is internally rotated until the closed packed position is achieved, then traction is applied along the long axis of the leg (Fig. 22.3.7A).
4. The athlete is instructed to take three to four deep breaths, and then cough forcibly. As the athlete coughs, the clinician tugs along the long axis. The leg is then returned to the midline position.

Muscle Energy for Inferior Innominate Shear
Most Applicable Conditions: Osteitis pubis.

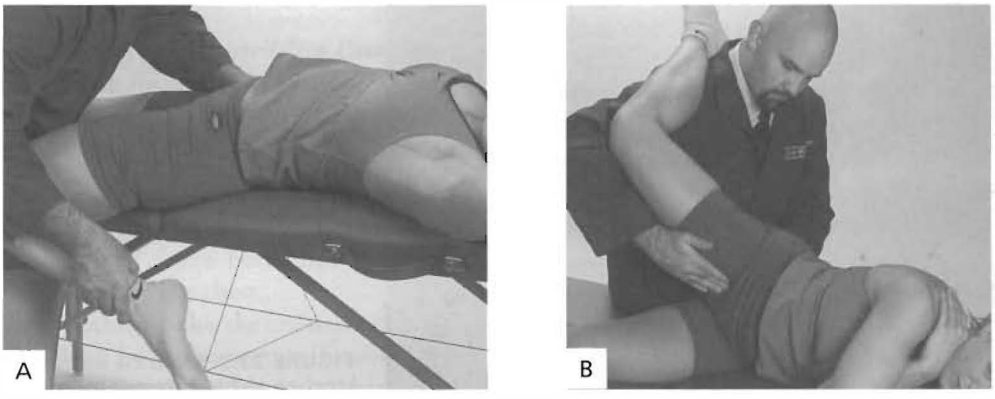

FIGURE 22.3.7. Muscle energy for superior innominate shear **(A)** and inferior innominate shear **(B).**

FIGURE 22.3.8. High-velocity, low-amplitude technique for posterior ilial dysfunction.

1. The athlete is placed in the lateral recumbent position with the dysfunctional side up.
2. The clinician sits behind the athlete and places the ipsilateral leg on his or her shoulder.
3. One hand is placed on the athlete's pubic and ischial rami. The other hand is placed on the ischial tuberosity and posterior inferior iliac spine of the dysfunctional side.
4. The clinician laterally distracts the innominate bone as the athlete relaxes. The clinician maintains a constant distracting force as the athlete takes deep breaths, relaxing between each breath (Fig. 22.3.7B).

High-Velocity, Low-Amplitude Technique for Posterior Ilial Dysfunction

Most Applicable Conditions: Osteitis pubis, hip pointer.

1. The athlete is placed in the lateral recumbent position with the dysfunctional side up.
2. The clinician stands at the side of the table facing the athlete.
3. The clinician flexes the athlete's superior hip and knee until motion is felt at the lumbosacral angle, then the *foot is placed in the popliteal fossa of the lower legs.*
4. Using the athlete's lower arm, the clinician rotates the upper body until all spinal rotation is removed down to the lumbosacral angle.
5. The clinician engages the athlete's torso with his or her cephalad forearm and the caudad forearm engages the athlete's superior ilium.
6. The rotational thrust is directed through the dysfunctional ilium (Fig. 22.3.8).

High-Velocity, Low-Amplitude Technique for Anterior Ilial Dysfunction

Most Applicable Conditions: Osteitis pubis, hip pointer.

1. The athlete is placed in the lateral recumbent position with the dysfunctional side up.
2. The clinician stands at the side of the table facing the athlete.
3. The clinician flexes the athlete's superior hip and knee until motion is felt at the lumbosacral angle, then the *superior leg is dropped off the table.*
4. Using the athlete's lower arm, the clinician rotates the upper body until all spinal rotation is removed down to the lumbosacral angle.
5. The clinician uses his or her cephalad arm to maintain the position while placing his or her caudad forearm over the superior ilium. The thrust is rotational and slightly downward through the dysfunctional ilium along the axis of the femur (Fig. 22.3.9).

Counterstrain for Iliopsoas Dysfunction

Most Applicable Conditions: Osteitis pubis, hip pointer, snapping hip syndrome.

1. The athlete is supine while the clinician sits on the ipsilateral side distal to the hip.
2. The clinician flexes the athlete's hip and knee so that the leg is resting on the shoul-

FIGURE 22.3.9. High-velocity, low-amplitude technique for anterior ilial dysfunction.

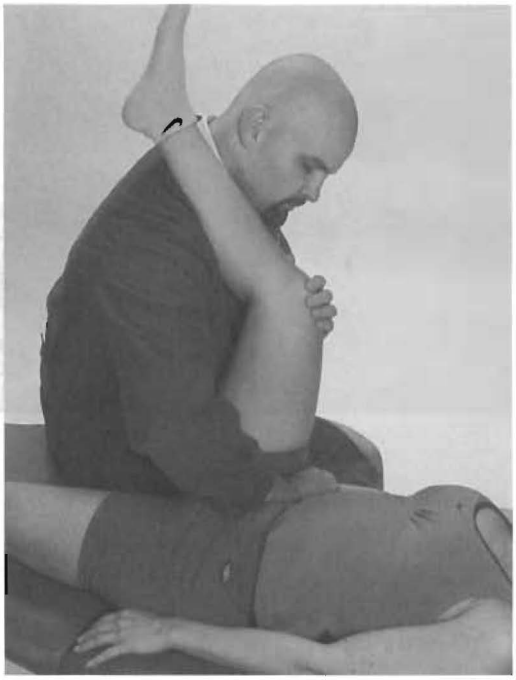

FIGURE 22.3.10. Counterstrain for the iliopsoas.

der and the iliopsoas is relaxed in a position of ease.
3. The clinician puts pressure on the iliopsoas and holds for 90 seconds (Fig. 22.3.10).
4. Retest.

STRETCHES FOR THE HIP AND PELVIS

Hamstring (Including Semitendinosus, Biceps Femoris, Semimembranosus). Sit on the floor with the legs split apart. Place both hands on one knee then slide the hands toward the foot. Engage the restrictive barrier and hold for 30 seconds (Fig. 22.3.11A). Repeat on the other leg. Good stretch for sports like track and field, basketball, football, softball/baseball, soccer, and hockey.

Iliopsoas Stretch. See Chapter 21.3, Lumbosacral Spine: Common Conditions (Fig. 21.3.16C.)

Iliotibial Band, Gluteus Medius, Tensor Fasciae Latae, Gluteus Minimus. Stand with the right hand on the wall for support and the right leg behind the left leg. Push down on the left hip. You should feel a stretch in the lateral part or side of the right thigh if the muscle is tight or shortened (Fig. 22.3.11B). Good stretch for running, tennis, handball, and racquetball.

Variation of Iliotibial Band, Gluteus Medius, Tensor Fasciae Latae, Gluteus Minimus. The athlete is lying on the right side with the left knee and hip bent and supported on pillows. The athlete places the left hand on the left knee to keep lumbar flexion and protect the spine. The clinician grabs the athlete's right leg and pulls upward toward the ceiling or in adduction (Fig. 22.3.11C). The clinician should feel stretch along the lateral portion of the right thigh. Stretch can also be done with the knee flexed.

Gluteal Stretch. The athlete lies on the table supine. The ipsilateral lower extremity is

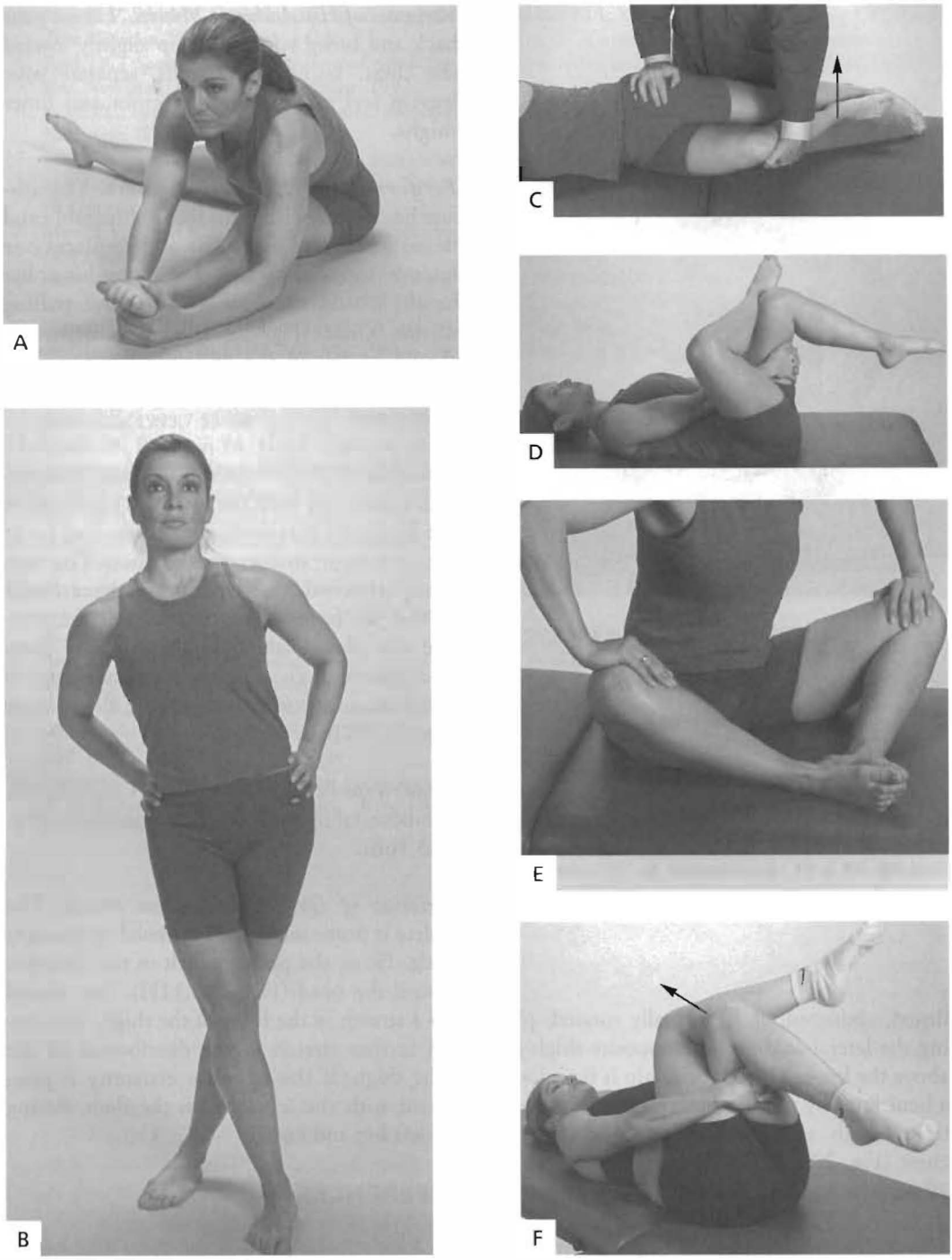

FIGURE 22.3.11. Stretches. **A,** Individual hamstrings; **B,** iliotibial band, gluteus medius, tensor fasciae latae, gluteus minimus; **C,** iliotibial band (IT) band adduction stretch with clinician; **D,** gluteal stretch; **E,** hip adductor stretch; **F,** piriformis muscle.

FIGURE 22.3.11. (*continued*) G, hip external rotators; **H,** variation of quadriceps femoris muscle.

flexed, abducted, and externally rotated, placing the lateral ankle on the opposite thigh just above the knee. The opposite hip is flexed with a bent knee by placing both hands on the posterior thigh and pulling the leg toward the chest (Fig. 22.3.11D). The stretch is held for 20 seconds and repeated three times.

Hip Adductor Muscles. Sitting up with your feet together and knees bent, separate your knees to feel a stretch in the groin and inner thighs, holding for 30 seconds (Fig. 22.3.11E). Good stretch for skaters, hockey, football, runners, skiing, dancers, and gymnasts.

Variation of Hip Adductor Muscles. Lie on your back and bring your knees up slightly toward the chest. Using your hands, separate your legs to feel a stretch in the groin and inner thighs.

Piriformis/Hip External Rotators. The athlete lies supine with both feet on the table and flexed knees and hips. The athlete places one knee on top of the other, then places his or her hands behind the bottom knee and pulling to the chest (Fig. 22.3.11F). This stretch should be felt in the midgluteal region. The stretch is held for 20 seconds and repeated three times.

Variation of Piriformis/Hip External Rotators. The athlete sits with the opposite leg flexed at the knee and externally rotated so that he or she is almost sitting on the foot. The side being stretched has the hip and knee flexed so that the foot can be crossed over the midline and placed lateral to the opposite knee. The athlete then uses the opposite arm to create an adduction force on the flexed knee (Fig. 22.3.11G).

Quadriceps Femoris Muscle. See Chapter 21.3, Lumbosacral Spine: Common Conditions (Fig. 21.3.16B).

Variation of Quadriceps Femoris Muscle. The athlete is prone with a belt fastened to the right ankle. He or she pulls the belt in the direction toward the head (Fig. 22.3.11H). You should feel a stretch in the front of the thigh. You may feel further stretch in the distribution of the right thigh, if the opposite extremity is positioned with the left foot on the floor, flexing the left hip and knee.

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23

THE KNEE

23.1

Anatomy

GAIL A. SHAFER-CRANE
WILLIAM M. FALLS

Four bones participate in the knee joint: the femur, tibia, fibula, and patella. The distal femur and proximal tibia form the main articulation. The patella, a sesamoid bone within the quadriceps tendon and the patellar ligament, provides anterior stabilization; and the fibula, which articulates proximally and distally with the tibia, allows leg rotation without sacrificing stability. Anatomy of the knee is presented in detail in major anatomical textbooks (1–8).

Important clinical bony landmarks are palpable about the knee joint. The femur flattens and widens just superior to the knee, forming the lateral and medial epicondyles. The articular femoral condyles, rounded ends of the femur, are palpated anterolaterally and medially as the knee is flexed. The quadriceps tendon is palpated just superior to the patella, and the patellar ligament is palpated just inferior to the patella, where it inserts onto the tibial tuberosity. When the knee is extended and relaxed, the patella can be glided laterally and medially on the articular surface of the condyles.

The anterior surface of the proximal tibia is palpated by pressing anterior to posterior with the fingertips along the inferior patellar border on either side of the patellar ligament. The tibial tuberosity is the bony protuberance that provides the insertion for the patellar ligament. It is palpated on the central anterior surface of the proximal tibia. The fibular head is palpated by sliding the thumb lateral to the patellar ligament along the tibial plateau, and then grasping the fibular head with the thumb and fingertips. The examiner can mobilize the head of the fibula in an oblique, anterior-to-posterior plane.

The tibiofemoral joint is the articulation between the articular condyles of the femur and the corresponding tibial condyles located on the superior surface of the tibia, or tibial plateau. This synovial hinge joint permits flexion, extension, and anterior-posterior translation and can bear body weight through approximately 120 degrees of range of motion. The femoral condyles are smooth articular surfaces, covered with hyaline cartilage, that wrap around the distal femur.

The patellofemoral joint is the articulation between the posterior patella and the anterior femoral condyles (Fig 23.1). The patella glides over the anterior articular surface of the distal femur during knee flexion and extension. In knee extension, the patella locks between the condyles to add stability to the knee.

The proximal tibiofibular articulation between the proximal lateral tibia and the proximal medial fibula is a synovial, condyloid joint supported by a fibrous capsule and anterior and posterior ligaments encircling the joint (Fig. 23.1.2). Dorsiflexion and plantarflexion of the foot create slight motions about the joint. The common fibular nerve and the nerve to the popliteus muscle innervate the tibiofibular joint.

The proximal attachment of the fibrous capsule is superior to the femoral articular condyles, and distal to the edges of the articular surface of the tibia. Its posterior attachment is within the intercondylar fossa. The popliteus muscle

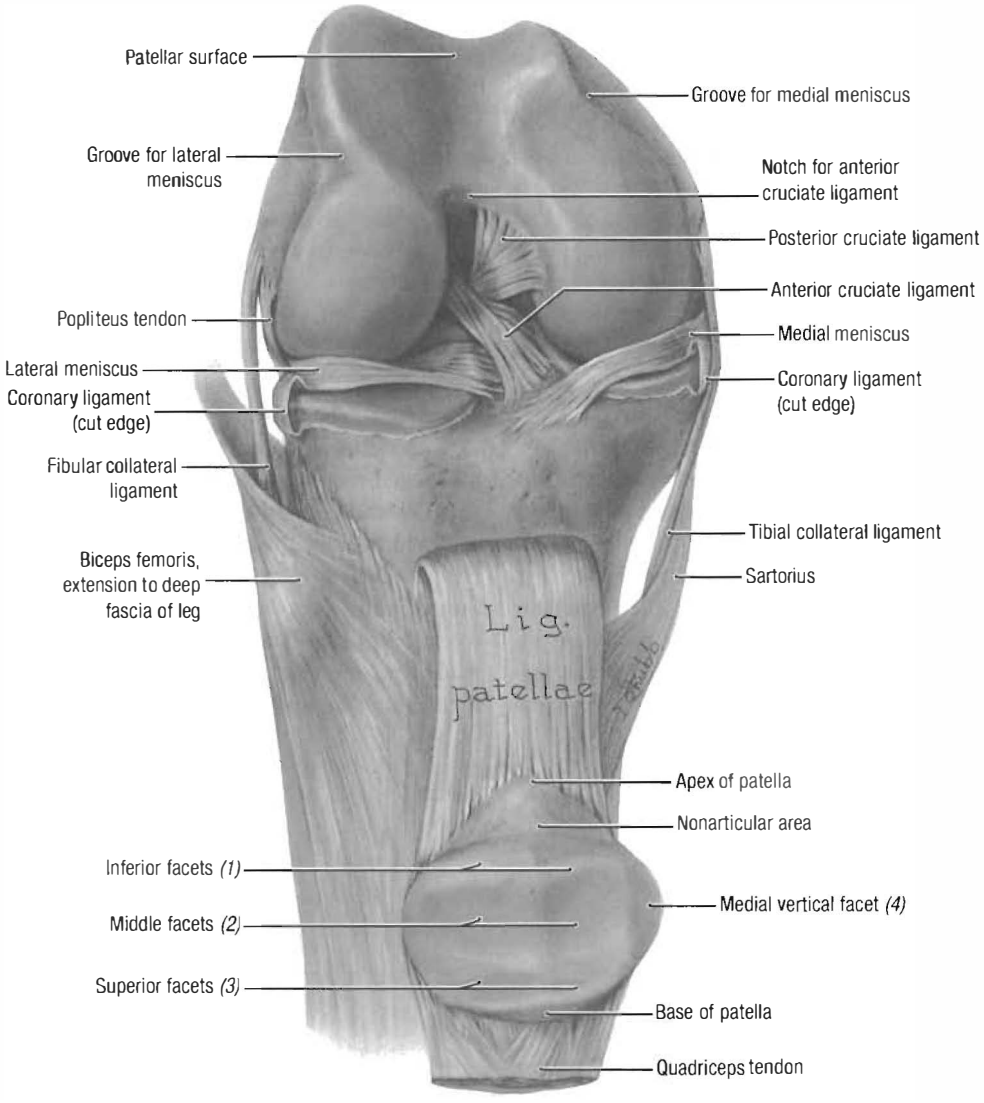

FIGURE 23.1.1. Anterior view of the knee with patella flipped down and out of the trachlea. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy*, 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

(description of the muscles follows) inserts on the tibia through an interruption in the lateral fibrous capsule. The lateral and medial menisci are semicircular fibrocartilaginous discs that lie between the fibular condyles and tibial plateau. Synovial membrane lines the fibrous capsule from the superior attachment of the fibrous capsule to the cruciate ligaments, and wrapping around to attach to the edges of the patella and menisci.

Extracapsular ligaments include the tibial and fibular collateral, posterior meniscofemoral, anterior and posterior cruciate, and transverse ligaments (Fig. 23.1.3). The patellar ligament is considered extracapsular, although it supports the joint capsule. The aponeurosis of the

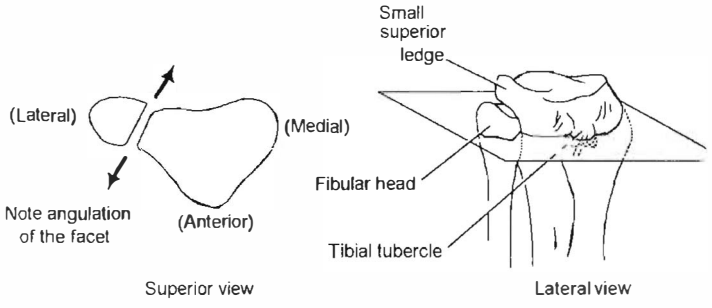

FIGURE 23.1.2. Proximal tibiofibular joint. (From Ward RC, ed. *Foundations of Osteopathic Medicine,* 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2003.)

quadriceps muscle blends with the patellar ligament, reinforcing the fibrous capsule.

The femoral, saphenous, tibial, obturator, and fibular nerves all provide branches that supply the knee. Since they also innervate the hip, knee pain is often referred proximally to the hip.

Muscles that act on the knee are divided into flexor and extensor groups. The knee extensors are the quadriceps femoris muscles. These include rectus femoris, vastus lateralis, vastus medialis, and vastus intermedius. The distal attachment of all of the quadriceps muscles is into the quadriceps tendon and aponeurosis and then the base of the patella, then to the tibial tuberosity through the patellar ligament. The extensor muscles of the knee are innervated by the femoral nerve (L2-L4).

Knee flexion is accomplished by thigh muscles (hamstrings, gracilis, sartorius, and popliteus); and lower leg muscles (gastrocnemius, soleus, and plantaris). Hamstring muscles include the long head of the biceps femoris, semimembranosus, and semitendinosus and act as

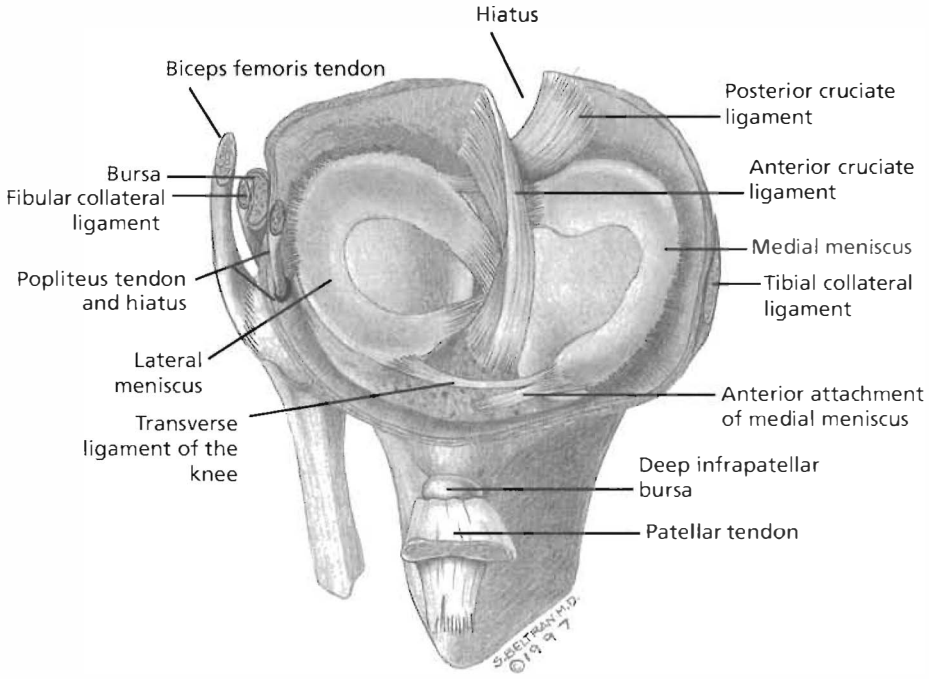

FIGURE 23.1.3. Anterosuperior view of the cruciate ligaments and menisci. (From Stoller. DW, *MRI, Arthroscopy, and Surgical Anatomy of the Joints.* Baltimore: Lippincott Williams & Wilkins, 1999.)

primary flexors of the knee, and hip extensors. They cross both the knee and the hip and are innervated by the tibial division of the sciatic nerve (L5, S1-S2). The short head of the biceps femoris does not cross the hip, and receives innervation from the fibular (peroneal) division of the sciatic nerve (L5, S1-S2); therefore, it is not technically a hamstring muscle. The gracilis is a medial thigh muscle. This straplike muscle works to adduct the thigh and flex the knee. The sartorius takes an oblique course across the thigh from superolateral to inferomedial. The semitendinosus, gracilis, and sartorius muscles join to make their distal attachment on the pes anserinus, a tendinous structure that attaches to the superior medial tibia (Fig. 23.1.4). A continuation of the semitendinosus tendon also reflects posteriorly to form the oblique popliteal ligament. The popliteus muscle is a small, deep triangular muscle in the posterior knee originating from the lateral femoral condyle and lateral meniscus, and inserting into the posterior, medial surface of the proximal tibia, superior to the soleal line (4).

The calf muscles are also secondary knee flexors due to the two gastrocnemius heads crossing the posterior knee and inserting onto the medial and lateral femoral condyles. Although the soleus and plantaris lie deep to the gastrocnemius muscle, they have a limited role in knee flexion. The soleus muscle makes its proximal attachment at the posterior fibular head, superior quarter of the posterior fibula, and the medial edge of the tibia. The plantaris muscle is a thin tendinous muscle that attaches proximally to the inferior lateral supracondylar line of the posterior femur and the oblique popliteal ligament. All three muscles insert onto

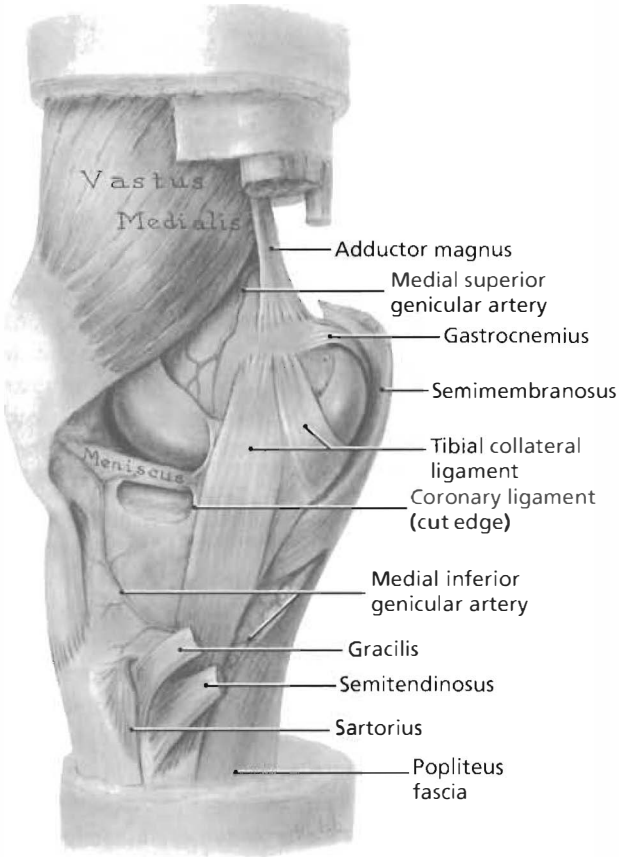

FIGURE 23.1.4. Medial dissection of the knee. Note the relationships among the medial collateral ligament, medial meniscus, and pes anserinus, where the gracilis, semitendinosus, and sartorius attach. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy*, 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

the posterior calcaneus or heel bone of the foot and are innervated by the tibial nerve (S1-S2).

The posterior knee is framed by the popliteal fossa (Fig. 23.1.5). The inferior borders of this diamond-shaped hollow are the heads of the gastrocnemius muscle as they attach to the lateral and medial condyles. It is defined superolaterally by the biceps femoris muscle and superomedially by the semimembranosus and semitendinosus muscles as they make their distal attachments to the lateral and medial femur. The adductor hiatus, the opening of the adductor (Hunter's) canal, which conducts the popliteal artery and vein through the thigh, is within the superior medial border. The sciatic nerve divides into the tibial and common fibular nerves in the superior confines of the popliteal fossa, and the medial sural cutaneous nerve splits from the tibia at the inferior border. The small saphenous vein, the posterior cutaneous nerve of the thigh, and lymph nodes and vessels are also contained within this space.

The synovial capsule and bursae form an extensive network within the synovial joint capsule. Lobes of this network are named for the adjacent tendon attachments. The suprapatellar bursa lies beneath the quadriceps tendon, and communicates directly with the anterior synovial joint capsule. The gastrocnemius and semimembranosus bursa communicate with the posterior portion of the synovial joint capsule, as does the popliteus bursa. The deep infrapatellar bursa lies

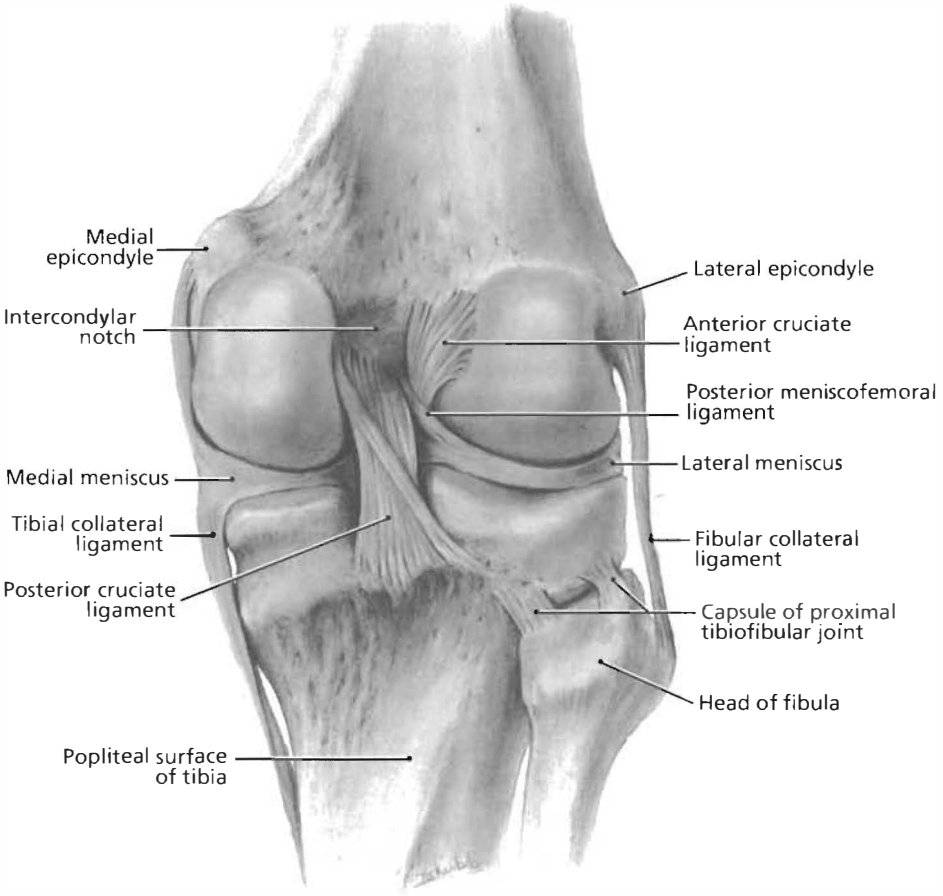

FIGURE 23.1.5. Posterior view of the knee. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy*, 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

between the patellar ligament and the tibial tuberosity. The tendons that attach at the pes anserinus, gracilis, sartorius, and semitendinosus are separated by the anserine bursa. There are subcutaneous bursae anterior to the patella and patellar ligament.

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23.2

Physical Examination

STEVEN J. KARAGEANES

OBSERVATION

Observe the athlete standing, looking for asymmetry in the knees. The important characteristics to look at and note are listed in Table 23.2.1. Arthritic knees can undergo bony remodeling and changes in alignment, so compare the condyles and tibial plateaus. Note the *knee and foot alignment*. Genu valgum, or knock-knees, has different implications in knee pain etiology than genu varum, or bowlegs. Genu recurvatum (hyperextension) should also be noted, of the knee, so look for this as well. Always include a foot examination with the knee examination and look for pes planus or cavus, hindfoot and forefoot valgus or varus, and restrictions in range of motion. Again, asymmetrical knee alignment may be a tip-off to degenerative knee changes.

Look at the *patellar alignment* (1). The winking kneecaps, where each patella faces more medial than normal, can indicate femoral anteversion. Note how lateral the patella rests in the intercondylar sulcus. A patella resting over the edge of the lateral femoral condyle may develop a mechanical tracking disorder.

Patellar position should also be checked (1). *Patella alta* is a condition in which the patella is anatomically too high in comparison to the femur. In full extension, only the inferior patellar pole should contact the sulcus. If it is above the sulcus, the athlete may have patella alta. *Patella baja* is when the patella rests too low. Radiographs can rule out both conditions.

Look for obvious *signs of injury or trauma*, such as ecchymosis, effusion (global or localized), erythema, and abrasion. Loss of landmark distinctions, such as the patella, fibular head, and tibial plateau suggest joint effusion. Fullness in the popliteal fossa may be from a Baker's cyst, so remember to look there (2).

Upon gait analysis, observe the range of motion in the injured knee compared with the uninjured one, such as excess flexion, internal rotation, and instability (Chapter 25 on gait analysis discusses this further).

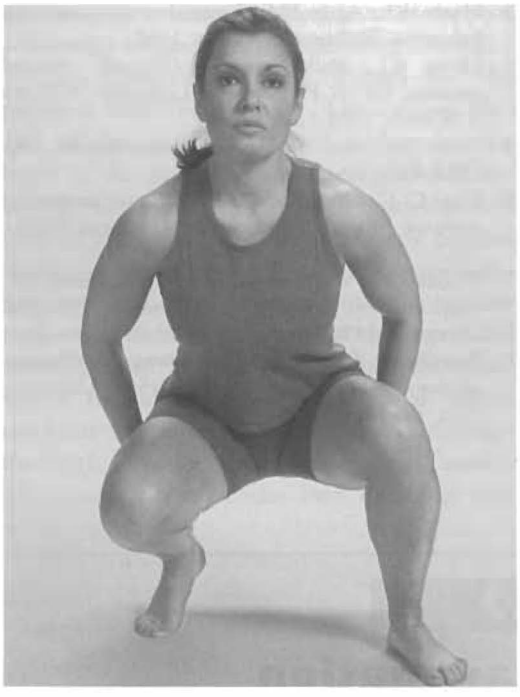

FIGURE 23.2.1. Duck-walk.

TABLE 23.2.1. WHAT TO LOOK FOR IN KNEE APPEARANCE

Alignment (genu valgum/varum/recurvatum)
Femoral anteversion
Effusion
Popliteal swelling
Signs of trauma or acute injury
Patellar position and alignment

GROSS KNEE TESTING

Duck-walk Test. In this screening test, the athlete moves from a standing to full squat position, with the thigh and calf in contact. From there, ask the athlete to "duck-walk" about 10 feet (Fig. 23.2.1).

Positive test: Pain in the knee, inability to perform the test.

Indicates: Evidence of knee pathology that warrants further testing.

PALPATION

Examine the athlete supine on an examining table. The examiner palpates the knee in four regions: anterior, medial, lateral, and posterior.

Anterior. Palpate the patella and feel for fluid superficial to the patella (prepatellar bursitis, or water on the knee), deformity (fracture or tendon rupture), or bony tenderness. Differential pre-patellar fluid contained in the bursa from an intra-articular effusion.

Feel superior and inferior to the patella for the quadriceps and patellar tendons, respectively. Look for fluid, tissue bogginess, tendon defects, and tissue tenderness.

Palpate the medial and lateral peripatellar regions, noting tenderness and tissue texture abnormalities. Since normal connective tissue is not tender to the touch, any such findings may be indicative of pathology.

Posterior. This part of the examination is usually done best with the athlete prone. Palpate in the popliteal fossa, noting fullness or tenderness. A Baker's cyst can form here and cause pain, while possibly indicating meniscus pathology. Pain along the posterior joint lines may indicate posterior horn injury of the menisci.

The popliteus tendon can be palpated at its lateral femoral origin and tibial attachments. Palpate for pain in these two regions. A posterolateral capsular injury has a limited window of opportunity to be surgically repaired, so look for signs of injury.

Medial. Palpate the medial joint line and note any tenderness or tissue texture abnormalities. Bogginess suggests an acute capsular, ligamentous, or medial meniscal injury. Slide up to the medial femoral condyle, feeling the medial collateral ligament (MCL) attachment. Palpate for the same on the medial tibial plateau.

Just slightly posterior and inferior to the tibial MCL attachment is the pes anserinus and its associated bursa. Tenderness here can indicate a pes anserine bursitis or tendon injury.

Lateral. First note the lateral joint line. Palpate the lateral femoral condyle, noting the lateral collateral ligament attachment and Gerdy's tubercle, which can play a role in iliotibial band syndrome.

Next, palpate the fibular head, the peroneal nerve just behind, and the proximal tibiofibular joint. Palpate the nerve gently, noting any tissue texture abnormalities or deformity.

NEUROVASCULAR EXAMINATION

(*Note:* In an acute knee injury, the neurovascular exam should be performed quickly to assess nerve damage and vascular compromise before any provocative tests are performed.)

1. *Vascular:* Palpate the popliteal artery and check for pulse. Check for abnormal swelling or ecchymosis in the region. Note any varicosities, and palpate for tenderness, which may indicate superficial thrombophlebitis. Homans' test and calf palpation may be necessary if the index of suspicion is high for a deep venous thrombosis.
2. *Patellar tendon reflex:* Ask the athlete to sit, legs relaxed and knee exposed. The examiner uses a reflex hammer to gently strike the midportion of the patellar tendon. The reflex is graded on a +4 scale.

Muscle Strength Testing

1. *Quadriceps:* Can be tested various ways. For gross testing, the athlete can sit on a table with the legs relaxed. The athlete extends the leg against a downward examiner-applied force onto the tibia, with the knee either at 90 degrees of flexion or at neutral (Fig. 23.2.2A). For a more accurate and specific measurement, the Cybex machines can be used.
2. *Hamstring:* Most commonly tested with the athlete prone on a table. The knee is flexed 20 to 30 degrees, and the athlete contracts against resistance (Fig. 23.2.2B).
3. *Gastrocnemius:* The athlete lies supine or prone, and the knee is in full extension. The athlete plantarflexes the foot against the examiner's resistance (Fig. 23.2.2C).

Note: From this point on in the examination, the athlete is supine on the table, unless alternate techniques are specifically described.

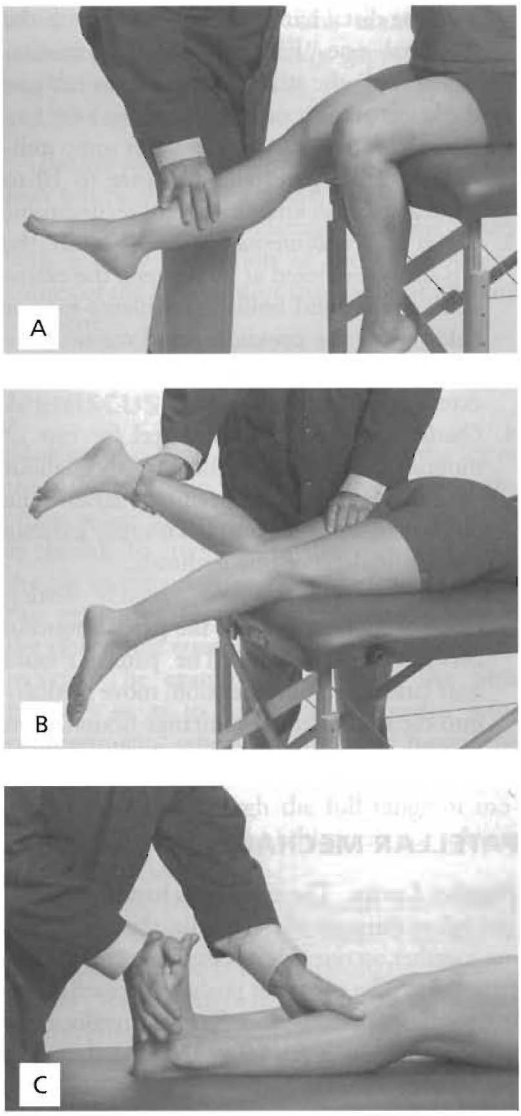

FIGURE 23.2.2. Strength testing. **A,** Quadriceps. **B,** Hamstring. **C,** Gastrocnemius.

RANGE OF MOTION

1. *Flexion.* With the athlete supine and the hip flexed at 45 degrees, bend the knee until the restrictive barrier is met or pain is felt. Normal range is 145 to 150 degrees (1).
2. *Extension.* The athlete is supine, and the examiner firmly holds the distal thigh with one hand, while the other hand firmly holds the distal tibia proximal to the ankle. Lift

with the distal hand, while holding with the proximal one. For goniometric measurements, slide the athlete down on the table so the knees are just off the edge. The knee can typically extend to 0 degrees, but some individuals can hyperextend the knee to 10 to 15 degrees, also known as genu recurvatum.

3. *Tibial external/internal rotation.* With the hip and knee flexed at 90 degrees, the examiner's distal hand holds the athlete's foot or ankle while the proximal hand controls the knee. The ankle is rotated internally, then externally (1).
4. *Quality of range of motion.* Feel for ease of motion and crepitus in the knee throughout flexion and extension, specifically around the patellofemoral mechanism. This may indicate chondromalacia or osteoarthrosis.
5. *Patellar mobility.* When passively testing range of motion, observe the patellar motion between the condyles. The patella should start laterally in full extension, move medially into the sulcus during midrange flexion, then again laterally as the knee is fully flexed.

PATELLAR MECHANISM

Patellar Laxity. The examiner's hands are above and below the poles of the patella, thumbs touching together on one side, index fingers on the opposite side. The examiner pushes the patella with the thumbs medially and fingers laterally until tissue restriction is felt (Fig. 23.2.3A). Laxity is graded on a +3 scale.

Patellar Apprehension. When testing patellar laxity (Fig. 23.2.3A) to the point of restriction, ask the athlete if the maneuver provokes any discomfort or sensation of instability.

Positive test: Athlete responds apprehensively, feels uncomfortable, or feels a sensation of giving out or dislocating.

Indicates: Previous patellar dislocation or severe instability.

Patellar Compression. The examiner places his or her hand over the patella and gently pushes toward the table (Fig. 23.2.3B).

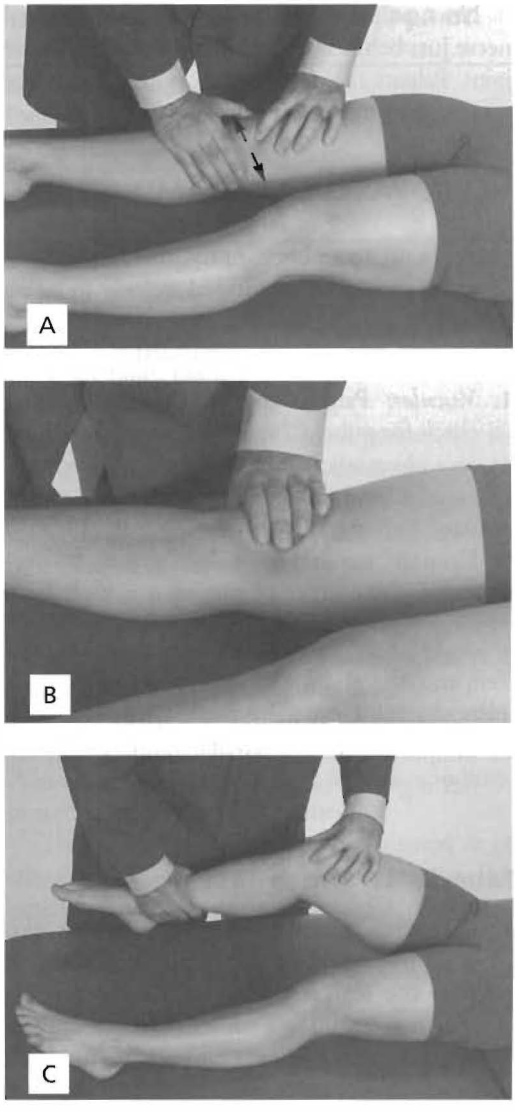

FIGURE 23.2.3. Patellar testing. **A,** Apprehension test. **B,** Patellar compression test. **C,** Patellar glide.

Positive test: Pain around or under the patella with compression.

Indicates: Injury or inflammation to the patellofemoral articulation.

Patellar Glide. The examiner notes how smooth the patella translates through the intercondylar sulcus during flexion and extension (Fig. 23.2.3C).

Positive test: Palpable or audible crepitus; pain under or around the patella.

Indicates: Chondromalacia patellae, osteoarthrosis, active tracking disorder.

COLLATERAL LIGAMENTS

Valgus Stress Test

At 30 Degrees Flexion. The examiner places one hand on the lateral thigh just above the lateral joint line. The athlete's leg is lifted, and the lower leg is placed and held against the examiner's side between the rib cage and humerus, leaving both hands free while the knee and hip are flexed roughly 30 degrees. With both hands firmly holding the proximal tibia, the examiner translates the tibia *medially*, gapping the medial joint line (Fig. 23.2.4A). Check the opposite knee for comparison. The examiner assesses for laxity, quality of end point, and painful response.

Positive test: Increased laxity, soft or absent end point, pain with maneuver.

Indicates: Medial collateral ligament disruption.

At Neutral. The examiner performs the test with the athlete's knee at full extension. Laxity suggests medial collateral ligament injury with a likely disruption of the anterior cruciate ligament.

Varus Stress Test

At Neutral. The setup is the same as for the valgus stress test in neutral, except that the examiner's hands are switched, and the proximal one is on the medial thigh. The examiner then pushes the lower leg *medially*, gapping the lateral joint line (Fig. 23.2.4B). Perform at neutral and 30 degrees of flexion.

Positive test: Laxity with gapping of the joint line, soft or absent end point, pain.

Indicates: Lateral collateral ligament injury with a likely disruption of the posterolateral corner of the knee and/or the posterior cruciate ligament.

MENISCUS

McMurray's Test. The examiner lifts the leg of the supine athlete, one hand controlling the ankle, while the other hand controls the knee, as shown. To stress the medial meniscus, start the leg in extension and flex the knee. During this maneuver, the clinician internally rotates the tibia while stressing the knee in a varus direction. The examiner is stressing the tibia medially so as to compress the medial joint compartment, while the rotation forces the condyles on and against the medial meniscus. This continues through the full range of motion (ROM) or until the athlete cannot tolerate it (Fig. 23.2.5A).

To test the lateral meniscus, the maneuver is reversed, but the clinician's hands can stay in the same position. During flexion, the clinician rotates the tibia externally and puts valgus stress on the knee, which compresses the lateral joint compartment (Fig. 23.2.5.B).

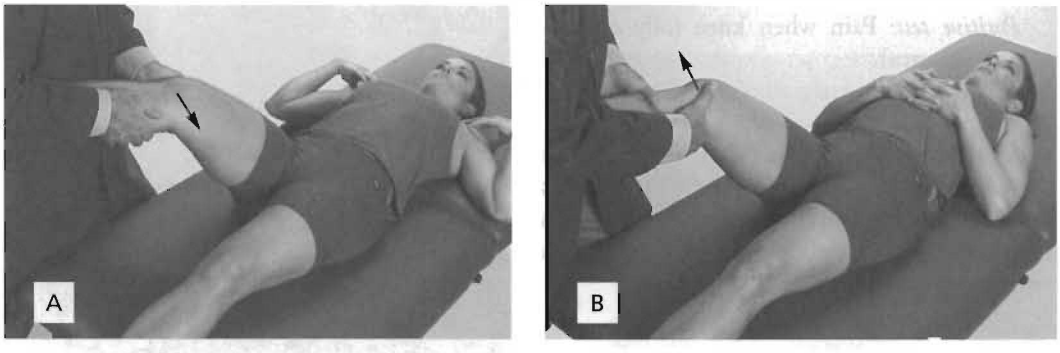

FIGURE 23.2.4. Collateral ligament testing. **A,** Valgus stress test at 30 degrees. **B,** Varus stress test at 30 degrees of flexion.

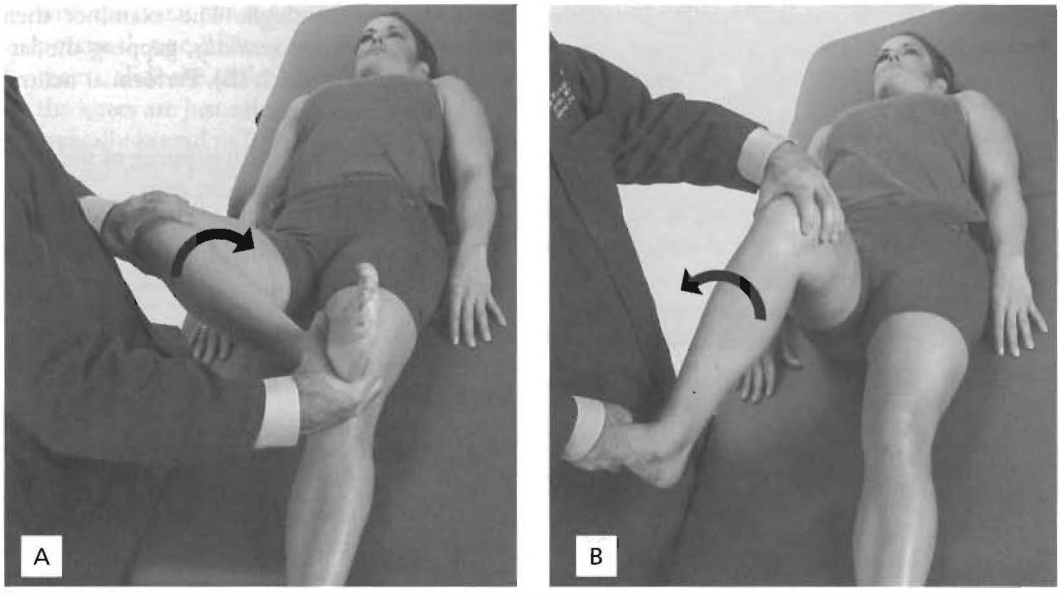

FIGURE 23.2.5. McMurray's test. **A,** Varus and internal rotation for the medial meniscus. **B,** Valgus and external rotation for the lateral meniscus.

Positive test: A click or clunk sensation; pain in a specific point of ROM that can be mechanically reproduced.

Indicates: Meniscus pathology.

Bounce Test. The clinician takes the relaxed supine leg with one hand on the proximal tibia and the other hand just above the ankle. The clinician first elevates the leg about 6 in., flexes the knee about 10 degrees, then lets the knee fall back toward the table, bouncing into extension. The key is to perform this in a relaxed fashion and with only minimal force (Fig. 23.2.6).

Positive test: Pain when knee fully extends (on the bounce).

Indicates: Meniscus pathology.

Apley's Grind Test. The athlete is prone with the knee flexed 90 degrees. The clinician pushes down on the foot toward the table and rotates internally to its end point (Fig. 23.2.7). This is repeated in external rotation.

Positive test: Pain with either internal or external rotation.

Indicates: Meniscus pathology.

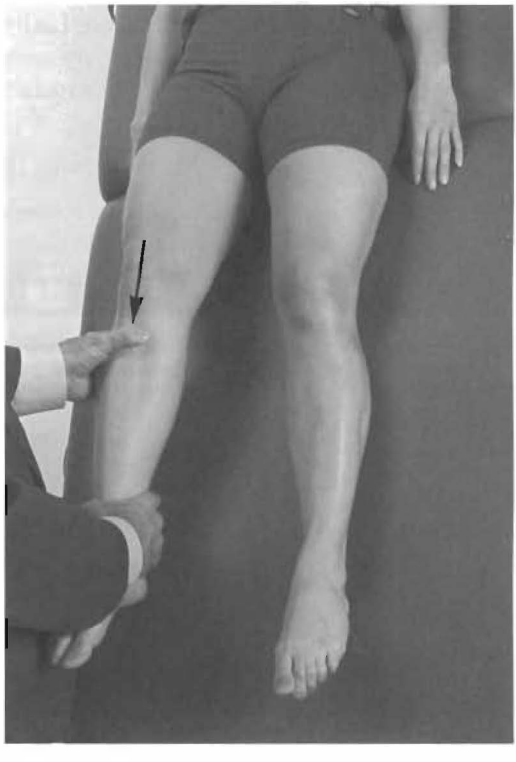

FIGURE 23.2.6. Bounce test.

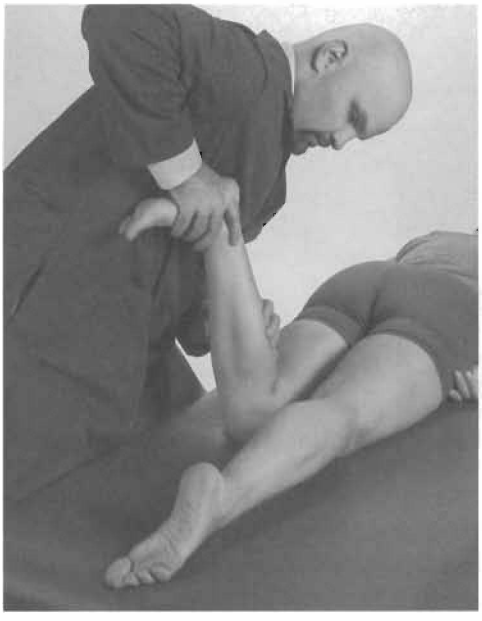

FIGURE 23.2.7. Apley's grind test.

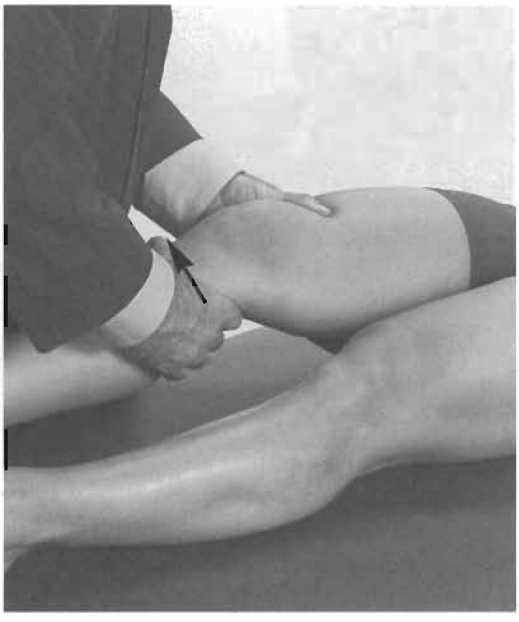

FIGURE 23.2.8. Lachman's test.

ANTERIOR STABILITY

Lachman's Test. While the athlete is supine, the clinician places the proximal stabilizing hand around the distal thigh just above the patella. The distal mobilizing hand grasps the proximal tibia. Flexing the knee 30 degrees, the distal hand pulls on the tibia away from the table, while the proximal hand stabilizes the leg. This pulling translates the tibia anteriorly (Fig. 23.2.8).

Positive test: Increased laxity, particularly compared with the opposite joint; soft or absent end point.

Indicates: Anterior cruciate ligament insufficiency.

Anterior Drawer Test. The knee is flexed to 90 degrees with the athlete's foot on the table. The clinician sits on the foot to stabilize the lower leg, while both hands reach around the proximal tibia and translate the tibia anteriorly (Fig. 23.2.9).

Positive test: Excessive translation, particularly in comparison with the opposite joint.

Indicates: Anterior cruciate ligament insufficiency.

Pivot Shift Test. The athlete is supine, while the clinician holds the knee with both hands. The knee is held in 20 degrees of flexion and neutral rotation. The athlete is asked to relax the leg, which allows the femur to sag back when the anterior cruciate ligament is disrupted. Knee is first moved into extension with applied valgus and internal rotational stress. Then the knee is flexed to 40 degrees, causing a forward subluxation of the tibia during the sudden change in direction (Fig. 23.2.10). This test is difficult to elicit in the clinical

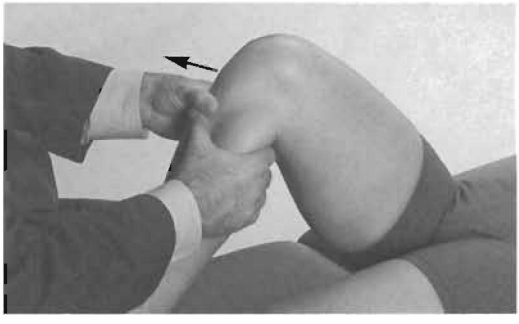

FIGURE 23.2.9. Anterior drawer test.

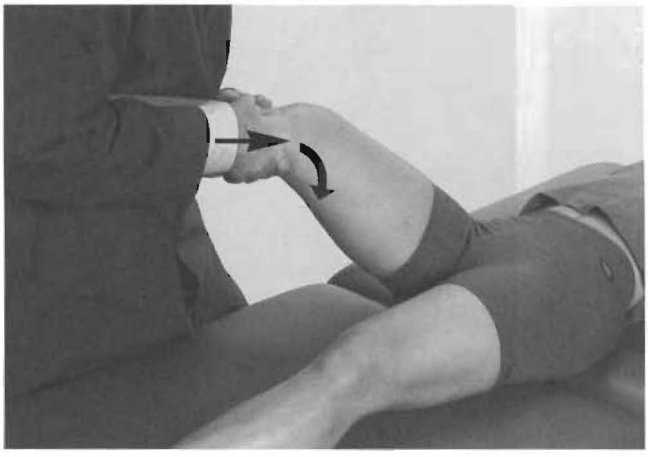

FIGURE 23.2.10. Pivot shift test.

setting, which is why it is often performed in the operating room under sedation to verify the diagnosis.

Positive test: Tibial subluxation (lateral greater than medial) at 20 to 40 degrees of flexion, shifting or "clunking" of the tibia against the femur.

Indicates: Anterior cruciate ligament deficiency.

POSTERIOR STABILITY

Posterior Drawer Test. The knee is set up in the same position as for the anterior drawer test. The clinician grasps the knee and translates the tibia in a posterior direction (Fig. 23.2.11).

Positive test: Excessive translation, particularly in comparison with the opposite joint.

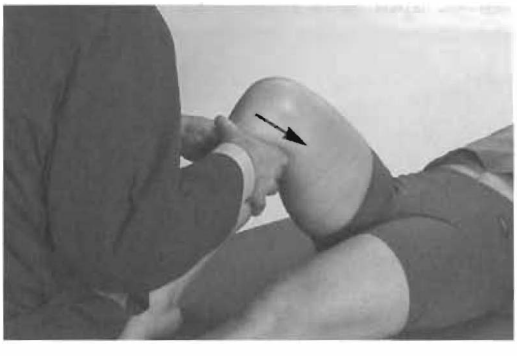

FIGURE 23.2.11. Posterior drawer test.

Indicates: Posterior cruciate ligament deficiency, posterior capsular injury or disruption.

Sag Test. Both knees are set up as for the anterior drawer position. The clinician stabilizes both feet and observes the prominence of the medial tibial plateau that should be anterior to the medial femoral condyle. Both knees are assessed for symmetry.

Positive test: Tibial plateau of affected side sagging more posteriorly than the opposite knee; loss of 1-cm stepoff between medial femoral condyle and medial tibial plateau.

Indicates: Posterior cruciate ligament deficiency.

Reverse Lachman's Test. The position of the hands and knee is the same as for Lachman's test. The proximal hand stabilizes the leg while the distal hand pushes the tibia toward the table in a posterior direction (Fig. 23.2.12). This test can be done right after the Lachman's test during the examination.

Positive test: Increased laxity, particularly in comparison with the opposite joint; soft or absent end point.

Indicates: Posterior cruciate ligament deficiency, posterior capsular instability.

Quadriceps Active Test. This test is most effective when the sag test is positive. The athlete's knee is flexed 60 degrees and the clinician

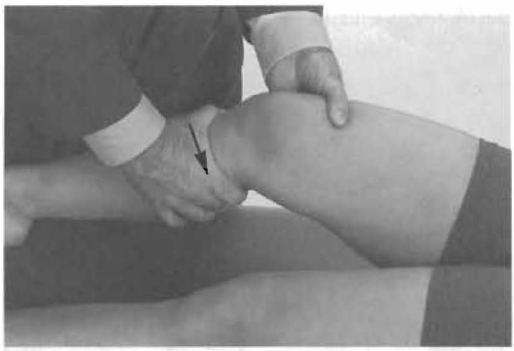

FIGURE 23.2.12. Reverse Lachman's test.

FIGURE 23.2.14. Dynamic posterior shi't test.

secures the foot on table. The athlete attempts to extend the knee with an isometric contraction (Fig. 23.2.13).

Positive test: Anterior tibial translation that recreates the 1-cm stepoff between medial femoral condyle and medial tibial plateau.

Indicates: Posterior cruciate ligament deficiency.

Dynamic Posterior Shift Test. The knee and hip are flexed at 90 degrees. The athlete slowly extends the knee to full extension (Fig. 23.2.14). With posterior cruciate ligament deficiency, the tibia is subluxed posteriorly throughout its range of motion, then it is reduced at full extension.

Positive test: Clunk near full extension.

Indicates: Posterior cruciate ligament deficiency.

External Rotation Recurvatum Test. The clinician lifts both supine legs by the great toes.

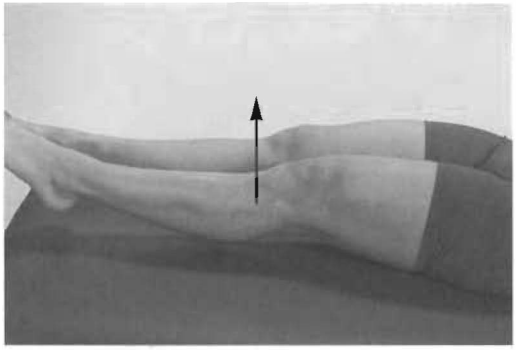

FIGURE 23.2.13. Quadriceps active test.

Positive test: Knee drops into varus alignment and hyperextension compared to uninjured side. Recurvatum bilaterally suggests generalized ligamentous laxity.

Indicates: Posterolateral instability, posterior cruciate ligament deficiency.

POSTERIOR CAPSULE

External Rotation Dial Test. While the athlete is prone, the foot is passively externally rotated, measuring rotation with the thigh-foot angle, then comparing with the opposite knee. Perform with the knee flexed in 30 and 90 degrees of flexion (Fig. 23.2.15A and B).

Positive test: 10-degree difference between knees in 30 degrees of flexion.

May indicate: Posterolateral capsular injury.

Reverse Pivot Shift Test. With the athlete supine, the clinician applies a valgus force to the knee flexed in 90 degrees. The knee is then slowly extended with the valgus force still applied (Fig. 23.2.16).

Positive test: Visible shift or clunk near 30 degrees of flexion.

Indicates: Posterolateral capsular injury or posterior cruciate ligament deficiency (3).

POPLITEUS MUSCLE

This muscle deserves special recognition because it often goes unexamined on a standard

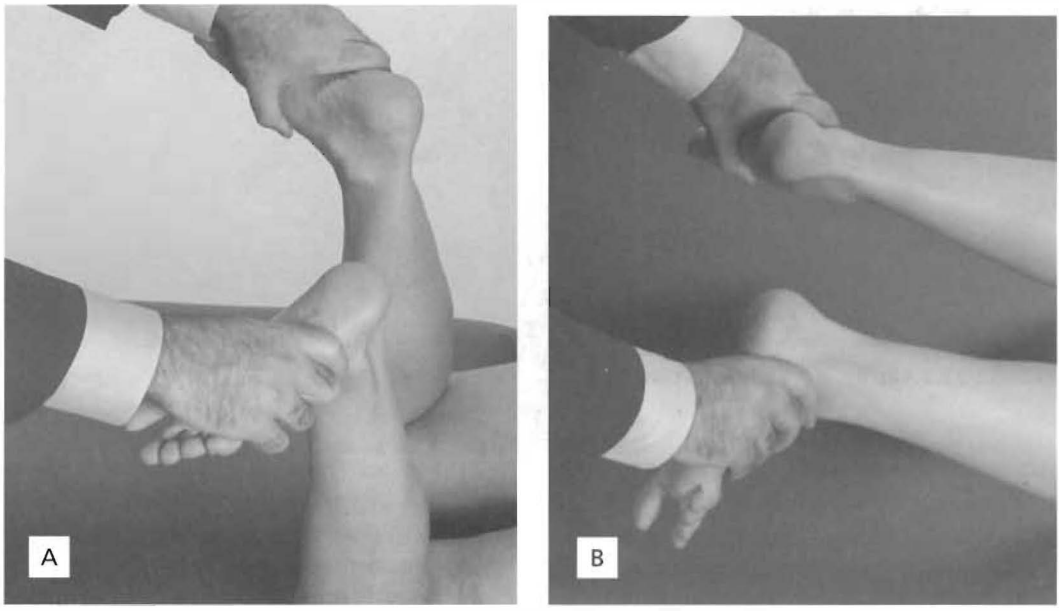

FIGURE 23.2.15. External rotation dial test. **A,** 90 degrees of knee flexion. **B,** 30 degrees of knee flexion.

orthopedic examination. The popliteus originates from the lateral femoral condyle and capsule of the knee, traverses the popliteal fossa, and inserts into the posterior surface of the tibia. Its origin is just inferior to the femoral lateral collateral ligament attachment, and a bursa separates the tendon from the ligament and capsule.

The popliteus muscle internally rotates the knee, which is important in bringing the knee out of full extension. By virtue of its location and blend with the arcuate ligament complex, the muscle also stabilizes the posterior knee by preventing posterior tibial translation on the femur, as well as the lateral meniscus.

Garrick's Test. While the athlete lies supine, the clinician flexes the hip and knee 90 degrees, internally rotating the leg. The athlete holds the leg in this position while the clinician applies external rotatory resistance (Fig. 23.2.17).

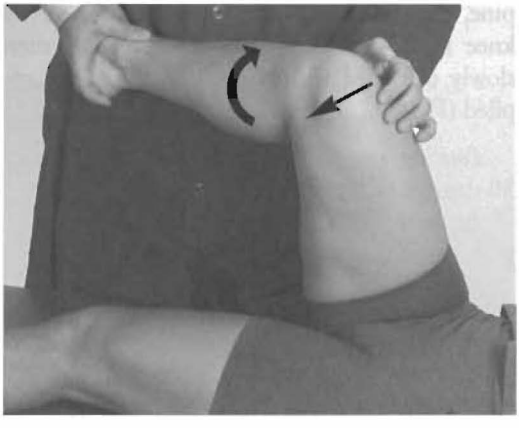

FIGURE 23.2.16. Reverse pivot shift test.

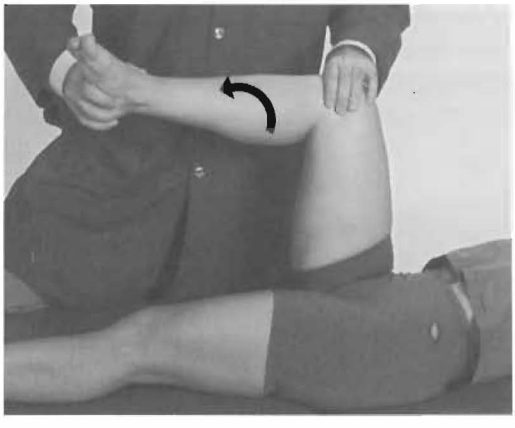

FIGURE 23.2.17. Garrick's (popliteus) test.

Alternate Method. The clinician introduces passive external rotation to the tibia, putting traction on the popliteus muscle. The athlete does not resist force.

Positive test: Pain with muscle contraction.
Indicates: Popliteus tendon or muscle injury (2).

JOINT PLAY

Patellofemoral Joint

a. Cephalic glide
b. Medial glide
c. Lateral glide
d. Rock
e. Caudal glide

This examination can be done with the athlete supine and the legs flat on the table. Cephalic, caudal, medial, and lateral glide refer to the direction that the patella is mobilized. Rocking can be done with the index and middle fingers of each hand on one patellar pole, then alternating mobilizing the patella with a posteriorly directed cephalic, then caudal force (4) (Fig. 22.2.18).

Femoral-tibial Joint

1. *Medial tilt.* The setup is the same as for valgus/varus stress tests, but the knee is only flexed 2 to 3 degrees to unlock the knee. Medial force is introduced to move the femoral condyles medially and gap the medial joint (Fig. 23.2.19). The mobilizing force is gentle and only tilts the plateau up to the anatomic barrier.
2. *Lateral tilt.* Same setup as for the medial tilt, but the femoral condyles are instead forced laterally, gapping the lateral joint (Fig. 23.2.19).
3. *Anteroposterior glide.* The athlete's knee is flexed 45 degrees (similar to the anterior drawer position), while the clinician sits on the foot to stabilize the leg. The clinician grasps the proximal tibia and translates it anteriorly (A), then posteriorly (B) (Fig. 23.2.20). Again, the operator is feeling for intrinsic motion, or play, not ligamentous

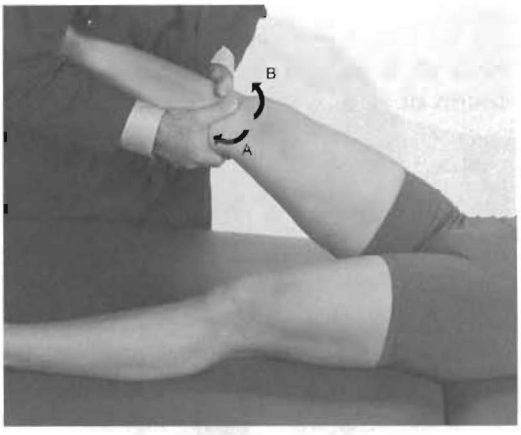

FIGURE 23.2.19. Femoral-tibial joint: medial tilt.

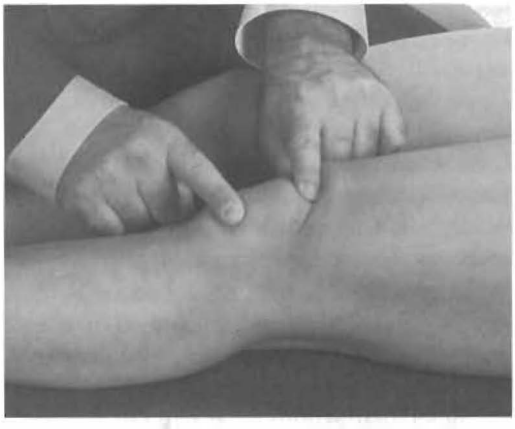

FIGURE 23.2.18. Joint play: patellofemoral joint.

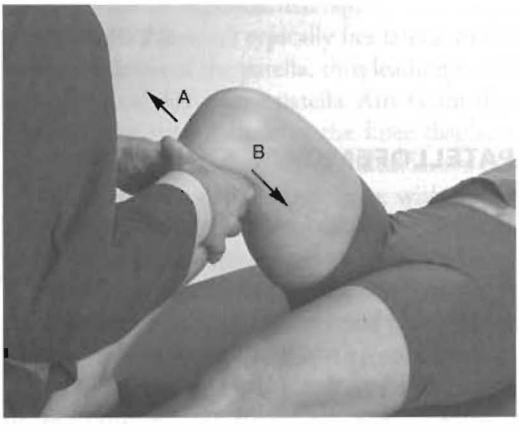

FIGURE 23.2.20. Anteroposterior glide.

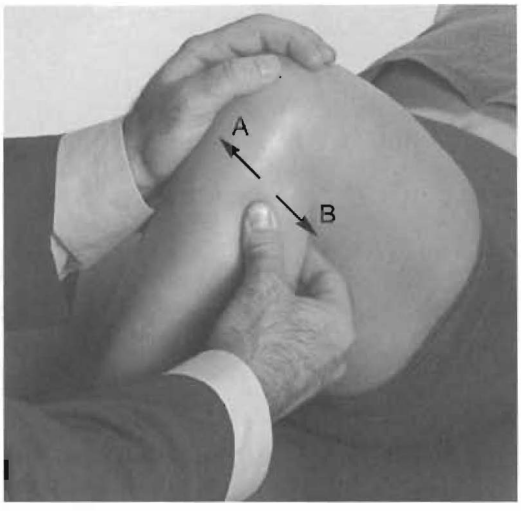

FIGURE 23.2.21. Joint play: proximal tibiofibular joint.

disruption (e.g., anterior drawer), so the translating force is subtle.

Proximal Tibiofibular Joint

a. Anterior glide
b. Posterior glide

The clinician sets up the knee flexed at 45 degrees. The fibular head can be grasped in two ways: (a) all fingers posterior and thenar eminence anterior, or (b) index and middle finger posterior and thumb anterior. (Fig. 23.2.21). The clinician mobilizes the fibular head anteriorly (A) then posteriorly (B). Note the amount of motion and quality of feel, and compare with the opposite side (4,5). For example, a fibular head that resists posterior mobilization is usually in an anterior position (5).

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23.3

Common Conditions

STEVEN J. KARAGEANES

PATELLOFEMORAL SYNDROME

Patellofemoral pain is the most common injury seen by sports medicine physicians. It is primarily an overuse injury that can be debilitating if untreated. However, effective treatment can be quite elusive, as many factors affect the articulation between the patella and the femoral condyles. These problems are common due to various inherent anatomic factors, but overuse plays a major role in most cases. The amount of force transmitted through the patella during jumping can reach up to 20 times body weight, which when coupled with anatomic and biomechanical factors can lead to overload and breakdown (1).

Classification

Patellofemoral syndrome (PFS) can be classified into overuse and traumatic injuries. Traumatic compression of the patella against the

condyles can cause damage to the joint surface. Osteochondritis dissecans causes focal loss of articular cartilage and discomfort when the patella contacts the defect, while long-standing degenerative changes lead to chondromalacia and osteoarthrosis of the patellofemoral joint. Patellar contusions can inflame the infrapatellar Hoffa's pad (also called fat pad syndrome), which can generate significant pain and mimic PFS. This is an important distinction to make, because patellar taping can help a tracking disorder but exacerbates a fat pad syndrome.

Patellar dislocations can occur with significant twisting and sudden quadriceps contraction. Subluxations occur more often, but can cause lingering pain, feelings of giving way, and instability. This can alter patellar excursion, generate effusions, and limit stair climbing and knee bending until treated appropriately. Meniscal tears can cause effusion and anterior pain occasionally, so this must be ruled out.

Overuse usually leads to inflammation of the patellar mechanism, at either the tendon or the patellofemoral articulation. This pain worsens with climbing stairs and negotiating hills or inclines. In children, these forces can lead to inflammation of the patellar tendon insertion at the tibial tuberosity (Osgood-Schlatter disease) or inferior patella (Sindig-Larsen-Johannsson disease) (2).

Pertinent Anatomy

As described in the physical examination of the knee (see Chapter 23.2), the patella is embedded in the tendon extending from the quadriceps to its insertion into the tibial tuberosity. It sits just lateral to the trochlea with the knee at full extension. When the knee flexes, the patella moves medially between the medial and lateral femoral condyles. The patellar articular surface contacts the condyles at around 30 degrees. Near 130 degrees, the patella shifts laterally to rest in the intercondylar notch. The lateral patellar retinaculum is much stronger than the medial, so the patella tends to shift laterally when destabilized. The prominent lateral femoral condyle couples with the iliotibial band and vastus lateralis to provide lateral support, while the medial retinaculum resists lateral translation.

Mechanism

The patella increases the efficiency of the extensor mechanism by as much as 50%. Its embedded position in the patellar tendon makes it act like a pulley dispersing force as it tracks between the medial and lateral femoral condyle. Climbing stairs increases loading force through the patella by three times body weight, and squatting increases force by seven to eight times body weight. In jumping, this force can reach 15 to 20 times body weight.

The patella also directs the divergent forces of the multiple quadriceps muscles to the patellar tendon. The balance of strength among the four muscle heads can dictate the direction the patella tracks because of the common union at the patellar tendon. Anything that alters the muscle balance can change patellar excursion.

Factors

Patellofemoral syndrome is challenging because of the number of potential factors involved (Table 23.3.1). The vastus medialis obliquus (VMO) stabilizes the patella by counterbalancing the vastus lateralis (VL) during quadriceps contraction. Poor development of the VMO is accepted as a cause of patellar tracking disorders, but slower VMO firing—when compared to VL firing—may be just as negative. This can either be congenital or acquired.

The shortest distance between two points is a straight line, but the line between the quadriceps origin (anterior superior iliac spine) and insertion (tibial tuberosity) typically lies lateral to the resting position of the patella, thus leading to the natural lateral drift of the patella. Any factor that increases the valgus angle at the knee displaces the patella further away from the ideal lateral position when extended, so any knee with excess valgus alignment is at higher risk for poor tracking. This measurement of valgus deviation of the knee is occasionally referred to as the Q-angle.

Overpronation causes internal rotation of the tibia and leads to an increased valgus moment at the knee. Duffey et al. identified pronation during the first 10% of the stance phase of the gait cycle as a specific predictor of patellofemoral pain (3).

TABLE 23.3.1. PREDISPOSING FACTORS FOR PATELLOFEMORAL SYNDROME

Muscle restriction
Rectus femoris
Hamstrings
Gastrocnemius
Iliotibial band
Adductors
Muscle dysfunction
Vastus medialis obliquus insufficiency
Hip abductors
Hip external rotators
Foot mechanics
Pes planus
Overpronation
Equinus contracture
Soft tissue restriction
Lateral patellar retinaculum
Iliotibial band
Anterior hip capsule restriction
Postsurgical
Anterior cruciate ligament reconstruction
Achilles tendon repair
Biomechanical
Femoral anteversion
Genu valgum
Female (wider) pelvis

Internal femoral torsion (femoral anteversion) increases external rotation of the tibia, which increases valgus. This can occur congenitally or through soft tissue compensation. Femoral anteversion also causes anterior hip tightness. Restricted motion in the hip and knee increases force through the patella, so restrictions in the anterior hip capsule, rectus femoris, tensor fasciae latae, and iliopsoas must be identified.

A tight iliotibial band causes overactivity in the tensor fasciae latae, which lengthens the gluteus medius. Elongated muscles are harder to recruit and become weaker, while shortened muscles, such as the tensor fasciae latae, become hypertonic and restricted. A weakened gluteus medius increases medial (internal) rotation at the hip, which causes the pelvis on the opposite side to drop, dynamically increasing the Q-angle (4).

The lateral supportive structures of the patella prevent excessive lateral translation. When these structures are too restrictive, they limit medial translation. Specifically, iliotibial band and vastus lateralis tightness limit medial glide, while the lateral retinaculum restricts medial tilt. Because medial glide occurs as the patella slides by the condyles into the intercondylar notch throughout knee flexion, restriction forces the patella onto the lateral femoral condyle, creating friction.

Hamstring restriction is a culprit in many athletic injuries, and its effect on the patellofemoral mechanism is seen during gait. A tight hamstring increases knee flexion during the heel strike phase of the gait cycle. Since the knee does not extend well, ankle dorsiflexion must increase to allow the body to come across the planted foot. When maximal dorsiflexion is reached, the foot pronates, causing a dynamic change in valgus alignment (4,5).

In addition, neurologic factors are being implicated as well. Sanchis-Alfonso correlated anterior knee pain with the number and size of sensory nerves in the anterior capsule, where the most severe pain correlated with the largest nerves, and moderate pain correlated with the highest neural area and number of nerves (6). Neural markers such as S-100 protein, neurofilament protein, substance P, and neural growth factor also showed a positive relationship to anterior knee pain (7).

The Athlete

Sports that involve running and jumping, such as track and field, soccer, basketball, volleyball, ballet, and recreational running, are more prone to developing patellofemoral syndrome. (5). However, most every sport places demands on the knee, and any sport that places repetitive eccentric load on the patellofemoral joint will likely trigger joint pain. Running tends to bring out PFS symptoms because of the repetitive mechanical nature of the activity, while basketball and volleyball are the types of sports that have higher eccentric load and affect the patellar tendon more than running.

Standard Treatment

In traditional treatment, the first step is to control the inflammation (Table 23.3.2). Physical therapy modalities are effective adjuncts to ice

TABLE 23.3.2. STANDARD TREATMENT FOR PATELLOFEMORAL SYNDROME

Reduce inflammation (ice, nonsteroidal anti-inflammatory drugs, electrical stimulation, ultrasound)
Correct foot biomechanical abnormalities (insoles, orthotics)
Patellar taping
Patellar bracing
Vastus medialis retraining/strengthening
Activity modification

and nonsteroidal anti-inflammatory drugs (NSAIDs). If poor foot mechanics, such as overpronation and hindfoot inversion, have a causal relationship to the anterior knee pain, then they can be corrected with orthotics.

Patellar taping aids in holding the patella closer to proper position as it moves through flexion and extension. This is particularly effective with significant lateral tilts. Taping also causes earlier firing of the VMO, which aids in stabilization, particularly with tight lateral support structures. Anterior tilt and excessive medial glide can be aided as well. Those who do not react well to taping (allergy, irritation, dampness) can try a patellar stabilization brace, which often relieves pain despite research showing that most patellar braces do not significantly alter patellar position or tracking. Neither treatment has been shown to consistently improve patellofemoral maltracking syndrome in studies.

Biofeedback improves the firing of the VMO compared with the vastus lateralis. Simple home techniques include placing the hand on the quadriceps when actively contracting the muscle. More complex techniques can provide visual and auditory feedback on muscle contraction.

Quadriceps srrengthening is an integral component of physical therapy protocols. Quadriceps strength has been shown to be the most positive indicator of long-term patellofemoral pain, so restoring strength and reducing muscle inhibition are essential to recovery. Suter et al. found that correcting sacroiliac dysfunction with manipulation improves quadriceps strengrh (8), so manipulation may be an effective adjunct to strength training by reducing muscle inhibition. Also, recent electromyographic studies demonstrate that exercises targeting VMO recruitment are no more effective than overall quadriceps exercises. Therefore, treatment should instead focus on overall quadriceps strength.

Care is needed to avoid further irritation to the patellar mechanism. Full range of motion open-chain VMO strengthening exercises are common in recreational strength programs despite the excessive load placed on the patella as it articulates with the femoral condyles. To avoid irritating the tracking mechanism, flexion should not exceed 20 degrees, and the knee should be held in full extension 5 seconds per repetition. After this point, the patella slides inferiorly into the trochlea, which can trigger patellofemoral pain.

Surgery is rarely indicated in patellar tracking disorders. Only when all conservative measures are exhausted should surgery be considered. Release of the lateral patellar stabilizers (retinaculum, iliotibial band) relaxes the excessive tightness that alters patellar tilt and glide. Tibial tuberosity transposition is sometimes done with chronic patellar subluxations or intractable maltracking to shift the patella medially so it glides directly superior-inferior between the femoral condyles. Chondroplasty may be performed on clinically evident chondromalacia patellae to improve tracking and glide.

Manual Techniques

Myofascial Release to the Lateral Patella. This technique is applied to release tight fascia and connective tissue, such as the lateral patellar retinaculum, iliotibial band, and hamstrings. The clinician applies force transverse to the direction of the tissue, sustaining pressure on the parella, subluxing medially to laterally. Hold for 5 seconds (Fig. 22.2.1).

Muscle Energy

These techniques resrore range of motion at restricted joint by lengthening the antagonist muscle and resetting the muscle memory.

Hamstring

1. The athlete is supine, with hip flexed to 90 degrees.

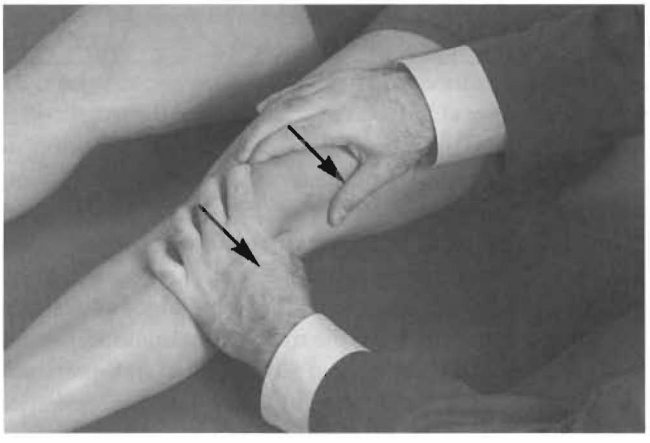

FIGURE 23.3.1. Lateral patellar myofascial release.

2. With the lower leg resting on the clinician's shoulder, the clinician leans in toward the leg with both hands locked on the anterior thigh (Fig. 23.3.2A).
3. The athlete gently flexes the knee against resistance, pushing down on the clinician's shoulder.
4. Rest, reposition, repeat, and reassess.

Anterior Hip Capsule

1. The athlete is prone; the clinician has his or her first hand on the proximal femur just distal to the gluteus maximus, and the other hand cupping the flexed knee from underneath.
2. The clinician lifts the knee off the table, gently introducing hip extension.
3. The athlete pushes the knee gently into the table against the clinician's resistance (Fig. 28.3.2B).
4. Rest, reposition, repeat, and reassess.

Mobilization Variation: Anterior Hip Capsule. The clinician's mobilizing hand introduces a series of eight to ten impulses anteriorly (into the table) against the proximal femur. During the impulses, the clinician internally and externally rotates the femur while varying the impulse direction medially and laterally.

Note: The number of impulses can vary according to the amount of restriction noted by the clinician. After eight to ten impulses, stop and retest, and repeat if necessary.

Supine Muscle Energy Variation for Anterior Hip Capsule

1. The athlete lies in the Thomas test position (see Fig. 22.2.4) with the affected thigh off the table.
2. With the opposite leg held securely by the athlete, the clinician ensures that the pelvis is not tilted posteriorly (if so, relax the hold on the opposite knee to minimize hip flexion).
3. The clinician has one hand on the distal thigh, and the other hand on the opposite leg for stability.
4. The athlete gently flexes the hip against resistance.
5. Rest, reposition, repeat, and reassess.

Variation of Hip Capsule Stretch. After finishing mobilizing impulses, the clinician exerts an anterior stabilizing force to the proximal femur while extending the hip into the restrictive barrier. Hold for 30 seconds.

Quadriceps/Rectus Femoris

1. With the athlete supine, perform the Thomas test (see Fig. 22.2.4) by holding the unaffected knee to the chest, letting the affected leg relax on the table.
2. The dysfunctional rectus femoris restricts passive knee flexion (range should be near 90 degrees).
3. The clinician places one hand superior to the knee for stability, while the other hand

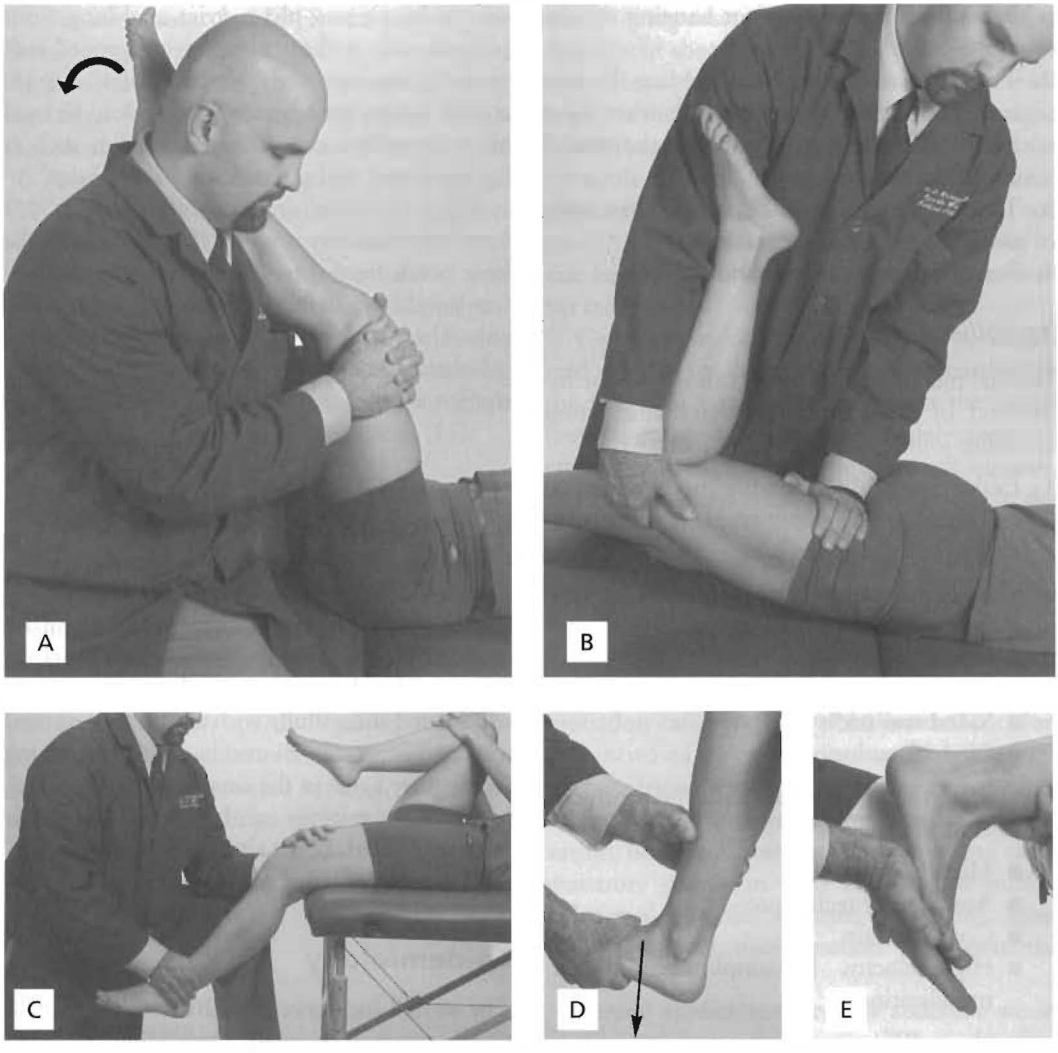

FIGURE 23.3.2. Muscle energy techniques. **A,** Hamstring; **B,** anterior hip capsule; **C,** quadriceps/rectus femoris; **D,** soleus; **E,** gastrocnemius (knee is fully extended).

grabs the tibia at midshaft. *Variation:* The clinician places his or her own foot under the dangling foot of the affected leg, using his or her own tibia to resist the athlete's extension during the technique.

4. The clinician introduces knee flexion until the barrier is met, while the athlete extends the knee gently against resistance (Fig. 23.3.2C).
5. Hold the contraction for 5 seconds, and then rest for 1 second. The clinician repositions the tibia further into the barrier and repeats as necessary.

Calf-Soleus

1. The athlete is sitting while the clinician holds the distal tibia with one hand and the forefoot with the other hand.
2. The clinician dorsiflexes the ankle and asks the athlete to gently plantarflex against resistance (Fig. 23.3.2D).
3. Rest, reposition, repeat, and reassess.

Calf-Gastrocnemius

1. The setup is the same as for the calf-soleus muscle energy technique, but the ath-

lete is prone with the foot hanging off the table.
2. The clinician has one hand holding the foot from the plantar side, while the other hand holds the ankle just proximal to the medial and lateral malleolus.
3. The athlete gently plantarflexes against resistance (Fig. 23.3.2E).
4. Rest, reposition, repeat, and reassess.

Sacroiliac Dysfunction

Manual medicine techniques can reduce the inhibition of knee extension that comes from sacroiliac joint dysfunction and pain (8).

1. Evaluate the athlete during the lumbosacral examination (see Chapter 21.2) for sacroiliac dysfunction. Check for
   - Anteriorly or posteriorly rotated innominate
   - Upslip innominate
   - Sacral shear
   - Sacral torsion
   - L5 dysfunction
2. Correct the dysfunction with the technique of choice:
   - Muscle energy
   - Articulatory techniques
   - Counterstrain
   - High-velocity, low-amplitude (HVLA) mobilization

Stretching

Stretching is the mainstay of the exercise program. Stretches should be performed daily in the morning, and before and after exercise.

1. Quadriceps
2. Iliopsoas
3. Hamstring
4. Iliotibial band
5. Gastrocnemius and soleus
6. Lateral patellar stabilizers

Strength Exercises

Quadriceps weakness is the primary predictor of anterior knee pain, so building quadriceps strength is crucial. Exercises such as sitting wall squats, leg presses, plyometrics, and lunges can be used only if the athlete has progressed successfully through early rehabilitation. For instance, lunges are advanced and should be used after the athlete can do basic exercises such as leg raises and wall squats without difficulty.

Open-chain full range of motion (ROM) knee extension may overload the patella while the knee bends from 0 to 100 degrees, and the athlete should avoid full ROM if it is uncomfortable. The athlete may plateau or regress in physical therapy from this overload, so limit knee motion to no more than 20 degrees of flexion.

ILIOTIBIAL BAND FRICTION SYNDROME

Iliotibial band friction syndrome (ITBFS) is a common cause of lateral knee pain, particularly among runners, military personnel, and cyclists. It is considered an overuse syndrome that is usually treated successfully with a multifaceted conservative approach. Biomechanical and training factors play a role in the development of ITBFS, but its exact etiology can be elusive. Surgery is rarely indicated, but it should be considered after 3 months of failed conservative treatment.

Epidemiology

The overall incidence of ITBFS in the population is not well reported. Depending on which population is examined, the incidence ranges from 1.6% to 52% (9). The incidence varies with the target population's type and intensity of activity. For instance, Linenger and Christensen (10) reported that in 12 weeks of Marine basic training, ITBFS was the most common specific injury and accounted for 22.2% of all lower extremity injuries. Twelve percent of all runners' overuse injuries are caused by ITBFS.

Mechanism

ITBFS typically is observed in vigorous exercisers. Like most overuse conditions, the generated stress breaks down tissue faster than the body can repair it.

The stress point in ITBFS occurs along the lateral femoral epicondyle. During knee flexion, the iliotibial band (ITB) moves posteriorly along the lateral femoral epicondyle. Contact against the epicondyle is highest between 20 degrees and 30 degrees (average 21 degrees), so when the ITB is excessively tight or stressed, it rubs more vigorously.

In runners, friction occurs near or just after foot strike during the contact phase of the gait cycle. Downhill running reduces the knee flexion angle and can aggravate ITBFS, while sprinting and fast running increase the knee flexion angle and are less likely to cause the syndrome (11).

Etiology

Several extrinsic factors cause ITBFS. It is usually caused by overuse, mostly due to errors in training. Single-session errors cause ITBFS as often as reperitive deficiencies. Sudden changes in surface (i.e., soft to hard, flat to uneven, or incline to decline), speed, distance, shoes, or frequency can be culprits in triggering ITBFS. Interval training, crowned surfaces, and excessive cycling during cross-training can also be precipitating factors (9).

Intrinsic factors include

- Leg-length discrepancy
- Genu varum
- Tibia vara
- Overpronation
- Hip abductor weakness
- Myofascial restriction

Studies examining leg-length discrepancies provide conflicting conclusions as to whether a direct correlation exists. Leg-length inequalities do cause changes in hip abduction during the gait cycle, sacral leveling, and pelvic tilt, which is believed to increase tension on the ITB and tensor fasciae latae. However, other studies did not find a direct correlation between length inequality and ITBFS.

From an osteopathic perspective, asymmetrical leg lengths cause the body to make compensatory changes to keep the sacrum balanced. Often, the pelvis compensates first by changes in innominate position or sacral torsion. From there, compensatory patterns can manifest throughout the entire spine. For example, a person whose right leg is longer than the left one by a quarter inch can compensate by using the right innominate to rotate posteriorly, which lifts the acetabulum superiorly and achieves a dynamic shortening of that leg. If the compensation is not enough, the sacrum then is unleveled, leading to side bending at L5 and the beginning of a lumbar curve.

Genu varum, or bowlegged knees, is considered a risk factor due to the increased tension on the ITB as it is stretched more over the lateral femoral epicondyle. This is a widely accepted etiology, although it has little empirical support.

Overpronation is controversial as well. In the running cycle, the lower limb strikes the ground with a rigid supinated foot. As the leg moves forward, the tibia internally rotates over the planted foot, "unlocking" it into a pronated-everted position, which allows for weight bearing. Pronation and internal rotation stress the ITB. Excessive pronation causes quicker tibial internal rotation and increased hip adduction, stressing the ITB over the lateral femoral epicondyle.

James (2) implicated a combination of genu varum, heel varus, forefoot supination, and compensatory pronation with ITBFS, but further videography studies disputed that claim. Most studies support overpronation as a potential cause.

Several studies support hip abductor weakness as an important factor. When the foot strikes the ground, the femur adducts against the eccentric load of the abductors (gluteus medius and tensor fasciae latae). These muscles move from eccentric to concentric through the support phase and into the propulsive phase of gait. The gluteus medius also externally rotates the hip, while the tensor fasciae latae internally rotates the hip.

When the hip abductors are weakened or fatigued, runners have increased adduction and internal rotation at midstance. This generates more valgus force at the knee, which Fredericson et al. postulate as increasing tension and friction on the ITB (13).

Myofascial restrictions and inflexibility can increase stress on the posterior ITB and tensor

fascia latae. Restrictions in hip extension (iliopsoas), flexion (gluteus maximus), and rotators (piriformis, gluteus medius, tensor fasciae latae) shift more load to the abductors and adductors, leading to hip abductor failure.

History and Physical Examination

Pain usually localizes along the lateral knee, but it also can include the hip, particularly over the greater trochanteric bursa. Pain is provoked with downhill running and becomes worse with activity after a pain-free start. Most athletes experience pain only during activities; however, some individuals may experience pain with walking as the syndrome progresses.

The examiner should observe for genu varum (bowleg) as opposed to genu valgum (knock-knee). Observe for any gait abnormalities, such as abnormal adduction of the leg. Tenderness is felt over the lateral femoral condyle, approximately 1 to 2 cm proximal to the lateral joint line. Pain can be reproduced with active flexion-extension of the knee within the first 30 degrees of ROM. Crepitus may be felt. The soft tissue at this point may be boggy and swollen, particularly if there is inflammation in the lateral synovial recess.

When testing range of motion, restriction in hip adduction indicates tightness in the ITB and tensor fasciae latae. Also look for restrictions in the iliopsoas, rectus femoris, gastrocnemius, and soleus. Examination commonly reveals restriction of hip adduction and weakness of the gluteus medius.

Myofascial restrictions can mimic ITBFS, and these restrictions can be identified by a careful examination. Trigger points along the vastus lateralis, biceps femoris, and gluteus minimus can refer pain to the lateral knee, while fascial adhesions of the posterior ITB can generate pain as well. Careful palpation of these trigger points can differentiate ITBFS from myofascial trigger points.

Provocative Tests

Ober's Test. See Chapter 22.2, Hip and Pelvis: Physical Examination (Fig. 22.2.10).

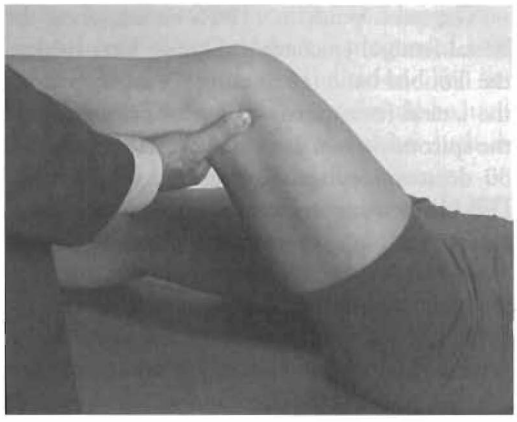

FIGURE 23.3.3. Noble's test.

Positive: Thigh remains suspended off the table.

Indicates: Shortened iliotibial band.

Noble's Test

1. The athlete is supine with the knee at approximately 90 degrees of flexion.
2. The examiner applies firm digital pressure on or around the lateral femoral epicondyle while passively extending the knee (Fig. 23.3.3).

Positive test: Pain at approximately 30 degrees of flexion over the lateral femoral epicondyle, reproduction of symptoms at location.

Indicates: Iliotibial band irritation or inflammation.

Renne's Creak Test. The athlete stands on a step stool and supports all of his or her weight on the affected leg. The clinician's thumb is placed over the lateral femoral condyle, and pressure is applied while the athlete bends the knee into 30 to 40 degrees of flexion. The posterior fibers of the ITB will be directly over the lateral epicondyle (Fig. 23.3.4).

Positive test: Reproduction of pain with pressure.

Indicates: Iliotibial band friction syndrome.

Note: Some literature lists the creak test separately. The creak test is the same as the Renne test, but the clinician's thumb is not placed on

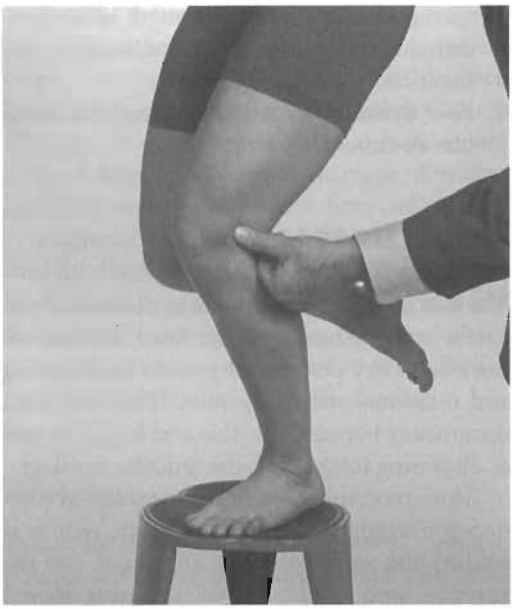

FIGURE 23.3.4. Renne's creak test.

the athlete. Pain when the athlete bends the knee 30 degrees on one leg is a positive test.

Thomas's Test. This test is used to evaluate the iliopsoas, rectus femoris, and tensor fasciae latae and/or ITB. (See Chapter 22.2, Hip and Pelvis:) Physical Examination (Fig. 22.2.4.)

Standard Treatment

Acute treatment includes the following:

1. The RICE protocol (rest, ice, compression, and elevation) should be used immediately to decrease inflammation.
2. Relative rest in runners, that is, reduce mileage by 50%. Most other athletes will improve by ceasing provocative activities.
3. NSAIDs.
4. Corticosteroid injections may be considered. Injections should be superficial or deep to the ITB, not in the ITB tissue.

Treatment for *subacute* ITBS includes several measures.

1. Physiotherapy is effective in improving flexibility, strengthening the hip abductors, and restoring balance to the lower extremity. Slow, progressive, pain-free return to running is essential to recovery. Increasing running too quickly during rehabilitation can flare the injury and create discouraging setbacks.
2. Address biomechanical foot issues. Consider the use of heel lifts, orthotics (prefabricated or custom), metatarsal pads, and stretching programs.
3. Correct environmental and training issues.

Surgical treatment involves posterior resection of the iliotibial band, either in a triangular or elliptical shape, which takes the pressure off of the band that overlies the lateral femoral condylar tubercle (14). Resection of the synovial tissue into the lateral femoral condylar recess is typically not indicated. Surgical treatment is rarely needed with appropriate conservative therapy (14).

Manual Medicine Techniques

Muscle Energy Technique (right hip) for Tensor Fasciae Latae Restriction

Rationale: Treats restricted tissue that limits adduction and increases tension over the greater trochanteric butsa.

1. The athlete is set up in the Ober's test position (see Fig. 22.2.10), with the dysfunctional hip up.
2. The clinician is behind the athlete and brings the leg into the adduction barrier.
3. The athlete contracts against the clinician's and holds for 3 to 5 seconds.
4. Relax, reposition, repeat, and reassess.

Muscle Energy for Restricted Hip Flexion. See Chapter 22.3, Hip and Pelvis: Common Conditions (Fig. 22.3.4A).

Friction Massage to Distal Iliotibial Band

Rationale: Cross-friction massage can loosen restricted tissue, particularly near the friction point at the lateral femoral condyle.

Technique. The clinician uses his or her elbow or fist to put direct pressure on the area of restriction, going against the grain of the tissue (Fig. 23.3.5). The elbow or fist can be held for

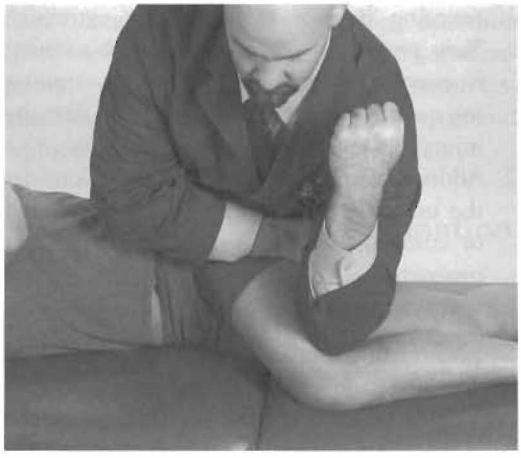

FIGURE 23.3.5. Friction massage to the distal iliotibial band.

90 seconds and turned into a counterstrain technique, as long as the leg is moved into a position of ease.

Prevention

Strength Training

1. Tensor fasciae latae
2. Gluteus medius

Important Stretches

1. Hip adductors
2. Hip abductors
3. Iliopsoas and quadriceps

Preventing injury also requires adjustments to training to avoid many of the factors discussed earlier. Intrinsic factors can be helped with an exercise program, proper shoes with pronation control, heel lifts, and manipulation. Exercises are especially important if a short leg syndrome is identified, since muscular shortening and restriction are likely to be found.

Areas to Check and Address

1. Restricted ankle dorsiflexion, caused by subtalar dysfunction or an equinus ankle.
2. Restricted hip extension, caused by tight iliopsoas and quadriceps muscles, or a posteriorly rotated innominate.
3. Compensation from leg-length discrepancies, including lumbar and sacroiliac dysfunction.
4. Foot dysfunctions, such as a dropped navicular or cuboid.

MENISCUS TEARS

The two semilunar menisci lie in the medial and lateral compartments of the knee attached to the tibia. They perform important load-bearing and rotational stabilizing roles. They allow for congruency between the tibia and femur, as well as dispersing force across the articular cartilage.

Motion occurs through the meniscus in rotation and weight bearing. For instance, Vedi, et al studied the weight-bearing knee, and saw the anterior horn of the medial meniscus moved through a mean of 7.1 mm and the posterior horn through 3.9 mm, and there was 3.6 mm of mediolateral radial displacement. In the lateral meniscus, the anterior horn moved 9.5 mm and the posterior horn moved 5.6 mm, and there was 3.7 mm of radial displacement (15).

Acute meniscus tears usually occur in the flexed weight-bearing knee while absorbing a high-velocity rotational force. The medial meniscus is three times more likely to tear than the lateral meniscus. Several types of tears are commonly seen: (a) bucket-handle, (b) longitudinal, (c) horizontal or cleavage, (d) radial, and (e) parrot beak. Other variations, such as a flap or oblique tear, are seen.

Meniscus tears also occur from chronic degeneration and wear, coupled with cartilaginous aging changes. These menisci are less hydrated, more brittle, and more easily torn. Often, small and complex tears accompany osteoathritic bony changes such as subchondral sclerosis, cortical irregularity, osteophyte formation, and bone remodeling.

Common symptoms seen with meniscus tears include joint line pain, giving way, pain with extension or flexion, and locking. Longitudinal tears most commonly cause locking, for the free flap of cartilage torn away from the body or horn can flip up into the joint space and mechanically block femoral movement on the tibia.

Physical examination reveals limited range of motion in either end-range flexion or extension, often causing sharp pain. The bounce, McMurray's, and Apley's grind tests (see Chapter 23.2) are designed to provoke pain from meniscus pathology. In a locked knee, range of motion is limited, as the new barrier is firm and painful.

Magnetic resonance imaging is the gold standard for diagnosing meniscus tears. Accuracy can be enhanced by gadolinium contrast injected into the joint. Ultrasonography can detect peripheral tears, but accuracy is limited by its unavailability to detect more internal meniscal anatomy.

Athletes

Except for direct trauma to the knee, meniscus injuries are common in any athlete who is required to perform sudden changes of direction or generate force from the lower extremities in a nonlinear direction. This includes such contact sports as football, soccer, baseball, and basketball. The repetitive loading and twisting mechanism of the meniscus such as used in football and basketball, can lead to gradual degenerative changes along the meniscus.

Treatment

Acute meniscus tears can lead to instability, locking, catching, and giving way. Some are small enough to be left alone, but when mechanical symptoms occur, arthroscopic repair is the preferred treatment of choice, depending on the specifics of the tear. The prognosis improves if the tear lies in the vascular outer one third of the meniscus as opposed to the relatively avascular inner two thirds. Often the fragment is shaved down or resected. Occasionally, a flap of meniscus can be anchored back down onto the tibia.

For degenerative tears, conservative treatment is the mainstay of management. This includes physical therapy to strengthen muscles supporting the knee, NSAIDs and corticosteroid injections, and bracing, particularly if there is evidence of unilateral joint compartment collapse.

There is some controversy as to whether arthroscopic débridement of degenerative tears in osteoarthritis, often referred to as a "wash-out" procedure, is effective (16,17). It is generally accepted that a mildly arthritic knee with mechanical symptoms such as locking, catching, or giving way may be suitable for arthroscopy if conservative therapy has failed.

Manual Medicine Techniques

Articulatory Technique

Rationale: A flap of cartilage from a torn meniscus can "lock" the knee if it flips up into the joint space, blocking full range of motion. Acutely, this can be debilitating and painful. Articulatory technique can be used in this situation to help relocate the flap tear back into its original position, or at least out of the way of tibiofemoral motion.

For a Medial Meniscus Tear

1. The athlete is supine while the clinician holds the distal leg between his or her chest and upper arm and with the hands holding the knee. Both thumbs are on the medial joint line.
2. The clinician flexes the knee slightly while the lateral hand compresses the distal femur medially, gapping the medial joint.

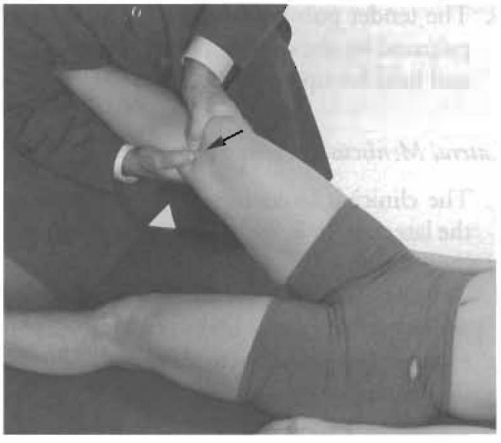

FIGURE 23.3.6. Articulatory technique for treating a displaced medial meniscus tear.

FIGURE 23.3.7. Counterstrain to the lateral meniscus or hamstring.

3. The clinician keeps his or her thumbs on the medial joint line, pushing posterolaterally (Fig. 23.3.6).
4. The athlete's leg is then moved into extension.
5. The manipulation may need to be repeated.

Counterstrain

Medial Meniscus

1. The athlete is supine with the affected leg hanging down in about 60 degrees of flexion.
2. The clinician uses the stabilizing hand to hold the ankle, slightly adducting and internally rotating the tibia so the ankle is almost under the table.
3. The tender point on the medial joint line is palpated by the clinician's other index finger and held for up to 90 seconds (17).

Lateral Meniscus or Hamstring

1. The clinician looks for the tender point on the lateral joint line or near the biceps femoris insertion.
2. The clinician holds the ankle with one hand, while applying slight abduction and external rotation (this requires monitoring and a feel for the tension of the knee).
3. The clinician puts the other thumb or index finger on the tender point and holds for up to 90 seconds (Fig. 23.3.7).

STRETCHES FOR THE KNEE

Most of the stretches that impact the knee are the same as stretches for the hip and pelvis (see Chapter 22.3, Stretches for the Hip and Pelvis), since most of the mobilizers of the knee originate from there. These include the following:

1. Quadriceps (hip and pelvis) (see Fig. 22.3.11H).
2. Hamstrings (hip and pelvis) (see Fig. 22.3.11A).
3. Gastrocnemius (foot and ankle) (see Fig. 24.3.11A).
4. Soleus (foot and ankle) (see Fig. 24.3.11B).
5. Hip flexots (lumbosacral spine) (see Fig. 21.3.16C).
6. Hip abductors (hip and pelvis)
7. Hip adductors (hip and pelvis) (see Fig. 22.3.11E).
8. Gluteals (hip and pelvis) (see Fig. 22.3.11B and D).

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24

FOOT AND ANKLE

24.1

Anatomy

GAIL A. SHAFER-CRANE
WILLIAM M. FALLS

SKELETAL

Three bones participate in the ankle joint: the tibia and fibula of the leg, and the talus bone, a tarsal bone. The tibia widens distally, forming the medial malleolus, while the distal fibula also enlarges to form the lateral malleolus. The tibia and fibula articulate distally in the distal tibiofibular joint. The inferior surface is a deep concave, articular surface that becomes the mortise of the ankle joint. The medial and lateral malleoli are bony landmarks that are easily palpated, and provide reference points for palpation of the rest of the ankle. The wedge-shaped rounded trochlea (Latin, pulley) of the talus fits within the mortise formed by the malleoli, creating a synovial hinge joint (1–6).

Osteology of the foot includes the tarsal bones, talus, calcaneus, navicular, cuboid, three cuneiform bones, five metatarsal bones, and the phalanges. (Fig. 24.1.1). The transverse talar joint is formed by the anterior articulation of the calcaneus and cuboid bones and the talus and navicular bones. The subtalar joint is between the talus and calcaneus. The tarsometatarsal joint is between the cuneiform bones, the cuboid bone, and the metatarsals. The five metatarsals articulate with the proximal phalanges forming the metatarsophalangeal joints. The joints of the toes are the proximal and distal interphalangeal joints.

The articulations of the tatsal and metatarsal bones and their ligaments form the transverse and longitudinal arches of the foot. The cuboid and cuneiform bones and the metatarsal bases form the transverse arch. The calcaneus, talus, navicular, three cuneiforms, and medial three metatarsal bones form the medial longitudinal arch; and the calcaneus, cuboid, and lateral two metatarsals form the lateral longitudinal arch. These arches permit significant shock absorption by flattening out and springing back during loading of the foot.

The distal tibiofibular joint is felt by placing the fingers anterior to the lateral malleolus, thumb posterior, and gently pinching. The fibula can be mobilized in an oblique anterior-posterior direction. The anterior ankle joint can be felt as a depression between the tendons as they cross the ankle. The talar trochlea is palpated by applying pressure, gently, in this depression as the foot is extended. The prominence formed by the head of the second metatarsal as it articulates with the second cuneiform bone lies just lateral to the long medial extensor tendon, extensor hallucis longus, and deep to the dorsalis pedis artery. The tuberosity of the fifth metatarsal is palpated along the lateral border of the foot.

JOINTS

The distal tibiofibular joint is a fibrous joint, or syndesmosis. Very little mobility is present at this joint; however, as the foot is dorsiflexed,

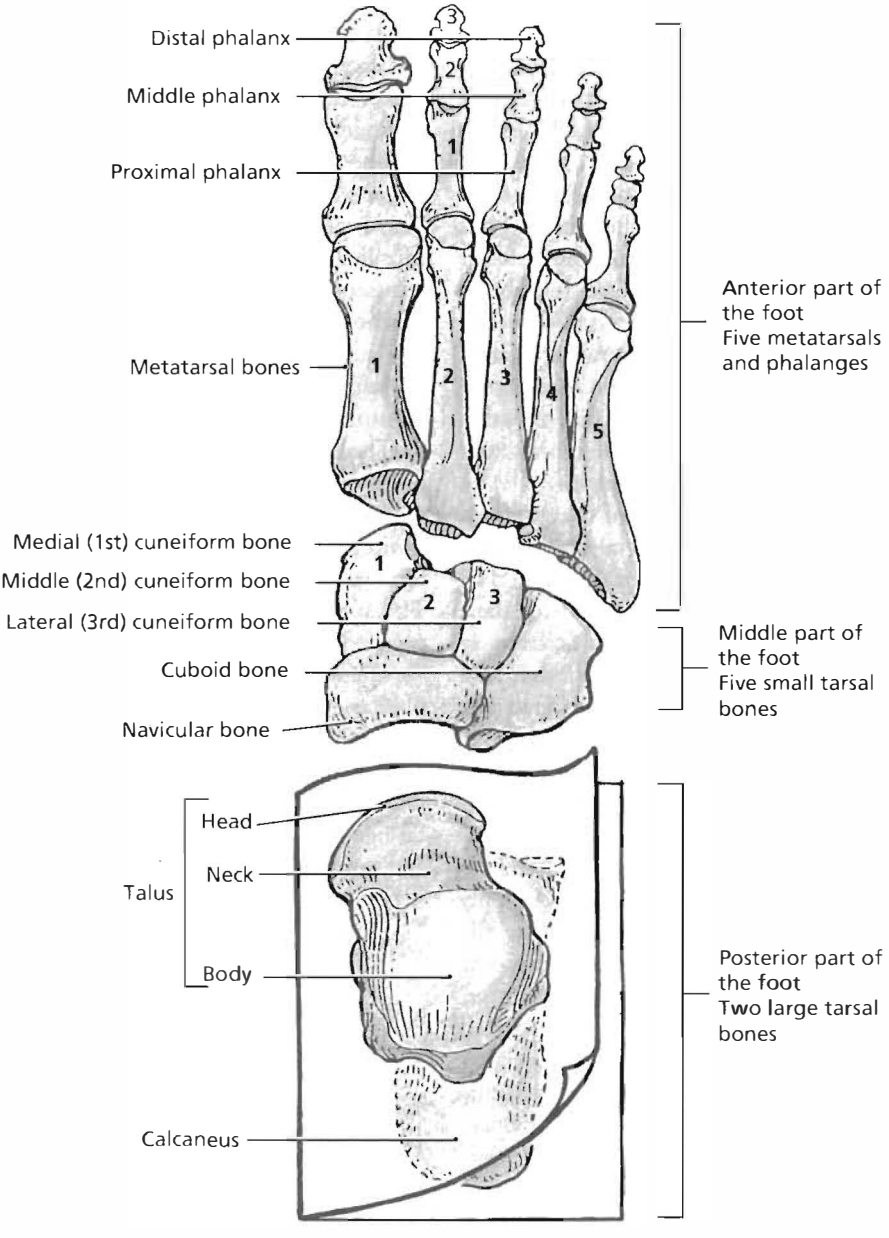

FIGURE 24.1.1. Bony anatomy of the foot. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy*, 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

the wedge-shaped talus exerts enough pressure to open this joint slightly. For this reason, the ankle joint is more stable in dorsiflexion than in volar flexion.

The interosseous membrane is a tough connective tissue sheet between the tibia and fibula. This membrane separates the leg into the anterior and posterior compartments, and then continues to become the interosseous ligament of the distal tibiofibular joint. Anterior and posterior inferior tibiofibular ligaments provide further reinforcement.

The tibiotalar joint is the mortise joint, formed by the distal tibiofibular joint as it articulates with the trochlea of the talus. Plantarflexion (extension) of the ankle is permitted as the talus rocks anteriorly, and dorsiflexion (flexion) occurs as the talar trochlea rocks posteriorly, slightly separating the distal tibiofibular joint. The posterior wall of the mortise joint is formed by a continuation of the inferiot tibiofibular ligament, the transverse tibiofibular ligament (Fig. 24.1.2). Blood supply to the tibiotalar joint is from the perforating branch of the fibular artery, and from the medial and malleolar branches of the anterior and posterior tibial arteries. The deep fibular, tibial, and saphenous nerves provide innervation.

There are three articulations of the proximal tarsals. The transverse tarsal or talocalcaneonavicular is a synovial ball-and-socket joint. The subtalar and calcaneocuboid joints are plane synovial-type joints. These joints collectively permit inversion and eversion of the foot through gliding and rotational motions between the tarsals. Intertarsal ligaments are named for their proximal and distal attachments to the tarsals. The spring, or talocalcaneonavicular, ligament is an important structure because it supports the transverse arch of the foot. These joints receive their blood supply from the posterior and anterior tibial, posterior fibular, and lateral tarsal arteries. The medial and lateral plantar nerves innervate the plantar aspect of these joints, while the deep fibular nerve innervates the dorsum.

The distal tarsals articulate with the bases of the metatarsals forming tarsometatarsal joints. These plane joints are synovial with fibrous capsules formed by the dorsal, plantar, and interosseous ligaments. Limited gliding between the bones is permitted. Blood supply is from the lateral tarsal artery, which is a branch of the dorsalis pedis. The deep fibular, lateral plantar, and sural nerves provide innervation.

The bases of the metatarsals articulate with one another in the intermetatarsal joints. These are plane-type synovial joints, which permit limited gliding. The dorsal, plantar, and interosseous ligaments form the fibrous capsules. The lateral tarsal artery provides the blood supply, and the innervation is by the digital nerves.

The metatarsals are long bones that form the plantar surface of the foot. Their heads articulate with the proximal phalanges in the metatarsophalangeal joints. These plane-type synovial joints allow limited gliding. Collateral and plantar ligaments form the fibrous capsules. The lateral tarsal artery provides the blood supply. Innervation is by the digital nerves.

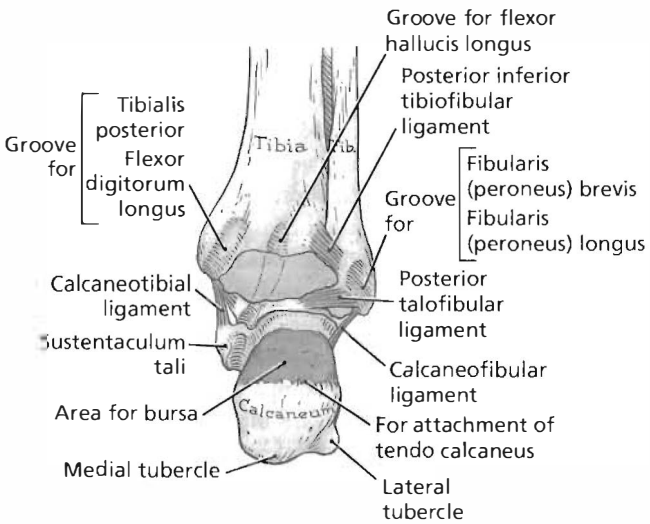

FIGURE 24.1.2. Posterior ankle joint. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy*, 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

LIGAMENTS

The lateral ankle is stabilized by three ligaments. The anterior talofibular ligament attaches the lateral malleous to the neck of the talus. The posterior talofibular ligament attaches the malleolar fossa to the lateral tubercle of the talus. The calcaneofibular ligament is a round cord that attaches the tip of the lateral malleolus to the lateral calcaneus.

The medial deltoid ligament has three components, the tibionavicular, anterior and posterior tibiotalar, and tibiocalcaneal ligaments. The apex of this triangular ligament makes its proximal attachment at the medial malleolus, and then fans out to attach to the navicular, talus, and calcaneus tarsal bones (Fig. 24.1.3A-B). This ligament supports the ankle during eversion, acts to prevent subluxation, and assists in maintaining the medial longitudinal arch of the foot. Blood supply to the tibiotalar joint is from the malleolar branches of the fibular and anterior and posterior tibial arteries. The deep fibular and tibial nerves innervate this joint.

TOES

The bones of the toes are the proximal, middle, and distal phalangeal bones. The great toe has only two phalanges, while the others have three. The interphalangeal joints are hinge-type synovial joints that allow flexion and extension. The collateral and plantar ligaments form the fibrous capsules. The superficial fibular nerve forms the digital nerve. These provide cutaneous innervation to the skin of the dorsum of the foot, and all toes except the lateral surface of the little toe and the adjacent surfaces of the great toe and the second toe. Superficial branches of the lateral and medial plantar nerves, which are continuations of the posterior tibial nerve, supply the plantar surfaces of the toes, and intrinsic muscles of the foot.

MUSCLES

Muscles of the ankle and foot are divided into extrinsic and intrinsic muscles. The extrinsic ankle dorsiflexor and toe extensor muscles are housed in the anterior compartment of the leg. The tibialis anterior muscle makes its proximal attachment on the lateral tibial condyle and superior lateral surface of the tibia and interosseous membrane. It narrows into a long tendon that traverses the ankle beneath the extensor retinaculum to attach on the medial and inferior surfaces of the medial cuneiform and base of the first metatarsal. Innervation is the deep fibular nerve (L4-L5). The extensor digitorum longus muscle runs just lateral to the tibialis anterior. Its proximal attachment is to the lateral tibial condyle, and proximal three fourths of the interosseus membrane and fibula. Its long tendon attaches to the middle and distal phalanges of the lateral four toes. The extensor hallucis longus attaches proximally to the middle of the fibula and interosseous membrane. The long tendon attaches to the distal phalange of the great toe. The fibularis tertius is a short muscle that makes its proximal attachment to the distal fourth of the interosseous membrane and fibula, crosses the ankle beneath the extensor retinaculum, and then attaches to the dorsum of the fifth metatarsal. The deep fibular nerve (L5-S1) innervates these three muscles.

These ankle dorsiflexor and toe extensor muscles pass beneath the extensor retinacula of the ankle. These structures are thickenings of the deep fascia of the leg. The superior retinaculum is a Z-shaped structure arising from the medial tibia and medial malleolus and crossing over the extensor tendons to attach on the fibula superior to the lateral malleolus and the cuboid bone. The inferior extensor retinaculum arises from the cuboid, and then crosses from lateral to medial over the long extensor tendons to attach to the first cuneiform bone. A second component arises from the first cuneiform bone, and crosses from medial to lateral to attach to the second metatarsal. Each extensor tendon is housed within a synovial sheath formed as the tendon emerges from the muscle and extending distal to the retinacula. These fluid-filled sacs provide lubrication and fluid pressure to limit impingement by the retinacula.

Plantarflexors of the ankle and flexors of the toes are housed in the posteriot compartments

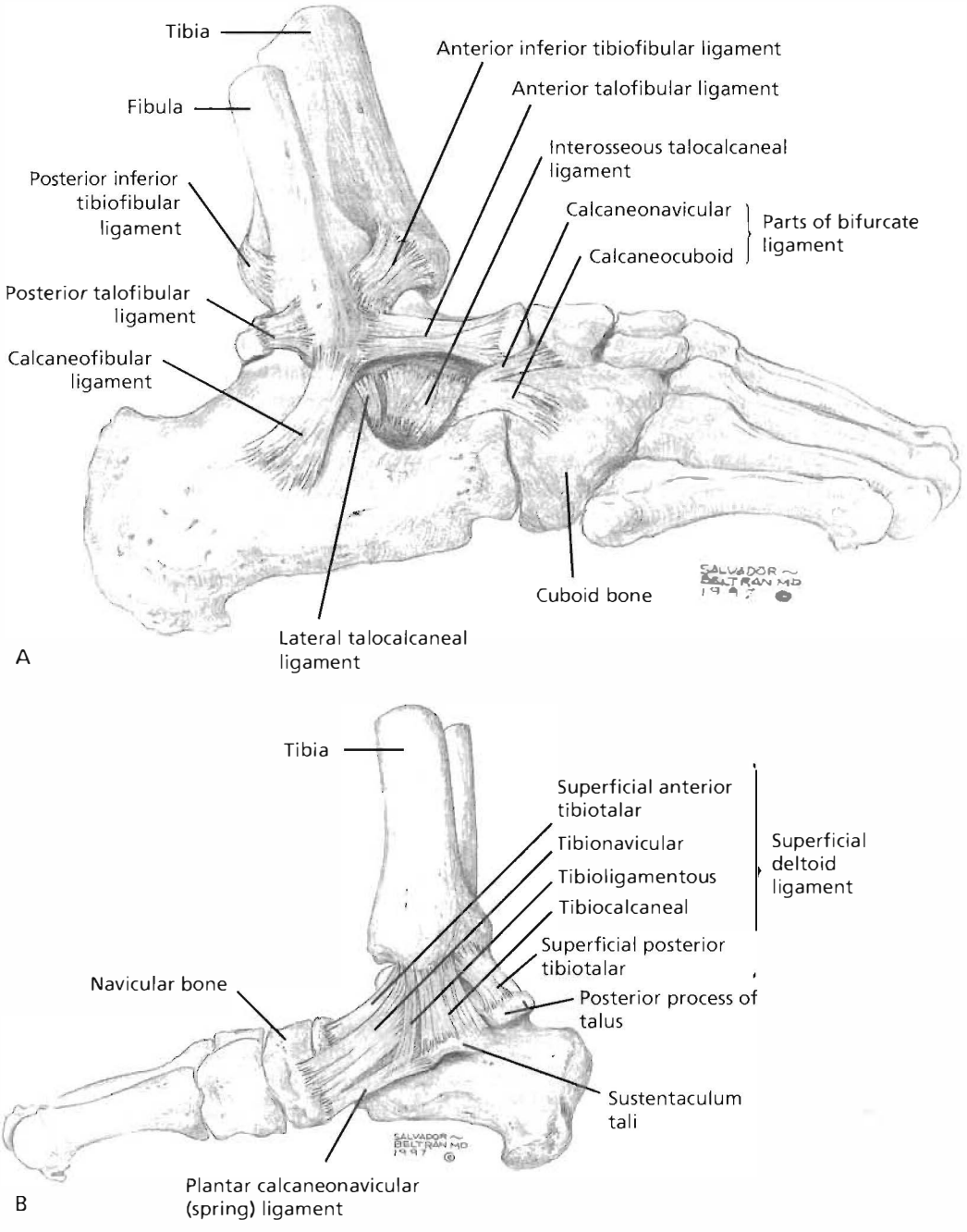

FIGURE 24.1.3. Lateral **(A)** and medial **(B)** ligaments of the ankle. (From Stoller DW. *MRI, arthroscopy, and surgical anatomy of the joints.* Baltimore: Lippincott Williams & Wilkins, 1999.)

of the leg. The plantarflexors are within the superficial posterior compartment. All of these muscles are innervated by the tibial nerve (S1-S2) and insert into the posterior surface of the calcaneus bone. The gastrocnemius splits forming the inferior borders of the popliteal fossa at the knee. The medial head attaches to the popliteal surface of the femur and just superior to the medial femoral condyle. The lateral head attaches to the lateral femoral condyle. The action of the gastrocnemius depends on the position of the knee. It plantarflexes the foot during knee extension to raise the heel during ambulation and is also a secondary knee flexor. The soleus muscle lies beneath the gastrocnemius within the superficial dorsal compartment of

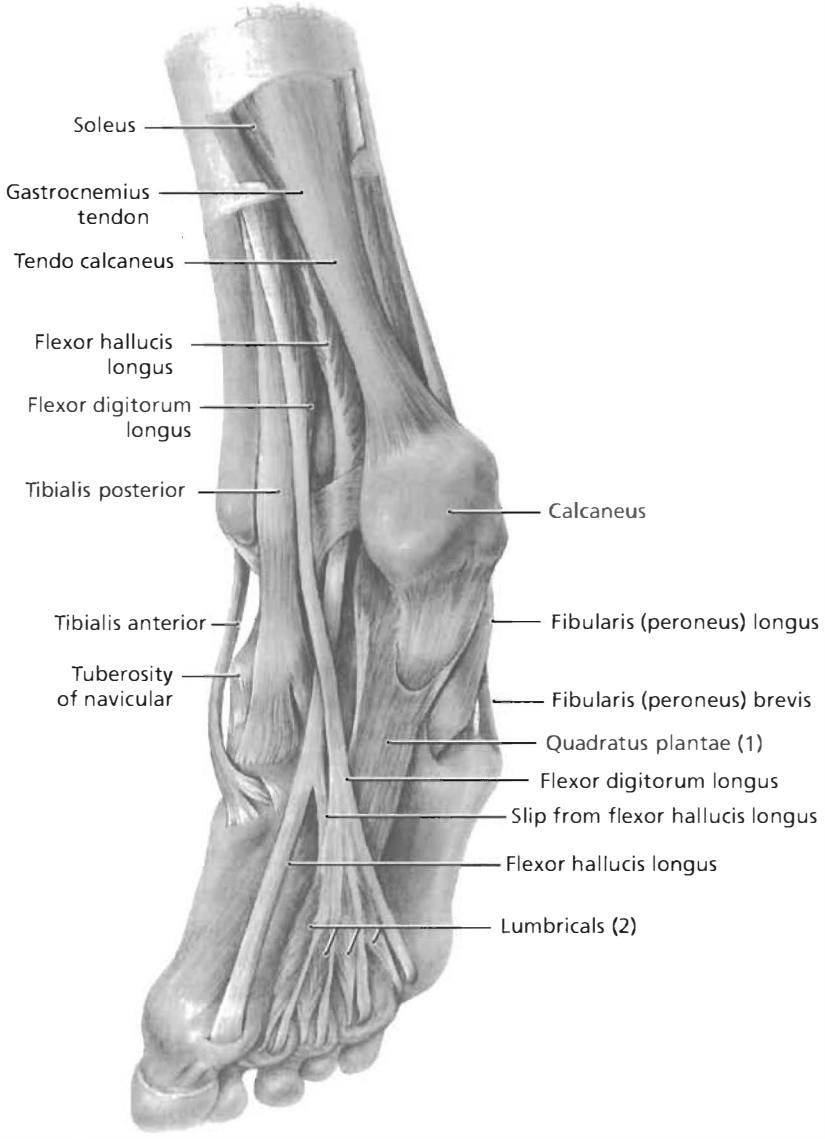

FIGURE 24.1.4. Muscles and tendons in the sole of the foot. (From Agur AMR, Lee ML. *Grant's Atlas of Anatomy*, 10th ed. Baltimore: Lippincott Williams & Wilkins, 1999.)

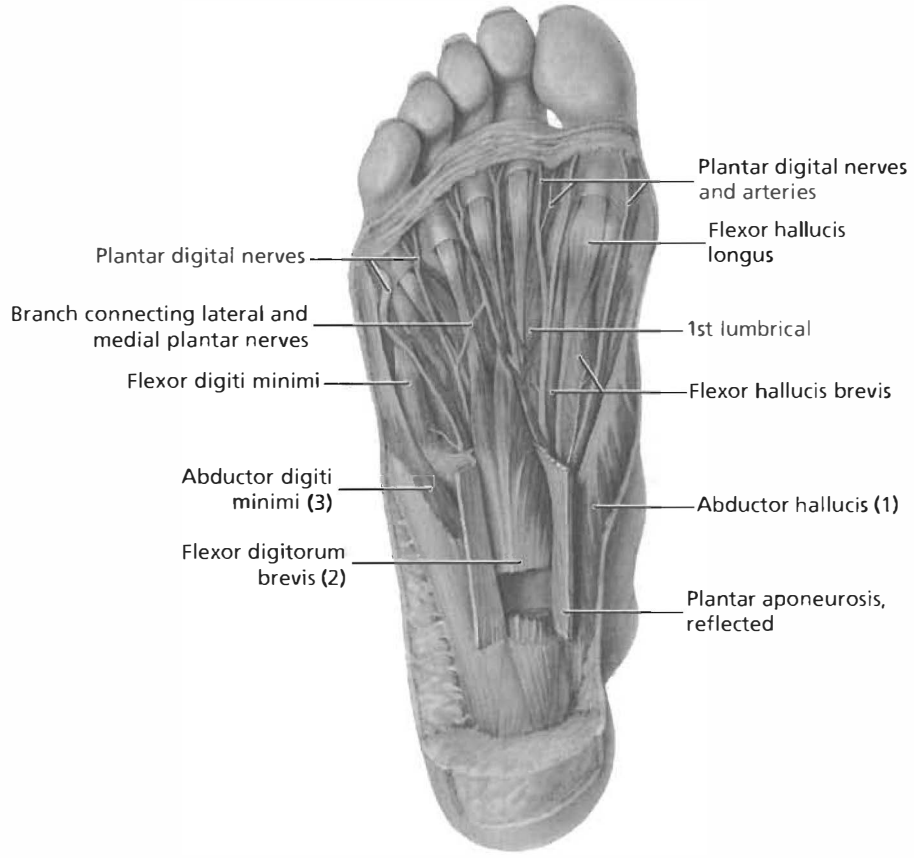

FIGURE 24.1.4. (*continued*)

the leg, plantarflexes the ankle, but acts independently from the knee. The plantaris muscle lies between the gastrocnemius and soleus. It is a very thin, long, triangular-shaped muscle that terminates into a very thin, cordlike tendon. Its proximal attachment is above the knee at the lateral supracondylar line of the femur and into the oblique popliteal ligament.

The toe flexors occupy the deep posterior compartment of the leg. The flexor hallucis longus (FHL) attaches proximally to the distal two thirds of the fibula, and distally to the base of the distal phalanx of the great toe. The flexor digitorum longus (FDL) lies medial to the FHL. It attaches proximally to the middle posterior surface of the tibia distal to the attachment of the soleus, and to the fibula by a broad tendon. Its long tendon passes beneath the tendon of its medial neighbor, the tibialis posterior before entering the flexor retinaculum and inserting onto the bases of the distal phalanges of the lateral four toes. The tibial nerve (S2-S3) innervates the FHL and FDL.

The tibialis posterior is the most medial of these three deep muscles, arising from the interosseous membrane, the posterior surface of the tibia distal to the origin of the soleus muscle, and the posterior surface of the fibula. It crosses the ankle beneath the flexor retinaculum between the tendons of the FDL and FHL before inserting on the tuberosity of the navicular, cuneiform, and cuboid bones and onto the bases of the middle three metatarsals. The tibial nerve (L4-L5) innervates the tibialis posterior.

The tarsal tunnel is an arch formed by the medial malleolus and calcaneus bones as its floor, and the flexor retinaculum as its roof. The long tendons of the FDL, tibialis posterior, and FHL traverse the ankle with the posterior tibial artery and the tibial nerve. The tendons within this compartment are housed within synovial sheaths.

Two muscles, the fibularis (peroneus) longus and brevis, evert the foot and weakly plantarflex the ankle. They are extrinsic muscles housed within the lateral compartment of the leg. Their proximal attachments are the head and superior and inferior two thirds of the lateral fibula, respectively. The fibular nerve (L5-S2) innervates these extrinsic muscles.

The intrinsic muscles of the foot make their proximal and distal attachments distal to the ankle. The plantar surface of the foot is organized in four layers (Fig. 24.1.4). The intrinsic muscles and the tendons of the extrinsic muscles are interwoven through these layers. Synovial sheaths encase the flexor and extensor tendons of the toes. Sesamoid bones are carried in the flexor tendons of the great toe.

Innervation of the sole of the foot is by the medial and lateral plantar nerves, which branch into deep and superficial branches. Their terminal branches, the plantar digital nerves, supply the toes.

The skin of the plantar surface of the foot is very thick. Just deep to the skin is a tough, thick layer of plantar fascia. The plantar aponeurosis is a deeper layer of connective tissue with longitudinal fibers running from the medial calcaneus to the distal phalanges.

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24.2 Physical Examination

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FOOTWEAR

Any athlete who has a foot and ankle problem should always bring his or her footwear into the examination as well. Shoe defects are recognized as a common source of foot injury (1). Overall wear should be initially assessed, and though many guides exist for how long an athlete can use a shoe, each situation must be evaluated individually. Injury prevention starts with assessing the type and quality of shoes (2). Heavy runners will typically wear down a shoe faster than a lighter runner, but a basketball player may not wear shoes as hard as a competitive tennis player. Shoes can lose more than 40% of their shock-absorbing capacity after 250 to 500 miles. The rule of thumb is to change shoes every 6 months. This will vary with the sport and usage, so knowing the essentials of footwear will help a clinician to

determine the appropriate type of shoe and when to replace it.

Uppers

Uppers consist of the material attached to the sole that encloses the foot. The material is typically much thinner than cross-training shoes to allow for evaporation of sweat and a lighter shoe. Look at the medial portion and see if the athlete's foot hangs over the sole. A curved (adductus) foot in a straight shoe may not fit properly and lead to problems. An overpronating foot will compress the arch and break it down faster. This problem is worse in a curved shoe, so most overpronators do best in a straight shoe with motion control and firm midsole support (Fig. 24.2.1). Conversely, a straight foot in a curved shoe may have excess bulging off the lateral aspect, leading to more pressure on the little toe and fifth metatarsophalangeal (MTP) joint. Look for abnormal friction or peel-away from the sole everywhere. Excessive wear on an upper may indicate contact with the opposite foot, which can result from a narrow base, crossing over of the other foot, an uncompensated equinus contracture, or hamstring restriction.

Counter

The counter is the hindfoot of the shoe. It attaches to the uppers and embeds its base into the rear of the sole. Unlike the uppers, the counter is stiff and designed to limit mobility. Look for the alignment or tilt and note if it tilts medially (pronation) or laterally (supination). Note any wear on the inside of the counter, which can be caused by excessive heel motion in a shoe too big (Fig. 24.2.2).

Check the counter itself. It should be firm and minimally mobile. A mobile counter is a sign of a cheap shoe with excessive subtalar and talar motion. Particularly in runners, shoes need to minimize talar motion and exhibit more control of the talus; otherwise, the eccentric load on the Achilles tendon, posterior tibialis, and peroneus muscles will increase, leading to mechanical fatigue and breakdown.

The portion of the outsole under the counter is usually constructed with a gel pocket or air cushion inside the sole. This should be given special attention during the examination. Defects in this structure, such as pad rupture, deflation, or fracturing, can alter talar mechanics during heel strike and lead to multiple problems. Make sure each pocket is inflated evenly by comparing the two shoes side by side. Push down on the counter to watch the pocket compress. Any significant asymmetry should be a signal to replace the shoes. Rock the shoe on the counter back and forth, noting any excessive or asymmetrical roll (1).

FIGURE 24.2.1. Anatomy of the running shoe.

FIGURE 24.2.2. The counter.

Insole

This removable portion of the shoe is extremely variable and interchangeable. The quality of insoles has improved over the years for shoes that demand more shock absorption and support. However, a broken-down insole does little for the athlete, and its condition should be evaluated at the time of any foot and ankle injuty. Note the elastic energy-absorbing properties of the forefoot and hindfoot, as well as the medial support along the arch. Many times, a shoe that claims to be motion control has only a built-up medial insole that breaks down after mild wear.

Examination of the insole can shed light on foot mechanics. Look at the impression on the sole and see how centered it is. A lateral impression implies a foot too rectus (straight) for the shoe, while a medial impression suggests an adductus (curved) foot. Note abnormal wear patterns, specifically at the MTP joints. The deepest impression should be near the first MTP, through which almost 60% of the body weight is transferred. Deeper impressions elsewhere may indicate abnormal weight distribution from poor foot biomechanics or other structural problems (equinus, pes cavus or planus).

Midsole

The midsole is between the forefoot and the counter/hindfoot. Its primary job is to provide stability and cushion. Take the shoe with one hand on the counter and the other on the forefoot. Twist the shoe forefoot clockwise, hindfoot counterclockwise and then the other way and look for excessive motion. A strong midsole should be stiff and resist twisting. A weak midsole, by design or breakdown, causes the counter to excessively invert and evert.

The material that makes up the midsole affects stability and shock absorption. A denser midsole gives stability and support, while a lower-density midsole absorbs more shock. Again, the athlete should define his or her needs, and then choose the shoe and the material appropriately. Some shoes are made with little midfoot support, so athletes should be educated about what they need from shoes. The wrong shoe can lead to needless injury.

Outsole

The outsole is the tread of the shoe that holds the athlete to the ground (Fig. 24.2.3). Many types of outsoles exist, so the athlete needs to choose the appropriate one for the playing surface. For instance, waffle patterns on the outsole work better on softer surfaces, so this type of shoe on a harder surface like concrete would wear down much faster. The types of material used in outsoles differ as well. Carbon outsoles are harder and more durable, while "blown" rubber is softer and more giving, yet less durable.

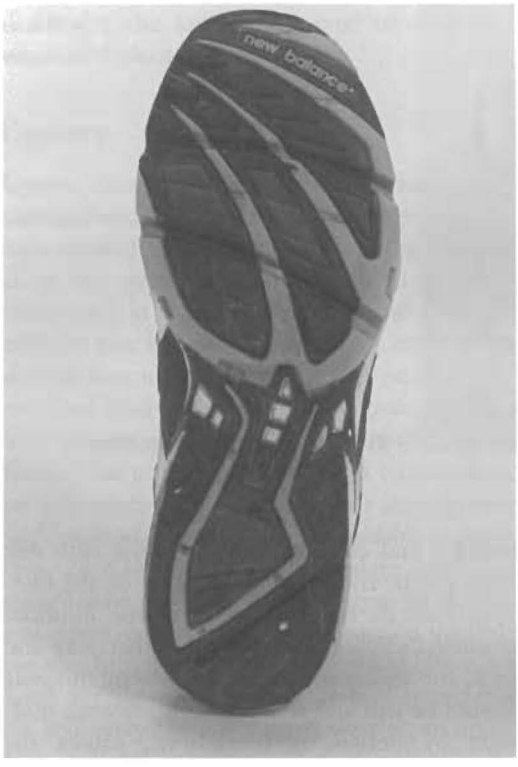

FIGURE 24.2.3. Outsole.

Note the type of outsole the athlete wears and consider the desired activities.

Look for wear on the outsole, including the pattern, amount, and its distribution. Specific areas of wear may lead to problems. For instance, heel wear is typically seen lateral to the midline. This is not abnormal, but if the heel wear is excessive, the counter may evert too much during gait, destabilizing the ankle and stressing the supportive soft tissue. Excess toe wear can be seen on the outsoles of sprinters, but it may also correlate with a possible equinus contracture.

Wear patterns can identify flaws in biomechanics. A worn lateral outsole indicates that the foot stays inverted and supinated during gait. It may also indicate a narrow base, where the feet are too close during gait, or hindfoot varus. On the other hand, excessive medial outsole wear comes from a heavy pronator with a widened base of gait. The medial shoe will typically collapse quicker, leading to abnormal wear. Asymmetrical wear between the left and right shoes may indicate a leg-length discrepancy.

The outsole itself may be the primary source of injury. The waffle tread on some insoles may have a prominent stud located under a sesamoid or a sensitive part of the foot, focusing enough pressure to inflame the foot. Structural flaws can form from even minimal use or may be present at purchase.

FOOT AND ANKLE OBSERVATION

Look first at the athlete's posture while standing and perform a static assessment. Note asymmetries in size, swelling, skin coloring, and shape. Make sure the bony and tendon landmarks are visible and within normal limits.

Look for any skin lesions, such as tinea pedis, nevi, and onychomycosis. Discoloration at the distal toes may indicate ill-fitting shoes. If the athlete is diabetic, the foot must always be examined at every physician visit and checked for discolored lesions and circulation.

Alignment is crucial. Note deviations in the toes and contact points among them. "Hammer toes" can be painful and affect performance, so early detection is helpful. Hallux adductus, especially in the first MTP, leads to bunion formation. Note the callus pattern, for it indicates weight distribution. Skin changes along the medial first MTP joint may signify a friction point and the beginning of a bunion.

Note the hindfoot and its position in reference to the forefoot. The heel may move into valgus or varus upon stance. The arch may be high (cavus) with a rigid supinated foot, or low (planus) with a flexible pronated foot. The forefoot may be rectus (straight) or adductus (curved), which can play a factor in injury if the athlete wears wrong-fitting shoes.

To observe pronation, have the athlete walk away and toward you without socks, shoes, and long pants. Look for depression or collapsing of the arch, rotation of the talus, and shifting of the calcaneus into valgus or varus alignment. More discussion on gait analysis can be found in Chapter 25.

PALPATION

Note the bony landmarks and assess for tissue texture changes and tenderness. Progress systematically, from distal to proximal, or vice versa. Structures to always include in the palpatory examination are the medial and lateral malleoli, calcaneus, fifth metatarsal base, first MTP joint, and the medial plantar fascia insertion on the calcaneus.

In the forefoot, palpate the MTP joints and the distal interphalangeal and proximal interphalangeal joints, noting swelling and tenderness. Palpate along the shafts of the metatarsals and their interspaces. Fullness and tenderness between the third and fourth metatarsal shafts may indicate Morton's neuroma.

Midfoot sprains can be debilitating and lead to long-term consequences, so palpate the first and second metatarso-cuneiform joints, noting swelling, bogginess, and tenderness on both the dorsal and plantar sides. Moving more proximally, palpate the calcaneocuboid and talonavicular joints. Also find the tarsal navicular tubercle, as the posterior tibialis tendon inserts there.

Note the sinus tarsi location and check for tenderness (Fig. 24.2.4). Sinus tarsi syndrome can be overlooked if the examination is incomplete. Areas of possible impingement are the region anterolateral to the lateral malleolus and the anteromedial talus region (Fig. 24.2.5). Palpate these areas for tenderness or tissue changes.

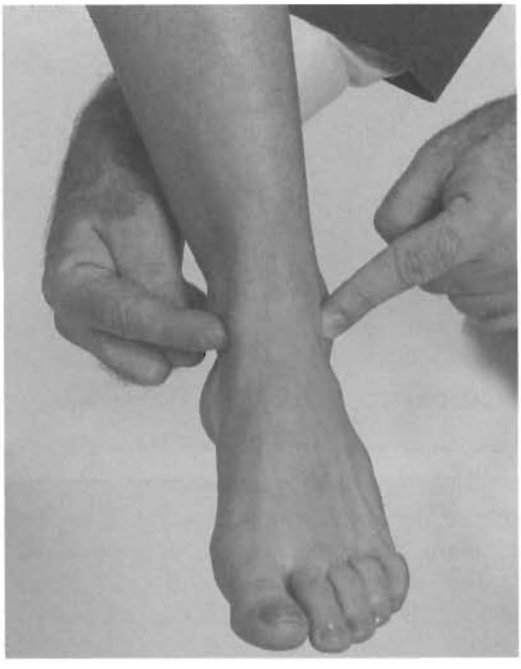

FIGURE 24.2.5. Anterolateral and anteromedial talar regions of impingement.

Palpate the lateral (anterior and posterior talofibular, calcaneofibular) (Fig. 24.2.6) and medial deltoid ligaments of the ankle (Fig. 24.2.7). Note any tissue bogginess or swelling, remembering that normal ligaments are not tender to palpation. Move your fingers

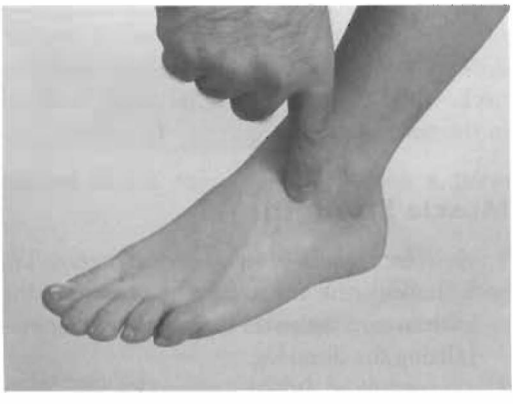

FIGURE 24.2.4. Sinus tarsi.

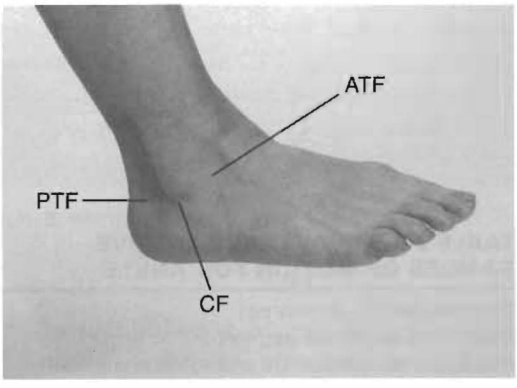

FIGURE 24.2.6. Lateral ligaments of the ankle. ATF, anterior talofibular; PTF, posterior talofibular; CF, calcaneofibular.

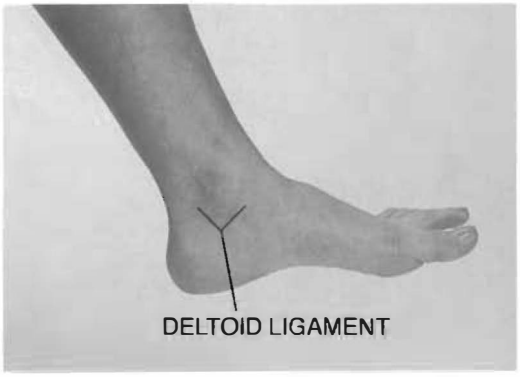

FIGURE 24.2.7. Medial ligaments of the ankle.

toward the anterior aspect and check the syndesmosis, medial to lateral, distal to proximal.

Feel along the tendons and their sheaths, specifically the Achilles, peroneal, and posterior tibialis tendons. Note swelling, defects, and tenderness. Pay close attention to the tarsal tunnel and its structures.

RANGE OF MOTION

1. *Active range of motion* (ROM). Active ROM is assessed with athlete sitting on the table with the legs hanging over side. The examiner asks the athlete to move the foot into plantarflexion, dorsiflexion, inversion, and eversion.
2. *Passive range of motion* (Table 24.2.1). Passive ROM is assessed with athlete sitting and the ankles relaxed. The same motions as above are checked (Fig. 24.2.8A to D). The examiner stabilizes the distal tibia with one hand while the other hand maneuvers the foot.

TABLE 24.2.1. AVERAGE PASSIVE RANGES OF MOTION FOR ANKLE

Dorsiflexion: 15–20 degrees
Plantarflexion: 55–65 degrees
Pronation (coupled ankle and subtalar motion): 5 degrees
Supination (coupled ankle and subtalar motion): 20 degrees

3. *Equinus contracture.* The ankle cannot achieve neutral dorsiflexion, so the foot stays more in some degree of plantarflexion. This triggers a cascade of biomechanical decompensation. Causes include neurogenic (cerebral palsy, neurapraxia), gastrocnemius or soleus injury, prolonged immobilization in plantarflexion, and surgical repair of the Achilles.
4. *Subtalar motion.* The athlete lies prone with the knee flexed to 135 degrees. The foot is brought into dorsiflexion, then moved through inversion and eversion. Overall motion at the joint is approximately only 10 to 20 degrees (Fig. 24.2.9).
5. *Inversion of the subtalar joint.* The athlete stands and rises on his or her heels. The heel will promptly invert, the longitudinal arch will rise, and the leg will rotate externally if the foot is functioning normally (Fig. 24.2.10).
6. *Eversion of the subtalar joint.* The athlete stands as the examiner observes him or her from behind to evaluate the tilt of the calcaneus. The calcaneus should slant to one side. If the calcaneus tilts inward, this is considered hindfoot valgus. The tarsal navicular is dorsiflexed and abducted on the head of the talus. The forefoot is supinated in relationship to the hindfoot.

NEUROVASCULAR EXAMINATIONS

Pulses. The examiner uses the pad of the index finger to palpate the dorsalis pedis and posterior tibial pulses. The dorsalis pedis is on the dorsal side of the foot around the anterior talar neck, while the posterior tibial artery is found in the tarsal tunnel.

Muscle Strength

1. *Anterior tibialis* (deep peroneal nerve, L4, L5). With the athlete supine or sitting, the examiner resists ankle dorsiflexion while stabilizing the distal leg.
2. *Gastrocnemius* (tibial nerve, S1, S2). With the athlete supine and the knee extended,

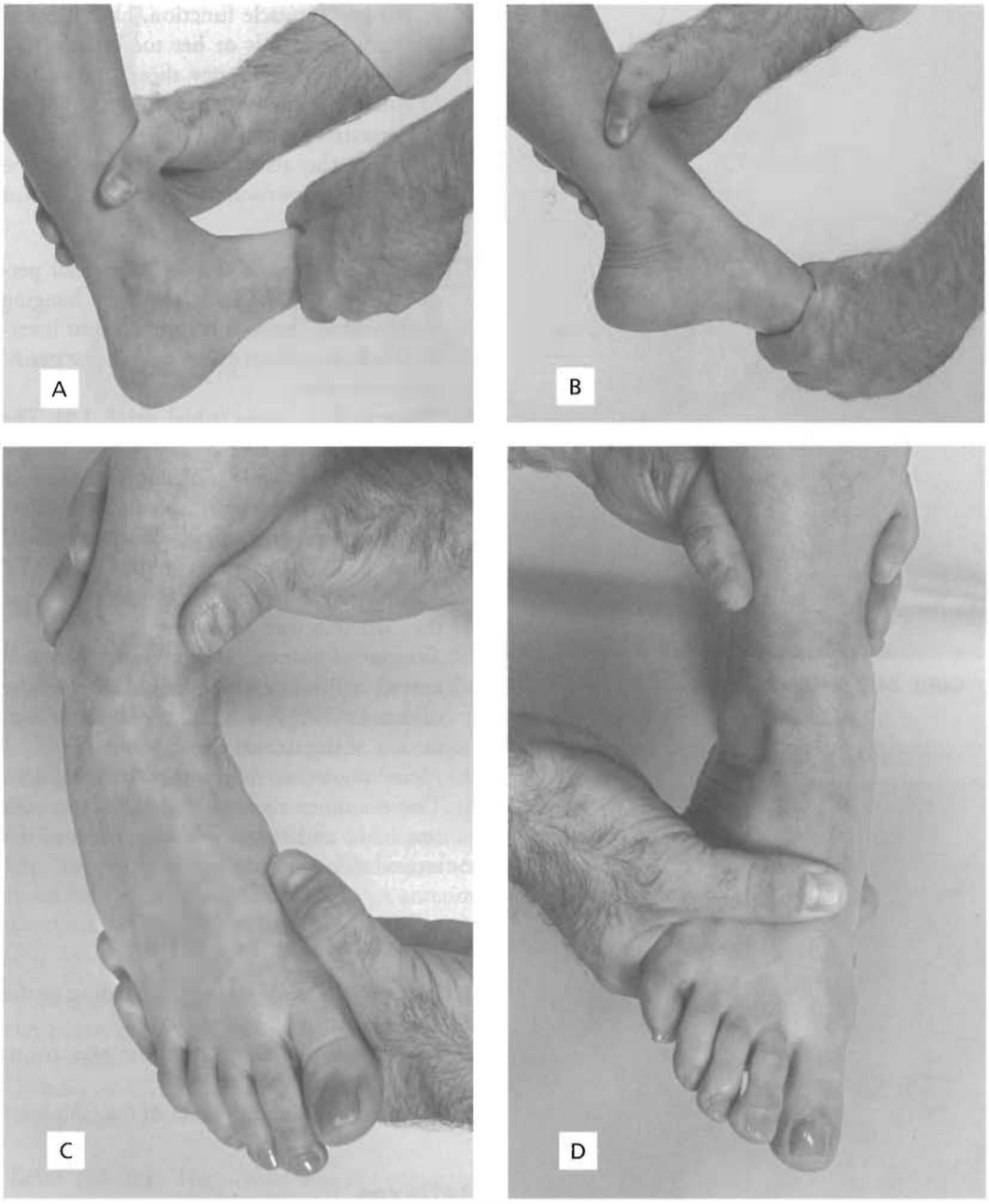

FIGURE 24.2.8. Passive range of motion. **A,** Dorsiflexion; **B,** plantarflexion; **C,** inversion; **D,** eversion.

the foot is brought into dorsiflexion, then pushed against the examiner's resistance.

Note: The following tests can be done with the athlete sitting up on the edge of a table.

3. *Soleus* (tibial nerve, S1, S2). With the athlete sitting on the edge of a table with the leg hanging, plantarflexion is again tested against the examiner's resistance.
4. *Posterior tibialis*

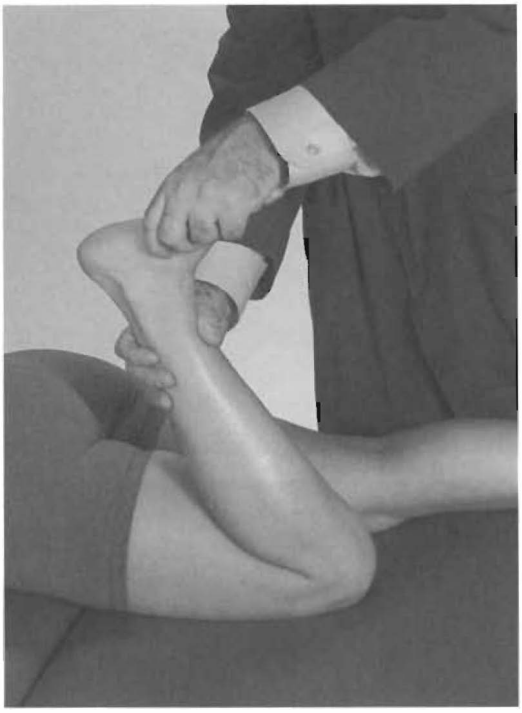

FIGURE 24.2.9. Subtalar motion.

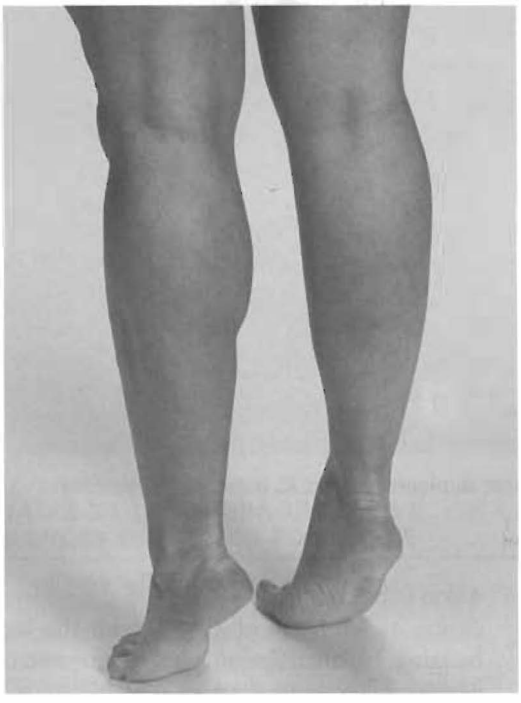

FIGURE 24.2.10. Inversion of the subtalar joint.

   a. For gross muscle function, have the athlete walk on his or her toes around the room. Any deficiency should be visible, either by pain, inability to stay up, or exaggerated pronation.
   b. While the athlete is sitting, bring the foot into eversion or neutral and resist inversion.

5. *Peroneus longus and brevus* (superficial peroneal nerve, S1). With the foot hanging from a table, the foot is brought into inversion and the athlete everts against the examiner's resistance.
6. *Flexor hallucis longus* (tibial nerve, L5). The examiner resists first MTP plantar flexion with one hand while securing the calcaneus with the other.
7. *Extensor hallucis longus* (deep peroneal nerve, L5). The examiner resists first MTP dorsiflexion with one hand while securing the calcaneus with the other.
8. *Extensor digitorum longus.* (deep peroneal nerve, L5). The examiner controls the calcaneus with one hand and resists dorsiflexion of the second through fifth digits.
9. *Flexor digitorum longus* (tibial nerve, L5). The examiner controls the calcaneus with one hand and resists plantar flexion of the second through fifth digits.

Sensorium (Fig. 24.2.11)

1. L4: medial side of the foot extending to the medial first MTP joint.
2. L5: dorsum of the foot from the great toe to the fourth toe.
3. S1: lateral foot and dorsum of the fifth toe.

Reflexes

Achilles (S1). While the athlete sits on a table edge, the examiner holds the forefoot and gently dorsiflexes the foot. The other hand uses a reflex hammer to tap the midportion of the Achilles tendon gently.

If the ankle or tendon is too tender to tap, bring the foot into dorsiflexion with the examiner's finger pads pushing against the MTPs

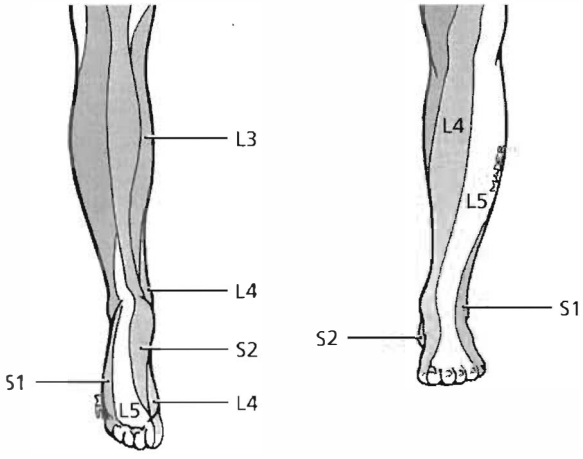

FIGURE 24.2.11. Foot and ankle sensorium. (From Ward RC, ed. *Foundations of osteopathic medicine,* 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2003.)

on the plantar side, then strike the fingers. This is best done with the athlete prone.

Stability

Anterior Drawer Test. The athlete sits on the edge of a table while the examiner, facing the athlete, controls the distal leg with a stabilizing hand. The mobilizing hand grasps the calcaneus posteriorly, while the thumb is placed over the anterior talus (in some cases, the thumb can be free if the examiner is comfortable controlling the calcaneus posteriorly). The examiner translates the calcaneus and talus in an anterior direction, noting laxity, end point, and pain (Fig. 24.2.12).

Positive test: Excessive anterior laxity and soft end point, particularly compared with the opposite ankle.

Indicates: Disruption of the anterior talofibular ligament.

Talar Tilt Test. The athlete and the examiner are in the same position as for the anterior drawer, but the mobilizing hand holds the calcaneus from the inferior direction, one finger on the lateral talus to monitor motion. The ankle has 10 to 20 degrees of plantarflexion. The examiner then inverts the talus to its end point of motion. The examiner must be careful not to invert the foot, as subtalar motion will be assessed (Fig. 24.2.13).

Additional Tests. Bilateral mortise view radiographs can be taken with the talus stressed in inversion. The tilt angle is measured and compared.

Positive test. Excessive tilt, particularly compared with opposite foot, more than 2-mm difference in tilt on radiographs.

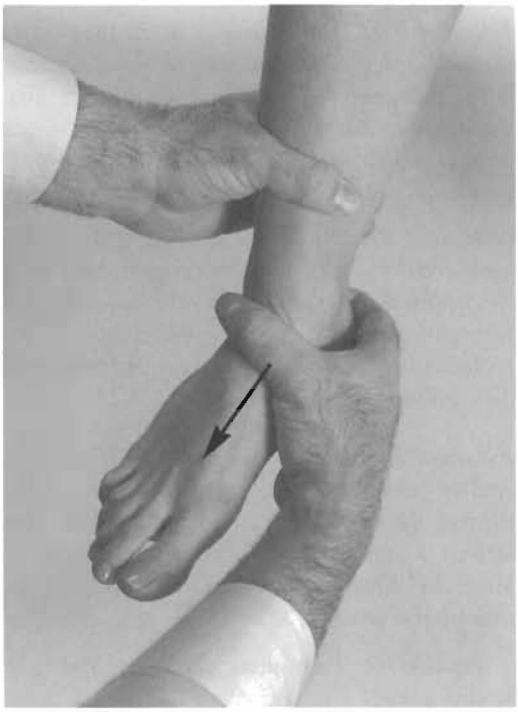

FIGURE 24.2.12. Anterior drawer test for anterior talofibular ligament integrity.

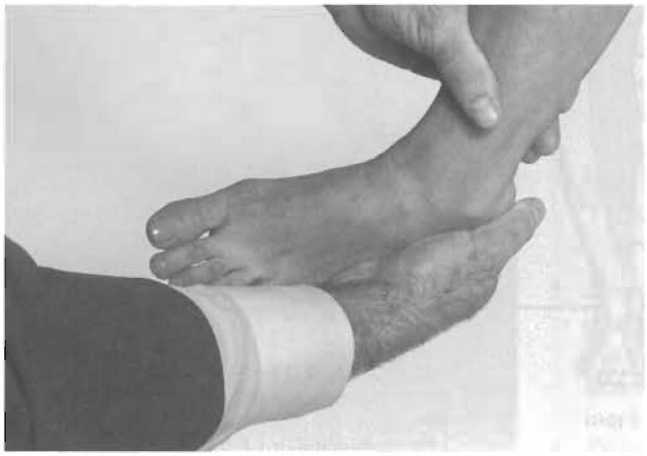

FIGURE 24.2.13. Talar tilt test.

Indicates. Anterior talofibular (ATF) and calcaneofibular (CF) ligament disruption.

Note: Increases in talar tilt usually occur with both ATF and CF disruption. When the ankle is injured in dorsiflexion, the ATF ruptures primarily, and the CF secondarily. Isolated CF injuries rarely cause increased talar laxity.

Eversion Test. The examiner holds the athlete in the same position as for the talar tilt test. With the proximal hand stabilizing the tibia, the opposite hand holds the midfoot from the plantar aspect and everts up to the anatomic barrier (Fig. 24.2.14).

Positive test: Pain with end range of motion into eversion, increased laxity compared with the opposite side; results should be correlated with palpation of the medial ankle.

Indicates: Medial deltoid ligament injury or disruption.

External Rotation Stress Test. With the foot and leg held in the same position as for the talar tilt test, the examiner externally rotates the foot against a stabilized lower leg. The idea is to force the talus to rotate as well, stressing the tibiofibular joint (4).

Positive test: Pain along the syndesmosis or medial ankle.

Indicates: Syndesmotic injury or disruption, injury to the deep fibers of the medial deltoid ligament.

Peroneal Tendon Stability Test. The examiner holds the athlete's foot with one hand, while the opposite hand gently palpates the peroneal tendons just posterior to the lateral malleolus. The examiner moves the foot into end-range inversion, then asks the athlete to evert against

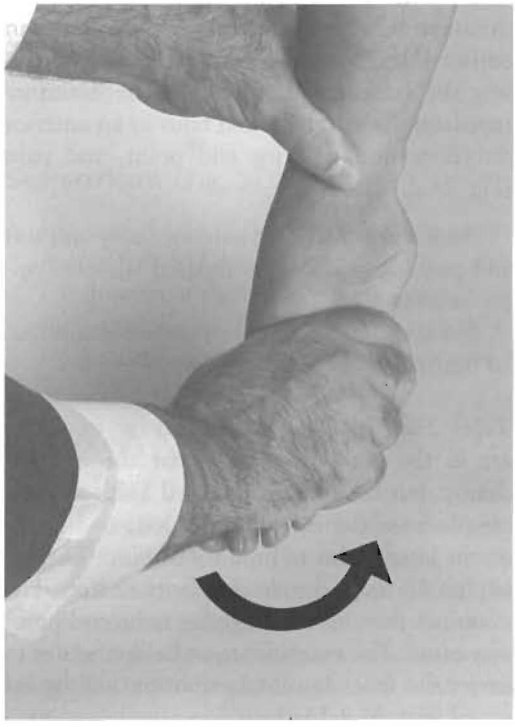

FIGURE 24.2.14. Eversion test.

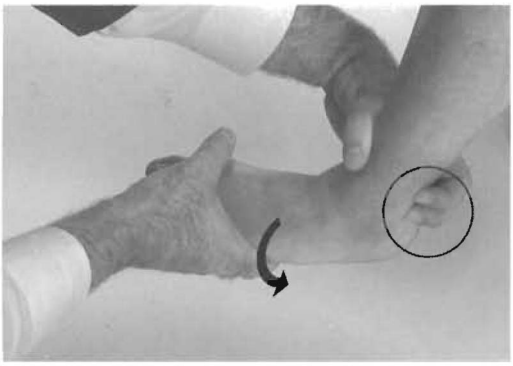

FIGURE 24.2.15. Peroneal tendon stability test.

resistance. The other hand monitors the peroneal tendon (Fig. 24.2.15).

Positive test: The tendon palpably subluxes over the malleolus; pain associated with excessive tendon excursion, clicking, or catching behind the malleolus.

Indicates: Peroneal tendon retinaculum disruption, lateral malleolar fracture.

Squeeze Test. With the athlete relaxed and sitting, the examiner locks his or her fingers behind the distal third of the tibia and places the thenar eminence along the tibial and fibular shafts. The examiner squeezes the hands together and holds for about 2 seconds (Fig. 24.2.16).

Positive test: Pain with squeezing at the level of the syndesmosis.

Indicates: Syndesmotic injury or disruption (4).

Kleiger's Test. The athlete flexes the knee to 90 degrees, dorsiflexing the ankle to 90 degrees and gently externally rotating the foot (Fig. 24.2.17) (3).

Positive test: Pain at the level of the syndesmosis and ankle mortise.

Indicates: Injury or disruption of the syndesmosis.

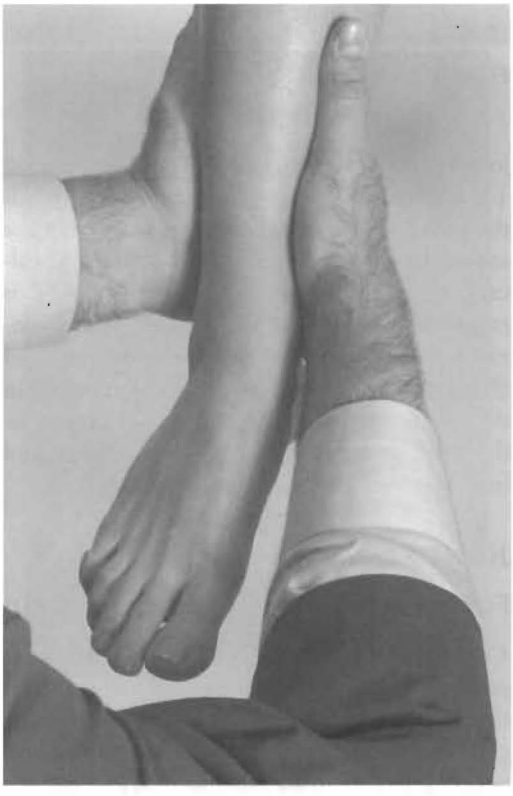

FIGURE 24.2.16. Squeeze test.

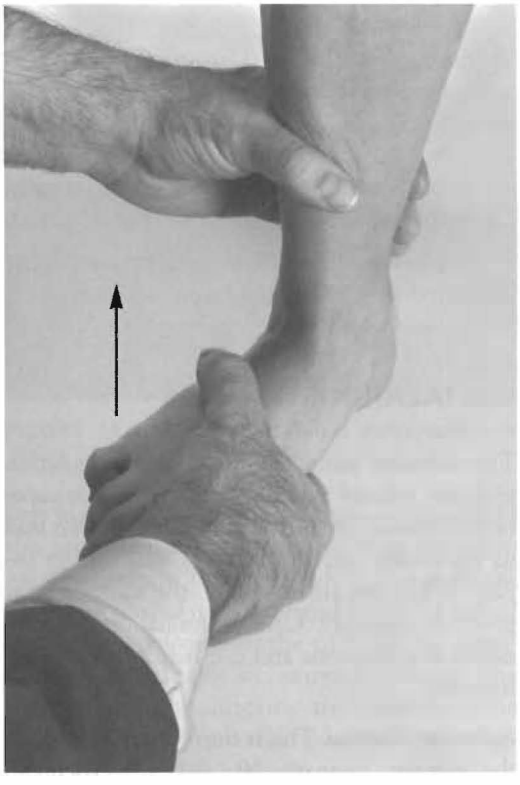

FIGURE 24.2.17. Kleiger's test.

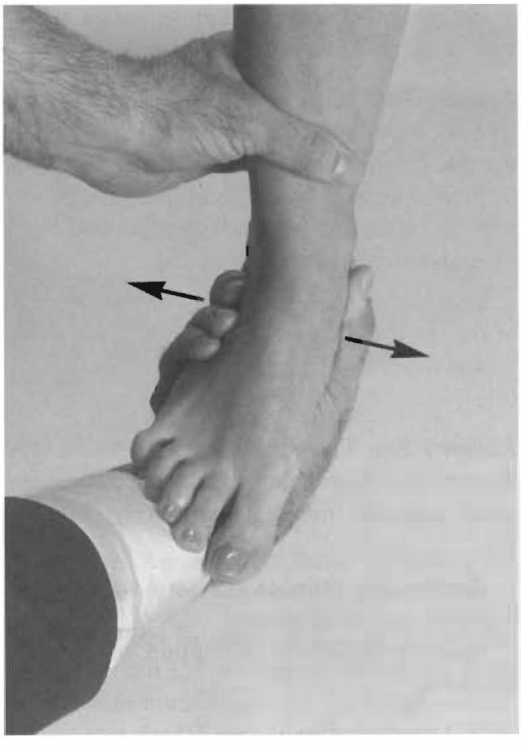

FIGURE 24.2.18. Tibiotalar shuck test (Cotton's test).

Tibiotalar Shuck Test (Cotton's Test). The examiner holds the athlete's lower leg with the stabilizing hand, while the mobilizing hand cups the talus from underneath. The examiner then translates the talus medially and laterally, noting pain and perceived laxity (Fig. 24.2.18).

Positive test: Pain with translation, laxity compared to the uninjured side.

Indicates: Syndesmotic disruption (4).

SUBTALAR JOINT

The subtalar joint represents the articulation with the inferior side of the talus and the superior calcaneus. Dysfunction of this joint can lead to significant gait disturbances and further injury. When the athlete walks, the subtalar joint pronates naturally. Careful evaluation helps to establish a diagnosis and can help prevent other injuries.

Subtalar Neutral. This is the foot position when the subtalar, talonavicular, and calcaneocuboid joints are reduced and congruous in the foot. The athlete is sitting while the examiner holds the fifth MTP joint between two fingers on the mobilizing hand. The other hand holds the calcaneus for position testing. The fifth MTP is raised until the foot is placed in the neutral position by centering the navicular on the talus (Fig. 24.2.19). After the foot has been placed in the neutral position, it is possible to determine the relative varus/valgus of the hindfoot and forefoot.

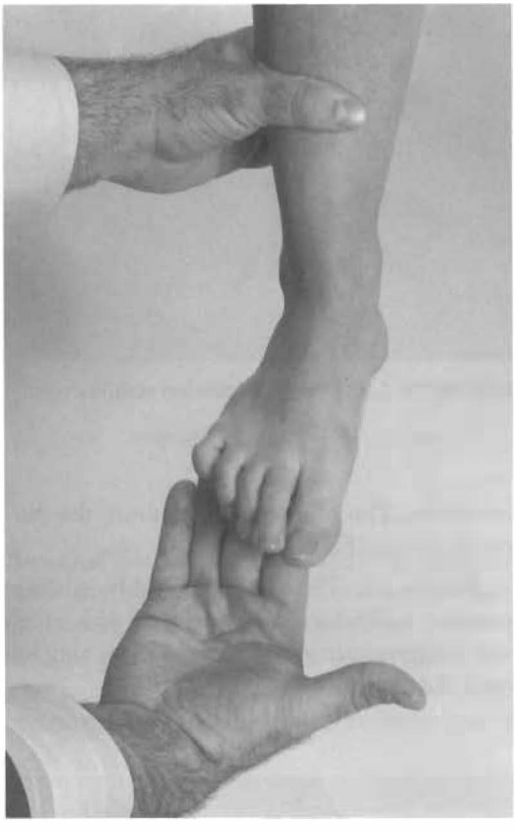

FIGURE 24.2.19. Subtalar neutral.

JOINT PLAY

Foot

Calcaneocuboid Joint. The athlete is supine on the table with the examiner at the end near the feet. Palpate for the plantar side of the cuboid, noting tenderness, tension, and prominence of its tuberosity when compared with the

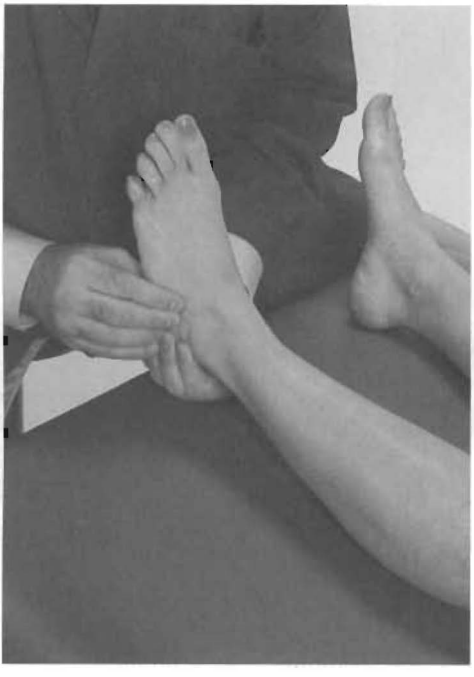

FIGURE 24.2.20. Joint play: foot, calcaneocuboid joint.

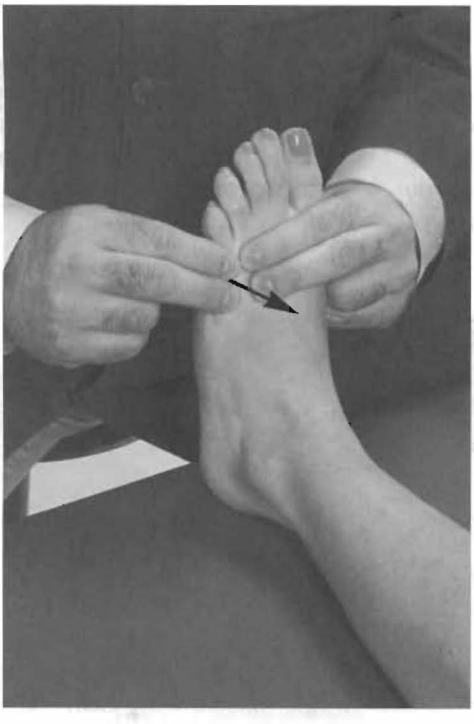

FIGURE 24.2.21. Metatarsal anteroposterior glide.

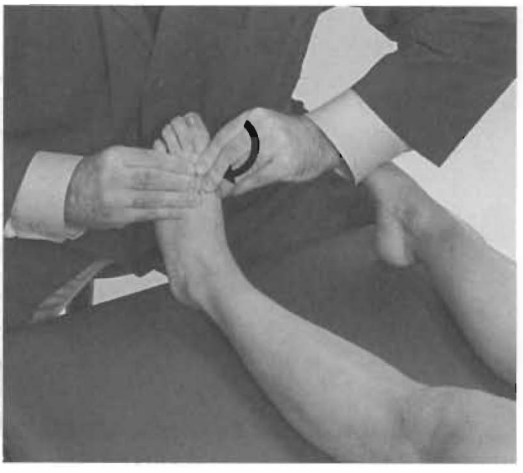

FIGURE 24.2.22. Forefoot (metatarsal) rotation.

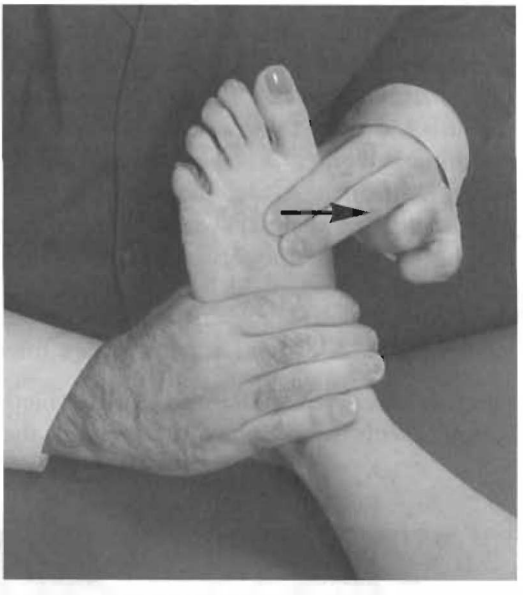

FIGURE 24.2.23. Tarsometatarsal anteroposterior glide.

opposite foot. The examiner tests the motion of this joint by using one hand to clasp the calcaneus posteriorly, holding the foot at a right angle to the tibia. The other hand holds the lateral foot so as to encompass the cuboid. The examiner can internally or externally rotate the forefoot while monitoring the motion at the calcaneocuboid joint, then compare to the opposite side (Fig. 24.2.20) (5).

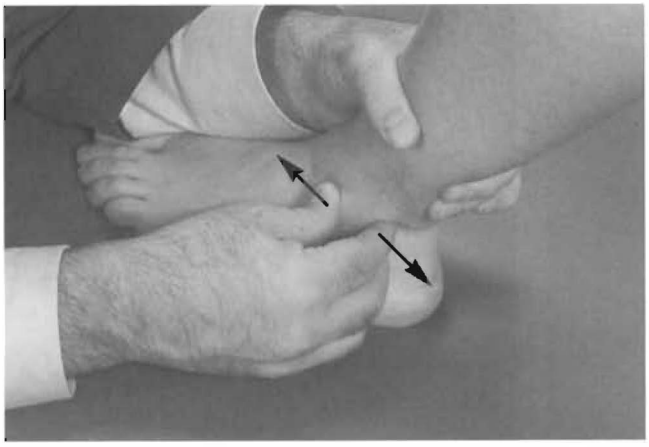

FIGURE 24.2.24. Joint play: ankle, distal fibula.

Metatarsal Anteroposterior Glide. The athlete is supine with the examiner at the end of the table. The examiner faces the plantar aspect of the feet, using one hand to stabilize the fourth metatarsal (MT) head between the thumb and finger pads. The other hand holds the fifth MT head and translates it slowly and smoothly in both a dorsal and plantar direction. The hands then slide down one and repeat, translating each MT head. For the first MT head, stabilize the second MT head, and translate with the opposite hand (Fig. 24.2.21) (5).

Forefoot (Metatarsal) Rotation. The athlete is supine with the examiner at the end of the table. The metatarsals are held in the same way as for metatarsal glide. The second MT head is stabilized while the first MT head is mobilized, but instead of anteroposterior glide, the examiner uses a shoulder swing to rotate the first MT head on the second, in both a clockwise and counterclockwise direction (Fig. 24.2.22) (5).

Tarsometatarsal Anteroposterior Glide. The athlete is supine, while the examiner stabilizes the midfoot tarsal bones with the left hand and holds the distal metatarsal neck and head with the left. The operator introduces anterior and posterior force to the metatarsal head noting play and end-feel (Fig. 24.2.23).

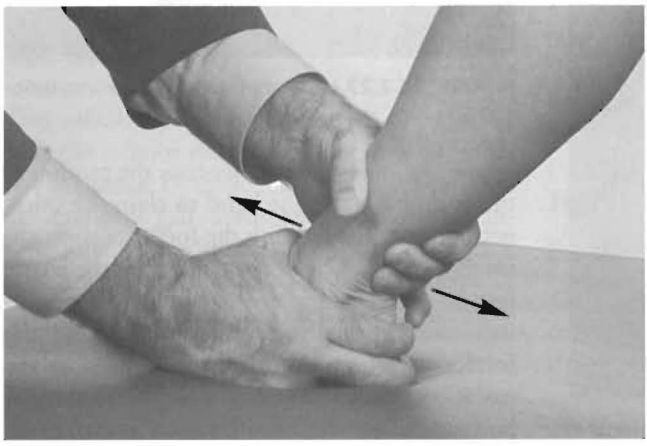

FIGURE 24.2.25. Anteroposterior talar glide.

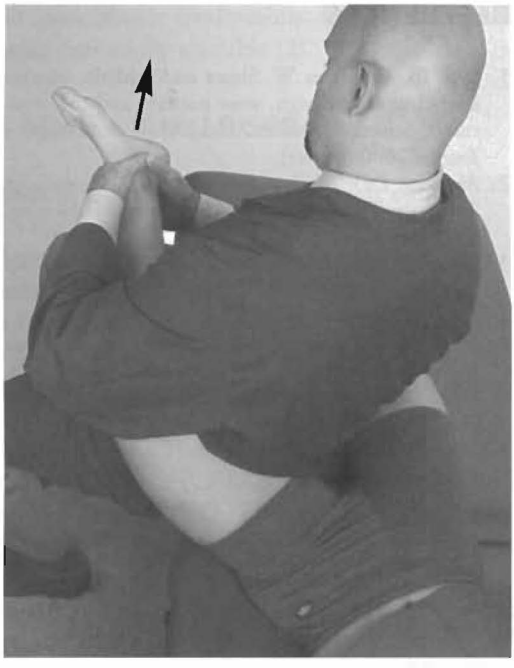

FIGURE 24.2.26. Long axis extension at the mortise.

ANKLE

Distal Fibula. The examiner's stabilizing hand holds the distal tibia firmly, while the thumb of the mobilizing hand is placed on the anterior lateral malleolus and its index and middle fingers on the posterior side. The examiner translates the fibular head anteriorly and posteriorly, assessing both directions for restriction (Fig. 24.2.24). In athletes with syndesmotic disruption, distal fibular joint play may be increased in the posterior direction (5).

Anteroposterior Talar Glide. The athlete is supine with the knee and ankle at 90-degree angles. The examiner grasps the lower leg just proximal to the malleoli with the mobilizing hand, while the stabilizing hand grasps the midfoot and dorsum of the foot. The mobilizing hand translates the distal tibia and fibula anteriorly, then posteriorly. The stabilizing hand holds the foot at a 90-degree angle (Fig. 24.2.25) (5).

Long Axis Extension at the Mortise. This is useful to assess a dysfunctional talar joint, particularly after prolonged ankle immobilization. The athlete is supine with the hip abducted and flexed to 90 degrees, while the examiner sits on the table in between the athlete's legs. The examiner grasps the foot with one hand, the thenar web placed over the anterior talus, while the other hand has its thenar web against the posterior calcaneus. The examiner leans against the athlete's thigh with his back, while the hands maintain the foot at a right angle to the tibia, then distracts the talus from the tibia with a short impulse (Fig. 24.2.26). This technique crosses both the subtalar and talotibial joints (5).

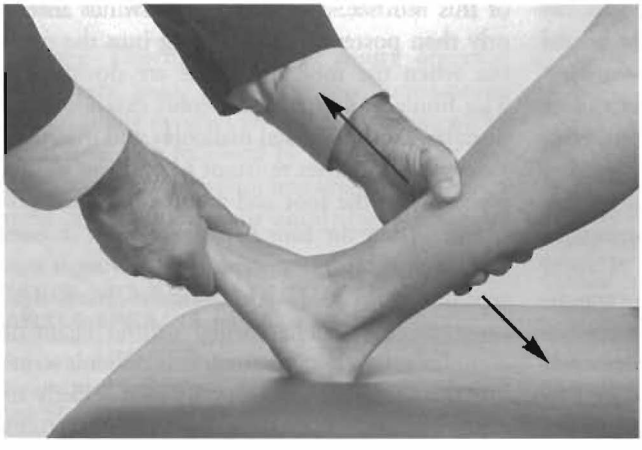

FIGURE 24.2.27. Isolated mortise joint movement.

Isolated Mortise Joint Movement. The athlete lies supine with the knee and ankle at right angles. The examiner grasps the lower leg approximately 15 cm proximal to the malleoli. The other hand stabilizes the sole of the forefoot, so that the foot rests upon the posterior-inferior angle of the calcaneus. From here, the examiner pushes the lower leg upward and downward, rocking the foot and ankle mortise, while the stabilizing hand holds the forefoot (Fig. 24.2.27). Any restriction felt during this maneuver indicates pathology involving the mortise joint.

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24.3

Common Conditions

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The ankle is the most common site for musculoskeletal injuries, and acute ankle sprains account for up to 75% of these injuries (1). Every year one million patients, mostly young athletes, present to physicians with acute ankle injuries (1). Although ankle sprains are common, their seriousness is often underestimated. Chronic pain from what appeared to be an uncomplicated acute injury may persist for several months or even years. Proper diagnosis and exclusion of more serious pathology, adequate control of injury-related inflammation, early mobilization, and comprehensive sport-specific rehabilitation are essential for early pain-free return to sport and prevention of chronic pain.

This part discusses two basic types of acute ankle sprains: (a) the more common, inversion-plantarflexion injury, often referred to as the lateral ankle sprain, and (b) the eversion–external rotation injury with associated syndesmosis disruption, known as the high ankle sprain.

RELEVANT ANATOMY

The anatomy of the talocrural joint is composed of the boxlike mortise formed by the distal tibia and the fibula. The anterior and posterior tibiofibular ligaments and the fibrous interosseous membrane maintain the integrity of this mortise. The talus, being wider anteriorly than posteriorly, locks snug into the mortise when the foot and ankle are dorsiflexed. The fibula and lateral malleolus extends more distally than the medial malleolus and thus provides a bony barrier resistant to eversion injury, especially in the foot and ankle dorsiflexed position. When the foot is plantarflexed, it loses that snug mortise, talocrural fit, leaving it susceptible to inversion and resultant lateral ligamentous injury. The medial deltoid ligament complex is broad and strong. It is difficult to injure this complex, but an injury to it is likely an eversion–external rotation force that disrupts

the tibiofibular syndesmosis, leaving the ankle joint potentially unstable (2).

LATERAL ANKLE SPRAINS

Lateral ankle sprains are seen most commonly in basketball, football, soccer, and running (2). Inversion-plantarflexion injuries typically occur when an athlete plants the foot on an uneven surface, such as stepping in a hole or on another player's foot. The foot tends to invert and plantarflex, causing collapse of the ankle and excessive load on the lateral ligaments, anterior talofibular ligament (ATFL), calcaneofibular ligament (CFL), and posterior talofibular ligament (PTFL). The injury is categorized from grade 1 to 3. A grade 1 injury to the lateral ankle ligaments involves some swelling and disability but no instability. There is injury to the ATFL and possibly the CFL complex. The athlete should be able to ambulate, albeit with a slight limp. A grade 2 injury is characterized by severe swelling over the lateral ankle, some mild instability, an antalgic gait, demonstrable mild ligamentous laxity, and some loss of motion and/or function. Grade 3 injuries involve disruption of the three lateral ligaments, marked loss of function, and static instability on clinical examination.

Factors. There are several extrinsic and intrinsic factors that may put athletes at risk or predispose them to a lateral ankle sprain. Little consensus exists in the literature with respect to many potential risk factors. The more supported factors are listed in Table 24.3.1

History. The mechanism of injury observed with lateral ankle sprains, as mentioned previously, usually involves an athlete stepping on an uneven surface followed by plantarflexion and inversion. Recurrent sprains without adequate rehabilitation may occur simply by planting the foot and attempting to cut. An audible "pop" is frequently heard. An athlete will often say "I rolled my ankle."

TABLE 24.3.1. RISK FACTORS FOR ANKLE SPRAINS (9,26)

Previous ankle sprains
Height and weight
Use of ankle braces

Physical Examination

Swelling and ecchymosis occur over the lateral ankle structures within hours. The athlete's ankle is tender to palpation over some or all of the lateral ankle ligaments, depending on the degree of injury. Range of motion is limited by the degree of swelling and pain. An antalgic gait is common, but the inability to bear weight suggests a higher-degree sprain or possibly a concomitant fracture. Swelling of the entire ankle joint, as opposed to simply the lateral structures, also suggests more serious intra-articular injury.

The anterior drawer test is positive in second- and third-degree sprains, while the talar tilt test is seen only in third-degree sprains. Fibular head tenderness may indicate a concomitant fracture (Maisonneuve fracture).

The uninjured ankle should be examined, first allowing a more accurate estimation of the amount of laxity in the injured ankle. A side-to-side difference in the anterior drawer test of greater than 5 mm or a talar tilt of greater than 10 degrees suggests a grade 3 ligamentous injury (2).

Athletes with ankle strains need evaluation of the kinetic chain to facilitate recovery. Dysfunctions above and below the joint will limit and restrict ankle function. Table 24.3.2 lists the most important areas to examine and treat.

The foot on the injured side should also be examined. Palpating for swelling and tenderness over the base of the fifth metatarsal, midfoot, navicular, talus, malleoli, and peroneal and Achilles tendons helps to identify other injuries that may be inadvertently overlooked.

TABLE 24.3.2. OTHER AREAS TO EXAMINE FOR ANKLE SPRAINS

Cuboid and navicular
Fibular head
Hip extension
Hip rotation
Sacroiliac (innominate rotation or shear)
Lumbar

Diagnostic Studies. Anteroposterior, lateral, and mortise view ankle radiographs should be obtained using the Ottawa ankle and foot rules as guidance. These guidelines have been extensively studied and found to be almost 100% sensitive for ruling out fractures of the acutely injured ankle in adults (1). In skeletally immature athletes and in situations in which clinical suspicion overrides these guidelines, radiographs should be obtained.

The use of stress-view radiographs has no role in the acute setting, and gives no information about future functional stability (2).

Standard Treatment

Standard treatment of lateral ankle sprains begins with PRICE (*P*rotection with crutches and/or splinting, *R*elative rest, *I*ce, brace, tape, or Ace wrap with a U-shaped *C*ompression pad applied around the lateral malleolus, and *E*levation of the extremity above the level of the heart). Nonsteroidal anti-inflammatory drugs (NSAIDs) can be offered and active range of motion initiated as soon as pain permits. Weight bearing as tolerated on crutches may be needed if pain is severe or the gait is markedly impaired. Functional rehabilitation should be initiated and progressed as soon as possible. Air-filled or gel-filled ankle braces have been shown to facilitate recovery and protect against injury (1).

SYNDESMOTIC (EVERSION–EXTERNAL ROTATION) ANKLE SPRAIN

Commonly referred to as the high ankle sprain, syndesmotic injuries occur in 11% to 18% of all ankle sprains (3). It is most common in high-impact sports such as football, rugby, ice hockey, and occasionally slalom skiing. Unlike the more common lateral ankle sprain, high ankle sprains typically require significant recovery time before return to play (3,4).

History. The mechanism of injury involves external rotation of the foot and ankle. This can occur with or without the foot in hyperdorsiflexion. A lateral blow to the knee with the planted foot forced into external rotation is a common mechanism (1,3,4). This results in a sprain of the deltoid ligament complex. This can also cause a malleolar or proximal fibula fracture (Maisonneuve fracture). The tibiofibular syndesomosis ligament complex and interosseous membrane are injured. If they are completely torn, instability of the ankle mortise results.

Physical Examination

The physical examination reveals pain, but at times surprisingly minimal swelling (3). The athlete should have all bony structures about the ankle and fibula palpated. Pain along the medial malleolus, deltoid ligament, and tibiofibular syndesmosis is common and suggestive of a high ankle sprain. The athlete typically has an antalgic gait, especially limited by toe push-off. Kleiger's test will be positive for syndesmosis derangement. The squeeze test also exacerbates pain at the distal syndesmosis. Most athletes with a high ankle sprain are unable to perform a one-leg hop test without significant pain (4). Anteroposterior (AP) and lateral radiographs of the ankle should include mortise views to look for tibiofibular widening. External rotation stress views are also helpful, if tolerated by the athlete. A tibiofibular clear space on AP or mortise views measuring more than 6 mm is suggestive of syndesmosis disruption and likely ankle instability (3). Computed tomography and magnetic resonance imaging (MRI) are excellent additional studies if initial radiographs are normal but clinical suspicion is high. MRI is a highly sensitive and specific tool for assessing syndesmotic injuries (3).

Standard Treatment

High ankle sprains involve the same rehabilitative principles as lateral ankle sprains. However, non–weight bearing with or without a semirigid ankle brace is recommended for the first week (3). Alternatively, a walking cast or cam walker device may be used for the initial 2 to 6 weeks (2). If there is clinical and/or radiographic evi-

dence of a disrupted (unstable) syndesmosis, then surgery should be strongly considered in the first 3 weeks to minimize long-term complications (2,3). Stable high ankle sprains commonly take anywhere from 2 to 8 weeks to heal. A progressive rehabilitation protocol is essential for full recovery and prevention.

A basic rehabilitation approach to ankle sprains is as follows (9):

Stage 1: Acute (rest, ice, compression, and elevation—RICE)

a. Reduce inflammation and swelling (modalities, NSAIDs, horseshoe taping)
b. Maintain proper weight bearing (crutches, walking boot)

Stage 2: Subacute (Pain-free range of motion)

a. Initiate non–weight bearing exercises
b. Proprioceptive training (Biomechanical Ankle Platform system board, wobble board–seated)
c. Increase ankle strengthening exercises (alphabet drawing, isometrics)
d. Gait training as tolerated
e. Restore proper muscle length
f. Ice or contrast baths (heat/cold alternate)
g. Joint mobilizations

Stage 3: Restorative

a. Single-leg balance
b. Heel raises/toe raises
c. Pronation and supination exercises
d. Initiate sagittal plane exercises
e. Manual resistive isotonics
f. Concentric and eccentric ankle strengthening

Stage 4: Activity and function

a. Initiate agility drills/frontal plane exercises (carioca)
b. Frontal plane reactive drills
c. Heel walks and toe walks
d. Return-to-sport training (circles and figure 8s)
e. Manual proprioceptive neuromuscular facilitation
f. Reactive exercises

Manual Medicine Techniques

There are several manual medicine techniques that may be used to treat an acute ankle sprain. Before attempting these techniques, fractures and significant intra-articular injuries should be ruled out clinically and/or with diagnostic studies.

Direct Talar Mobilization (talar tug technique)

Rationale: In injury mechanisms with significant ankle plantarflexion and inversion the talus may become misaligned in regard to its position in the mortise. Restriction of ankle motion and pain may persist until this misalignment is corrected. By correcting this problem with a high-velocity, low-amplitude (HVLA) technique, restoration of motion and release of restricted venous and lymphatic fluid may be achieved.

1. The athlete is placed lying flat on the examination table in the supine position and instructed to hold onto the top of the table.

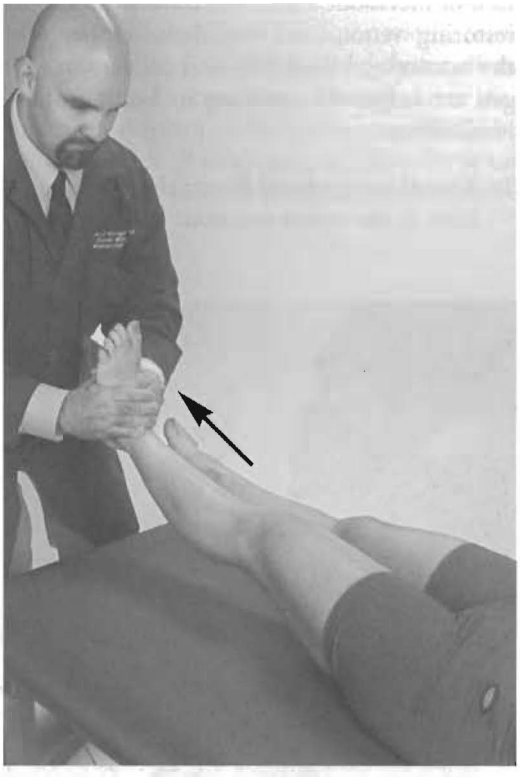

FIGURE 24.3.1. Talar tug technique.

2. The clinician gently grasps the forefoot, palm touching the sole of the foot, with one hand and cups the heel with the other hand, pads of the fingers touching the retrocalcaneal region.
3. The ankle is dorsiflexed to 90 degrees and traction is gradually applied while maintaining the dorsiflexion. The traction should just be enough to take up the slack (Fig. 24.3.1).
4. The athlete is informed that a short, quick tug will occur. A gentle HVLA tug on the foot and ankle is performed in the same axis as the leg. A painless audible pop may be heard. The athlete should not be jerked off the table by the applied force.
5. Recheck ankle active range of motion and document the results in the progress notes.

Lymphatic Pump

Rationale: Acute ankle sprains often result in significant ankle joint motion restriction and soft tissue edema. Venous and lymphatic drainage is impaired and results in accumulation of metabolic waste and cellular debris. By restoring venous and lymphatic drainage to the extremity, blood flow and delivery of oxygen are improved resulting in healing of injured tissues.

1. The athlete is placed flat on the examination table in the supine position.
2. The clinician gently grasps the foot and ankle by wrapping both hands around the sole of the foot and interlocking the fingers over the dorsum of the forefoot.
3. The athlete is informed that the clinician will induce a gentle plantarflexion and dorsiflexion repetitive motion to the ankle joint.
4. The clinician cyclically pumps the foot and ankle into plantarflexion and dorsiflexion. Stay in the range of motion arc that the anatomic barriers permit (Fig. 24.3.2).
5. The pumping action helps to restore ankle joint motion and milks the fluid from the soft tissues distally to the central venous and lymphatic systems. Passive calf muscle contraction and stretching simulate physiologic activity that normally propagates venous and lymphatic return.

HVLA Technique for Cuboid Release

Rationale: Ligamentous attachments from the plantar fascia run laterally toward the calcaneocuboid joint. Increased tension along the fascial tissue can increase inflammation and decrease flexibility of the midfoot. Dysfunctions in the calcaneocuboid joint can be the culprits or this tension may cause a dysfunctional cuboid that excessively pronates (internally rotates). The athlete may not be acutely aware of the tension but will have tenderness and fullness with palpation on the plantar side of the

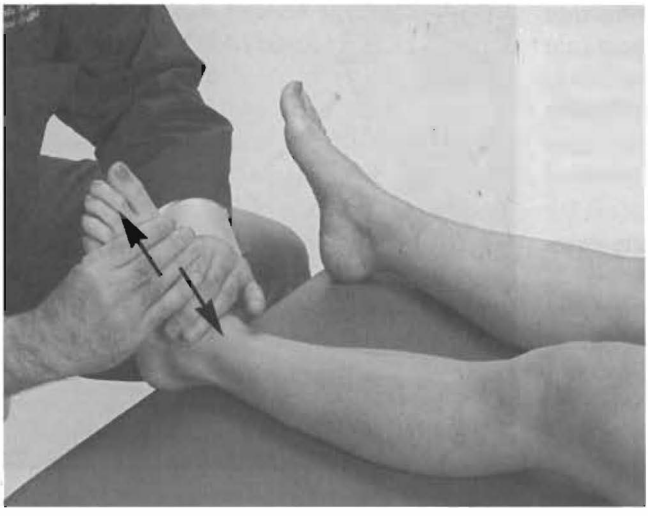

FIGURE 24.3.2. Lymphatic pump (both hands on one foot).

cuboid. This limits flexion through the calcaneocuboid joint, which is a fulcrum point for pushing off in running.

1. The athlete is prone with the affected leg off the table and the clinician facing cephalad on the same side.
2. The clinician grasps the affected foot with both hands while placing the lateral hand's thumb on the plantar side of the cuboid and the medial hand's thumb on top of it.
3. The athlete relaxes the leg as the clinician swings the leg off by holding the foot, plantarflexing it repeatedly and rhythmically.
4. The clinician then engages the barrier through plantarflexion and whips the foot down to the ground, keeping pressure constant on the cuboid with the thumbs (Fig. 24.3.3).
5. Reassess.

Peroneus Deep Tissue Release

Rationale: When the foot and ankle invert and plantarflex as a result of stepping on an uneven surface, the peroneus muscles are elongated or stretched. The peroneus muscles react to this elongation by forcefully contracting in an attempt to evert the ankle. This eccentric load often results in tenderness along the muscle and musculotendinous structures. The goal of the soft tissue technique is to gently knead and stretch the muscles in an attempt to remove metabolic debris, improve venous and lymphatic flow, and reduce lower lateral leg pain.

1. The clinician locates the lateral malleolus of the ankle and the peroneus muscles just proximal to the lateral malleolus.
2. The clinician kneads the band of muscles and musculotendinous structures of the lateral leg moving distal to proximal, gently using the pads of his or her fingers and moving all the way up to the head of the fibula. Some cases may require the olecranon to get direct force into the tissue (Fig. 24.3.4).
3. The clinician starts back at the distal lower lateral leg and once again kneads the tissues from distal to proximal up the lateral leg.

HVLA Technique for Fibular Head Dysfunction

Rationale: The peroneus muscles forcefully stretch then contract as the foot and ankle roll into plantarflexion and inversion during a lateral ankle sprain. This reflex forceful contraction may pull the fibular head posteriorly, causing lateral knee pain, fibular head dysfunction, and potentially common peroneal nerve entrapment. A proximal fibular fracture should be ruled out with all ankle sprains, clinically or radiographically, prior to using any manipulative techniques.

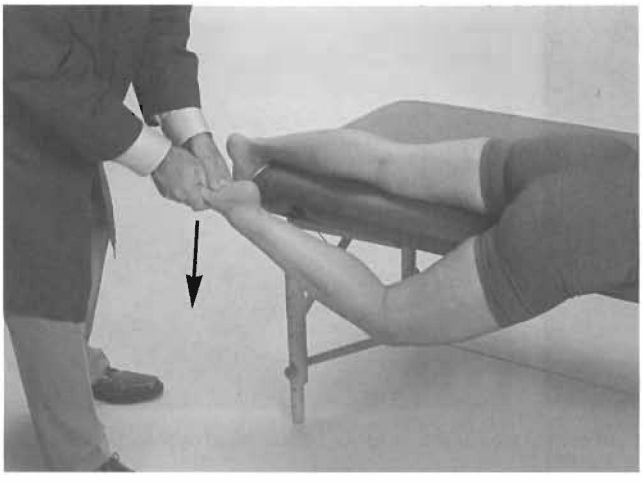

FIGURE 24.3.3. High-velocity, low-amplitude technique for cuboid release.

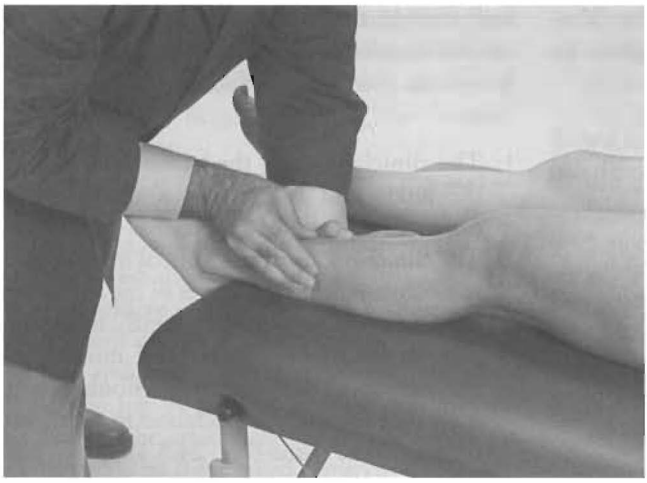

FIGURE 24.3.4. Peroneus deep tissue release.

Anterior dysfunction (head is positioned anteriorly)

1. The athlete is supine while the clinician stands at the foot of the table facing the athlete.
2. The athlete's affected leg is internally rotated slightly so the fibular head is more prominent.
3. The clinician places the mobilizing thenar eminence on top of the fibular head.
4. The clinician introduces an impulse thrust posteriorly into the table on the fibular head (Fig. 24.3.5A).
5. Reassess.

Posterior Dysfunction (head is posteriorly positioned)

1. The athlete is supine while the clinician stands at the foot of the table facing the athlete.
2. The clinician places the index and middle fingers in the posterolateral corner just behind the fibular head while the knee and hip are flexed.
3. The clinician gently brings the knee into more flexion so as to engage the index and middle fingers, then introduces an impulse thrust on the tibia to further flex the knee (Fig. 24.3.5B).
4. Reassess.

Rib Dysfunction

Rationale: Ipsilateral rib dysfunction may be observed on the side of the injured ankle. This may be due to reflex muscle contractions that occur as an athlete attempts to recover balance while falling.

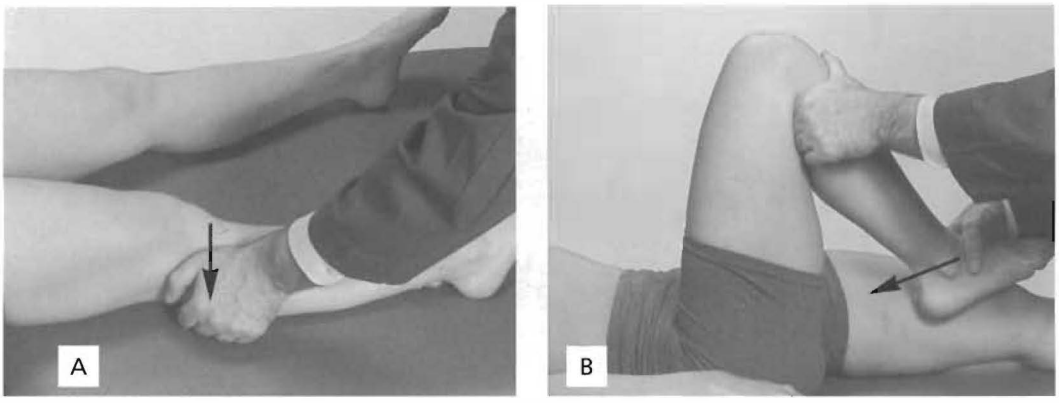

FIGURE 24.3.5. High-velocity, low-amplitude technique for the anterior fibular head dysfunction **(A)** and the posterior fibular head dysfunction **(B)**.

TABLE 24.3.2. PREVENTION OF ANKLE SPRAINS

Proprioceptive training
Strength training
Taping
High-top shoes
Semirigid brace

Technique: See Chapter 20.3 Thoracic Spine.

Prevention

Verhagen and collegues have extensively studied and critically reviewed the literature on the prevention of ankle sprains (Table 24.3.2) (5). Overall, proprioceptive training and semirigid braces have been found to be the most effective methods of reducing the incidence of ankle sprains (5). The benefit of high-top shoes is still unclear. The combination of taping and high-top shoes effectively reduces ankle sprains in athletes with a history of prior sprains (6,7). Athletic tape appears to prevent injuries by proprioceptive feedback rather than providing stability. A comprehensive and progressive rehabilitation program, along with a semirigid brace for at least 6 months, helps prevent recurrent ankle sprains in those athletes who have sustained a moderate to severe injury (6,7).

Return to Play

Return to play for athletes with either lateral or high ankle sprains can be difficult to predict. The rate of recovery for an individual athlete varies on the type of ankle sprain, degree of injury, the residual effects from prior injuries, the compliance and motivation of the athlete, and the demands of the athlete's sport (8). Manual medicine techniques can often speed recovery and return to play.

Guidelines for return to play are as follows:

1. Full active range of motion.
2. Minimal or no swelling.
3. Ankle strength should be at least 90% of the uninjured ankle.
4. Able to do 10 toe raises and be able to hop on the affected side without pain.
5. Able to do sport-specific drills without pain.
6. Proprioception should be assessed and restored to minimize the risk of reinjury.

PLANTAR FASCIITIS AND ACHILLES TENDINITIS

Plantar fasciitis and Achilles tendinitis are conditions caused by repetitive submaximal overload forces to previously normal tissue. The basic mechanism for both conditions is one of overuse. Plantar fasciitis is the result of accumulative impact loading and repetitive microtrauma to the plantar fascia, particularly at the insertion into the calcaneus. In Achilles tendinitis the injury is believed to be more of a repetitive tensile overload. This repetitive microtrauma causes an inflammatory response, which then produces heel pain either in the medial plantar fascial insertion or the posterior Achilles insertion. This mechanism is not unlike most repetitive overuse tendinopathies.

Athletes

Runners and basketball and volleyball players are at highest risk for these conditions because of the repetitive stress their activities generate on the foot and ankle. Repetitive eccentric loading leads to tissue breakdown if the tissue does not recover properly. Runners training for long distance can have an especially difficult time if they attempt to maintain their high mileage while the fascia or tendon is inflamed. Jumpers abruptly load their fascia and tendon eccentrically when landing, so time off from the sport may need to be part of the treatment.

Factors

Intrinsic etiologic factors for plantar fasciitis and Achilles tendinitis can include areas of decreased vascularity, aging, and degeneration of the plantar fascia, Achilles tendon, or their respective attachments (10). Ischemia of the lower limb has been reported as an etiology for Achilles tendon

rupture (11). Another concern is the potential risk for tendon injuries in patients who are taking certain medications. Although relatively rare, there have been reports of Achilles tendon ruptures in patients taking a fluoroquinolone antibiotic (12,13). Spontaneous Achilles tendon ruptures have also been reported in patients taking systemic corticosteroids (14). One must always consider the general health and medical conditions of athletes with plantar fasciitis or Achilles tendinitis (15).

There are multiple *extrinsic* etiologic factors, and each needs to be thoroughly explored. One simple technique is to review the "six S's of running injuries" (16):

- Shoes—evaluate any abnormal wear pattern in the tread or shoe.
- Surface—investigate the firmness and topography of the running surface.
- Speed—a sudden increase in speed or distance can be a precipitating factor.
- Stretching—observe for lack of appropriate flexibility, or hypermobility.
- Strength—muscle imbalances should be identified.
- Structure—check for anatomic malalignment, or somatic dysfunction.

Common mechanical issues in both conditions include restricted hip flexors and poor gastrocsoleus flexibility, both of which restrict dorsiflexion during running. A varus position of the heel and/or supination of the forefoot can produce overpronation of the foot during running. An excessively planus (pronated) or cavus (supinated) foot can increase strain on the plantar fascia. All of these factors can lead to increased strain on the plantar fascia as well as the Achilles tendon.

Lower extremity pathology is often exacerbated by leg-length discrepancies (functional or static), pelvic dysfunction, or repetitive biomechanical imbalance (17). In keeping with a holistic approach, any foot and ankle injury in an athlete needs a biomechanical evaluation going up past the pelvis.

Shoe defects are implicated in many injuries, including plantar fasciitis (18). Shoe tilt can prolong supination or pronation, depending on the direction of tilt. This irregularly loads the talus, which can lead to stress fractures and anterior knee pain.

Another significant (and increasingly more common) problem is obesity. Athletes who measure significantly above their body mass index (BMI) and have lower extremity problems such as plantar fasciitis and Achilles tendinitis should be counseled for weight loss. Notwithstanding the multiple health risks, obesity is a leading risk factor for degenerative osteoarthritis and frequently contributes to multiple lower leg tendinopathies. Weight loss should be presented as a preventive measure for such problems as plantar fasciitis and Achilles tendinitis.

History and Physical Examination

Athletes can suffer these injuries at any age, depending on how much and what type of stress the foot and ankle handles. Progressive Achilles pain can result from years of degeneration and tendinosis, and this can set the stage for an acute rupture. Acute ruptures are typified by a pop behind the heel and the sense that someone has kicked the person. Plantar fasciitis classically has an insidious onset, but can occasionally be precipitated by a traumatic event. One of the hallmark signs is exquisite pain with the first step in the morning, or with walking after a period of inactivity. The pain may then resolve somewhat with further walking and stretching. Achilles tendinitis has a similar onset, but with pain at the posterior aspect of the calcaneus where the tendon inserts, or within the tendon itself.

The classic examination finding in plantar fasciitis is tenderness to palpation of the plantar fascia at the medial calcaneal insertion. However, pain can be elicited anywhere in the plantar fascia. For Achilles tendinitis, pain is felt on palpation of the tendon or its insertion. Pain *behind* the Achilles tendon is more consistent with a retro-Achilles bursitis. Other pertinent examination findings to note include pes cavus, pes planus, or other malalignment. Foot mechanics should be evaluated in both weight-bearing and non–weight-bearing positions as well as during walking. Specific areas to examine for somatic

dysfunction include the cuboid, cuneiform, navicular, and talus. The fibula, both proximal and distal, is also important to evaluate for somatic dysfunction. The rest of the leg, knee, hip, and pelvis should also be evaluated for restrictions and somatic dysfunctions.

Standard Treatment

Acute Phase (approximately 1 week). The same principles apply as for ankle sprains, but for those athletes who have significantly more pain and inflammation in the acute phase, injection therapy can be considered as another form of relief. Injection therapy with either a steroid or anesthetic (or a combination of both) allows physicians to more effectively treat localized severe pain and inflammation associated with plantar fasciitis. Injections are rarely done in tendons, and some of the literature suggests that plantar fascia rupture can occur after injection of glucocorticoids.

Stretching should involve the following (12):

- Gastrocnemius-soleus complex and Achilles tendon.
- Plantar fascia with either a rolling pin (rolling the foot on top of the pin) or a frozen water bottle (which also provides analgesia and anti-inflammation).
- Night splint implementation to maintain foot and ankle dorsiflexion to help keep the Achilles tendon and plantar fascia on stretch throughout the night.

If the pain associated with plantar fasciitis is extremely severe, the athlete may want to consider using crutches (12). This is usually not necessary, but should be kept in mind when the condition has progressed to this level of impairment.

Subacute or Repair Phase (approximately 1 to 3 weeks). The athlete should be gaining full pain-free weight bearing with walking at this phase. The type of footwear and support should be modified (12). Pain management, as previously described, may be continued along with ultrasound and cross-friction massage. Ultrasound and massage techniques also help to increase blood flow to the injured area and speed the recovery process (12).

Runners should continue stretching and use of a night splint in active conditions. Once flexibility exercises no longer produce pain, weight bearing may gradually increase as tolerated.

Shock-absorbing heel cups are another valuable tool. These unload the medial plantar fascia insertion yet still allow the athlete to comfortably bear weight. Arch tapings may also help to significantly decrease excessive pronation and the subsequent pain it produces (12). Athletes with Achilles tendinitis should use heel cups with more lift to increase the slack in the tendon, which reduces the traction forces and provides pain relief.

Custom semirigid foot orthotics reduce hyperpronation. One study showed that custom orthotics significantly reduced the pain experienced with weight bearing (19). Along with a proper heel counter in supportive footwear, orthotics are crucial in managing the biomechanical factors of plantar fasciitis. Although the cost of such equipment may initially alarm athletes, proper biomechanical control in plantar fasciitis management may be hard to achieve otherwise.

Remodeling Phase (approximately 2 weeks). Athletes should be at full pain-free weight bearing with jogging and running (12). Although an athlete has progressed into different phases of treatment, previously used modalities are still acceptable. Flexibility exercises are integrated into normal training routines, along with toe flexor strengthening.

The runner should follow a gradual progressive training module. The training principles that have been previously described remain of utmost importance when returning to activity from plantar fasciitis. Key criteria that must be met before returning to running include (12)

- Proximal arch and heel are pain-free.
- Proper Achilles and plantar fascia flexibility.
- Maximal strength within the lower leg.
- Pain-free running.
- Psychologically ready for competitive running.

The use of injections is sometimes effective in recalcitrant cases. An injection of a corticosteroid around the insertion of the plantar fas-

cia is often beneficial, but steroid-induced necrosis of the calcaneal fat pad can be a negative side effect (20). Injecting the Achilles tendon sheath is done less often for controlling tendinitis, but has been reported to be effective; human studies show that *peritendinous* injections offer a significant reduction in pain without an increase in tendon ruptures (21). Injecting the Achilles tendon is not recommended due to the risk of rupture. There have been case reports of tendon rupture following tendon injection, but this finding is not supported by convincing data from controlled clinical trials (21,22). Animal studies have, however, demonstrated deleterious effects as a result of intratendinous corticosteroid injection (23). Operative treatment for both plantar fascial release or Achilles tendon débridement—are typically reserved for athletes who fail conservative therapy.

Newer therapies are being investigated, including extracorporeal shock wave therapy for plantat fasciitis and sclero therapy for neovascularization (which has been suggested as an etiology for pain in Achilles tendinitis). However, conclusive efficacy has not been demonstrated for either of these therapeutic modalities (24,25).

Manual Medicine Techniques

Many areas of somatic dysfunction are classically found in plantar fasciitis and Achilles tendinitis. In line with kinetic chain principles, each link should be examined and treated to optimize recovery. Manual techniques are effective in facilitating the recovery of repetitive soft tissue injuries in conjunction with standard treatment.

Direct Muscle Energy for a Plantarflexed Talus

Rationale: The talus is positioned in plantarflexion (will not dorsiflex). Restoring normal talar motion will lessen strain on other components of the foot, lessening the cumulative microtrauma on the plantar fascia.

1. The athlete is sitting on the table with the clinician in front. Using the lower hand on the athlete's ankle, apply a caudad force down on the anterior talar body and neck.
2. With the other hand, grasp the forefoot and apply a cephalad force to dorsiflex the foot (Fig. 24.3.6A).
3. Have the athlete gently (1 to 3 lb of force) plantarflex against your resistance, and then relax.
4. Dorsiflex the talus to the new barrier and repeat the contraction-relaxation phase three to four times.

Direct HVLA Technique for a Plantarflexed Talus

Rationale: The talus is restricted in plantarflexion (will not dorsiflex). Restoring normal talar motion will lessen strain on other components of the foot, lessening the cumulative microtrauma on the plantar fascia.

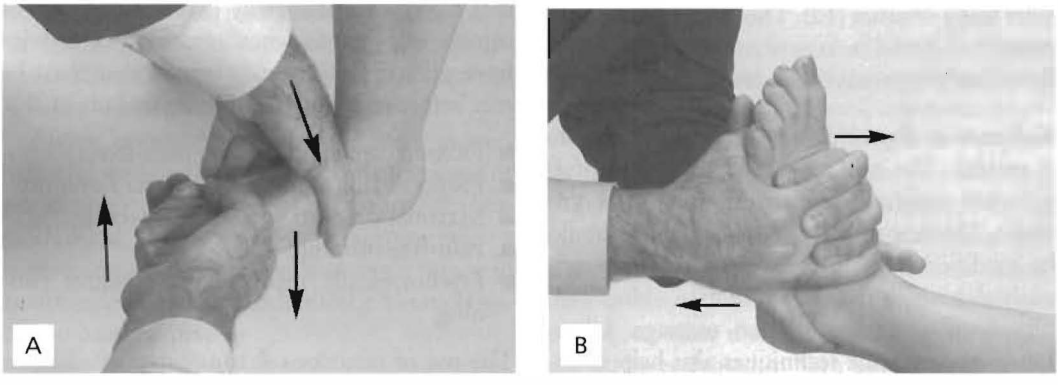

FIGURE 24.3.6. Plantarflexed talus treatment. **A,** Direct muscle energy for a plantarflexed talus. **B,** Direct high-velocity, low-amplitude technique for a plantarflexed talus.

1. Grasp the athlete's foot with the fingers interlaced around the head of the talus.
2. Dorsiflex the ankle to the barrier and apply a short, quick pull in a caudal direction (Fig. 24.3.6B).

Direct HVLA Technique for an Anteromedial Talus

Rationale: The talus moves anteromedially on the calcaneus and is restricted in posterolateral motion. The lateral malleolus may be posterior. Restoring normal talar motion will lessen strain on other components of the foot, lessening the cumulative microtrauma on the plantar fascia (Fig. 24.3.7A).

1. The clinician grasps the athlete's heel with the palm of his or her hand.
2. With the other hand, press the hypothenar eminence against the head of the talus on the medial side of the foot.
3. Apply a short, quick thrust against the talar head posterolaterally while thrusting the calcaneus medially (Fig. 24.3.7A).

Alternative Method. An indirect method may also be used.

1. The clinician grasps the heel with the palm of the hand.
2. Traction is applied in a caudal direction.
3. Use the thumb and index finger on the talar head to monitor and move the talus to disengage the restrictive barrier.
4. Hold until a release is felt (Fig. 24.3.7B).

Direct HVLA Technique for a Posterolateral Talus

Rationale: The talus moves posteromedially on the calcaneus and is restricted in anterolateral motion. The lateral malleolus may be anterior. Restoring normal talar motion will lessen strain on other components of the foot, lessening the cumulative microtrauma on the plantar fascia.

1. The clinician grasps the heel with the palm of the hand.
2. With the other hand, press the hypothenar eminence against the head of the talus on the lateral side of the foot.
3. Apply a short, quick thrust against the talar head anteromedially while thrusting the calcaneus laterally (Fig. 24.3.8A).

Alternative Method. An indirect method may also be used.

1. The clinician grasps the heel with the palm of the hand.

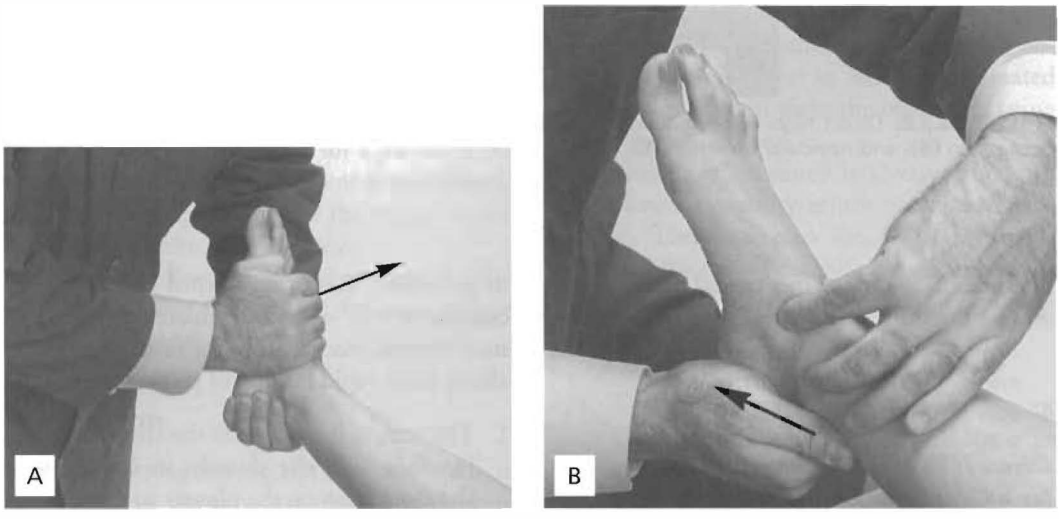

FIGURE 24.3.7. Anteromedial talus treatment. **A,** Direct high-velocity, low-amplitude technique. **B,** Indirect method.

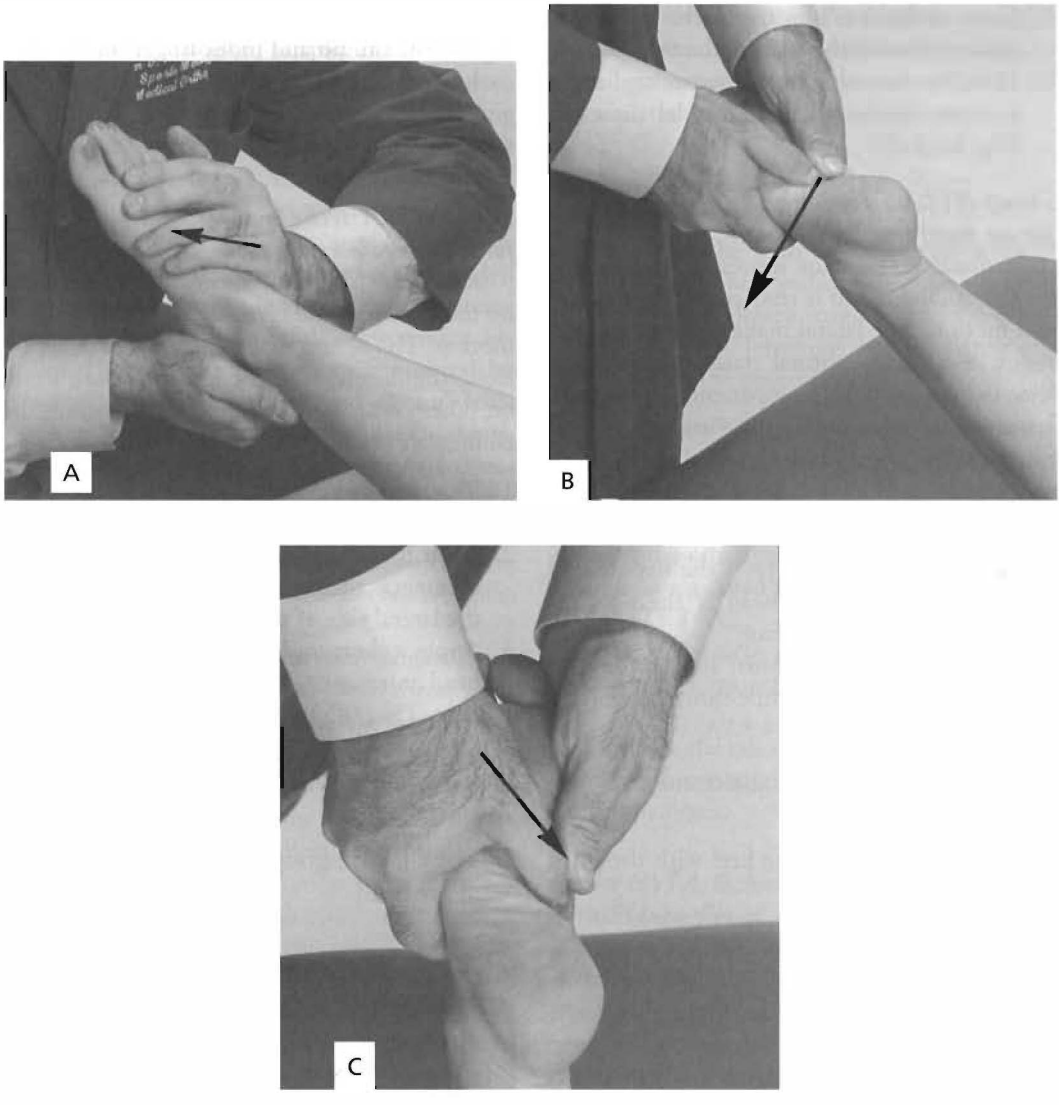

FIGURE 24.3.8. Direct high-velocity, low-amplitude technique for a posterolateral talus **(A),** cuneiform depression **(B),** and navicular inversion **(C).**

2. Traction is applied in a caudal direction.
3. Use the thumb and index finger on the talar head to monitor and move the talus to disengage the restrictive barrier.
4. Hold until a release is felt.

Direct HVLA Technique for a Cuneiform Depression

Rationale: The cuneiform is positioned more toward the plantar surface and is tender to palpation. Restoring normal motion to the cuneiform will lessen the athlete's pain and restore normal mechanics of the foot. This in turn allows more rapid healing of plantar fasciitis.

1. The athlete lies prone as the clinician grasps the foot with the thumbs in contact with the cuneiform on the plantar surface.
2. The clinician moves the foot from medial to lateral and halfway between the medial and

lateral barriers, applying a short, quick thrust downward (Fig. 24.3.8B).
3. Retest.

Direct HVLA Technique for a Navicular Inversion

Rationale: The navicular is held with the plantar surface more medial and is tender to palpation. Restoring normal motion to the navicular will lessen the athlete's pain and restore normal mechanics of the foot. This in turn allows more rapid healing of plantar fasciitis.

1. The athlete lies prone while the clinician grasps the foot with the thumbs in contact with the navicular on the plantar surface.
2. The clinician moves the foot from lateral to medial and applies a short, quick thrust against the navicular as the medial barrier is reached (Fig. 24.3.8C).

Counterstrain to the Plantar Fascia

Rationale: The fascia becomes tight and fibrotic from repetitive stress and chronic inflammation. Trigger points can develop as well. The specific area to treat is found by palpating and locating specific trigger points in the plantar fascia.

1. If the trigger point is located in the midportion of the plantar fascia, the clinician passively plantarflexes the arch to shorten the fascia.
2. The clinician then "wraps around" the trigger point, placing the athlete in a position of maximal comfort (Fig. 24.3.9).
3. This position is then held for a period of 90 seconds, and the trigger point is monitored. When in proper position, the trigger point should be reduced or painless.
4. The area is slowly returned to neutral position. This can be repeated for each trigger point found.

FIGURE 24.3.9. Counterstrain to the plantar fascia.

Prevention

Exercises. Strengthening the foot and ankle is best started in the subacute phase and continued as pain lessens and normal function is restored. Examples of these exercises follow.

1. *Towel grasp.* The patient sits in a chair and has a towel spread out on the floor with the foot resting on the towel. A thin towel will work best at first. The athlete repeatedly tries to bunch up the towel by grasping it with the toes (Fig. 24.3.10A). Repetitions and sets can be determined individually.
2. *Marble pick-up.* Marbles or other small objects can be laid out in front of the seated patient, who then picks the objects up using only his or her toes.
3. *Proprioception.* Balance is always crucial in any lower extremity injury prevention program. These exercises should be instituted toward the end of rehabilitation and continued onward. Most of these exercises are similar to an ankle sprain program.
4. *Toe raises*
   a. The athlete starts on a flat surface, or ideally a step, with the heels off the edge of the step.
   b. The athlete then performs simple toe raises with both feet in a controlled manner, with the eyes open, holding onto the

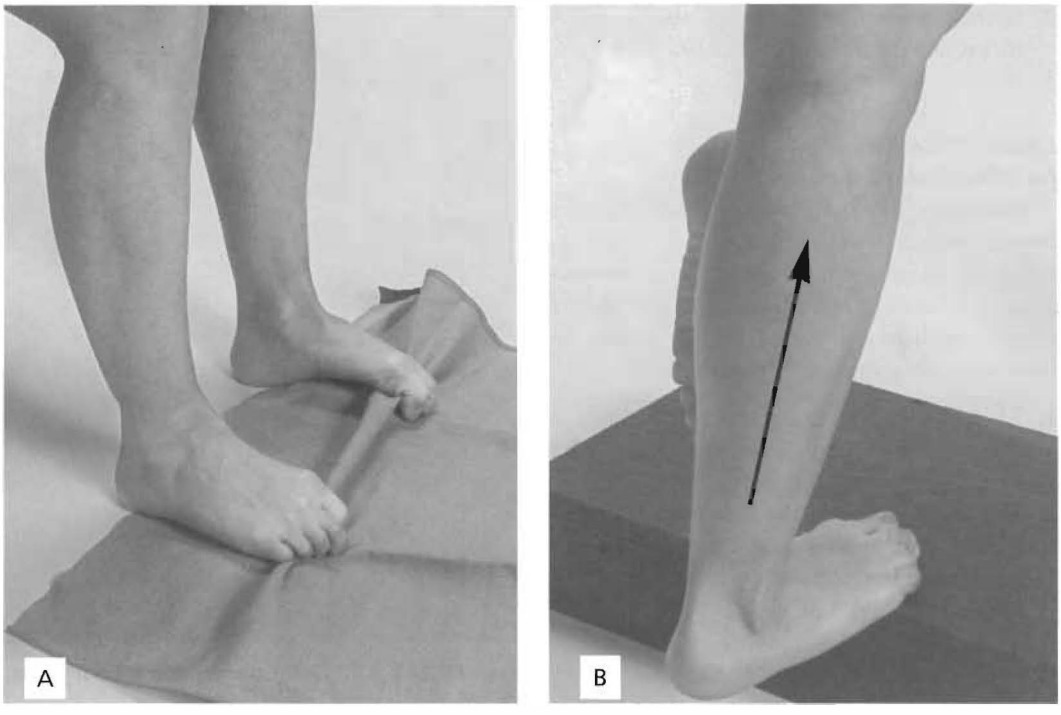

FIGURE 24.3.10. Exercises. **A,** Towel grasp **B,** Proprioception: toe raises.

wall or a banister for balance, as needed (Fig. 24.3.10B).

c. The athlete then performs the same toe raises, but using one side only.
d. The athlete closes his or her eyes and performs toe raises using both feet and holding onto a wall or banister, as needed for balance. This will start developing proprioception.
e. The athlete continues to progress in a stepwise fashion until he or she is able to do unilateral toe raises with the eyes closed and without help.

Other Factors

Athletes may need to make certain modifications, such as their shoes (and possibly orthotics), running surface, avoiding excessive hills, reduction of their training schedule, or changes in their pattern. Weight loss is imperative for overweight patients. Appropriate maintenance of strength, flexibility, and proper mechanics are key components of preventing recurrence of this notoriously chronic and persistent problem.

FOOT AND ANKLE STRETCHES

Gastrocnemius Muscle, Posterior Tibialis Muscle, Plantaris Muscle. The athlete puts the hands against a wall with one foot behind and one foot forward. The back foot stays flat on the ground, while the back leg is fully extended. The athlete leans in toward the wall, dorsiflexes the back ankle, and holds the stretch for 20 to 30 seconds (Fig. 24.3.11A). A variation of this includes pointing the foot in and out to stretch medial and lateral gastrocnemius heads. Good stretch for runners, basketball, volleyball, hand/racquetball, tennis, dancers, and gymnasts.

Soleus Muscle. The setup is the same as for the gastrocnemius stretch, but the back foot is closer to the wall, and the back knee is bent

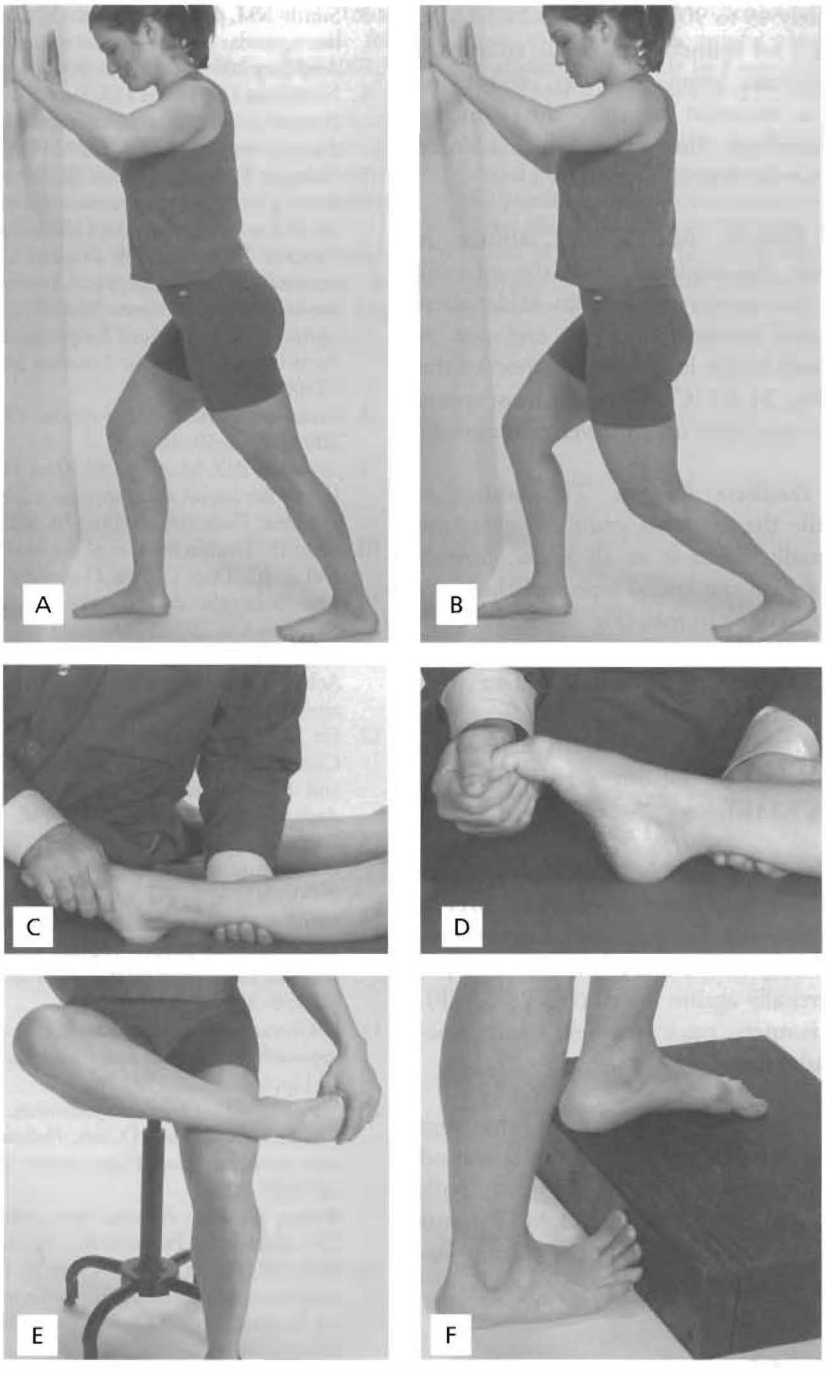

FIGURE 24.3.11. Stretches for foot and ankle. **A,** Gastrocnemius, posterior tibialis, plantaris muscle. **B,** Soleus. **C,** Anterior tibialis. **D,** Extensor hallucis longus. **E,** Leg and foot extensors. **F,** Plantar fascia, flexor hallucis brevis, flexor hallucis longus, flexor digitorum brevis, flexor digitorum longus, and lumbricals.

approximately 45 to 90 degrees (Fig. 24.3.11B). Good stretch for runners, basketball, volleyball, hand/racquetball, tennis, dancers, and gymnasts. In a variation of this, the clinician forcibly dorsiflexes the prone athlete's ankle with the knee bent at 90 degrees.

Anterior Tibialis Muscle. The athlete is supine while the clinician holds the athlete's right foot and moves the foot in ankle plantarflexion and eversion or down and out. A stretch should be felt in the lateral aspect of the right leg (Fig. 24.3.11C). Good for most sports that involve repetitive use of lower extremities.

Extensor Hallucis Longus. The athlete is supine while the clinician grabs the great toe and maximally flexes it at all joints. Stretch should be felt in the lateral aspect of the right leg or dorsum of right foot (Fig. 24.3.11D).

Extensors of the Leg and Foot. Sitting with right foot on the left leg, the athlete flexes the toes of the right foot with the left hand, so a stretch is felt on the dorsum of right foot and leg (Fig. 24.3.11E).

Plantar Fascia, Flexor Hallucis Brevis, Flexor Hallucis Longus, Flexor Digitorum Brevis, Flexor Digitorum Longus, and Lumbricals. The athlete keep all five toes of the right foot positioned vertically against a wall (Fig. 24.3.11F). Good for runners, track and field events, soccer, and basketball.

Variation. The athlete sits with the legs straight out in front and wraps a towel around the forefoot and toes. The athlete gently pulls the towel to bring the ankle and MTP joints into extension, thus stretching the plantar fascia and the flexor tendons.

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25

GAIT ANALYSIS

PAUL TORTLAND

A well-known physical therapist once said, "When the foot hits the ground, the mud hits the fan." His point was that there are inextricable links between foot mechanics, gait, and human performance. Volumes have been written about gait analysis, and a complete discussion of this topic is beyond the scope and purpose of this chapter. Rather, the goal here is to discuss functional foot and gait mechanics and the influence on—and by—the spine within an osteopathic framework.

To understand how gait disturbances can play a role in sports injuries (and how more proximal injuries can affect gait mechanics), one must first have a basic understanding of the gait cycle and normal gait mechanics.

THE GAIT CYCLE AND NORMAL MECHANICS

The Gait Cycle

The gait cycle is routinely divided into two phases, swing and stance. Alternatively, gait may be divided into single-support and double-support phases. The gait cycle is defined as the processes that occur between two successive occurrences of one of the repetitive events of walking (1). The start of the gait cycle is generally accepted as beginning with heel strike, which initiates the stance or initial double-support phase. The swing phase begins with toe-off of the same limb, and ends with heel strike, thus completing one cycle. This is illustrated in Figure 25.1 (2).

In normal walking 60% of the gait cycle encompasses the stance phase, and 40% is the swing phase. Ten percent of the gait cycle consists of double support, that portion between heel contact of one limb and toe-off of the opposite limb when both limbs are in full contact with the ground. This ratio, however, can be dramatically altered depending on the speed of walking or running. In fact, the disappearance of double support marks the transition from walking to running (1).

Foot Mechanics

The key joints of the foot related to gait are the talocrural joint (tibia-fibula–talar dome articulation), the subtalar joint, the midtarsal joints, and the metatarsophalangeal (MTP) joints.

Dananberg has described three "rocker" mechanisms that occur in sequence in the foot during the gait cycle (3). The first is the rounded bottom of the calcaneus that "pivots" during and through heel strike. The second is the dorsiflexion that occurs at the talocrural joint, described subsequently (3). The third is the extension that occurs at the MTP joints.

During gait, the talocrural joint must allow for 10 degrees of dorsiflexion from the point of full ground contact in midstance in order for the body's center of gravity to pass forward normally. The talar dome is wider posteriorly than anteriorly. As the joint moves into dorsiflexion, the fibula moves laterally and proximally to allow the mortise joint to widen to accommodate the wider posterior portion of the talus. At the same time, the distal tibia-fibula syndesmosis stores energy, preparing for the ensuing plantarflexion.

The subtalar joint (the articulation between the talus and the calcaneus) may be viewed as a type of differential gear, capable of converting limb rotation into pronation and supination (3). During heel strike, the tibia, femur, and

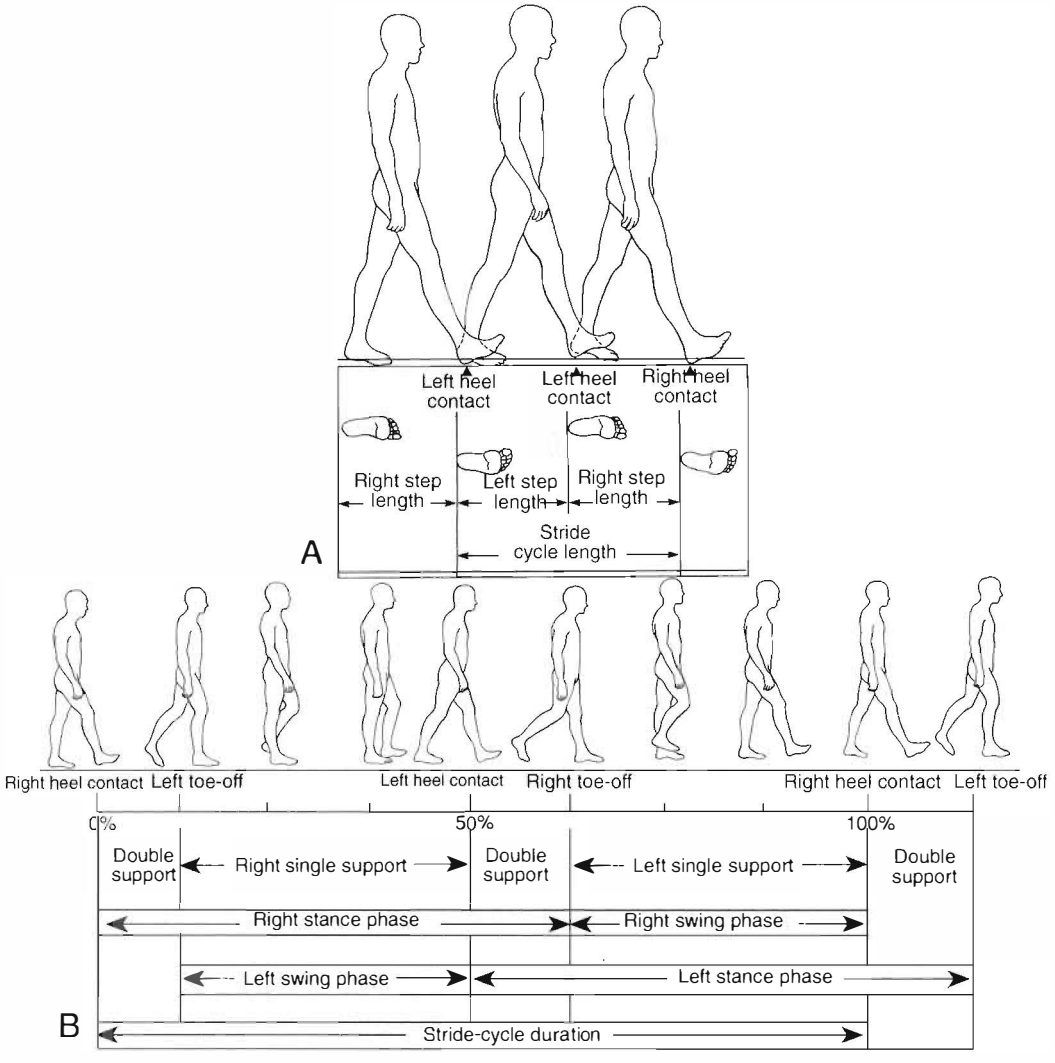

FIGURE 25.1. The gait cycle. (From Shumway-Cook A, Woollacott MH. Control of normal mobility. In: *Motor control*. Baltimore: Williams & Wilkins, 1996).

pelvis are rotating internally. Under this load the subtalar joint pronates.

Much ado is made in the clinical and popular recreational sports literature about pronation. Pronation is a combination of talar eversion, forefoot abduction and dorsiflexion, along with flattening of the medial, anterior, and lateral arches. It is a *normal* occurrence that begins after heel strike and ends just before midstance, comprising no more than 25% of the gait cycle (4). Pronation ends once the leg and foot begin to externally rotate in preparation for toe-off. Pronation is necessary to transform the relatively flexible and accommodating foot into a more rigid platform from which to push off.

Likewise, supination is a normal mechanical event that begins at midstance. The limb that was rotating internally at heel strike begins to rotate externally as the opposite limb pulls forward and rotates the pelvis. The subtalar joint therefore moves into supination (a combination of talar inversion, forefoot adduction, and plantarflexion) to facilitate this transition.

The midtarsal joints act as a mechanical link between the ankle–subtalar complex and the metatarsophalangeal joints. They also act as termination points for most of the extrinsic and intrinsic muscles acting on the foot and ankle. Finally, they act as close-packing mechanical links when loads are applied, facilitating the transition of the foot from a flexible accommodating structure to a rigid propulsive structure, as will be seen later in the chapter.

Finally the MTP joints form the third and final pivot point of the normal rocker motion of the foot during gait.

The first MTP joint is the most important component of this final pivot. The plantar fascia has its strongest distal attachment (50% of its entire thickness) at the base of the proximal first phalanx. Also the motion of the first MTP joint is critical for normal gait to occur. The normal total available range of motion at the joint is 65 degrees. This amount of motion is necessary to account for normal movement of the torso and leg. As the body's center of gravity passes through midstance, the hip extends 20 degrees. At heel lift, the ankle plantarflexes 20 degrees and the knee flexes 45 degrees. The first MTP joint must flex accordingly to accommodate these movements and allow the body to pass smoothly through toe-off.

The Role of Muscles

Conventional wisdom maintains that the legs are the motive force in locomotion, bringing the passive torso along for the ride. However, more recent and detailed electromyographic analysis has demonstrated that the muscles of the lower extremity fire predominantly in an eccentric contraction, rather than the concentric contraction one would expect if these muscles were prime movers (5).

Gracovetsky proposed the theory of the *spinal engine* to describe the central role of the spine in the gait process. Essentially, the spine initiates gait by generating pelvic rotation. Accordingly, the lower extremity follows in a pendulum manner, and the muscles of the leg generally fire eccentrically, acting as a "brake" to slow down the limb movement and to generate stability. At heel strike, forces are transmitted up through the leg into the knee, hip, sacroiliac joint, and spine. The entire process is a carefully orchestrated pattern of movements involving the muscles, joints, and myofascial elements of the spine, pelvis, and lower extremity (6).

GAIT DYSFUNCTION

The average adult completes 2,500 stance–swing cycles per limb per day, assuming an average of only 80 minutes per day of weight-bearing activity. This equates to approximately 1,000,000 steps per year for each limb. These numbers can go significantly higher in athletic populations, especially in sports involving running, where the actual amount can double or even triple (7). Any perturbations in normal gait mechanics may result in cumulative adverse structural and performance compensations.

Pes Planovalgus

Much ado is made about "overpronation" and flat feet as a cause of gait disruption (Fig. 25.2). Pronation is a normal and even necessary event in proper gait mechanics. Nonetheless, excessive pronation can be problematic. Abnormal pronation continues past midstance through toe-off.

One of the major effects of pes planovalgus is that it alters the normal timing of events in the gait sequence by retarding the return of the foot

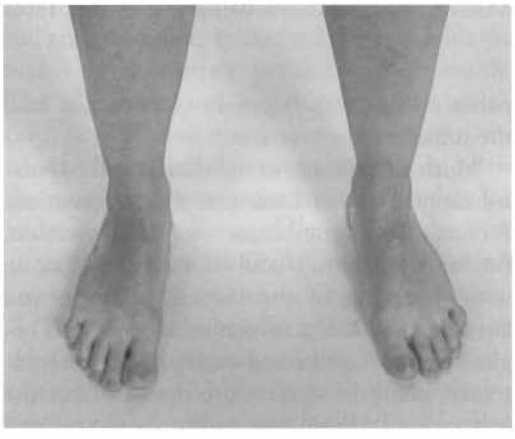

FIGURE 25.2. Pes planus, or flat feet.

to supination to enable toe-off. The altered timing changes the forces acting on the proximal joints. Altered forces can strain the sacroiliac and lumbosacral joints, leading potentially to instability and abnormal thickening of the inguinal ligament (4). At the knee it can create a valgus stress.

In addition, flattening of the medial longitudinal arch internally rotates the lower extremity and hip, tightens the iliopsoas, and induces anterior innominate rotation and an increased sacral angle and lumbar lordosis. The sacroiliac joint can rotate downward, stretching the pitiformis and sacrospinous ligament, potentially causing piriformis trigger points or sciatic nerve entrapment.

Increased fascial and muscle tension can also produce myofascial symptoms. For example, the tibialis posterior muscle attaches along the posteromedial aspect of the tibia. Its tendon runs distally behind the medial malleolus and continues under the medial arch to insert on the plantar surfaces of the medial aspects of the cuboid, navicular, first and second cuneiforms, and sustentaculum tali.
stress on the tibialis posterior and can result in tendinitis or periostitis. From an osteopathic perspective, this myofascial strain can continue proximally into the medial thigh and perineal region.

In a similar manner, pes planovalgus can lead to a chronic shortening of the Achilles tendon and gastrocnemius-soleus complex, and tightness in the hamstrings, tensor fasciae latae, erector spinae, and quadratus lumborum. Because this is a recurring functional problem, the muscle tightness will not respond appreciably to stretching regimens until the culprit is addressed.

Lastly, due to the loss of shock absorption in pes planovalgus, increased stress on the osseous structures of the lower extremity can increase the risk of stress fractures.

Pes Cavus

Pes cavus, or high-arched foot, represents a combination of subtalar inversion forefoot adduction, and plantarflexion, with an elevated medial longitudinal arch. A cavus foot is more rigid and therefore poorly accommodating to the impact forces directed into the foot. In addition to compromised shock-absorbing capacity, a cavus foot stresses the lateral aspect of the knee joint, increases tension through the tensor fasciae latae and iliotibial band, and functionally shortens the Achilles tendon–gastrocnemius-soleus complex.

The effect on gait from a cavus foot can be varied. Knee pain (both from direct stress and from influence from the tightened tensor fasciae latae/iliotibial band), hip pain, and low back pain all can result.

Sagittal Plane Blockade

One of the most overlooked causes of gait dysfunction is sagittal plane blockade. As noted previously, sagittal plane motion of the foot (the three rockers) is critical to normal gait. Any conditions that restrict the motion of one or more of these rockers serve to alter gait mechanics, more than a million times a year with nothing more than regular walking.

Anterior Tibia on Talus

Failure of the talocrural joint to achieve the 10 degrees of dorsiflexion during midstance is one form of sagittal plane blockade. One common reason for this is the restriction in which the tibia is driven forward relative to the talus. This can occur, for example, when a soccer player attempts to kick the ball at the same time that an opposing player also kicks the ball from the opposite direction. The foot is suddenly decelerated, yet the momentum of the lower leg continues to drive the tibia forward.

As the tibia slips forward on the talus, the ability of the talocrural joint to dorsiflex becomes restricted. This initiates an impediment to sagittal plane motion through midstance. Compensatory mechanisms may be engaged, such as early knee flexion or activation of the calf muscles to help "lift" the foot past the restriction. Gradual onset of pain may be experienced in the foot and ankle, Achilles tendon and calf region, or knee. Clinically, there is typically decreased and/or painful passive ankle dorsiflexion.

High-velocity, Low-amplitude Technique for Anterior Tibia on Talus

1. The athlete lies supine, with the clinician standing at the foot of the table facing the athlete.
2. The clinician grasps the heel of the involved leg by cupping the heel with one hand, and gently moves the ankle into mild dorsiflexion.
3. The clinician places the heel of the opposite hand directly over the anterior aspect of the distal tibia, just above the ankle joint.
4. Then the clinician gently pushes down on the tibia, taking up the "slack" in the soft tissue, until a firm end point is appreciated. He or she delivers a high-velocity, low-amplitude (HVLA) thrust by quickly pushing down on the distal tibia (Fig. 25.3). An articulatory pop may be appreciated but is not necessary for correction.

Talocrural Restrictions

The talocrural joint is formed by the distal tibia and fibula and the talar dome. It is also known as the "mortise" joint and represents the ankle joint proper. Restrictions of this joint can likewise impede the normal sagittal plane motion of the joint as the leg comes into and through midstance.

The most common cause of restrictions to the talocrural joint is inversion ankle sprains. In an inversion sprain, the foot typically is in a plantarflexed and inverted position. The talus follows and often becomes functionally restricted in this position.

Manual Technique: Talar Release. The goal of treatment is to restore the functional motion of the talus in the mortise joint (see Chapter 24.3, Foot and Ankle: Common Conditions).

Tarsal Navicular Restriction

The tarsal navicular occupies an important position as the "keystone" of the medial longitudinal arch. An intact and kinetically functional medial arch is crucial for normal gait mechanics, as it is through this arch that most of the forces acting through the lower extremity and foot are transmitted. The arch must sequentially serve first as a flexible shock absorber, followed by a rigid force transmitter to enable toe-off.

A common somatic dysfunction involves the navicular dropping or rotating inferomedially, resulting in partial functional collapse and dysfunction of the normal arch mechanics. Most navicular dysfunctions are asymptomatic, so an understanding of functional gait mechanics and a high index of clinical suspicion are necessary to consider navicular lesions (see Chapter 24.3, Foot and Ankle: Common Conditions).

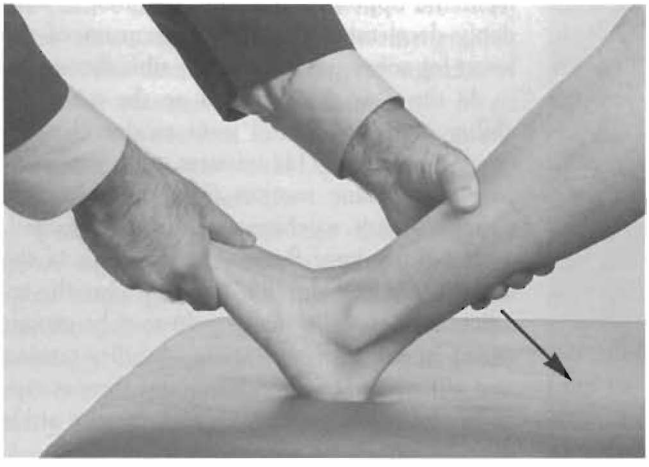

FIGURE 25.3. High-velocity, low-amplitude technique for anterior tibia on talus.

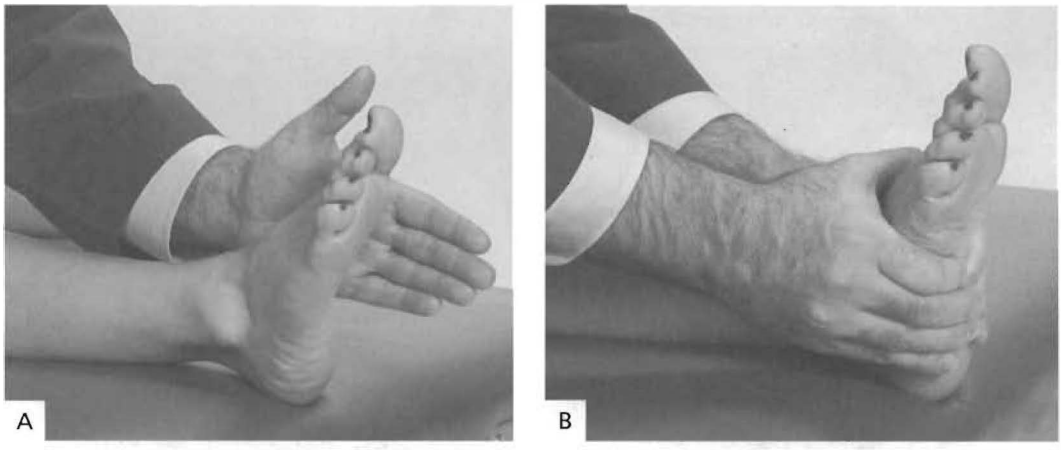

FIGURE 25.4. Treatment for tarsal navicular restriction. **A,** Proper position of the hypothenar eminence against the tarsal navicular. **B,** Proper hand position to introduce distracting impulse thrust.

Manual Treatment: Tarsal Navicular Restrictions

1. The athlete lies supine on the table while the clinician stands at the side of the table of the affected limb facing the feet.
2. The clinician identifies the navicular and places the hypothenar eminence of the hand closest to the table directly over the navicular, with the forearm parallel to the athlete's shin (Fig. 25.4A).
3. The clinician grasps the foot comfortably with both hands, maintaining contact over the navicular. Keeping the ankle in neutral (neither eversion nor inversion), he or she brings the ankle into dorsiflexion (Fig. 25.4B).
4. Using the forearm and hand, the clinician applies axial traction directed inferiorly, distracting the medial longitudinal arch. Once all the "slack" is taken up and the end point is reached, he or she applies a quick HVLA thrust. If the navicular is restricted, an articulatory pop is typically noted.

Functional Hallux Limitus

As pointed out earlier, the last, and perhaps most important, rocker mechanism in the foot facilitating sagittal plane motion is the first metarsophalangeal joint. Functional hallux limitus (FHL) represents a locking of the normal joint dorsiflexion. Accordingly, the ability to allow forward movement while simultaneously close-packing and aligning the medial arch column never materializes. This results in a sagittal plane block, effectively preventing the body's center of gravity from moving forward. As an analogy, imagine pushing a wheelbarrow when suddenly the wheel hits a rock. This suddenly blocks forward movement.

FHL is distinguished from hallux rigidus in that FHL is a ***functional*** restriction; that is, it is only noted in a position of function (weight bearing), whereas hallux rigidus manifests in all positions.

The diagnosis of FHL is made with the athlete seated or supine, with the leg and foot in a non–weight-bearing position. The clinician grasps the first metatarsal and passively dorsiflexes the first MTP joint by pushing dorsally on the great toe. There should be up to 65 degrees of motion (Fig. 25.5A). Next, apply pressure on the plantar aspect of the first metatarsal head, moving it into dorsiflexion (simulating a weight-bearing position). Again, apply passive dorsiflexion to the great toe. A range of motion of 15 degrees or less indicates FHL (Fig. 25.5B).

The problem with FHL is that the athlete rarely, if ever, presents with symptoms involving the MTP joint. FHL manifests clinically

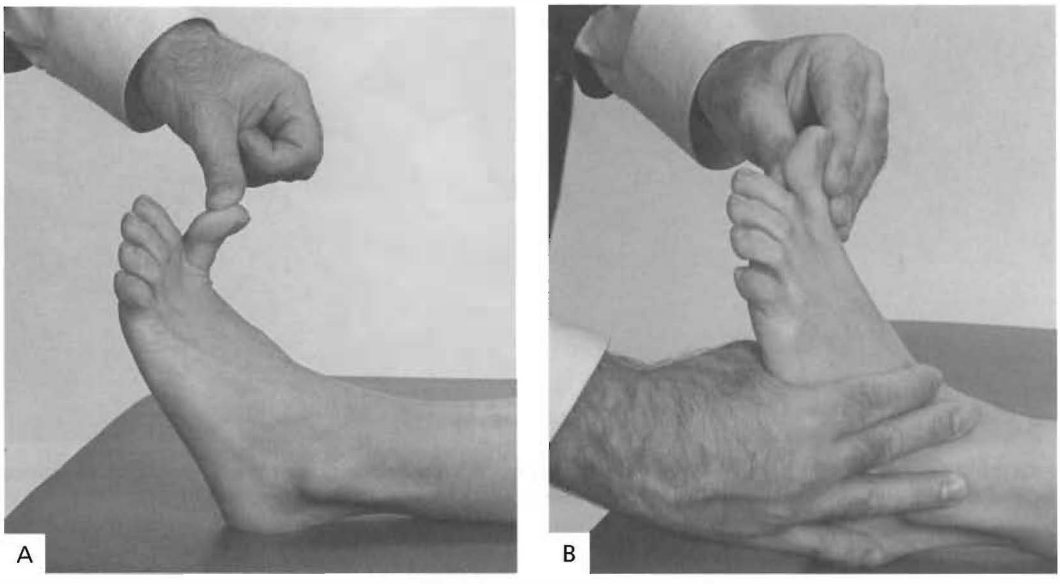

FIGURE 25.5. Treatment for dysfunction at the first metatarsophalangeal (MTP) joint. **A,** Normal range. **B,** Treatment position for functional hallux limitus at the first MTP joint.

through various compensatory mechanisms at alternative sites that attempt to provide the necessary motion for forward momentum (7). These sites can include the knee, hip, low back, upper back, and neck.

Because the general joint motion at these sites is primarily in extension during gait to maintain an erect torso, the MTP joints in general, and the first MTP joint in particular, provide the dorsiflexion necessary to permit forward movement of the body.

According to Dananberg, the most evident effect of FHL involves hip motion. During normal gait, there is approximately 15 degrees of hip extension by the end of the single-support phase (7). This extension allows the upper body to remain erect, allows for thrust against the support surface, ideally positions the limb to prepare for the swing phase, and reduces the angle between the back of the leg and the ischial tuberosity.

FHL, however, causes the hip to move into *flexion*, rather than extension. This causes lumbar flexion versus extension, thereby potentially creating both disc compression and activation or overuse of the lumbar paraspinal muscles. Loss of the thrust mechanism requires increased muscular activation of the hamstrings, gluteal muscles, and quadriceps. The swing mechanism is adversely affected by loss of hip extension also. The iliopsoas is inappropriately recruited to aid in lifting the lower extremity to facilitate forward swing, causing muscular fatigue and biomechanical compensation into the mid and upper back, neck, and shoulder girdle. Lastly, loss of "angle closure" between the posterior thigh and ischial tuberosity halts normal pelvic rotation and creates tension in the biceps femoris. Athletes often present with hamstring tightness that is not relieved by stretching.

In addition, postural changes may slowly develop. Because the sagittal plane blockade prevents the body's center of gravity from moving forward, often a gradual forward slouching posture may develop as a way of trying to shift the center of gravity more anteriorly to facilitate forward movement. Returning to the wheelbarrow analogy where forward movement is halted when the wheel hits a rock, to push the wheelbarrow

over the rock one typically leans forward to shift the center of gravity and gain more momentum to "drive" the wheelbarrow over the restriction. The slouching posture can instigate neck and upper back pain, and the scapular protraction can predispose to developing shoulder and rotator cuff pathology.

Manual Medicine Techniques

Since FHL is a functional problem, treatment is directed at promoting more appropriate first MTP joint motion and reducing the adverse influences on the systems noted earlier. Techniques such as mobilization, myofascial release, stretching, strengthening, and gait training all fail to address the problem.

FHL is best addressed by fabricating a custom foot orthosis that reduces the negative impact of the FHL. A specific adaptation is incorporated into the device whereby the orthotic is "clipped" at the first metatarsal head and a cutout is made in the padding under the first metatarsal head. This adaptation allows the first metatarsal head to drop slightly into plantarflexion and medial rotation. The result is that, when the foot moves into toe-off, the second through fifth metatarsals will begin to dorsiflex before the first MTP joint is engaged. This retards the onset of sagittal plane blockade until after the body's center of gravity has moved sufficiently forward.

OTHER GAIT-RELATED INFLUENCES

Not all gait dysfunction is the result of foot and ankle problems. Leg-length discrepancy, sacroiliac joint (SIJ) dysfunctions, and muscle imbalances in the trunk, pelvis, and lower extremity all can influence the biomechanics of the lower extremity, thereby influencing gait.

One of the most important—and often overlooked—areas of dysfunction is the SIJ. As the leg enters the swing phase of gait, the ipsilateral innominate rotates posteriorly relative to the sacrum, thereby increasing tension of the SIJ ligaments and preparing the joint for heel strike. Likewise, during the single-leg stance phase, the innominate starts to rotate anteriorly on the sacrum. The long dorsal sacroiliac ligament resists this motion (8). Therefore, loss of the ligamentous support of the SIJ leads to instability of the joint, adversely affecting the normal biomechanics of the pelvis and, eventually, the lower extremity. This may result in a variety of pain symptoms including foot and ankle pain, knee pain, hip and low back pain, and neck and upper back pain.

Along the same lines, loss of strength and control of the trunk and pelvic muscles can lead to myofascial instability through the pelvic girdle. This results in either increased stress on the SIJ ligaments, increasing the risk of premature ligament failure and subsequent SIJ dysfunction, or compensatory muscle activation, resulting in altered biomechanics. For example, SIJ dysfunction often leads to inhibition of the gluteus maximus, resulting in shortening of stride length. The hamstrings are then recruited and overused to compensate for loss of hip extensor power (8). The result can be a predisposition to recurrent hamstring strains, patellofemoral dysfunction (since the hamstrings play a major role in controlling the rotation of the thigh), or both.

Myofascial instability of the lumbosacral region can put increased strain on the iliolumbar ligament. The quadratus lumborum, which is a prime stabilizer of the lumbar spine, attaches inferomedially directly to the iliolumbar ligament and can become inappropriately activated if the ligament is compromised. By way of its inferolateral attachment to the ilium, the activated quadratus lumborum can alter normal innominate motion during gait, causing gait abnormalities.

Likewise, if other myofascial conditions give rise to quadratus lumborum activation, gait can similarly be affected. For example, habitual one-legged standing can cause asymmetrical activation of the trunk and pelvic muscles (9). Likewise, athletic activity that emphasizes asymmetrical muscle activation (such as the tennis serve or golf swing) can cause muscular, ligamentous, and myofascial imbalances, all of which can affect gait.

There is an extensive and continuous myofascial connection from the foot up through the lower extremity and SIJ/pelvis, and into the thoracolumbar region (10). Disruption anywhere along this kinetic chain can have adverse consequences for gait.

Treatment therefore is directed first at identifying the presence of any of these problems, and then second, employing appropriate osteopathic, rehabilitative, and corrective interventions.

CORRECTIVE FOOT ORTHOTICS

Treatment of both planus and cavus foot problems involves the use of corrective foot orthoses. Orthotics are more than just "arch supports." Ideally they are motion control devices, intended to restore both the normal timing of the sequence of events as well as the degree to which each event occurs.

For athletes with planovalgus, orthotics can prevent excess motion while still allowing normal pronation to occur. In athletes with cavus feet, orthotics serve to increase shock absorption and to help initiate normal motion.

Orthotics that are semirigid are preferred to rigid thermoplastic devices. Rigid devices prevent the foot from moving normally and can be counterproductive. They also tend to be more uncomfortable. Semirigid devices can control excess motion while still allowing normal motion.

CONCLUSION

Understanding and addressing gait-related issues involves awareness of the intricate interplay among biomechanics, muscle activation, and kinesiology. Disruption in any of these systems can affect gait, which then can disrupt any or all of the complementary systems.

The physician is therefore presented with two challenges. First is the need to persevere and remain diligent when the apparent cause of the problem is not readily evident or when the athlete is not responding as expected to treatment. Second is the realization that the root cause of the problem may be asymptomatic and/or located remotely from the site of the presenting complaint. Knowledge of the interrelationships of systems involved in gait and a high index of clinical suspicion are necessary. A holistic and comprehensive approach that considers the whole-body interrelationship of the mechanical, postural, and myofascial aspects of gait offers the best hope of identifying and correcting the precipitating factors.

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SECTION

IV

SPECIFIC SPORTS

26

BASEBALL

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OVERVIEW

Baseball is one of the oldest and most popular spectator sports today. It has been played in the United States since the mid-1800s, although pinpointing its exact origin is debated to this day. Supposedly, Abner Doubleday created the game in 1839, but there is little support for this story, not to mention evidence of versions of the game as far back as 2400 B.C. Egypt. Then called "seker-hemat") batting the ball) (1). What is certain is the first professional game was played on May 4, 1871 between Forest City and Fort Wayne, and baseball never looked back (2). Today, the game is played worldwide, most notably in the Dominican Republic, Cuba, and Japan. Professional baseball
tors to ballparks each year and entertains millions more through radio and television broadcasts.

THE ATHLETE

Baseball values all-around ability, particularly in the younger players, yet many are successful in performing one or two skills well. The two most essential skills of the game are throwing and hitting the ball. Most players who go on to play baseball competitively can do both reasonably well, but almost all ballplayers have to be able to hit to be successful. Both skills are extremely complex activities that use multiple abilities and faculties.

Players are divided into pitchers and fielders. Both hit in the lineup, and fielders are usually everyday players. Because of the stress on their arms, pitchers who start games throw every fourth or fifth day, while relievers may pitch every other day, but for fewer innings. In younger levels of baseball, pitchers may play in the field on nonthrowing days. In collegiate and professional baseball, pitchers usually just pitch and do not hit in games when they are throwing.

Players need to perform different tasks during a game, depending on the position. The following are tasks that the typical baseball player needs to do during a game:

1. Swing a bat to hit a pitched ball (hand-eye coordination).
2. Run fast from base to base or in the field to get to a hit ball (speed).
3. Catch a ball that is pitched, airborne, or hit on the ground (agility).
4. Throw a ball hard and accurate from the outfield, infield, or pitcher's mound (arm speed and torque).

The athlete's metabolism is primarily anaerobic, mixing sprints into significant periods of inactivity, either from sitting on the bench or standing in the field. Therefore, flexibility and muscle endurance are necessary to avoid muscle injuries. Off-season workouts help to develop this, but in-season flexibility and strength training must be done to keep off-season gains.

The repetitive swinging of a bat and pitching a ball thousands of times a season demands strong core strength, which involves the abdominal region and lower lumbosacral spine and pelvis.

THROWING

Basic Biomechanics of the Shoulder and Elbow

The primary motions that occur during throwing are abduction, horizontal adduction, inter-

nal rotation, and external rotation. Shoulder abduction is a motion that should stay constant throughout throwing. The arm stays abducted approximately 90 to 100 degrees until the ball is released. This has been identified as the ideal angle for the throwing arm, and variance in this angle alters the stress load and increases risk of injury. The shoulder has less hyperangulation forces with lower ball deliveries, which may explain fewer injuries with pitchers who deliver side arm, or "submariner" style. However, the side-arm delivery is not the optimal angle to release the ball because the ball's flight is flat and easier to hit. The change in trunk position also can lower the arm and change the abduction angle as well.

The motion of horizontal adduction is an important position relative to hyperangulation. There is approximately 30 degrees of motion between a horizontal abduction position and a horizontal adducted position. The elbow is flexed approximately 90 degrees and quickly extends to 20 degrees (3,4,5).

Wilk et al. have recently described a concept called total motion (5). Total motion is the thrower's maximal external rotation plus maximal internal rotation. Many have published findings that pitchers have greater amounts of external rotation than position playets do (6,7). However, this is compensated for by a decrease in internal rotation. Some theorize that the posterior capsule scars and tightens with repetitive throwing, leading to restriction of internal rotation, particularly if the athlete becomes a thrower later in the teen years. Others suggest that the glenoid itself remodels during bone growth to a point where internal rotation has a bony block. The concept of total motion takes this into consideration, since a pitcher's normal arc of motion is shifted compared with position players and his or her nondominant arm, but the total motion is nearly the same for both.

Throwing Phases

The baseball pitch can be categorized into specific phases, each with specific muscle firing patterns and biomechanical activities. From beginning to end, the average pitch takes under 2 seconds to fully complete. However, deficiencies in any phases can dramatically alter performance and increase the risk of injury.

Windup (Balance). The thrower assumes this readying position as he or she faces the target to prepare to throw. This phase generates little energy, and only minimally loads the shoulder. Pitchers take a step backward with the lead leg, keeping the push-off leg on the rubber. The athlete stays balanced, and the weight is placed over the push-off leg letting the trunk/core start to generate the arm into external rotation (early cocking). Windup is optimal when the weight is shifted from the push-off leg to the leading leg. The hands separate at the end of the windup, the body rotates 90 degrees, and the stride leg is elevated as the body faces the batter. The body should be balanced as the stride leg reaches maximal height and the late cocking phase. The forearm is slightly pronated, the shoulder slightly internally rotated, and the elbow flexed (Fig. 26.1A) (8). Muscle firing is low at this point.

Early Cocking (Stride). The stride motion begins at the end of the windup phase as the lead leg moves toward the target or plate. The key to high arm velocity is to keep the trunk coiled as long as possible and the delivery arm close to the body, to store the energy before the transfer. As the lead leg moves toward the target (the ball), the energy is uncoiled and the push-off leg begins to move the body forward. As the stride foot makes contact with the ground, the arm is semicocked (Fig. 26.1B). The stride length of a pitcher is slightly less than his or her body height (3,9).

Mechanics often break down at this point. If the stride foot is too closed, the pitcher will tend to throw across the body. If the stride foot is too open (flying out), then the stored energy dissipates. Good foot position is slightly closed to neutral when the lead foot hits the ground. Early activation of the deltoid occurs, while the supraspinatus, infraspinatus, and teres minor fire late.

Late Cocking. Hip rotation starts in late cocking to prepare for accelerarion. The shoulder is at

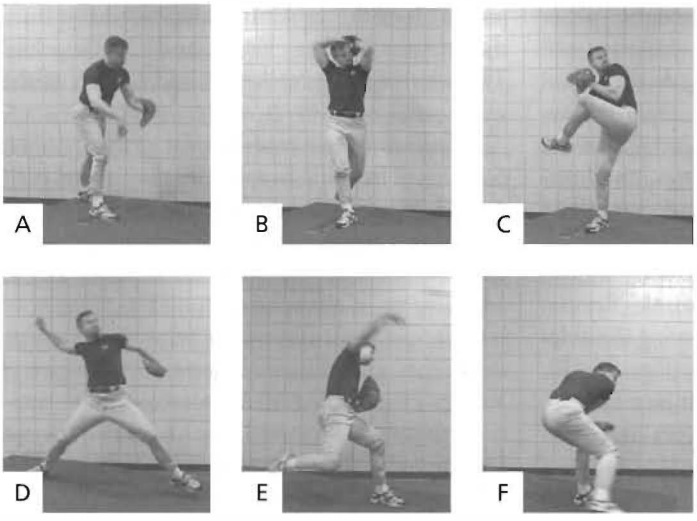

FIGURE 26.1. The phases of throwing: **A,** Windup. **B,** Early cocking. **C,** Late cocking. **D,** Acceleration. **E,** Deceleration. **F,** Follow-through.

maximal external rotation, which eccentrically loads and stretches the internal rotators. The shoulder has a large amount of energy available from the stored energy of the legs, hips, and trunk. At this point, tremendous stress is on the anterior shoulder. The forearm continues to stay pronated throughout this phase. The subscapularis and pectoralis major fire to form an anterior wall to prevent humeral subluxation.

The wrist begins to cock in preparation for the throw and final release. The elbow remains flexed between 60 and 75 degrees, while the extensor carpi radialis brevis and longus show high levels of activity. The medial elbow encounters high valgus load (Fig. 26.1C).

The deltoid firing decreases from early cocking, but the supraspinatus, infraspinatus, and teres minor reach maximal activity. The subscapularis, pectoralis major, and latissimus dorsi eccentrically fire to stabilize the humeral head as it moves posteriorly, then anteriorly in the later phases of throwing.

Arm Acceleration. The arm is ready to move to the target. The elbow begins to extend, closely followed by the beginning of humeral internal rotation. The trunk begins to flex as the medial elbow endures tremendous load, specifically the medial collateral ligament (MCL) and the pronator/flexor muscle group (3). The pronator teres, and flexor carpi radialis and ulnaris maximally fire at this point (Fig. 26.1D).

The triceps fires early in the phase, while the pectoralis major, latissimus dorsi, and serratus anterior fire late. The humerus moves through horizontal abduction to neutral, so the posterior capsule and rotator cuff absorb minimal shear stress. This may explain why some pitchers are still able to play with profound posterior cuff weakness or injury. Arm acceleration is completed when the ball is released.

Arm Deceleration. The most violent phase of throwing occurs after the ball is released. The arm and body continue to follow toward the target, and the leg kick helps to decelerate the body's momentum. The lead leg begins to extend as the push-off leg comes to meet it. Arm deceleration has reached its end point when internal rotation is at 0 degrees (Fig. 26.1E). The posterior rotator cuff and shoulder muscles fire eccentrically to slow internal rotation and prevent distraction at the glenohumeral joint, and the long head of the biceps adds ttaction to slow down the arm. Deceleration requires the largest amount of muscle activity from the elbow flexors. Significant eccentric activity occurs in the latissimus dorsi, subscapularis, and infraspinatus muscles.

Follow-through. The follow-through phase dissipates all the energy used to accelerate and decelerate the arm. It adds nothing to the velocity or control of the pitch, but a proper follow-through minimizes the risk for injury. Shoulder abduction holds at about 100 degrees, and horizontal adduction increases to 35 degrees. The follow-through should have a longer path to allow time to disperse energy. Large body regions should be involved, such as trunk flexion. Follow-through ends with the throwing shoulder and hand at the opposite knee (Fig. 26.1F). Shoulder distraction and risk for injury increase if the arm ends up toward the target instead of across the body at the opposite limb.

As stated previously, pitching delivery in the above phases is different mechanically from the throwing form of position players. Infielders often throw off-balance and side-arm rushing to get the batter or runner out. Their deliveries are usually much shorter, with quicker cocking and release phases. Outfielders often use a crow-hop delivery, which consists of a hop, skip, and throw. Crow hops can also be used as a drill for throwers having difficulty involving their core or trunk.

MUSCLE ACTIVITY

Muscle Firing

Electromyographic studies have reported that the supraspinatus is most active at late cocking, as anterior translation and migration off the glenoid are being controlled (10,11). The upper trapezius, supraspinatus, and deltoid are maximally recruited during the early cocking phase (12). The infraspinatus and teres minor are most active during the late cocking and follow-through phases. The serratus anterior and subscapularis muscles are also active from acceleration to follow-through. The rotator cuff is not a prime mover in throwers, but rather a stabilizer, particularly after 90 degrees of elevation. The latissimus dorsi, pectoralis muscles, and subscapularis muscle are the prime movers during the internal rotation and acceleration phase. The lower and middle trapezius continues to stabilize the scapula through acceleration. Furthermore, the core or trunk generates significant power, since the latissimus dorsi originates from the thoracolumbar fascia and lumbopelvis. Thus, latissimus stretches should be part of the warm-up.

Significant force couples exist at the shoulder complex. The joint couples are the rotator cuff/deltoid, subscapularis/infraspinatus, and trapezius/serratus anterior (13). The serratus and lower trapezius muscles work together at about 120 to 140 degrees of elevation to limit the amount of scapular posterior tilt, which helps the acromion clear the glenohumeral joint. Happee and Vander Helm noted 40% thoracoscapular muscle activity with forward arm movement versus 18% noted at the rotator cuff (14). During deceleration, the teres minor, lower trapezius, subscapularis, and posterior deltoid are recruited most to resist the amount of force moving forward toward the target (12).

Knowledge of concentric and eccentric muscle firing and points of peak activity allows the clinician to prevent injury, locate the source of pain, and treat injury more effectively. Part of the history with any throwing injury is knowing exactly at which phase of throwing the pain occurs, such as upon ball release versus follow-through versus late cocking. This information is crucial to diagnosis and treatment.

Ground Reaction Forces and the Kinetic Chain

Many have hypothesized that the breakdown in the kinetic chain is the reason for shoulder problems (14–16). The ground-reaction force is the beginning of the kinetic chain and defines the art of throwing, and disputes the contention that it is an arm (shoulder)-only activity. The transfer process in throwing starts from the ground, to the legs, then the trunk, the shoulder, next to the arm, and finally to the ball. Kibler and co-workers note that the hip and trunk provide the most energy (51% to 54%), while the shoulder provided only 13% of the energy and 21% of the force to the link in overhead activities (16,17).

MacWilliams et al. (8) evaluated ground-reaction forces and showed that push-off forces gradually increased during windup and reached

the highest before foot contact. After the lead foot hits the ground, the energy is then transferred through the body via the trunk and into the arm to deliver the ball. This study also suggested that the greater the push-off magnitude, the greater the kinetic energy into the upper limb. Hence, any lower extremity problems can break down energy transfer along the kinetic chain. Do not overlook the disadvantages of uncontrolled lower body power and shoulder girdle weakness. This creates shear forces that can overload and injure the shoulder complex.

COMMON INJURIES

Baseball injuries are usually a result of breakdown from overuse. Baseball has minimal player-to-player contact, very few off-days in the major and minor league seasons, and adolescent players who demonsttate velocity or precocious skills tend to play as much and as long as they can. Accumulation of wear and tear without ample opportunity to recover can lead to chronic injuries. This precept admittedly applies to almost any sport, but the repetitious nature of baseball subjects the body to consistent loads that challenge collagen tensile strength and muscle endurance. For instance, a major league pitcher who starts 35 games for a team and throws 90 to 100 pitches per start will have thrown up 3,500 pitches in a season, not including warm-ups, spring training, and in-between start throwing programs. Depending on the pitcher, those pitches can vary in difficulty and torsional force (fastball vs. split-finger). An average major league batter may swing at anywhere from 4 to 10 pitches each game, which can extrapolate to anywhere from 2,400 to 6,000 swings in roughly 600 plate appearances, and that does not include daily batting practice. This accumulation of yearly low-load stress can be difficult to tolerate if little effort is made in conditioning and flexibility.

Younger baseball players have significant injury risk as well. Lyman et al. studied 298 pitchers ages 9 to 12 over two seasons and noted the frequency of elbow pain at 26%, and that of shoulder pain at 32% (23). Elbow pain risk factors include increased age and weight. The six secondary ossification centers of the elbow are at highest risk for injury. Older pitchers tend to throw more often and harder, while extra weight increases strain on the extremity, which may explain how age and weight correlate (23). Trick pitches such as the sinker which the immature thrower learns may have a direct link to elbow pain as well.

Age and weight were not as much of a factor in shoulder pain, but arm fatigue and higher pitch counts were related. Pitch counts over 75 pitches were positively correlated to shoulder pain, while throwing pitches like the changeup and slider decreased shoulder pain (23). Lyman et al. later noted in a subsequent study of 476 pitchers that the curve ball was associated with a 52% increased risk of shoulder pain, and the slider with an 86% increase in elbow pain (24).

Injuries are a part of sports, but many baseball injuries can be limited or prevented by conditioning, training, and practice. Baseball players should use the off-season to make gains in flexibility, strength, muscle endurance, and core stability. In-season programs, years ago considered detrimental to performance, are now mandatory on major league teams. These are designed to maintain endurance and any off-season gains that were made, not necessarily to build strength. Programs need to be tailored to the position and athlete, depending on the athlete's skills and needs.

The throwing athlete must be an efficient mechanical machine reproducing optimal mechanics as often as humanly possible. Although we discuss specific injuries in the elbow, shoulder, and spine, all are interconnected and impact on each other. Shoulder instability can initially manifest as elbow pain, lumbar pain can limit follow-through and cause rotator cuff impingement, and midthoracic pain can limit retraction of the scapula, which is crucial to the cocking phase. This is the kinetic chain in action.

Common Areas of Breakdown

Throwing a baseball is an unnatural act for the shouldet. Even if an athlete is well prepared and healthy, he or she can still develop shoulder pain

just by throwing too much or without proper rest or warm-up. Cohen et al. describe four categories of causes for injuries to the pitching arm: (a) conditioning, (b) fatigue, (c) overuse, and (d) mechanical flaws (18). In all these cases, the essential underlying element is the failure of soft tissue constraints to withstand the stress loads from throwing pitches. Whether the cuff fatigues from too many pitches, the anterior capsule strains from the shoulder opening too quickly during the throwing motion, or poor conditioning causes the trunk and legs to tighten later in the season, the end result is an unstable and inefficient joint.

Changes in throwing mechanics often lead to shoulder and elbow injury, even from body regions far from the shoulder, as any injury or dysfunction can disrupt the kinetic chain (16,17). For instance, Achilles tendinitis can affect a pitcher's push off the mound. This limits the force generated to the trunk, which the shoulder and elbow try to replace. Often, the effort to compensate for a force deficit alters the natural motion and energy distribution in the kinetic chain. Complete windup with the lead leg reaching its highest point must happen to ensure optimal energy coil, as well as preventing a free fall toward the plate that would decrease velocity. Tissue has finite stress loads, thus structures such as the ulnar collateral ligament or the rotator cuff also break down when the kinetic chain fails.

Another example is improper foot planting during follow-through. The foot should be in front and pointing to home plate. A front foot planted outside the plate (toward first base for a right-handed pitcher) leads to early opening of the hips during delivery, hyperextending the shoulder and increasing the load on the shoulder and medial elbow as the ball lags through the delivery. The front foot planted too far in (toward third base) usually leads to early ball release and loss of accuracy. It could restrict hip rotation, limiting momentum and energy transfer, and cause the arm to be the main source of velocity (5).

Other examples of mechanical breakdown are as follows:

1. The forearm does not pronate fully before ball release. For right-handed pitchers, the ball should face the shortstop, while the left-hander should have the ball facing the second baseman before release.
2. Shoulder hyperangulation is another problem. If the arm moves out of the scapular plane and excessively abducts during late cocking, the humerus overrotates and increases internal impingement. This position during pitching is known as the slot, which is usually overhead or three-quarters. In either case, the humerus should ideally stay at 90 to 100 degrees to the scapula. Throwing outside of that range increases the risk of injury.

Medial Collateral Ligament Injury

In baseball, medial collateral ligament (MCL) tears are usually season-ending injuries. Complete tears require reconstruction, 12 to 15 months of rehabilitation before returning to the sport, and a 70% to 90% chance of returning to the previous level of competition, while partial MCL tears have a 42% failure rate of nonoperative treatment in returning to the mound (25).

Failure occurs usually after chronic repetitive overload of the ligament; very few ruptures occur acutely in baseball. This load comes from the tremendous valgus force generated by throwing a pitch. These are highest during late cocking and early acceleration, which is when the elbow moves from flexion to extension (26). Valgus stability comes primarily from the anterior bundle of the MCL and secondarily from the radiocapitellar joint. Repetitive valgus load from pitching explains such chronic changes as MCL thickening, radiocapitellar osteophytosis, and osteochondritic dissecans of the capitellum.

Once the ligament is attenuated and valgus laxity develops, several subsequent problems can occur:

1. Bony impingement of the olecranon and its fossa, which can lead to irritation, chondral lesions, and osteophytes, which leads to increased impingement.
2. Ulnar nerve traction injury, which can cause ulnar neuritis, nerve compression in the cubital tunnel, and even neuropathy. Some studies noted that 40% of patients with valgus instability had ulnar nerve symptoms (19,20).

3. Increased compression in the radiocapitellar joint, which can lead to reactive bony changes, osteochondritis dissecans, and degenerative osteoarthritis.

Valgus overload leads to attenuation and eventual failure of the MCL if nothing intervenes in the process, so early identification of medial symptoms is crucial. However, the factors relating to MCL injuries are usually seen away from the elbow. Common related factors include violent or inefficient throwing mechanics, excessive pitch counts with insufficient recovery, premature shoulder extension during transition to the acceleration phase, and poor shoulder stability.

The athlete with chronic injury presents with medial elbow pain while throwing, sometimes with radiarion around and down the ulna. Intensity and duration of pain may increase with more throws. Pain usually occurs during the early acceleration phase, and velocity may decrease. An acute-on-chronic injury typically has a defined moment with a "pop" and subsequent inability to throw hard. This may follow a period of time in which the athlete has minimal or mild medial elbow symptoms. An acute traumatic injury is rarely seen in baseball.

Physical examination should target several issues:

1. Etiology and reproduction of the pain and symptoms.
2. Irritability of the ulnar nerve.
3. Assessment of valgus stability and MCL competency.
4. Evaluate for posterior olecranon impingement.

Standard Treatment

Nonoperative management of MCL disruption in nonthrowing athletes is effecrive, even with acute traumatic complete ruptures. Less success is seen in the competitive thrower, with less than half recovering from the injury without surgery (25). Initially, the thrower is immediately shut down from throwing, and antiinflammatory treatments begin immediately. Rehabilitation then focuses on improving muscle endurance in the periscapular and shoulder girdle muscle, and strength in the wrist flexors, pronator, and rotator cuff muscles. When the athlete completes an intensive rehabilitation program without pain, he or she progresses to the throwing program, which eventually leads to return to the mound. Complete tears in throwing athletes typically do poorly and need reconstruction to return to throwing (20). Partial tears that fail a rehabilitation and throwing program are also candidates for reconstruction.

Manual Medicine Techniques

The elbow needs a smooth transition from flexion to extension during the acceleration phase. Reasons for restriction include proximal radioulnar joint dysfunction, distal bicipital dysfunction, and dysfunctional extension of the olecranon into its fossa. Arthritic changes may be resistant to some manual treatments, and many pitchers develop slight flexion contractures (usually less than 10 degrees) during their career, yet perform without difficulty, since the arm never truly reaches full extension during a pitch. Thus, every pitcher with a contracture or arthritic changes may not respond quantitatively to treatment.

Extension dysfunction can be treated by muscle energy to the biceps that engages the extension barrier. Radial head dysfunctions can be treated with muscle energy or high-velocity, low-amplitude (HVLA), while joint play techniques can improve medial or lateral tilt of the olecranon so as to allow unrestricted end-range extension.

The scapula is a crucial link for energy distribution in the kinetic chain, as noted in Chapter 17.3, in the section on scapular dyskinesia. The scapula must retract fully during cocking, then protract during acceleration. Restriction in either motion destabilizes the shoulder and invites compensation from the surrounding muscles to accomplish the task. Invariably, the force not dissipated by the restricted and unstable scapula gets transmitted to the elbow and increases valgus load. The cumulative effect of this load over time is ligament failure. Counterstrain, myofascial release, and muscle energy techniques can be

applied to muscles that restrict the scapula, as discussed in Chapter 17.3). Specific trouble areas to evaluate are the levator scapulae, latissimus dorsi, and the midthoracic spine and the muscles that originate from that area. Counterstrain trigger points can be easily identified and treated.

Collateral ligaments with scar tissue or fibrotic tissue with palpable thickening and myofascial restriction can be treated with deep tissue massage or myofascial release. Muscle energy to the flexor muscle bundle can also help decrease tissue tension around the MCL. This does not help an incompetent ligament, but this approach can be helpful as the athlete progresses through the throwing program and moves from the subacute phase of treatment to a more functional level.

Any thrower with elbow pain recalcitrant to standard treatment should have a complete musculoskeletal assessment done to diagnose and treat any somatic dysfunctions along the kinetic chain, as well as a specific orthopedic evaluation to assess the integrity of the MCL and return to play.

Thoracic Dysfunction

Many players complain about stiffness or discomfort between the scapulae. Throwers use the thoracic spine between T1 and T8 to anchor the medial scapula. During the cocking phase the scapula is fully retracted against the spine, so any limitation to retraction affects the scapular position and destabilizes the shoulder from cocking into acceleration. As the upper body and scapula cock, the thoracic spine extends, then flexes through late acceleration, deceleration, and follow-through.

Additionally, the batting swing violently torques the thoracic spine, and although vertebral motion is limited due to the rib cage and multiple ligamentous and muscular attachments, dysfunctions and muscle strains occur. Thoracic extended segments are also generated by weightlifting, specifically the bench press. Any lifting that forces the thoracic spine into extension may promote extended vertebral dysfunctions.

These are not typically serious injuries that disable a player long-term, although the possibility of a herniated disc or thoracic vertebral fracture must be entertained in significant long-standing pain resistant to conservative therapy. The thoracic spine frequently becomes dysfunctional, restricts motion, and can affect muscle firing and scapular retraction.

Manual Techniques

Soft tissue should be prepped with massage techniques to loosen up the muscles and subcutaneous tissue. Myofascial techniques should then be applied to the scapula, including scapular rotation myofascial release. Muscle energy to the levator scapulae, trapezius, and C7-T1-first rib complex (using the scalenes) should follow. Dysfunctions in glenohumeral articulation or the cervical spine should be diagnosed and treated with a preferred technique. Dysfunctions in the lumbosacral region should also be addressed.

The use of HVLA in the thoracic spine is acceptable and can restore mobility and give relief. Paravertebral muscle spasm would make HVLA more difficult, so single segmental dysfunctions can be treated easily with muscle energy or counterstrain. Lumbar articulatory technique often treats the lower thoracic segments as well near the end range of rotation.

Glenohumeral Instability

Throwing a 90-mph fastball is not a natural act for the shoulder. When the small rotator cuff muscles break down from overuse or injury, the glenohumeral joint destabilizes, and throwing becomes difficult. The four rotator cuff muscles work with the static shoulder stabilizers (capsule, glenohumeral ligaments, labrum) to form a kinematic, not anatomic, ball-and-socket joint. These kinematics allow the humerus to rotate enough to allow a throw yet, offer limited stability, as compared to the bony hip ball-and-socket joint. Any breakdown in these soft tissue constraints, as mentioned earlier, disrupts these kinematics. Various factors can cause glenohumeral instability, such as the following:

- Anterior instability (pitchers).
- Posterior instability (hitters).
- Acquired capsular laxity vs. posterior capsule scarring, leading to anterior instability.
- Humeral subluxation/dislocation (sliding/diving).
- SICK scapula syndrome (see the discussion in Chapter 17.3, The Shoulder: Common Conditions).
- Rotator cuff weakness secondary to tendinitis, superior labrum anterior-posterior (SLAP) tear (can be due to overuse, excessive distraction forces).
- Labral pathology (SLAP, posterior impingement syndrome).
- Internal impingement (posterior-superior labral pathology).

Instability in a baseball player is classified into groupings based on whether the cause is from (a) microtrauma, (b) hyperlaxity, or (c) a traumatic event (21). Identifying the category for the athlete aids in developing an effective treatment program.

Any pain from the shoulder needs to be treated to avoid inhibition of the rotator cuff, which may mean no throwing for several days. A painful rotator cuff will become weak and allow the humeral head to move out of its stable position in rhe glenoid fossa, primarily due to imbalance with deltoid contraction.

Standard Treatment

In general, rhe underlying causes of instability must first be diagnosed and treated. This is paramount, because early instability is the time when nonsurgical injuries can become surgical problems if not addressed by the coaches, trainers, or doctors.

Next, rehabilitation moves in phases, which is adjusted if the athlete needs surgery for stabilization. Some of the basic aspects are as follows:

- Establishing full range of motion.
- Rotaror cuff eccentric strength (first isometric, then isotonic, then end ranges of motion, then functional proprioceptive neural facilitation patterns).
- Scapular stabilization (lower and upper trapezius, forced couples, serratus anterior, eccentric control of the rhomboids).
- Rhythmic stabilizations using dynamic proprioception exercises.
- Throwing program (see Chapter 14) before return to play.

In particular, scapular stabilization is crucial due to its effects along the kinetic chain. For instance, a thrower with poor rhomboid strength requires the lumbar paraspinal muscles to work too hard to compensate. This weakens important scapular stabilizers (serratus anterior, upper and lower trapezius). Every muscle off the coracoid process then tightens, which increases protraction and anterior tilt, which inhibits muscle firing of the posterior scapular muscles, making the scapula, and the whole shoulder, less stable. Further discussion of glenohumeral instability is beyond the scope of this text.

Manual Medicine Techniques

Batters and pitchers need to be able to transfer energy from their legs and trunk into their arms and hands, and manual medicine techniques should be used to facilitate this. No amount of manipulation can repair structural damage or eliminate cuff tendinitis, and proper recognition of its limitations is important, particularly in the elite baseball athlete. Manual medicine is most effective during the early onset of symptoms, near return to play, and maintaining homeostasis in a long season. Because baseball seasons are longer than in any other sport, the accumulation of repetitive stress over months can cause fatigue, microtrauma, and compensatory changes that affect the kinetic chain. Manual medicine can catch and eliminate these changes before dysfunction becomes injury, or in severe cases, before surgery is required.

Manual medicine techniques are effective in the following areas:

1. Latissimus dorsi, trapezius, and levator scapulae inflexibility, which increases supraspinatus impingement.

   Techniques: Stretching, muscle energy, counterstrain, functional, massage.

2. Glenohumeral mobility. Loss of internal rotation and posterior capsular tightness.

   Techniques:

   - Anterior, inferior, and posterior capsular stretching.
   - Muscle energy for internal rotation restriction.
   - Seven stages of Spencer for glenohumeral mobilization.
   - Counterstrain to the posterior rotator cuff, latissimus, and teres major.
   - Thoracic mobilization.
   - Scapular myofascial release (rhomboids, serratus, trapezius).
   - Articulatory release to the C7-T1-first rib complex.

3. Thoracic vertebral dysfunction. The scapula is not able to retract properly.

   Techniques:

   - HVLA thoracic mobilization.
   - Muscle energy or counterstrain.

4. Excessive shoulder protraction from a tight pectoralis minor.

   Techniques:

   - Myofascial release or muscle energy to the pectoralis minor.
   - Counterstrain to weak posterior scapular muscles.

5. Lumbosacral dysfunction. This often manifests as pain in extension, localized at the left or right posterior superior iliac spine, and lower lumbar stiffness. The components of this dysfunction usually include a sacral torsion, rotated or sheared innominate, L5 dysfunction, ipsilateral gluteus medius trigger point, iliopsoas or anterior hip capsule restriction, and unilateral hip internal rotation limitation. Erector spinae spasm, piriformis dysfunction, iliolumbar ligament pain, and pubic symphysis dysfunction may also be noted.

   This problem can affect a pitcher in late cocking and early acceleration, and somewhat in deceleration and follow-through if there is muscle tightness limiting flexion. A batter can have problems with the end range of the swing, limiting available torque power, and potentially limiting the player from reaching out for pitches. The techniques preferred by the author for this scenario are the following (reevaluation is done after each technique, so that steps are not performed unnecessarily if the lesion has already resolved) or does not exist. Pubic symphysis release and piriformis stretching → lumbar articulatory technique → muscle energy for sacral torsion (treat with posterior inferior lateral angle (ILA) down) → muscle energy inferior lateral angle (ILA) L5 (and higher) dysfunction → counterstrain to gluteus medius, sacrotuberous ligament and/or iliolumbar trigger points → muscle energy and/or joint play mobilization for iliopsoas and anterior hip capsule → muscle energy for rotated innominate or shear, or HVLA for an upslip innominate → treat dysfunctions elsewhere found on the screening examination.

PREVENTION

Throwers need to be proactive in taking care of their arms, and compliance is essential. A basic throwing injury prevention program includes self-stretches for the capsule, shoulder girdle, neck, and core; overall conditioning of the entire player; proper warm-up and cool-downs during workouts and games; pitch counts to monitor fatigue and limit overuse; and strengthening and proprioception training of the rotator cuff, shoulder girdle, and scapular stabilizing muscles, both off-season and maintenance during the season. The in-season program is typically less intense than in the off-season. The thrower's ten exercises described by Andrews and Wilk are designed to strengthen those muscles intrinsic to throwing (see discussion of thrower's exercises in Chapter 14).

In July 2003, *USA Baseball* updated their recommendations for prevention of youth baseball injuries (22). One of the most important points made was using and enforcing pitch counts. Pitchers 9 to 10 years old should not throw more than 50 pitches in a game, while

young pitchers aged 11 to 12 and 13 to 14 can throw up to 75 per game. Weekly, seasonal, and yearly counts should be kept and monitored as well. Also, breaking ball pitches should not be thrown until at least 13 years old, and only if the young athlete is taught by a coach. Teaching proper mechanics should also be emphasized in preference to merely learning new pitches.

With regard to specific pitches, *USA* Baseball recommends that a fastball and changeup should be the only pitches used between ages 9 to 14 (21). A curve ball can be learned after age 14. Sliders, knuckleballs, and forkballs are not recommended before ages 15 to 16, although there is debate as to whether high school pitchers should throw them at all. The author believes that they should be eliminated from the high school repertoire, and the curve, changeup, and fastball should be the only pitches allowed. If a pitcher cannot master these three pitches, adding the forkball or knuckleball will likely not improve his or her performance. Rather, the likelihood of injury will only increase.

Eliminating high pitch counts, halting the premature use of breaking pitches, and emphasizing proper throwing mechanics would dramatically cut down on youth injuries (6), but coaches and parents need to take the initiative to implement these recommendations. Young athletes need credible resources and strong role models, and the hope is that through continuing education and the dedication of coaches and parents, their needs will be met.

CONCLUSION

Baseball is a unique sport that requires multiple skills and remarkable endurance. The highly repetitious nature of the game causes many overuse and fatigue-related injuries. Throwers have their own subset of unique injuries that require an accurate diagnosis at the site of injury and a comprehensive musculoskeletal examination to ferret out compensatory changes and dysfunctions that may have led to the injury itself. The kinetic chain and manual medicine principles should be applied in the osteopathic approach to the baseball player in order to prevent and limit injury.

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BASKETBALL

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The sport of basketball has grown worldwide and has surpassed many traditional sports in popularity across the United States and worldwide. The highest level of competition, the National Basketball Association (NBA), has continued its remarkable growth following the Larry Bird and Magic Johnson era of the 1980s. The game has reached a global scope as international players are drafted frequently and Olympic competition has become more evenly matched.

The growth of basketball at the elite level also includes the collegiate, high school, Amateur Athletic Union (AAU), and recreational levels. An estimated 40 million play the game in the United States alone (1). The National Collegiate Athletic Association (NCAA) basketball championship tournament is one of the most watched events in the world. Basketball provides recreational and professional opportunities for participants of all races, religions, colors, and genders. Size, strength, speed, intelligence, and individual commitment often are the determining factors for which level a player may achieve.

Unfortunately, injuries also have a role in player development and achievement. Basketball injuries range from the acute traumatic to the chronic overuse variety. The highly publicized anterior cruciate ligament injuries, Achilles tendon ruptures, and significant ankle sprains capture the media attention and are replayed over and over again on television news stations nightly. The chronic stress changes to the lower extremity (stress fracture, shin splints) are actually more common. Combined with back, hip, and foot and ankle pain, these insidious conditions sideline more basketball players typically for longer periods of time than do acute traumatic injuries.

The purpose of this chapter is to review many of the chronic injury patterns and a few acute events found in basketball and discuss how to integrate manual medicine techniques with standard treatment. A holistic approach that follows kinetic chain and osteopathic principles will hopefully improve recovery.

EPIDEMIOLOGY

In an analysis of intercollegiate basketball in Canada, Meeuwisse and associates (27) noted injury rates at 4.94 injuries per 1,000 athlete-exposures for all injuries. The ankle had the highest overall injury rate (1.22 per 1,000 athlete-exposures), followed by the knee (0.87), thigh (0.46), and foot (0.39). This is consistent with previous studies reviewed by Meeuwisse. However, the knee had the highest rate of injuries, which resulted in seven or more sessions (practices and games) missed (0.25), followed by the ankle (0.21). Average time lost for a knee injury (18.25 days) was three times longer than ankle injuries (5.47 days) and over twice as long as foot injuries (7.82 days).

ENDURANCE AND STRENGTH APPROACH TO THE ATHLETE

Basketball requires a combination of energy from both the aerobic and anaerobic energy systems. The ability to deliver oxygen to the tissues during prolonged bouts of exercise requires a well-developed aerobic system to efficiently perform at peak levels for extended training sessions. The ability to perform faster and more explosively with less fatigue is based on anaerobic power (2).

Muscular power directly affects performance factors such as speed, agility, and explosiveness. Sudden changes of direction, alternating stop-and-start movements, jumping, driving, shooting, and rebounding require quick bursts of anaerobic energy. Therefore, the more powerful the muscle, the better an athlete can perform these tasks. Immediate energy is supplied by use of adenosine triphosphate (ATP). Typically, ATP energy bursts are short in duration, lasting from less than 1 to 3 seconds (2). As the duration of prolonged intensity persists, exhaustion of sufficient oxygen from anaerobic glycolysis ensues and stage II energy metabolism is required to maintain performance. This ultimately leads to elevated levels of blood lactate from the tissues, resulting in muscle fatigue and prolonged recovery. Limiting stage II energy requirements in competition is crucial to provide for rapid recovery and ultimately more effective and prolonged play.

Aerobic power is the ability to deliver oxygen to the tissues during prolonged bouts of exercise (VO_{2max}) (2). The ability to condition an athlete in aerobic training drills allows for play at higher-intensity levels and for prolonged periods of time. High-level aerobic training increases the anaerobic threshold, enhances lactic acid clearance, and delays the onset of fatigue.

Muscle strength and power provide explosive energy both to the upper and lower extremities. These components are essential to maximize performance in rebounding, positioning, jumping, sprinting, and driving to the basket. Muscular endurance is vital to allow for repeated power throughout the course of a play or longer. Aerobic endurance training in conjunction with muscle strengthening provides peak performance for short and long energy demands.

The basketball player faces excessive load over the length of a season, particularly to the lower extremities, and strength training throughout the season is now commonplace for many competitive teams. Upper extremity strength is emphasized more today since the myth of weight training adversely affecting shot accuracy has been largely dispelled. Core strength is probably the most important area that gets overlooked by athletes, but its importance in lumbar stability and lower extremity power generation has been documented.

SPECIFIC INJURIES

Lower Leg, Foot, and Ankle

The most common injury in the sport of basketball is the ankle sprain (3). Typically due to a plantarflexed and inverted foot and ankle landing on an uneven object (typically an opposing player's foot) or to a sharp, cutting, or pivoting movement, the inversion ankle sprain most frequently occurs as a result of inherent weakness and instability of the ankle joint. Chronic ankle sprains are seen more frequently from a combination of intrinsic and extrinsic factors. Muscle weakness, poor proprioception, and altered foot mechanics lead to instability of the subtalar joint. Improper shoewear may affect forefoot mechanics, predisposing to excessive pronation or supination and altered gait patterns. Each muscle group works jointly with the others in different phases of the gait cycle and stabilizes the subtalar joint through its motion. Observation of callus buildup medially or laterally on the plantar foot assists in determining the pronated from the supinated foot and their respective wear and stress patterns seen with walking, running, and jumping. A thorough biomechanical evaluation with a supervised rehabilitation program and well-fitted orthosis can assist in providing a neutral subtalar joint and balanced muscle patterns around the foot, ankle, and lower leg. Ankle manual medicine techniques are also discussed in Chapter 24, Foot and Ankle.

Other common, but less frequently assessed structural changes in the foot and ankle include cuboid subluxations, fibular head dysfunctions (proximal tibiofibular joint), distal tibiofibular joint alignment, the talocalcaneal articulation (subtalar joint), and midfoot and metatarsophalangeal joint restrictions. Cuboid subluxations typically present as midfoot pain along the lateral and plantar surface of the foot. Clinical examination includes a palpable cuboid prominence along the lateral plantar foot with associated localizing pain. Relative discomfort

and cuboid position versus the unaffected side are the hallmarks for diagnosis.

Manual Medicine Techniques. Manual medicine techniques are based on athlete tolerance and mobilization of the cuboid. High-velocity, low-amplitude (HVLA) muscle energy, and counterstrain techniques often adequately reduce the cuboid. Pain resolution and enhanced mobility at the articulation are signs of a successful reduction.

Dysfunctional tibiofibular articulations proximally, and less so distally, are commonly seen in inversion mechanism ankle sprains. The proximal articulation is intimately related to the knee and ankle. Restriction at the proximal articulation affects knee and ankle mobility. Typical anterior-posterior glide is directed by actions of the biceps femoris muscle and its local insertion (4). Evaluation of ankle injuries should include palpation, joint play evaluation, and articulatory techniques to the proximal and distal tibiofibular articulations (5). Standard treatment is rehabilitation with muscle stretching, and neural tension stretches should be maintained to ease biceps femoris tightness and contraction. Anterior and posterior restrictions should be manipulated, using HVLA and muscle energy techniques.

Restriction of the talus superiorly at the tibiofibular-talus articulation and distally at the tibiocalcaneal articulation (subtalar joint) significantly restricts ankle motion (4). Ankle stability is greatest in dorsiflexion, while limitation of motion in this plane affects ankle and foot function. Talus position affects ankle motion control, and dysfunctions at this articulation may lead to recurrent sprains due to altered mechanics. Mobilization techniques include muscle energy and HVLA maneuvers. Assessment of subtalar motion is critical following all acute and chronic events to ease ankle mobility and limit mechanical alterations in the gait cycle.

Stress Injuries of the Lower Leg

Lower leg injuries often include shin splints and stress-related changes to the tibia or fibula. Medial tibial stress syndrome involves an overuse mechanism to the soleus and deep muscular fascial attachments along the middle and posterior distal tibia (3). The actual pathology is somewhat unclear, but is believed to be due to fasciitis or possibly a local periostitis. Treatments follow conventional RICE protocol (rest, ice, compression, and elevation) with cross-training to tolerance. Structural assessment of foot biomechanics, fibular head and subtalar motion, and muscle tightness through the plantar fascia, gastrocnemius-soleus complex, and into the hamstrings is essential. Lengthening of specific tight muscle groups and strengthening of muscle imbalances should be keys to recovery. Assessment of shoewear to provide either additional motion control for the pronated foot or added cushion for the supinated foot deserves attention as well.

Stress-related changes to bone include periostitis, stress reactions, or the more common stress fracture. A result of many factors including overtraining, poor foot biomechanics, muscle imbalances, hormonal alterations, and dietary deficiencies, stress fractures typically present along the posterior medial aspect of the tibia and the second and third metatarsals of the foot in athletes involved in jumping and running sports. Rarely seen early on standard radiographs, local pain early into training with persistent discomfort at rest should raise suspicion for stress-related changes to the bone. Frequently, bone scan or magnetic resonance imaging (MRI) is required to confirm the diagnosis.

Anterior tibial stress fractures are less common but more ominous due to traction forces across the bone, poor bone remodeling, and lengthy time to recovery. Whereas typical tibia, fibula, and metatarsal stress fractures require 6 to 8 weeks before return to play, anterior tibial stress fractures may require greater than 6 months and frequently surgical intervention for complete healing. Fibular stress fractures typically present secondary to biomechanical changes following previous injury. In this instance, foot and ankle overcompensation applies undue stress laterally onto the lower leg and ultimately leads to stress changes on the fibula.

Metatarsal stress fractures follow the pathophysiology and overload characteristics of tibial injuries. Lack of mobility of the second and

third metatarsals puts them at greatest risk when overload and excessive stress forces are applied.

Standard treatment first includes changing the training environment or routine of the athlete, and relative rest. Strengthening and flexibility training should start in an organized therapy program. Correction of all extrinsic and intrinsic precipitating factors should be achieved prior to a return of functional exercise. Shoe insoles or orthotics may help distribute weight more effectively to where it should be loaded.

Manual Medicine Techniques. Manual medicine techniques should focus on the kinetic chain and treatment of the dysfunctions and restrictions along the leg. Second and third metatarsal restriction can be treated by anteroposterior joint play techniques, remembering to add HVLA at the restrictive barrier. Dorsiflexion and plantarflexion of the ankle can be treated with a talar release technique, then muscle energy to reset muscle lengths. Treat any other dysfunctions along the kinetic chain, including sacral torsions, hip flexor and anterior capsular restriction, and fibular head dysfunction.

Knee

Previously described as the "career-ending injury," the anterior cruciate ligament (ACL) tear has now become a part of sports culture and a relatively routine repair, allowing top-level athletes to return to previous levels of function and performance. The magnitude of the injury on a player and the team is often devastating, but with aggressive rehabilitation and functional bracing, men and women alike are returning to their sport more quickly and with fewer complications. The medial collateral ligament sprain and meniscal pathology found in high-level athletes have also affected careers, but typically for short durations and with less fanfare. Meniscal repairs and excision have provided a means to return athletes back to their sport relatively quickly. Secondary degenerative changes along bony articular surfaces have a greater impact on longevity. The advent of the osteoarthritis transfer system (OATS) repair and the use of viscosupplementation have progressed a once debilitating injury to one that may only minimally impact an athlete's career. Many athletes are now returning to sport with few repercussions.

Anterior Knee Pain

Despite all the notoriety that acute and traumatic knee injury has achieved, the most common and still troublesome knee complaint is anterior knee pain. It affects basketball players from all skill levels and accounts for the most frequent complaint of the knee in the clinical setting. Etiology for the disorder is multifactorial and therefore treatment requires a thorough review of biomechanics, leg length, patellar position, Q-angle measurement, muscle tone and flexibility, and lumbosacral and pelvic alignment. Less common are torsional deformities of the femur and tibia, femoral trochlea depth, and patellar misalignment.

The most common knee pathology seen in basketball is patellar tendinitis. It occurs as the eccentric load overwhelms the natural healing process of the connective tissue. This usually affects the distal patellar tendon, but it can also include the suprapatellar or quadriceps tendon. The classification of patellar tendinitis is as follows (26):

Phase 1: Pain after athletic participation only.

Phase 2: Pain during participation but no effect on performance.

Phase 3: Pain during participation that affects performance.

Phase 4: Tendon disruption.

Early phases demonstrate inflammatory changes such as microhemorrhage and edema (21). As the inflammation becomes chronic, fibrosis and tendinosis settle in. Poor quadroceps and hamstring flexibility is the most significant causative factor (20). Patellar tilt is also implicated as a factor (20). Richards and co-workers noted that other factors involved in patellar tendinopathy include high ankle inversion-eversion moments, high external tibial rotation and plantarflexion moments, large vertical ground reaction forces, and high rate of knee extensor moment development (25). Patellar

tendinitis can cause changes in the tendon seen on MRI or ultrasound that are often asymptomatic initially (19,21,22).

Clinical examination begins with assessment of foot biomechanics and correction of the pronated or supinated foot if indicated. Leg length by gross measurement or use of radiographic postural studies helps to correct structural alterations affecting asymmetries and ultimately loads on the entire lower trunk. Q-angle measurement determines the position of the patella relative to the hips. Exaggerated Q-angle measurements may preclude an athlete to lateral patellar tracking. Vastus medialis oblique (VMO) muscle strength and tone may predict patellar direction during muscle contraction. Weakness of the VMO leads to excessive lateral patellar glide due to the relative increased strength of the vastus lateralis muscle. Associated quadriceps, iliopsoas, piriformis, and hamstring muscle tightness also alter patellar motion and need attention for relative symmetry.

Standard treatment typically involves relative rest when the pain is acute, use of a patellar tendon strap, physical therapy modalities, and a progressive quadriceps strengthening program. Any rehabilitation program must include quadriceps and hamstring flexibility (18), while a medial patellar taping can help delay onset of symptoms (28). Orthotics and an ankle stabilization program should be used to limit motion. Chronic tendinitis may require more extreme techniques such as extracorporeal shock wave therapy (23). Recalcitrant cases failing conservative treatment may eventually require tendon débridement, which has moderate success on the whole (24). Athletes in phase 1 or 2 usually do well with conservative treatment, results for phase 3 are more variable, but phase 4 requires surgery to repair the tendon (27).

Manual Medicine Techniques. Manual medicine techniques for anteriot knee pain must first treat any dysfunctions away from the lower extremity, such as sacral torsions, rotated innominates, and lumbar dysfunctions. Leg-length asymmetries should be resolved by manipulation of the pelvis or by a heel lift. The foot should be treated for any cuboid or navicular dysfunction, and joint play in these areas should be assessed thoroughly. Muscle energy, myofascial release, and deep tissue massage can help with hamstring and quadriceps muscle flexibility, as well as pelvic muscle imbalances.

Massage or myofascial release can be attempted on the patella to assist in loosening a tight lateral patellar retinaculum. Cross-friction massage can also be used in this situation, but if the athlete has active or subacute inflammation, it may be painful.

Gluteal tone and strength are the cornerstone of pelvic stability, with substantial indirect effects on patellar tracking. A coordinated firing pattern of the hamstrings, gluteals, and quadratus lumborum muscles aids in hip and pelvis stability, ultimately affecting upper leg muscle tone and flexibility. These effects have a secondary control on patellar motion and can produce misalignment and tracking changes to the anterior knee mechanism.

Common structural findings higher in the skeletal chain include sacroiliac and pubic dysfunctions. As mentioned previously, hip and pelvis misalignment play a key role in inhibiting muscle firing patterns and contractile force. Iliosacral dysfunctions (innominate shears) from improper landing following rebounding and jumping drills are a common cause of lower back and lower extremity pain syndromes (4). For instance, an innominate rotated anteriorly makes that leg functionally longer. This increases the amount of force through the patella during the midstance phase of gait. Correction of sacroiliac and pubic dysfunctions should be corrected prior to work on shear dysfunctions. An accurate diagnosis of the shear cannot be made until these other corrections are achieved due to the unilateral relation of the ibium to the sacrum, which must be midline to assess relation of the ilium to the sacrum (4).

Anterior knee pain requires a thorough evaluation and often multiple levels of treatment to correct biomechanics, adjust leg-length asymmetry, balance improper muscle tone and flexibility, and achieve a level and coordinated sacroiliac and iliosacral base. Manual medicine treatments include muscle energy, HVLA, joint play, and counterstrain techniques. A detailed exercise pre-

scription is required to make the appropriate adjustments and maintain proper alignment.

Lumbosacral Spine, Hip, and Pelvis

The sacrum and corresponding lumbar spine are frequently affected by the forceful pushing and shoving seen in the low post as players position themselves for entry passes and rebounds. Sacral malalignment and lumbar somatic dysfunctions present a real challenge not only to correct, but more so to maintain due to training schedules and length of season. A number of frequently seen sacroiliac and iliosacral dysfunctions were mentioned previously. Prevention and stabilization of the lumbosacral spine in basketball require flexibility and therefore lengthening of the psoas, rectus femoris, piriformis, quadratus lumborum, and hamstring muscle complexes. Each of these muscle groups has their insertions and origins about the pelvis. Gluteus maximus and medius strengthening along with abdominal muscle retraining (rectus abdominis and obliques) provide core stability to assist in maintaining proper lumbosacral position and motion (4). Excessive and repetitive hyperextension of the lumbosacral spine has detrimental effects on the lower back aside from the typical somatic dysfunctions described earlier. Stress applied to the pars interarticularis of the lumbar spine due to overloading of this region with jumping and extension of the hips, pelvis, and lumbosacral junction may cause stress changes of varying degrees, described as *spondylolysis*. Instability at this site (typically L4-S1) can lead to neurovascular compromise and typically increased pain and lower extremity paresthesias. Movement of the upper vertebra over the lower segment is called *spondylolisthesis*. The degree of severity and often clinical symptoms are based on the degree of vertebral motion of the affected segment relative to the stable vertebra below. Diagnosis is made by standard radiograph with the level of acuteness confirmed by a positive single photon emission computed tomography (SPECT) scan. Often SPECT imaging may prove negative, suggesting a chronic spondylolysis and possible genetic etiology or previous injury rather then a true acute event.

Treatment of acute spondylolysis includes lumbosacral bracing to limit extension, a progression to flexibility exercises, and finally stabilization techniques to enhance the inherent core stability of the trunk once adequate bone healing and/or pain resolution has occurred. Chronic spondylolysis is treated similar to other mechanical low back pain with correction of somatic dysfunctions by manual medicine and core strengthening.

Thoracic Spine and Ribs

Thoracic somatic dysfunctions and associated rib dysfunctions often limit function and play. They occur less frequently but are debilitating when present. Whether acute in nature or secondary to compensatory changes from shoulder pathology, thoracic spine and rib dysfunctions affect not only muscular and skeletal motion, but also respiratory status and coordinated breathing.

Thoracic dysfunctions typically occur due to abnormal side-bent and rotated thoracic spinal segments during play or conditioning. Local midthoracic pain and associated muscle spasm inhibit motion to that segment and the supporting soft tissues (4). Rib dysfunctions are commonly seen in conjunction with thoracic changes. Following respiratory motion in inhalation and exhalation at both the lower and upper rib cage identifies areas of restriction. Restrictions of the "bucket-handle" or "pump-handle" motion are seen throughout all levels (4). During inhalation, the anterior rib segment moves superiorly like a pump-handle, while the lateral aspect follows a bucket-handle motion. In exhalation, the same motion occurs but in a caudad direction. Generally, upper ribs display a more pronounced pump-handle motion, while the lower ribs follow a bucket-handle movement. Restrictions at these segments require identifying the most involved rib and assisting the motion at that segment to facilitate improved glide and coordinated rib movement

with inhalation and exhalation (4). Treatment typically involves muscle energy and/or HVLA mobilization techniques and requires localization and mobilization of individual ribs in either the seated or supine position.

A common rib restriction is the elevated first rib. Commonly presenting as neck pain, headaches, and occasionally radicular arm symptoms, the elevated first rib can be palpated as an asymmetry and painful fullness in the thoracic inlet. This is often acquired due to sudden side bending and rotated neck motion with primary or secondary muscle tightness. Basketball has significant physical contact when rebounding missed shots, and most players are looking up, extending their cervical spines, while their arms are reaching high over their heads. This can set the athlete up for a C7-T1-first rib complex dysfunction. Treatment is directed at muscle energy and articulatory techniques, with HVLA manual medicine techniques used if the former techniques do not work. Repositioning of the first rib often relieves symptoms. Maintaining side bending and rotary stretches helps to reduce muscular tension at the site and limits the return of symptoms.

Upper Extremity Injuries

Shoulder, elbow, hand, and wrist injuries in basketball occur more often as a result of an acute process in practice or competition and less commonly from overuse or chronic effects. Rotator cuff pathology, acute shoulder subluxations and dislocations, and acromioclavicular ligament sprains all require a degree of rest, relative immobilization, and rehabilitation. Occasionally, surgical repair is required to correct severe and recurrent events. Despite the etiology of the shoulder injury, any injury may produce secondary structural restrictions delaying an athlete's return to play. Shoulder parhology often presents with secondary muscle tightness and weakness
scapular and thoracic restrictions affecting shoulder motion. Awareness of scapular position and glide with scapular stabilization testing and articulatory techniques helps to evaluate and treat restrictions allowing for improved mobility at the scapulothoracic articulations. Spencer technique stages work well for basketball players as well.

Injuries at the elbow, hand, and wrist frequently involve fractures, dislocations, and ligament disruptions. Each typically requires a degree of immobilization prior to return to play. Following any form of immobilization, joint motion becomes restricted and virtually all ranges of motion are affected. Using joint play techniques to mobilize restricted joint function at each individual immobilized segment improves range of motion and strength more effectively throughout the rehabilitation process.

PREVENTION

Everything starts with a thorough screening examination at the pre-participation physical. A detailed history for risks of sudden death is imperative when screening all athletes. A history of family members with sudden death under the age of 50; episodes of lightheadedness, fatigue, or syncope with exercise; and wheezing, cough, or early fatigue with aerobic training are all important points of emphasis in reviewing risks for sudden death, arrhythmias, and exercise-induced asthma, in that order.

Due to the tall, lean nature of most basketball and volleyball players, a focused cardiac, ophthalmologic, and musculoskeletal history and physical examination is essential to identify those athletes at risk for Marfan's syndrome. Marfan's syndrome is a connective tissue disorder that leads to production of defective fibrillin (3). Subsequently, cardiovascular alterations may occur, leading to dilation of the aortic root secondary to cystic medial necrosis. Life-threatening complications from this disorder include a ruptured aortic dissection or aortic aneurysm. Aortic dilatation on echocardiography will likely preclude an athlete from participation (3).

Injury prevention is stressed during the screening process with a focus on lower extremity biomechanics, and flexibility and core strength through the hips, gluteals, pelvis, and lumbar spine. In the upper trunk, a general re-

view of shoulder, thoracic spine, and supporting upper trunk musculature will provide a means to identify areas of concern. Muscle testing for inherent asymmetrical weakness, atrophy, and inflexibility helps to tailor a strength and conditioning program that will correct imbalances. Adherence to in-season workouts is important to maintain flexibility, muscle tone, and endurance.

Female ACL tears occur four to eight times more often than in males; this fact alone has triggered significant amounts of research over the last decade (6–10). Many reasons have been postulated, such as a narrow intercondylar notch (11,12), presence of the estrogen cycle (13), and planovalgus misalignment (14). However, research has shown differences in male and female muscle balance and firing (15). Females tend to be quadriceps-dominant, which antagonizes the ACL, while males are more hamstring-dominant, which stabilizes the ligament. The peak-torque ratios of the hamstring in females were slower than in males. This means that when the knee is destabilized, the hamstrings are slower to contract and protect the ACL in females.

Unlike most other factors, this can actually be improved with appropriate training. Plyometric training for female basketball players has been shown to decrease ACL injuries (16). Reinforcing the proper way to perform basketball-specific skills, such as landing from a jump, running and stopping, and performing layups, helps as well, and this takes coaching. A study looking at injury rates compared with skill level noted that the more elite (collegiate and professional) athletes are injured less often than players from high school and younger (17). By working on a lower-extremity plyometric program and practicing better basketball skill technique, a female player has a better chance of avoiding injury.

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28

CYCLING

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DELMAS J. BOLIN

Bicycling for transportation and fitness has enjoyed a resurgence in the United States in recent years. Once considered a children's toy, the bicycle is now a favorite fitness avenue for adults, from stationary spinning to mountain biking. With this surge in popularity has come an increase in the number of bike-related injuries, which account for over half a million emergency room visits annually (1). The actual number of injuries is certainly much higher, as many injured cyclists self-treat and never seek care. Of the approximately 800 riders who die each year, nearly two thirds are due to traumatic brain injury sustained by riders not wearing a protective helmet (1). Cycling's physical demands place the rider at risk for a variety of traumatic and overuse injuries. An osteopathic approach to many of these overuse injuries can reduce time away from training and improve rider performance.

HISTORY

Cycling is an ancient sport with origins dating back to 2300 B.C. in China. The modern era of cycling began with de Sivrac's Celerifere in France in the 1790s. This hobby-horse was a heavy, rigid, two-wheeled wooden machine with a central backbone. The rider propelled himself by paddling his or her feet on the ground. Initially used for gardening, these early bikes were later raced along the Champs Elysées. In perhaps the earliest example of cycling overuse injuries, young men frequently developed hernias from straining to steer the hobby-horse (2). In 1817, Baron Karl von Drais introduced the Dtaisienne, which had a steering bar connected to the front wheel. These rigid, solid-wheeled machines transmitted every bump in the road to the rider and were nicknamed bone-shakers.

In the 1830s, a Scottish blacksmith named MacMillan added foot pedals to the wheels. By the 1870s, the Penny Farthing or ordinary bicycle was introduced. The hallmark of this classic design was a very large front wheel with attached pedals paired with a small back wheel (3). The wheel ratio was the secret to the increased speed and efficiency of this bicycle; the larger the wheel, the greater the distance traveled with each revolution of the pedals. Almost as soon as the self-propelled bike was available, records began to be kept. In 1876, the first 1-hour cycling record (25.508 km) was recorded in England (4).

The Penny Farthing offered a tremendous mechanical advantage to earlier designs and made the bicycle an efficient mode of transportation but more difficult to ride than earlier designs. Riders were commonly injured when the front wheel of this model caught against the uneven stones or deep ruts in primitive roads. The sudden stop often propelled the rider head-first from a significant height onto his head, the origin of the phrase "taking a header" (3).

This problem was partially solved by the development of the safety bike, introduced first in the 1830s, which featured smaller, equal-sized wheels. During the late 1800s and early 1900s, improvements to the safety bike, including chain-drive systems, air tires, and gear-change technology, resulted in the modern bicycle. During this time bicycles were widely used for transportation. Bicycle racing became extremely popular and drew large crowds. Champion cyclists were celebrities and the highest paid athletes of the time (5).

TABLE 28.1. RELATIVE PERCENTAGE OF INJURIES TO MEN AND WOMEN PARTICIPATING IN A NATIONAL MOUNTAIN BIKE RACE

Injury	Men	Women	Men (%)	Women (%)
Fracture	26	13	16.6	28.9
Concussion	9	2	5.7	4.4
Abrasion	60	18	38.2	40.0
Contusion	30	9	19.1	20.0
Laceration	11	1	7.0	2.2
Sprain	12	2	7.6	4.4
Strain	7	0	4.5	0.0
Hematoma	1	0	0.6	0.0
Puncture	1	0	0.6	0.0
Totals	157	45		

From Kronisch R, Pfeiffer R, Chow T, et al. Gender differences in acute mountain bike racing injuries. *Clin J Sport Med* 1994;13:1–14.

The bicycle's basic efficiency was a main factor in its rise to popularity. Walking consumes 0.75 calories per kilometer, whereas a bicycle increases the speed fourfold while decreasing caloric expenditure fivefold.

The development of the motorcar as affordable and faster transportation during the early twentieth century resulted in the bicycle becoming primarily a children's toy. During the later part of the twentieth century and the beginning of the twenty-first century, cycling has enjoyed renewed interest due to physical, psychological, and ecological benefits. Cycling is now one of the largest participatory sports for recreational and competitive athletes.

EPIDEMIOLOGY

Injury patterns in biking are dependent upon numerous factors including the age and experience of the rider, terrain, time of day, speed, and the type of riding. Children are much more likely to be injured than adults, with the peak incidence of injury between the ages of 9 and 15 (6,7). Boys are two to three times more likely to be injured (7). Among adult riders, traumatic and overuse injuries are influenced by type of riding. Long-distance road biking predisposes the rider to unique overuse injuries including neuropathies of the hands, perineum, and feet. Mountain bikers are more prone to traumatic injury. These injuries peak in riders between 20 and 39 years of age and usually occur from loss of bike control on unfamiliar downhill rerrain at excessive speed (8). Prospective injury data have recently been reported for a major U.S. mountain bike race. In this competitive setting, women had nearly twice rhe risk for injury as men and were four times more likely to sustain a fracture. Table 28.1 shows the relative percentages of injuries in competitive male and female mountain bikers (9). The distribution of injuries is relatively unaffected by gender.

BICYCLE DESIGN

There are generally five types of bicycle design: racing, sport/touring, mountain, hybrid, and juvenile/specialty bikes (tandem, stunt bikes, etc). The design dimensions of the different bike types are subtly influenced by their function. Likewise, the proper fit of a bike to the rider is highly dependent upon what the rider intends to do.

Figure 28.1 shows the frame and main components of the typical bicycle. A road bike has steeper frame angles (e.g., between seat stay and chain stay) that produce a more upright geometry (10). Road cyclists usually ride on a lightweight bike in an aerodynamically advantageous crouched position on narrow, low-resistance tires. Road bike components are selected for

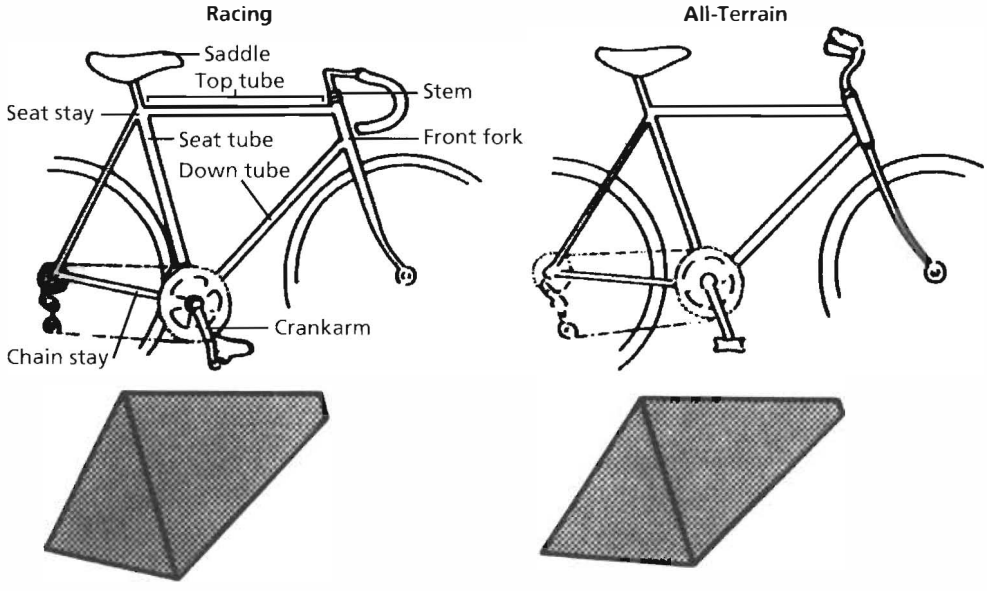

FIGURE 28.1. The dimensions of road and mountain bikes. The steeper angles of the road bike produce a more upright geometry. Main components are labeled. (Adapted from Burke E. Proper fit of the bicycle. *Clin J Sport Med* 1994;13:1–14.)

performance and speed. In contrast, mountain biking typically involves off-road riding on dirt roads or narrow trails (single track). The mountain bike has a heavier frame, which lowers the center of gravity, and is equipped with wider, deeper-treaded tires than a road bike. Mountain bike frames have more shallow angles (10), allowing the rider to sit back placing more weight on the back wheel for traction while climbing. Advanced components, such as front and rear suspension, may be required on more advanced single-track rides with steep descents and jumps.

Hybrid bikes are a combination of touring and mountain bikes. These bikes have the geometry of a road bike, but with a stronger frame and more heavily treaded tires. These characteristics permit their use on a variety of terrain, including paved, gravel, or dirt trails.

BICYCLE FIT AND PERFORMANCE

Proper bicycle fit not only prevents injury but also optimizes efficiency, whereas an improperly fitted bicycle predisposes the rider to injury. Every component of the bike from the saddle to the length of the crankshafts to the handlebar stem is important and should be customized for the rider to optimize performance.

The first step to ensuring proper fit of the bicycle is to determine the correct frame size. While straddling the bike, the top tube should be 1 to 2 inches (2.5 to 5 cm) below the crotch for road bikes, and 3 to 6 inches below the crotch for mountain bikes.

A number of different measurements for estimating seat height are available. The saddle should be even with the trochanter when the rider stands barefoot next to the bike (10). We have found that a rider can quickly estimate the correct position by sitting on a supported bike with the pedals in the 6 and 12 o'clock positions. When the seat is at the correct height, the leg should be nearly fully extended when the pedal is in the 6 o'clock position. At this height, power output is maximized and oxygen consumption and calorie expenditure are minimized (11,12). Seat height influences muscle recruitment, movement patterns, and joint forces during pedaling (13–15). Mountain bike saddle height is typically lower than that of a road bike, giving the rider a lower cen-

ter of gravity, greater maneuverability, and stability. Subsequent changes to saddle height should generally be in small (0.25 in.) increments spaced out over a few riding sessions (10). The saddle should be level, or tilted slightly downward to relieve pressure on the perineum during the ride.

On a road bike, the saddle should move fore or aft so that when the pedal is in the 9 o'clock position, a plumb line can be dropped from the anterior patella through the middle of the pedal. In contrast, the mountain bike saddle should be positioned so that the knee is 2.5 to 15 in. (1 to 6 cm) behind the axle of the pedal in the 9 o'clock position. This allows for more relative weight to be placed on the rear wheel when climbing (10). The ball of the foot should be centered over the pedal axle.

The handlebars are positioned at least 2 in. (5 cm) below the saddle height. Taller riders typically place the handlebars lower, to achieve the most aerodynamic riding position. The stem length may be customized for comfort. The proper handlebar position and stem length can be estimated by placing the tip of the olecranon against the saddle front; the fingertips should rest on the horizontal center of the handlebar.

The crankarm length influences knee and ankle angles, revolutions per minute (rpm), and leverage. The standard crankarm (35 ft [170 cm]) fits most riders from 5 to 6 feet tall. A shorter crankarm is better for low-gear, low-rpm pedaling such as climbing. Longer crankarms are better for pushing higher gears on flat surfaces (10). Power output studies show optimal output when crankarm length is proportionate to lower extremity length (trochanter to foot) (16). Higher-performance cyclists require longer than expected crankarm lengths to optimize power output at their usual higher pedal rpm (17).

AERODYNAMICS AND POWER

Aerodynamic drag and friction are significant slowing forces that the rider must overcome. Aerodynamic drag is the most important slowing force in road biking. At speeds greater than 13 kph (8 mph), common for both recreational and high-performance riders, it accounts for more than 80% of the total retarding force; at 40 kph (25 mph), drag is 90% of the slowing force (18). The power needed to overcome aerodynamic drag increases in proportion to the cube of velocity. Since the power-generating capacity of human riders is limited, most riders focus on decreasing drag to increase speed without expending more energy (4).

Drag can generally be reduced in four ways:

1. Shielding the rider and machine inside a shell can reduce drag by up to 80% (4). This is impractical for most recreational riders.

2. Decreasing the frontal cross-sectional area that the bike and rider present to the wind, which is more practical. Aerobars, which allow the rider to lean forward with elbows tucked and resting on padded handlebars, decrease the frontal area and place the rider in a streamlined position. This position reduces drag by nearly 30% when compared with upright riding positions (4) but does not alter the rider's VO_{2max} (19). Further reductions in drag can be achieved by optimal positioning of components and accessories. For example, placing the water bottle behind the seat post and placing the tire pump in a horizontal position both decrease the frontal cross-sectional area of these components. A water-bladder worn by the rider further decreases drag as the rider can maintain aerodynamic position with both hands on the handlebar while hydrating.

3. Smoothing out both bike and rider. Cables, rods, sharp cotners, and unnecessary components are hidden within the frame or removed. Clipless pedals offer less wind resistance than clips or ordinary pedals (4) and increase mechanical efficiency (20). Narrow, smooth-treaded tires offer less rolling resistance. This frictional component is more important at lower speeds. *Aerodynamic wheels*, either lens-shaped lenticular discs or three-spoke composite wheels, have significantly less wind resistance compared with standard 36-spoke wheels. In practical terms, a rider using these wheels gains 30 meters every kilometer (33 yards every mile) over an equal opponent using spoke wheels (21).

Skin-tight riding clothes and aerodynamic helmets dramatically reduce drag. Wind-tunnel studies demonstrate that a smooth tight skin-suit significantly reduces wind resistance. An equal opponent wearing loose clothing would lose 25 meters for every kilometer (27 yards for each mile) of the race (21). Racing helmets also reduce drag dramatically; a rider wearing a standard helmet would fall behind 19 meters every kilometer (21 yards for each mile) (21). Significant gains in performance can be realized by attention to technique and equipment independent of rider fitness.

4. Drafting. Those who draft follow the lead bike very closely, often with wheel gaps of only 15 to 30 cm (6 to 12 in.). Drafting cyclists consume 30% to 40% less energy than the lead rider. A group of riders, alternating in the lead position, can travel 2 to 6 kph (1.25 to 3.75 mph) faster than an individual riding alone (22). Drafting requires the concentration of all riders as a sudden change of speed or direction by the lead rider can result in multiple crashes.

TRAUMATIC INJURIES

Whether single-track mountain biking or drafting on a road bike, cycling's combination of high speed and technical difficulty predisposes the rider to accidents. In covering a cycling event, the physician's primary survey of a downed rider includes careful examination of the most frequently injured sites. Skin lacerations and abrasions, head injuries, and injuries to the upper extremity and abdomen are most common. Table 28.2 lists the common traumatic injuries seen in cycling.

The skin is at risk from falls and impact from trail brush while riding. Skin abrasions against the riding surface, commonly known as road rash, must be thoroughly cleaned to prevent traumatic tattooing. Initial treatment with lidocaine (Xylocaine) jelly anesthetizes the skin to make subsequent cleaning tolerable. Lavage with saline or surgical scrubbing may be necessary to cleanse the wound of small stones and debris to avoid traumatic tattooing of the skin. Antibiotic creams, such as 2% mupirocin, are as effective as oral antibiotics in treating and preventing cellulitis. Nonadherent dressings such as Secondskin (Spenco), Telfa, or a semipermeable dressing such as Tegaderm should be applied with tape and covered over with flexnet or a similar bandage. This ensures that the dressing remains in place during the remainder of the ride. Tetanus booster should be up to date.

TABLE 28.2. COMMONLY SEEN TRAUMATIC INJURIES IN BIKING

Skin abrasions (road rash)
Solar injury to skin
Head injury, concussion
Eye injury, corneal abrasions, trauma from insects, stones, dust
Phalangeal, metacarpal, distal radial fractures
Acromioclavicular separation
Clavicle fracture
Glenohumeral dislocation
Blunt abdominal trauma (liver, pancreas, and splenic laceration)

Unprotected skin is at risk for solar injury on rides of even moderate duration (1 hour). Darker-colored jerseys absorb more photoenergy and are more protective. Wet material is less effective, and therefore newer rapid-drying fabrics should be worn. Water-resistant sunscreens are available that protect against both ultraviolet A and B rays. A minimum sunblock of skin protection factor (SPF) 30–45 should be used under the clothing. These should be applied copiously 30 minutes before exercise and repeated after periods of intense sweating (23).

Head injuries are a significant source of both morbidity and mortality among cyclists. The initial evaluation of a downed rider must include a careful survey for concussion and head injury. The importance of head protection cannot be overemphasized as two thirds of cycle-related deaths result from head injuries. Helmet use could have prevented 80% of these deaths. Most head injuries occur in childten under 15 years old, with ages 5 to 9 at greatest risk (24). Helmet use is the single most important intervention to prevent cycling-related head injury, including concussion, skull frac-

ture, and intracranial hemorrhage. Patient education programs can be effective at increasing helmet use and decreasing traumatic head injury (25).

Eye injuries, including corneal abrasions or penetrating trauma, may result from falls or by impact with insects, stones, or dust thrown into the air. High speeds can cause tearing in unprotected eyes. Protective polycarbonate (shatterproof) lenses should be worn to protect the eyes from sun, wind, and airborne debris.

The rider's natural response to falling is to reach out a hand to catch himself. As a result, upper extremity injuries including the hand, wrist, shoulder, and acromioclavicular joint injuries are common. Impact fractures of the phlanges, metacarpals, and carpal bones as well as the distal radius are frequent. On-site care should focus on controlling bleeding and splinting in a neutral position. A roll of electrical or athletic tape and the tire pump are helpful for making temporary splints. Definitive treatment should be sought as quickly as possible, as many carpal fractures are prone to avascular necrosis. Impact fractures of the olecranon frequently occur when the bike slides out from under the rider during high-speed cornering.

Shoulder dislocations and clavicle fractures are common, particularly in high-velocity impacts with cars and in drafting accidents where many riders are injured at one time. Relocation of a dislocated shoulder should be done as quickly as possible, and numerous techniques have been described. If initial attempts are unsuccessful, anesthesia in the emergency department may be required to obtain adequate relaxation for relocation maneuvers. After reduction, radiographs should be obtained to rule out a glenoid fractures or Hill-Sachs lesion, which would predispose to later instability. Acromioclavicular separations or clavicle fractures should be immobilized in a sling, and prompt follow-up arranged. For mountain bikers, a spare inner tube can be used as a temporary sling until definitive treatment can be sought. Often, riders have a small saddlebag first-aid kit consisting of adhesive bandages, antibiotic cream, electrical tape, a multifunctional tool, and a spare inner tube.

Abdominal injury, although less common, is also a potentially significant source of injury. Blunt abdominal impact either from handlebars or surrounding environmental objects (such as tree limbs) should be thoroughly investigated. In one series over a 30-year period, bicycle accidents were the most common cause of pediatric abdominal trauma (26). Significant injuries including liver, pancreas, and splenic laceration have been reported. There is frequently little external bruising, even with significant internal trauma. This suggests that even in minor accidents, significant forces may be transmitted ro internal organs through the small cross-sectional area of the handlebar end (27). A high index of suspicion for abdominal trauma should be maintained when evaluating children involved in bike accidents, as these injuries may be life-threatening if not recognized promptly and aggressively treated (28).

OVERUSE INJURIES

Cycling is a sport that lends itself to overuse injuries. Most of the reports of overuse injuries are taken from surveys of participants in endurance events. A rider with suboptimal equipment and an improperly fitted bike may be predisposed to overuse injuries. As in other sports, anatomic malalignment, such as a valgus deformity of the knee, may contribute to overuse syndromes.

The Cervical Spine

The maintenance of aerodynamic posture by a cyclist places enormous strain on his or her neck and low back. It is not surprising that neck and low back pain are extremely common complaints among riders. Of 113 participants in a 500-mile bicycle tour, two thirds reported neck and shoulder complaints, with 20% labeling their discomfort as significant (29). Riders often develop painful trigger points in the neck or over the trapezius. These masses are exquisitely painful rubbery or ropy nodules in the muscle or fascia. Myofascial pain syndromes

are caused by trauma or overuse (30). On long rides, the trapezius and levator scapulae function as check reins, holding the neck in hyperextension for long periods. The left side is more commonly affected as the rider looks over his or her shoulder for overtaking traffic. An insertional tendinitis may develop on the superomedial aspect of the scapula (30). Multiple muscles in the neck and shoulder girdle can be involved.

Neck pain secondary to repetitive microtrauma, or multiple microwhiplash, can occur on long rides with the neck in a hyperextended position. The accumulated damage from multiple jarring motions is thought to contribute to muscle and tendon strains, fasciitis, muscle spasm, and the development of trigger points (30). Pain and disability may require temporary cessation of riding and weeks to months of rehabilitation.

In addition to osteopathic techniques discussed subsequently, treatment for neck injuries should include careful scrutiny of the rider's position on the bike. Adjusting the seat position, increasing handlebar height, and decreasing the handlebar reach place the rider in a more upright riding position, obviating the need for extreme neck hyperextension. Frequent changes of hand position may also help to reduce symptoms. The recreational rider may occasionally need to discontinue riding and cross-train for short periods until symptoms abate.

The Thoracic Spine and Shoulders

The chronically flexed posture necessary to maintain good aerodynamics during road cycling can result in overload syndromes of the midback and shoulders. This nearly horizontal and hunched position of the thoracic cage stresses the posterior myofascial elements. The loads placed through the upper extremities and shoulders are dependent primarily on handlebar type and hand position (standard drop bars vs. aero bars). The scapulae are literally being pulled away from the posterior thorax as a result of this positioning, placing tremendous stress on the scapular stabilizing structures, as alluded to earlier. Significant dysfunction can result at the cervicothoracic junction, in the periscapular region, and in the midthoracic spine. Osteopathic treatment of the resultant dysfunctions can provide symptomatic relief in addition to adjusting rider position, as previously described.

The Lumbar Spine and Pelvis

The low back is prone to overuse in cycling and particularly in road cycling. From a purely biomechanical perspective, the low back is the platform from which pedal force originates. In the ideal riding position, the ischial tuberosities are seated on the saddle and the pelvis maintained in a neutral position with the low back in a slightly lordotic posture. Most of the support muscles that maintain this position contract isometrically.

The risk for overuse injuries increases when the ideal pelvic and lumbar positioning is not maintained. Again, proper bicycle fit cannot be overemphasized. If the stem/top tube length is too great, the rider will be forced into an uncomfortable lordotic posture. If the handlebars are too low, regional lumbar flexion is exaggerated, placing increased pressure on the myofascial and osseous elements of the low back. If the stem/top tube length is too short, the lumbar spine is forced into flexion, increasing the pressure on the intervertebral discs (30).

Pelvic position is mainly dependent on muscle balance. Remember that both anterior and posterior muscle groups attach to the thoracolumbar fascia making this a key structure at risk due to muscle imbalance and overuse. Tight quadriceps pull the pelvis anteriorly, while tight hamstrings pull the pelvis posteriorly. The iliopsoas, used in pulling up the pedals, also tends to tilt the pelvis anteriorly. From above, the neutral pelvic position is dependent on the abdominal muscles, which assist in both flexion and extension of the lumbar spine. During periods of prolonged climbing or pushing large gears, the gluteus and hamstrings may become fatigued, leading to anterior pelvic tilt and painful hyperextension posture of the lumbar

spine (31). Wider, less inflated tires can diminish low back stress by decreasing road vibrations.

The Perineum

Compressive forces on the perineum during cycling predispose it to overuse injury. Anatomically, men have a lower pubic symphysis than women. Chronic pressure on this area against the saddle entraps the pudendal nerve, resulting in perineal numbness. In men, prolonged pressure may lead to venous leak from the penile vessels resulting in impotence. Impotence typically resolves over a period of hours, but prolonged dysfunction has been reported, usually associated with longer rides. Saddle position and padding can decrease chronic pressure on the perineum. A horizontal or slight nose-down saddle position opens the angle between the saddle and the pubic symphysis, reducing the pressure against the pudendal nerve. Frequent brief periods of standing while riding restores circulation and may help alleviate symptoms. Padding on saddles and padded cycling shorts provide further cushion. Newer saddles with central cutouts reduce direct pressure to this area and are associated with fewer symptoms (32). Figure 28.2 shows three standard saddle designs.

Saddle sores result from chafing that progresses to ulceration in the perineal area. Careful hygiene is necessary to avoid infection in this sensitive area. Padded saddles and cycling shorts can reduce pressure while healing. Sitz baths can be helpful to speed healing. Numerous topical skin products have been suggested to help saddle sores, but neither steroid nor salicylate creams prevented sore development (33). Creams may promote friction and saddle sore formation, as cyclists who used a topical cream developed more frequent sores than those who did not use it (34).

Repetitive microcompressive trauma can lead to aseptic necrosis of the superficial perineal fascia and nodule formation. The nodules form on either side of the perineal raphe and can cause discomfort to the rider. There is no specific treatment, and they have been reported only in elite male cyclists (35).

The Foot

Peripheral nerve compression resulting in interdigital neuroma can occur in the foot. Tight clips, straps, and narrow shoes with rigid forefoot compress the peripheral nerves and can lead to paresthesias. Clipless pedals and a widened toe box can relieve pressure and diminish symptoms. Occasionally, subtle cleat position adjustments (typically directly underneath the metatarsal heads) can be helpful. Orthotics with metarsal cookie padding can restore a metatarsal arch and relieve pressure on the affected interdigital

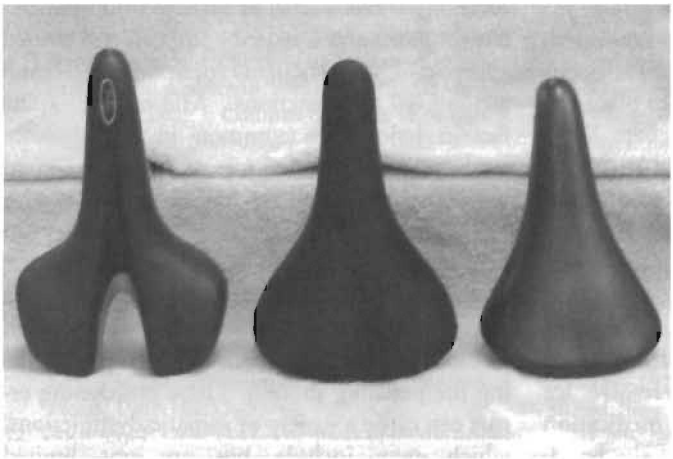

FIGURE 28.2. Saddles. Central cutouts (**left**) relieve pressure from the perineal area and the pudendal nerve. Alternative designs involve a well-padded saddle with central depression (**center**) or a standard leather saddle (**right**).

nerve. In competition, the shoe upper can be cut away to decrease pressure.

Compressive Neuropathies of the Upper Extremity

Handlebar neuropathies commonly affect cyclists. Paresthesias in the distribution of the ulnar nerve, so-called cyclist's palsy, are the most common and affect sensation of the fifth and ulnar side of the fourth digit. Ulnar nerve damage occurs from prolonged wrist extension with extreme pressure of the hypothenar eminence against the handlebars. The nerve is stretched and crushed against the bony margins of Guyon's canal, resulting in a traction neuropathy. Inexperienced road cyclists are prone to this injury because they do not change hand position in an effort to maintain a grip close to the brake levers. Drop handlebars and ill-fitting bikes with extended reach are two common contributors. On examination, riders have decreased sensation in the ulnar distribution and decreased fine motor control of the hand. Atrophy of the dorsal interossei and decreased strength in the abductor digiti minimi and adductor pollicus longus may be noted. Symptoms usually abate within 24 hours, but aggressive cyclists who continue to ride despite pain or discomfort may have prolonged symptoms (36). Partial claw-hand deformities, which resolved over several months, have been reported (37). Median nerve palsy related to cycling has also been reported (38).

Handlebar neuropathies are easily prevented. Proper bike fit with appropriate reach is essential. Excessive reach forces the rider to place too much weight forward on the handlebars. With drop handlebars, hand position should be shifted frequently to avoid prolonged hyperextension. Padded riding gloves and handlebars dampen vibrations and reduce neuropathy (36). For off-road riders, vibrations can be further dampened with a front suspension and wider, less inflated tires. In recreational riders, a hiatus from riding may be appropriate until symptoms resolve. Ice and nonsteroidal anti-inflammatory medication may help. Osteopathic treatment of the hand, wrist, and upper extremity complex can be beneficial if specific somatic dysfunction is identified. If symptoms persist, further workup including electromyography is warranted. Rarely, more aggressive treatment including steroid injection and surgical release may be necessary.

OSTEOPATHIC CONSIDERATIONS

Osteopathic considerations in cycling are driven primarily by the effect of the cycling position on the spine and pelvis. Trauma can occur and the resultant injuries must be dealt with appropriately, including osteopathic functional evaluation and treatment. Most injuries are of the overuse type and may have a biomechanical and/or anatomic basis (e.g., a short leg). The challenge for the clinician is to ascertain the relative contributions of the machine and the athlete to the injury being evaluated. For example, low back pain may be related to intrinsic pathology or be driven by an extrinsic stressor such as inappropriate seat height.

Regarding cycling-related somatic dysfunction, it has been our experience that most spinal somatic dysfunction is in the sagittal plane related to the chronic postural strain from the typically flexed posture a cyclist assumes. Most lesions are either flexion or extension dominant, depending on the region of the spine affected and the primary type of cycling performed by the athlete.

Cervical spine extension and extension with rotation lesions occur frequently (multiple microwhiplash) and must be evaluated and treated appropriately, particularly occipitoatlantal and atlantoaxial dysfunctions. Midthoracic spine flexion lesions are common in long-distance road cyclists. These lesions can be easily treated with both muscle energy and mobilization with HVLA impulse. Thoracic extension exercises can help stabilize this region and improve mobility.

Regarding the lumbar spine and pelvis, a common mistake is to position the seat too high or too low. This causes rocking of the pelvis during the pedaling motion. These positioning errors can cause a variety of somatic dysfunctions, which may include but are not limited to sacroiliac dysfunction, sacral dysfunction,

pubic symphysis dysfunction, innominate rotation, and lumbar facet syndromes.

Upper extremity dysfunction can occur, as previously described. The resultant compressive neuropathies are amenable to osteopathic techniques designed to decompress the carpal tunnel region. Always evaluate and treat the cervical spine in any upper extremity injury or dysfunction.

Junctional dysfunctions also commonly occur (i.e., cervicothoracic, lumbosacral), and they should not be left untreated. Recurrence of dysfunction, assuming exercises and stretches are done, can happen as a result.

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29

DANCE

RICHARD BACHRACH

Dancers are a unique form of athlete. Although most injuries seen in dancers are similar to those encountered in all athletes, particularly aerobic exercisers, runners, and gymnasts, many significant differences exist with regard to pathomechanics and approaches to management.

THE DANCER

The physical conditioning achieved by a professional ballet dancer in the corps de ballet is approached by that of only a few world-class athletes. To realize and maintain this razor-sharp neurology and conditioning, the ballet dancer must participate in a minimum of 20 hours of strenuous class work weekly. In a major dance company, this schedule is maintained year round with only brief respites. During rehearsal and performance periods, when the number of classes may be reduced, the schedule becomes even more trying. This environment discourages dancers to disclose injuries until too late, and this affects the dancer's psyche. The saying goes among ballet dancers: Miss one class and you know it, two classes and the teacher knows it, miss three classes and the whole dance world knows—and there goes the job.

For every ballet job available, there are hundreds of dancers ready and waiting in the wings. An even greater number of jazz and modern dancers await placement in dance worlds that have less support and financing (except for Broadway musicals, which are limited and ever shrinking in number). Therefore, dancers seeking treatment are usually not interested in prescribed rest. Recovery from an injury depends to a significant extent on their confidence in the physician. Confidence can be facilitated if the physician has a working knowledge of the basic dance positions and movements.

All ballet movements of the lower extremity evolve from one of five basic positions (Fig. 29.1A to E). The ability to assume these positions depends on the amount of external rotation (turnout) available at the coxofemoral joint (Fig. 29.2). Attempts to compensate for decreased range of external rotation at the hip by screwing the knee and/or everting and pronating the foot are the principal alignment fault and the major cause of dance-related injury (1–9).

The principal turnout muscles are the gluteus maximus, most hip adductor fibers, and the outward rotators deep to the glutei, which arise from the pelvis and sacrum, and insert into the greater trochanter of the femur. The principal structural determinants of the range of external rotation at the hip joints are the iliofemoral and pubofemoral ligaments (together forming the Y ligament of Bigelow, the strongest ligament in the body), and the angle between the neck and shaft of the femur when seen from above. This angle (version) ranges from +40 to −20. The hip with a greater angle has greater range of internal rotation, while less anteversion increases the range of external rotation (7,10). Because mature ligaments do not stretch without tearing, stretching a ligament compromises the integrity of the joint involved. For these reasons, an individual who starts dance training after the age of 10 or 11 cannot be expected to structurally increase turnout (7,11).

The following movements derive from one of five basic ballet positions (Fig. 29.1A to E). Their proper execution depends on the turnout effected at the hip and on proper alignment.

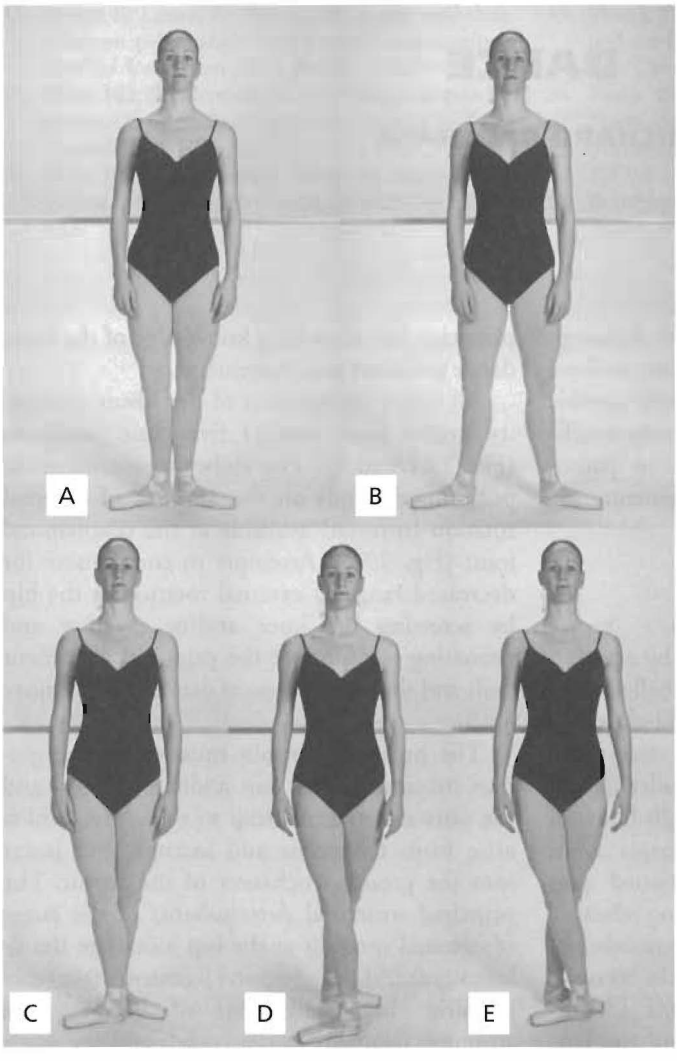

FIGURE 29.1. Positions of ballet. **A,** First position. **B,** Second position. **C,** Third position. **D,** Fourth position. **E,** Fifth position.

1. *Demi-Plié* (*pli*, fold [French]). (Fig. 29.3A). This movement may take place in any of the five basic positions on both or one leg. The knees, hips, and ankles are flexed, and the full foot rests on the floor. This lowers the center of gravity, stretches the soleus component of the triceps surae, and thus prepares for air work. It involves concentric contraction of the hip flexors, abductors, and external rotators. There is concentric contraction of the hamstrings at the knee and concentric contraction of the dorsiflexors of the foot and toes. The hip adductors and proximal hamstrings contract eccentrically. Contraction of the quadriceps (except the rectus femoris at the hip) and the peroneus longus, tibialis posterior, and flexor hallucis longus is also eccentric. The depth of the demi-plié is limited by the tension in the soleus muscle and by the abutment of the anterior margin of the distal tibia on the neck of the talus. The patella is compressed against the femoral trochlea. In a properly executed demi-plié, the center of gravity moves directly downward through the legs, the knees passing directly over the feet in the same plane.

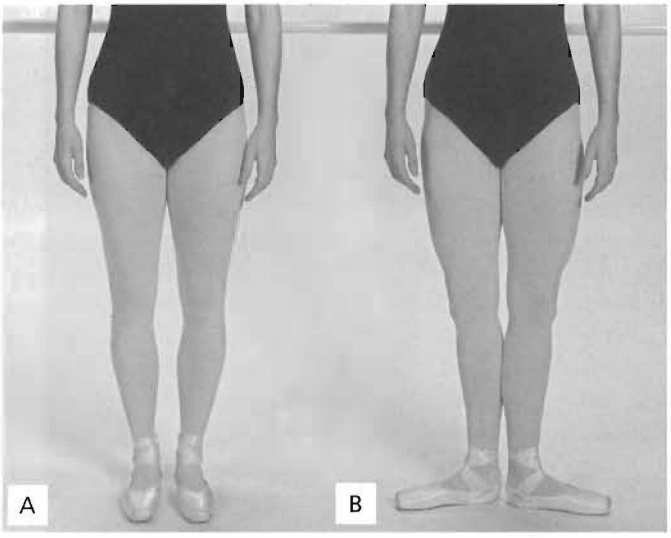

FIGURE 29.2. Turnout. **A,** Dancer standing in neutral. **B,** Femurs externally rotated.

2. *Grande-Plié* (Fig. 29.3B). In this movement, the center of gravity descends further. The amount of flexion at the knees, hips, and ankles is increased, and the heels are finally lifted from the floor. Extreme stress is put on the knee, particularly components of the patellofemoral and medial tibiofemoral compartments.
3. *Relevé* (Fig. 29.3C). While the plié is a vertically downward movement of the center of gravity, the relevé facilitates ascent of the center of gravity, at the same time shifting the body weight to the ball of the foot (in the case of advanced or professional female ballet dancers, to full pointe). It requires active concentric contraction of the long plantarflexors of the foot, toes, and ankle, and also of the quadriceps, because it is usually executed with the knee extended. There is simultaneous eccentric or lengthening contraction of the hamstrings. The deep external rotators and the glutei concentrically contract to stabilize turnout throughout the movement. The adductors eccentrically lengthen to allow for abduction at the hip.
4. *Pointe.* Unless a dancer is an aspiring professional female ballet dancer, she will infrequently confront this position. Nevertheless, full pointe is the end point of the relev. In the presence of a long posterior process of the talus or os trigonum tarsi, this position is difficult to attain and is painful in the posterior triangle. There is less strain on the triceps surae en pointe than in relev to half-toe. Ankle stability with the foot en pointe is provided by the flexor hallucis longus and posterior tibial and peroneal (stirrup) muscles.
5. *Tendu* (*Battement Tendu*) (Fig. 29.3D). This movement is a sequential plantarflexion of the foot as it slides along the floor, until only the tips of the toes remain in contact. (The heel leads the movement until it is forced to release from the floor.) The leg remains straight, with active eccentric contraction of the hamstrings. The leg returns to its starting position in a reverse sequence. (When the same movement is performed with the foot brushing off the floor, it is termed *dégag,* meaning to disengage.) These movements are executed also to the side and back and are essential elements of the ballet barre.
6. *Développé* (Fig. 29.3E). This movement is almost always performed with full outward

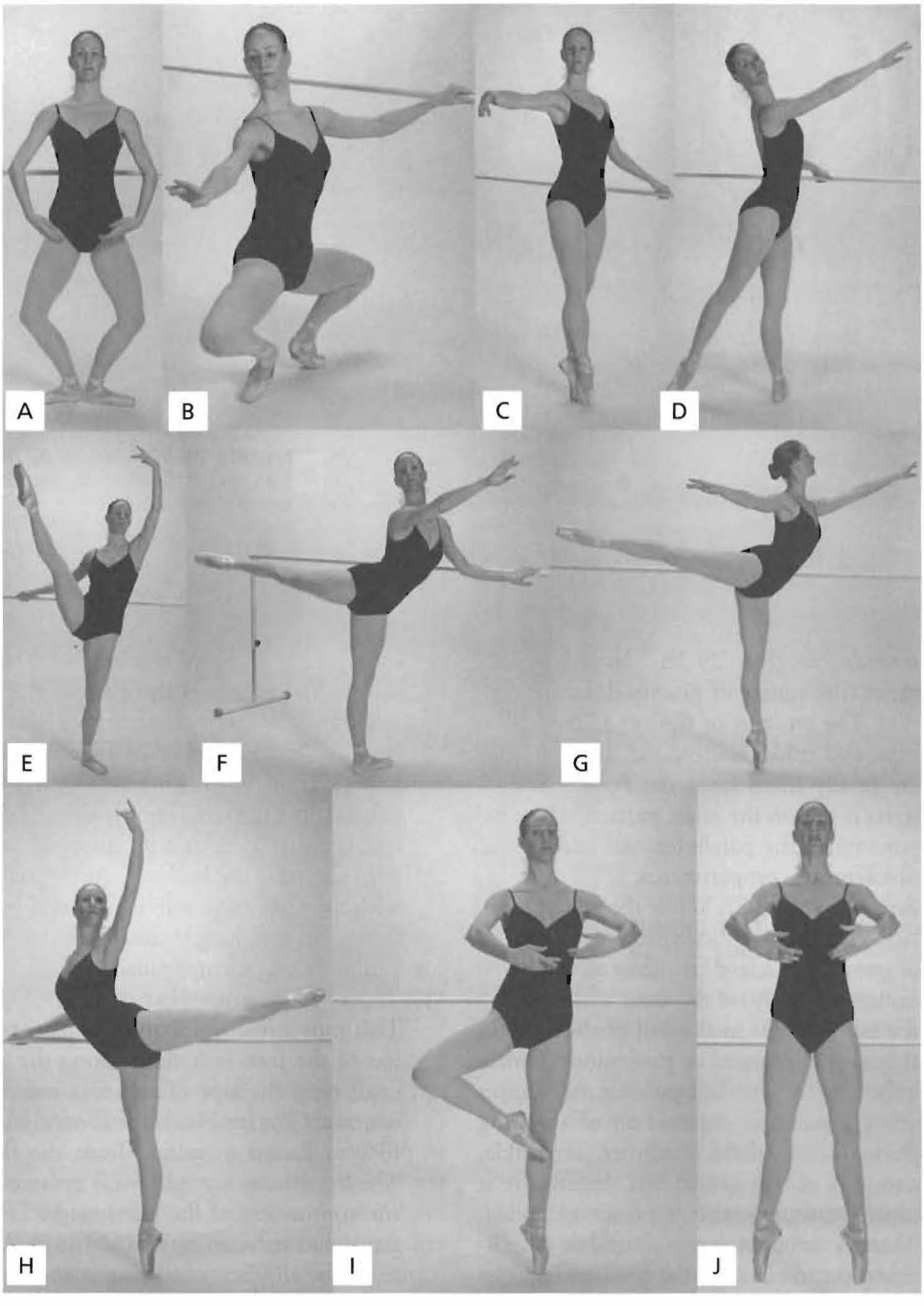

FIGURE 29.3. Basic dance movements. **A,** Demi-plié. **B,** Grande plié. **C,** Relevé. **D,** Tendu (or battement tendu). **E,** Développé. **F,** Grand battement. **G,** Arabesque. **H,** Attitude. **I,** Pirouette or tour. **J,** Jumps.

rotation at the hip. The thigh folds into 90 degrees of flexion at the hip. Simultaneously, the knee also flexes and the foot is drawn up the medial aspect of the standing leg to the knee. Retaining hip flexion, the knee is then extended fully, either to the front, side, or back. Développé front requires active concentric contraction of the hip flexors, in particular the iliopsoas and the rectus femoris. Of primary importance is the sartorius, whose concentric contraction assists in flexion at the hip joint, flexes the knee, and outwardly rotates the thigh during the preparatory action. The hamstrings first concentrically contract to flex the knee, and then eccentrically lengthen during the extension of the leg, as do the gluteals. The external rotators contract to maintain the outward rotation, both on the supporting leg and also on the gesturing leg.

7. *Grand Battement* (Fig. 29.3F). This is often referred to by the dancer as an extension and involves simultaneous flexion of the hip to at least 90 degrees with the knee maintained in extension. This movement is usually performed with some speed but may also be done slowly. It is essentially an amplified tendu and dégag, passing through these two movements with the leg sweeping straight up. Proper execution requires concentric contraction of the hip flexors, particularly the iliopsoas and rectus femoris. The quadriceps contract concentrically to straighten the knee, and the tticeps surae, tibialis posticus, flexor hallucis longus, flexor digitorum longus, and peroneals do likewise to plantarflex the foot. There is simultaneous lengthening of the hamstrings. Like the développé, this movement may be executed to the front, to the side, and to the back.

This may be a good time to introduce dance speak. When a dancer says I have a great extension, he or she means that the range of hip flexion with the knee extended is unusual. When a dancer speaks of a foot being flexed, it refers to dorsiflexion. Plantarflexion is referred to as pointing or extending. Pointing, combined with excessive lateral deviation of the forefoot, is referred to as winging, or sickling out. This represents a technique defect, as does its opposite (sickling in) (7,10,12).

8. *Arabesque* (Fig. 29.3G). This beautiful, graceful, and extremely difficult position is characterized by carrying the leg, which is fully extended at the knee, past 90 degrees posteriorly. The foot is pointed and the hip is fully extended in a position in which the iliofemoral ligament is most taut. The lumbosacral junction must be hyperextended, and there is marked torsion of the pelvis on the spine. On the supporting side, the knee is straight (unless plié is performed), and the foot is parallel to the pelvis. This is an extremely difficult position to attain and sustain, requiring concentric contraction of hip external rotators to maintain outward rotation on both legs. The hip flexors, including the rectus femoris, must eccentrically lengthen across the coxofemoral joint, while the latter joint, with the other quadriceps, must concentrically contract to extend the knee, while the hamstrings and gluteals contract concentrically at the hip. The hamstrings must lengthen across the back of the knee. Technique faults are recognized by excessive pelvic torsion and/or failure to keep the knee of the gesturing leg fully extended and the foot pointed.
9. *Attitude* (Fig. 29.3H). The path out to this position is similar to that of développé, except that full extension of the leg does not occur. Rather, the dancer reaches a point at which the leg is held with the hip flexed greater than 90 degrees and the knee flexed 90 degrees. This position can also be reached from grande battement, or from any other movement, and may be executed to the front, side, or back.
10. *Pirouette* or *Tour* (including *fouett, attitude*, etc.; Fig. 29.3I). These are turns executed on one foot usually beginning from a demi-plié in one of the five basic positions. The weight is then shifted over the ball of the foot of the supporting leg, simultaneously moving into relevé, beginning the

turn, and positioning the gesturing leg. The execution of the turn depends on how centered the dancer is over the supporting leg. This, in turn, is largely dependent on the strength and development of the stirrup muscles, that is, the flexor hallucis longus, flexor digitorum longus, and the tibialis posterior medially, and the peroneus longus and brevis laterally.

11. *Jumps* (Fig. 29.3J). Whether off one or both feet, the preparatory position for all jumps is the demi-plié. The higher the jump, the deeper the plié must be. Powerful contractions of the gluteus maximus extend the thigh and the hip; the quadriceps femoris extends the knees, and the triceps surae and other plantarflexors point the feet and toes. In landing, the dancer passes through the ball of one or both feet, releasing the lower extremities into flexion moving back into demi-plié.

There are many more ballet positions and movements, but a firm grasp of the abovementioned information will improve your ability to communicate with the injured dancer and help you to understand the demands placed on him or her. This should facilitate management of a wide range of orthopedic problems affecting the great number of people involved in dance.

DANCE INJURIES RELATED TO LUMBOPELVIC SOMATIC DYSFUNCTION

All dance injuries are the result of faulty technique, excluding those caused by adverse environmental factors, malfeasance of fellow dancers, and choreography involving movement beyond the range of human achievement (3). In turn, these are usually the result of maladaptive movement patterns secondary to nonfacilitative alignment. The principal sources of the last two are listed in Table 29.1.

Most dance injuries involve the foot, ankle, or knee. They are profoundly interrelated, both to each other and to dysfunction of the low back and pelvis. The most common site of muscular dance injury is the lower back (2). The majority of soft tissue back injuries are related to strength-length disparity between the trunk and hip flexors and extensors, and between the internal and external hip rotators. These imbalances may be secondary to lumbar articular facet joint capsule and sacroiliac ligament laxity, which in turn may be the cause or effect of intervertebral disc pathology (4).

TABLE 29.1. PRINCIPAL SOURCES OF FAULTY TECHNIQUE

Poor teaching
Incorrect anatomic/biomechanical information
Inappropriate imagery or technique corrections
Self-imposed inappropriate and inaccurate body image
Inappropriate physical compensations for intrinsic biomechanical limitations
Restricted external rotation at the hip joints (2,6–8,12,16).

Abdominal Wall

The abdominal muscles can be divided into three groups: (a) lateral, including the external and internal obliques and the transversus abdominis; (b) medial, including the rectus abdominis and pyramidalis; and (c) deep, situated anterior to the vertebral column, the iliopsoas (IP), and the quadratus lumborum (QL). The IP is important in all forward motion. The lateral and medial muscles serve to support the abdominal contents by forming the anterior wall of the abdominal cavity. They lower the ribs and elevate the anterior rim of the pelvis, assist in flexing the trunk on the hips, and assist in side bending or twisting. They also rotate the trunk with the lower extremities fixed and, most importantly, during respiration their contraction elevates intra-abdominal pressure, supporting the trunk anteriorly, thus assisting in maintaining verticality.

The QL is a quadrangular sheet of muscle arising from the posterior iliac crest, transverse processes of the lower lumbar vertebrae, and iliolumbar ligament. It extends to the twelfth rib and the costal processes of the upper lumbar verrebrae. It primarily stabilizes the neutral lumbar spine, lowers the twelfth rib, raises the

ipsilateral iliac crest, and flexes the body to the side of the contraction. Unlike the other abdominal muscles, bilateral QL contraction extends the spine (13).

The *psoas major* (PM) and *iliacus* (IL) (arising from the sacrum and the iliac fossa) are the primary flexors of the hip. They join together near the femoral head to form the ilopsoas. Secondary hip flexors include the rectus femoris, pectineus, sartorius, adductors, tensor fasciae latae, and anterior fibers of the gluteus medius and minimus. The PM is vitally involved in low back and postural mechanics and thus of primary concern not only in low back injury, but in almost all dance injuries. The PM arises in a series of slips from the vertebral bodies, intervertebral discs, and transverse processes of the last thoracic and the five lumbar vertebrae and the tendinous arches over the lateral bodies of L1-L4. It courses diagonally downward, forward, and slightly outward, crosses the front of the hip joint, and runs caudad and posteriorly to join the iliacus in a common tendon insertion into the lesser trochanter of the femur.

With the lower extremities fixed and the muscles acting from downward up, the PM and IL side bend the trunk, flex the trunk at the hip, and pull the lumbar vertebrae anteriorly, which increases the lordosis and extends the pelvis on the spine. Acting in the opposite direction (from above downward), the psoas flexes, externally rotates, and adducts the free thigh at the hip.

However, with the leg fixed (most significant in relation to dance injuries) working in concert with the internal rotators, the PM externally rotates the trunk on the standing femur so as to advance the opposite side in walking, in effect becoming an internal rotator of the ipsilateral femur. Thus, when the PM is shortened, it has the following effects in dancers:

1. Exerts a prolordotic action on the lumbar spine promoting vertebral facet joint approximation and impingement, which destabilizes the sacroiliac joints (SIJs) (14).
2. Restricts weight-bearing hip external rotation (turnout) particularly at the end range.
3. Restricts extension at the hip (promoting anterior pelvic rotation). This is of major significance in the causation of dance injuries (5).

Michele's Test (adapted from Arthur Michele) (9)

Rationale: This is a modified version of the Thomas test for iliopsoas tension, but with more sensitivity for dancers, since they typically have joint ranges of motion that render the Thomas test negative.

Contraindications to test: Low back or lower extremity pain strongly suggests lumbar disc pathology, while acute psoas spasm is an obvious contraindication to the maneuver (3).

1. The dancer lies with hips and knees flexed fully at the end of the table.
2. While the dancer grasps the left knee, clutching it as close as possible to the chest without discomfort, the examiner extends the right knee so the lower extremity is now as near to the perpendicular as feasible and comfortable.
3. The right leg is then extended on the hip, supported by the examiner's right hand, while the left hand monitors the anterior superior iliac spine.
4. The extended leg is lowered passively until the point at which anterior rotation of the innominate is palpated. At that point, the angle that the lower extremity makes with the horizontal is recorded as the degree of psoas tightness. The dancer should be able to attain 10 degrees of extension (below the horizontal) with the knee extended (Fig. 29.4).

Positive test: Inability to extend one hip to less than 30 degrees from the horizontal (without concomitant anterior rotation of the innominate), enough to cause symmetrical or asymmetrical anterior pelvic rotation with sacroiliac joint instability and dysfunction.

Indicates: Significant hip flexor tightness.

Iliacus Evaluation

Rationale: The iliacus is a hip flexor that is important in dancing, and its dysfunction can disrupt normal symmetrical function.

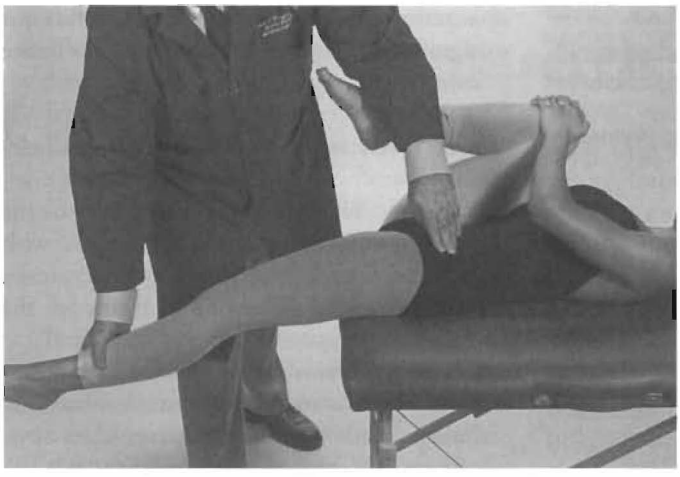

FIGURE 29.4. Michele's test.

1. The same position is taken at the end of the table as in the evaluation of the iliopsoas Michele's test), above. The dancer still maintains left hip and knee flexion.
2. While stabilizing the right innominate with his or her left hand, the examiner allows the right lower extremity to extend at the hip to a gravity-dependent end point, the "iliacus pre–angle measurement position." The examiner records the angle of hip extension (Fig. 29.5A).
3. The examiner presses on the right suprapatellar region to induce further hip extension, the amount of which is limited by palpation of tissue tension and resistance, "the iliacus post–angle measurement position." The examiner measures the new angle of hip extension (Fig. 29.5B).
4. Thus it should be possible to determine the extent of the contribution of the iliacus and the psoas major to hip flexor tightness and direct therapy appropriately (15,16).

Positive test: Any positive angle is a measure of iliacus restriction, so the higher the angle, the worse the restriction.

Indicates: Iliacus restriction.

Unilateral iliopsoas shortening usually results in relative downward and anterior rotation of the ipsilateral innominate. Bilateral iliopsoas tightness results in bilateral innominate and sacral nutation. This configuration destabilizes the sacroiliac joints (SIJs) and predisposes to SIJ dysfunction. In the attempt to stabilize the SIJs, the dancer loads the iliotibial bands, gluteals, hamstrings, and piriformis.

The hip flexors are countered by the gluteus maximus and hamstrings through caudad tension on the posterior pelvis and by the abdominals via a rostral influence on the anterior pelvic brim. Facilitative alignment exists in a state of static muscular balance, so the external auditory meatus sits over the shoulder, hip, knee, and midtarsals.

With iliopsoas dysfunction/insufficiency, however, the lumbar lordosis is increased, the hips are slightly flexed, and the pelvis is extended on the lumbar spine. The posterior vertebral elements now bear more weight and therefore are more subject to stress changes (4).

The *hamstrings* (the semimembranosus, semitendinosus, and biceps femoris) act as extensors of the thigh at the pelvis and complement the abdominals. They also counter the iliopsoas by pulling the posterior pelvis downward into counternutation. Because of their dual and opposite actions as knee flexors and as hip extensors, the hamstrings are particularly vulnerable to dance-related injury. Hamstring tightness, particularly of the long head of the biceps femoris on the side of predominant psoas tightness, is

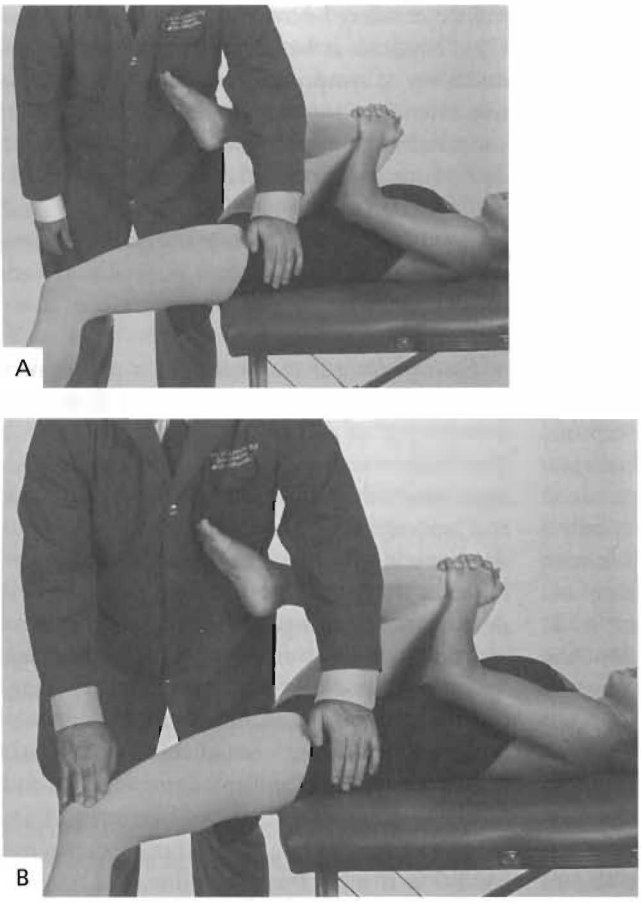

FIGURE 29.5. Iliacus evaluation. Pre-**(A)** and post-**(B)** iliacus angle measurement positions.

probably a result of the relative increase in distance between the origin and insertion of the hamstrings, often owing to anterior innominate rotation.

Through transfer of tension through the sacrotuberous ligament, nutation is suppressed, which invokes the self-locking mechanism of the sacroiliac joint (17). Sacroiliac joint stability depends on the maintenance of tension in the oblique abdominals, gluteus maximus, piriformis, thoracolumbar fascia, and particularly the biceps femoris (17,18) when the sacrotuberous, sacrospinous, and interosseous ligaments are lax. Abdominal and gluteal weakness is common in low back injuries with resultant sacral counternutation. Hence, treatment in such instances requires careful selectivity so that the hamstrings are not overstretched, thereby further destabilizing the sacroiliac joints and the lumbopelvic mechanism.

The *gluteus maximus* is an extensor of the hip and a powerful external rotator of the thigh. It acts, from below upward, to flex the pelvis on the lumbar spine. It is an abductor of the thigh and a stabilizer of the pelvis through its insertion into the iliotibial band. The *gluteus medius* and *gluteus minimus*, however, are the principal internal rotators of the hip, as they maintain the pelvis close to horizontal with the weight on one leg (13).

The *external rotators* of the hip are far stronger than the internal rotators, so the normal summation of forces results in external rotation of the free limb by approximately 5 to 10 degrees (13).

LOW BACK INJURIES

Dance injuries are seldom caused by one single trauma or physical factor; rather they are usually the result of repeated overload microtrauma superimposed upon multiple interrelated pathologic biodynamic factors (2). A significant percentage of dance injuries to the low back and lower extremity are related to restriction of the range of external rotation at the hip (turnout) and the attempt by the dancer to compensate for it (19). Dancers are aware of many cheats needed to increase this range. Hip flexion achieves relaxation of the anterior hip capsule, permitting increased range of hip external rotation. In the erect position, hip flexion alone would result in a forward-flexed trunk, hardly a stable attitude and certainly incompatible with the posture required by the dancer. Therefore, in order to preserve vertical alignment while achieving maximum hip external rotation, the pelvis rotates anteriorly, which increases the lumbar lordosis (7).

In the presence of the hyperlordosis generated to compensate for restricted external rotation at the hip, the zygapophyseal joints are approximated. Repetitive torsional and hyperflexion-hyperextension movements, integral to dance, may stress the articular pillars enough to develop a *spondylolysis* (stress fracture of the pars interarticularis). This condition is common in the skeletally immature spines of adolescent dancers, particularly those with eating and consequent menstrual disorders, often due to improper execution of arabesque or other movements posteriorly involving hyperextension at the lumbosacral junction.

Spondylolysis and Spondylolisthesis

Isthmic *spondylolysis* has a predilection for skeletally immature females. Early in the course, the dancer usually complains of a sharp, pinching pain in the low back occurring consistently with extreme lumbosacral extension on the affected side while standing on that leg. In the mature dancer, this usually signals articular facet impingement syndrome. Later, pain may also be present at the extremes of forward bending, and the dancer may be aware of a dull ache at rest (7). Diagnosis is based on the history; reproducibility of symptoms through active and passive extension; rotation into the side of the lysis combined with extension; radiographic demonstration of articular pillar lysis; and, finally, radionuclide bone scan confirmation. Frequently, an old asymptomatic lysis will show up on plain radiograph in a skeletally mature dancer. Bone scan, however, rules out acute pathology (5).

Spondylolisthesis may occur as a progression of bilateral spondylolysis augmented by the anterior pull of the PM on the vertebral bodies. This is often seen at L5 on S1, or, with degenerative facet arthrosis, L4 on L5 (1). Spondylolysis and spondylolisthesis are more prevalent in dancers and gymnasts; the frequency of spondylolysis is 10% to 12% in these groups as opposed to 6% in the general population. *Unilateral articular pillar lysis* is essentially a benign lesion without serious consequence (5,11). More common in female dancers, these lesions correlate with an earlier commencement of their training, nutritional and endocrine vagaries, and failure to achieve timely skeletal maturity. *Acute spondylolisthesis* in dancers is unusual; rather, the condition is more frequently due to a progression of articular pillar lysis related to repetitive hyperextension/flexion/torsion movements. The clinical picture is not dissimilar from that of spondylolysis described previously except that pain is more pervasive and constant.

Physical examination is significant for a step deformity, usually at the level of L5-S1. Active and passive extension is restricted and usually generates pain on the side of the lesion, particularly when the dancer is fully weight bearing on the leg on the affected side. The lumbar lordosis fails to reverse on forward bending, which also elicits pain on the side of the lysis. Lower extremity radiation may be present. This is often related to discogenic- or facet arthropathy–generated neural impingement, usually at the level above the anterolisthesis (3).

Management. Selective rest from extension is essential until activities of daily living are pain-free. This may require as long as 6 to 8 months.

Immobilization is facilitated by the use of a Boston-type antilordotic brace, initially worn all the time. The use of bracing in acute spondylolysis is somewhat controversial in the literature, as studies have shown successful healing with no bracing, in 4 to 6 weeks, and in 3 months (20,21). Current treatments and algorithms are not well defined, and each athlete should be assessed individually (22).

Manual Medicine Techniques. Manual therapy is directed toward decreasing tension on the hip flexors, anterior rotators of the pelvis, internal hip rotators, innominate derotation, and distraction at the lumbosacral junction. Indirect, counterstrain, and myofascial techniques are indicated. An antilordotic exercise program including iliopsoas stretching, and abdominal, gluteal, and hamstring strengthening is essential for prevention and adjunctive to treatment. A stable grade I spondylolisthesis or a stable spondylolysis should not necessarily prohibit a dancer from dancing following recovery (11). (See Chapter 21.3.)

Sacroiliac Dysfunction and Iliolumbosacral Instability

With this condition, the dancer usually complains of lower back, gluteal, sacroiliac, and/or proximal posterior thigh pain. Pain rarely radiates past the knee. Typically, the pain shifts from one side to the other and is associated with a feeling of instability or giving way of one leg, occasionally resulting in falling. This is often called the slipping clutch syndrome (23). Neurologic examination is within normal limits; however, muscle weakness, particularly of the hip abductors, gluteus medius, gluteus maximus, and hamstrings may be present. Muscle weakness is usually associated with lack of musculotendinous purchase or pelvic instability. Pain-producing positions or movements include pelvic torsion and lumbopelvic extension as in the performance of arabesque, attitude, and port-de-bras back. (See Chapter 21.2.)

Physical examination discloses positive tests for sacroiliac dysfunction, as well as iliopsoas shortening, usually unilateral and most frequently tight-sided. Forward bending in the erect position may reveal a positive forward-bending test, usually right-sided, associated with pain at the time of rostral rotation of the posterior superior iliac spine that, as forward bending progresses, either diminishes or disappears entirely. This helps to distinguish pain arising from sacroiliac joint dysfunction and associated myofascial pain syndromes from those of discogenic origin, which, under the same circumstances, increase in intensity and also probably inhibit the full range of forward bending. Myofascial trigger points may be found at either or both quadratus lumborum, iliopsoas motor points, and iliacus. In the presence of sacroiliac and/or lumbopelvic instability, myofascial trigger points frequently present at the piriformis, gluteus medius, and gluteus maximus with ligamentous entheses trigger points at the iliolumbar, sacrotuberous, and the posterior sacroiliac ligaments (3).

Piriformis Myofascial Pain Syndrome

A further consequence of sacroiliac dysfunction and instability due to ligamentous laxity is hyperactivity of the rotator cuff of the hip, principally the piriformis. This is a postural pelvic muscle that works along with the hamstrings, hip adductors, iliopsoas, and tensor fasciae latae to preserve the integrity of the pelvic girdle (24). Chronic piriformis shortening may also compress the sciatic nerve as it passes through or underneath the substance of the piriformis. These dancers will tend to present with symptoms suggestive of sacroiliac dysfunction or sciatica, such as low back and sacroiliac pain with radiation into the groin, buttocks, thigh, and occasionally past the knee. Sacroiliac instability may be accompanied by reflex inhibition of gluteals, hip adductors, abductors, extensors, and hamstrings.

Piriformis myofascial pain syndrome can be easily tested for and temporarily ablated by application of a sacroiliac belt. The clinician then has the dancer repeat contraction against resistance, noting any improvement in

strength. Other tests for piriformis involvement include resisted attempted external rotation and abduction in the lateral recumbent position. In the prone position, the hip is passively internally rotated to the end range and the dancer is asked to atrempt to externally rotate against resistance (3).

Management. Acutely, treatment is first directed toward correction of SIJ dysfunction, then stabilization. The order of treatment is (a) iliopsoas, (b) piriformis, (c) QL, and (d) gluteal release. Use strain–counterstrain and stretching techniques first, then high-velocity, low-amplitude (HVLA) technique or muscle energy to correct the SIJ dysfunction. Treating in reverse may reestablish SIJ dysfunction and instability by anteriorly rotating the innominate. In a significant number of cases, the dancer requires local anesthetic injection of the medial and lateral piriformis trigger points (identified most effectively by the transrectal or transvaginal approach) followed by passive, then active stretching.

SIJ dysfunction tends to be refractory to treatment unless all perpetuating factors including ligamentous laxity are addressed. The clinician may need to repeat trigger point injections several times. In chronic refractory cases, ligamentous stabilization by proliferative injection therapy (prolotherapy) can be done to the posterior sacroiliac, sacrotuberous, and iliolumbar ligaments (3).

Preventive and maintenance treatment includes lumbopelvic stabilization instruction using innominate derotation exercises (14). Stretching of the iliopsoas, quadratus lumborum, piriformis, and iliotibial band should be coupled with transverse and oblique abdominal, hamstring, and gluteal strengthening. Dance technique and alignment errors must be addressed with, hopefully, the help and guidance of dance teachers.

Lower Back Muscular Injury

The most common muscular injuries among dancers involve the lower back (9). Frequently they occur due to several factors:

1. Failure of the compensatory mechanisms for contracture.
2. Muscle shortening.
3. Failure of adaptive lengthening of the iliopsoas with resultant weakness.

Abdominal muscle weakness undermines the purchase of the hamstrings and gluteals on the bony pelvis, not only reducing their effectiveness as flexors of the pelvis on the spine, but also destabilizing turnout. Consequently, there is increased anterior pelvic rotation, lordosis, and iliopsoas tightness. The gluteal muscles are then loaded, further limiting forward movement at the hip. This effect is increased by the shortening and compensatory posterior pull of the erector spinae in the thoracolumbar segments. This further restricts trunk flexion, facilitates perivascular intramuscular nociceptors, and establishes the myofascial pain syndromes described earlier, thus accounting for most muscular dance-related low back pain (5).

Discogenic Low Back Pain

In the dance world, discogenic low back pain and/or radiculopathy is seldom acute in onset and is most frequently related to underlying dysfunctional postural mechanical factors, particularly hyperlordosis, upon which have been superimposed hyperflexion, hyperextension, and torsional and lifting microtrauma essential to dance movement (11).

Manual medicine techniques are directed primarily toward normalizing vertebral mechanics; counterstrain techniques applied to secondary myofascial pain syndromes, muscle energy, and myofascial release are particularly effective. On the other hand, HVLA is almost universally contraindicated.

Interspinous Ligamentous Injury (Enthesopathy)

This enthesopathy usually initially produces midline pain on flexion and/or extension of the spine, then secondary paravertebral muscle spasm, and is usually the result of a forceful hyperflexion or hyperextension injury, the

latter probably traumatizing the supraspinous ligament entheses (7). Extremes of flexion and extension movements are painful. There is usually extreme sensitivity of the spinous processes at the levels of involvement. These injuries usually occur at transitional spinal segments and the dancer may be predisposed to them by dysfunctional postural mechanics, described previously.

Treatment includes a local anesthetic, then a proliferant injection (prolotherapy) at the involved entheses. Adjunctive manual medicine treatments include paravertebral and accessory muscle myofascial release and facilitated positional release. Corticosteroid injections, despite the instant relief, should be avoided if possible.

HIP INJURIES

Low back pain is hardly the sole consequence of iliopsoas dysfunction/insufficiency. The principal intrinsic determinant of hip external rotation (turnout) is the degree of version of the femoral neck in relation to the shaft of the femur; therefore, the greater the degree of anteversion, the more restricted the turnout. The femur is internally rotated, and an anteroposterior radiograph indicates a foreshortened femoral neck. There is considerable evidence that in the newborn the hips are anteverted and that gradual retroversion takes place until early adolescence.

Dancers will do anything to increase turnout, such as adopting the frog position prone on the floor, attempting to retrovert the femoral neck (possibly of some limited value prior to age 11) (10). Some have even sought out elective femoral neck osteotomy to increase hip retroversion. Males usually have smaller ranges of hip external rotation due to narrower pelvic anatomy and initiation of dance training late enough (around 13 years old) so that the femoral neck is no longer malleable. Few dancers make the cut with external hip rotation ranges less than 60 degrees; those students with less than 45 degrees should probably look elsewhere for careers unless they are willing to suffer the multiple consequent injuries. Ideally, combined external rotation of the dancer's feet on the floor should approximate 180 degrees, 90 on each side. Approximately 15 degrees are available at the foot and ankle, less than 10 degrees at the knee. Therefore, less than 65 degrees at the hip makes the necessary 90 degrees virtually unattainable without predisposition to injury (19). Dancers with extremely limited hip external rotation usually select themselves out of the serious dance community (7,12). Internal rotation at the hip in excess of the range of external rotation is also incompatible with serious dance, although this is not absolute.

The degree of turnout is determined most effectively with the dancer prone, the hip in extension. The degree of turnout is most relevant with the hip in extension, as on the standing leg, since external rotation with the hip flexed, as with the gesturing leg, is increased simply by virtue of the hip being in flexion. To assess turnout, the knee is flexed to 90 degrees with the thighs adducted. The leg is grasped with the right hand, and is rotated medially, externally rotating the hip, while the left hand on the ipsilateral buttock monitors pelvic motion. Internal rotation at the hip is similarly determined by rotating the leg laterally. Hip extension may also be determined in the same position, giving an approximation of the degree of tightness in the front of the hip (5–7).

In the presence of iliopsoas dysfunction/insufficiency, the lumbar lordosis is increased, the hips are slightly flexed, and the pelvis is extended on the lumbar spine. The center of gravity at the pelvis is displaced anteriorly. In compensation, weight is shifted posteriorly accounting for an increased distance between the origins and insertions of the posterior compartment musculature, loading the hamstrings and triceps surae. Hip flexion is thus inhibited and further limited by the encroachment of the femur on the anteriorly tilted acetabulum. This also causes impingement of the intervening soft tissues, resulting in a decreased range of external rotation. Repetitive impingement of the proximal femur on the acetabular labrum affected by forced external hip rotation may result in labral tears and eventual hip arthrosis, a condition endemic to older dancers, especially

those with intrinsically restricted external hip rotation (1).

Femoral neck stress fracture may be another consequence of restricted hip external rotation and the attempts to repetitively reach a limited threshold or to forcibly increase this range (11). Abdominal muscle weakness undermines the purchase of the hamstrings and gluteals on the bony pelvis, not only reducing their effectiveness as flexors of the pelvis on the spine, but also destabilizing turnout. Consequently, there is further pelvic anterior rotation, increased lordosis, and iliopsoas tightness. The gluteals hypertrophy, further limiting forward movement at the hip, including flexion of the trunk at the coxofemoral joint (2).

Snapping hip syndrome is a common complaint in dancers and has many possible sources. One of the most common sources is the proximal iliotibial band (ITB). A tightened ITB may snap over the greater trochanter, often giving rise to the misperception of a dislocating hip. ITB tightness is usually related to an anteriorly rotated innominate (11). *Iliopsoas tendon tightness or inflammation* secondary to repetitive forceful contraction of the iliopsoas during flexion or to stretching during extension of the hip may result in friction over the iliopectineal eminence or over the lesser trochanter as it passes to its insertion. *Tightness of the long head of the biceps femoris* as it crosses over the ischial tuberosity may also produce anterior hip pain.

Anterior displacement of the femoral head due to *iliofemoral and/or pubofemoral ligament laxity* is the result of repetitive extreme forcing of turnout, particularly in the presence of femoral anteversion or other static restrictors of hip external rotation. The presence and degree of anterior ligamentous laxity may be determined by the anterior drawer test. *Intra-articular* sources of hip pain include anterior labral tears, loose bodies, and synovitis, but these may ultimately be traced to postural malalignment and technique faults.

Differential diagnosis of hip injury may be quite difficult. The more chronic cases may require thin-slice magnetic resonance or computed tomography scan to aid in the diagnosis, but since most of these conditions are related to dysfunctional mechanics, most can and should be diagnosed by history and examination. Keep in mind that L5 radiculopathy may be the source of pain at the anterior or lateral hip.

Management. Selective rest is the initial step in treatment. Management must combine analysis and correction of the related muscle imbalances and technique faults. Iliopsoas tendinopathy is particularly difficult to manage because contraction and/or stretching of that muscle is an essential component of all dance movement. Therefore, modified or non–weight bearing on the affected limb may be necessary to effect recovery. Manual medicine techniques include iliopsoas counterstrain and myofascial release and correction of coexisting sacroiliac dysfunctions, while muscle energy can treat shortening of the iliopsoas, tensor fasciae latae, and piriformis muscles.

KNEE INJURIES

Knee pain in dancers can also frequently be traced to muscular imbalances. The rectus femoris is more anterior to the quadriceps knee extensors. By virtue of its proximal origin (straight head) from the anterior inferior iliac spine, it flexes the thigh on the pelvis and extends the pelvis on the lumbar spine. Like the hamstrings, the rectus femoris crosses two joints, having opposite actions on the hip and knee; therefore, it is vulnerable to strains and other injuries. Rectus femoris tightness in concert with a shortened iliopsoas tends to anteriorly rotate the innominate, which loads the hamstrings. The resultant abnormal stress on the quadriceps mechanism increases tension on the patella and quadriceps tendon, while compressing the patella asymmetrically into the femoral trochlea.

The dancer forces external rotation at the foot and ankle as a tool to compensate for restricted hip external rotation (19). Subsequent foot pronation creates internal tibial (and to a lesser extent femoral) rotation, patellofemoral malalignment, and valgus stress at the knee (7).

Therefore, dysfunctional postural mechanics and technique faults load the medial collateral ligament of the knee, predisposing to degenerative tears of both that ligament and the medial meniscus to which it is attached. Pes anserine bursitis refractory to treatment is also a common finding (25).

The dancer with anterior knee pain syndromes and anterior horn medial meniscal tears avoids full extension of the knee. As a result, atrophy of the vastus medialis obliquus rapidly develops. This increases lateral-medial quadriceps imbalance, leads to increased lateral pull, and serves to exacerbate patellofemoral incongruity. Gluteus maximus and tensor fasciae latae hyperactivity also occur with pelvic hyperextension, loading the iliotibial band and its lateral patellar retinaculum extension. The combination of these factors tends to promote lateral patellar excursion, chondromalacia, patellofemoral subluxation, dislocation, extensor mechanism strain, quadriceps and patellar tendinopathies, and ultimately, patellofemoral osteoarthrosis.

FOOT AND ANKLE INJURIES

Injuries to the foot and ankle frequently relate to similar muscle and postural imbalances. Maximum hip flexion achieves relaxation of the anterior hip capsule, permitting increased range of hip external rotation. To increase demi-pli (dorsiflexion at the ankle), the dancer turns out at the ankle and the foot everts and overpronates (7). This may excessively stress the plantar fascia and the posterior tibial and flexor hallucis longus tendons, leading to consequent tendinopathies and, commonly among dancers, the *cuboid dysfunction syndrome*. Anterior impingement syndromes may develop with spurring of the distal tibia and grooving of the talar neck. This repeated microtrauma causes a decrease in accessory motions at the ankle, contributing further to decreased range of ankle dorsiflexion and shock-absorption capacity (12,26). Although these injuries may occur in other athletic activities, their significance and treatment are often different than in dancers.

Anterior Impingement Syndrome. The dancer complains of pain over the dorsal aspect of the talotibial articulation occurring most frequently at the bottom of the demi-pli (at full weight-bearing dorsiflexion of the ankle) and feels as if there is something in the way (8,12). The effort required over many years of intensive dance training to increase the depth of demi-pli causes anterior talotibial impingement. The source of this impingement syndrome is the attempt by the dancer to compensate for a decreased range of external rotation at the hip (19). In some, a deep groove is formed in the neck of the talus, increasing range of ankle dorsiflexion. In the unluckier dancers, usually late starters, this results in osteophytic proliferation with further restriction of dorsiflexion range, necessitating increased foot pronation to achieve the desired turnout and demi-plié.

Consequently, there is a marked decrease in accessory motions at the ankle. The mortise is shifted anteriorly, stressing the posterior ligamentous structures, particularly the posterior talotibial and calcaneofibular ligaments, and eliciting posterior ankle pain in plié. Talotibial impingement and restricted dorsiflexion range may increase stress on the midfoot and forefoot at heel-off and overload the talonavicular and metatarsophalangeal articulations. This can lead to localized arthrosis and stress fractures of the metatarsal shafts (1,8,12).

A typical situation is a dancer who has been told that she is rolling in (hyperpronating the foot) in demi plié and probably has chronic restricted hip turnout. Physical examination shows diminished external rotation at the hip while prone, and decreased hip flexion while supine. In the weight-bearing parallel position, a relative decrease in the range of dorsiflexion of the ankle may be noted, with pain over the dorsal aspect of the talus. A small, tender projection may be palpated at the anterior border of the distal tibia. Radiographs of the ankle in lateral projection may show roughening of the anterior border of the tibia and/or an osteophyte of varying size projecting dorsally from the neck of the talus. Another lateral weight-bearing film in demi-plié demonstrates the impingement with associated gapping of the

posterior talotibial joint space, stretching the posterior talotibial ligaments.

Anterior impingement leads to compensatory overpronation to avoid pain while achieving maximal ankle dorsiflexion. This is a technique error that requires exercises to increase hip external rotation while decreasing turnout at the ankle. This cannot be accomplished through sheer admonition; since it is part of the chain of pathomechanical events, correction depends on total movement pattern modification.

Effective treatment includes manual mobilization of the foot and ankle to correct the associated subtalar and talar dysfunctions. Hip external rotation restriction can be treated with muscle energy, myofascial release, and counterstrain if the problem is not intra-articular. A significant number of cases require surgical débridement of the exostosis, a procedure that is not always successful. The most effective treatment is prevention.

Posterior Impingement Syndrome. The dancer complains of pain in the back of the ankle when pointing the foot and a sense of something in the way of motion. The ankle requires at least 90 degrees of plantarflexion, and 10 to 15 degrees more to compensate for genu recurvatum. Since a significant amount of plantar and dorsiflexion comes from motion at the subtalar joint, a combination of mild forefoot pronation and abduction is necessary to loosen the subtalar joint (12). Lateral radiographs of the ankle with the foot in forced plantarflexion show either an elongated talar posterior process or an ossicle (os trigonum) connected by fibrous tissue to the talus (7).

The history in these cases is usually diagnostic. The pain is posterolateral rather than medial, which is seen in flexor hallucis longus tendinitis. It can also be reproduced by passive compression of the calcaneum against the tibia. Some confusion may arise when the dancer describes the sudden onset of posterior triangle pain and swelling following a hard landing from a jump. This can signify a fracture of either the posterior process of the talus or of an os trigonum. In either case, the bony element is caught like a nut in a nutcracker. Frequently, impingement caused by an os trigonum can be temporarily reduced by manipulation and then sustained by self-manipulative techniques involving primarily distraction of the talocrural articulation.

The diagnosis can be confirmed by injecting 0.5 to 0.75 mL of 1% lidocaine into the posterior triangle behind the peroneal tendons. Manipulation, physical therapy, along with technique corrections (particularly the error of jamming the calcaneum into the posterior tibia when pointing the foot), combined local anesthetic, and short- and long-acting corticosteroid injection may result in permanent relief. An os trigonum may coexist with flexor hallucis longus tendinopathy, and the latter may be due to forced hyperplantarflexion of the hallux in the presence of obstruction in the posterior triangle. Occasionally, surgical excision is necessary (12).

Achilles Tendinopathy. This is usually related to a rigid cavus foot and tight calf combined with adverse environmental factors (i.e., hard floors, poorly fitted footwear) or with major and sudden upward changes in training intensity. Physical examination is characterized either by tiny nodules around the tendon or on its surface or in the case of a more severe, acute strain, by a tender fusiform swelling around the tendon 2 to 2.5 cm proximal to the calcaneal insertion. The dancer may complain initially of swelling and pain when working on a hard floor, particularly during jumps. Later, there may be a scraping sensation on takeoff, with pain progressively increasing in severity with dancing. Later, the tendon is hot, there may be crepitus with active motion, and pli is restricted and painful. The posterior compartment musculature including the gastrocnemius/soleus and hamstrings is tight, as is the plantar fascia (in reality, the tendinous extension of the triceps surae). In female ballet dancers, Achilles tenosynovitis may also be a result of tying the toe shoe ribbon too tightly.

Treatment includes a combination of manipulative corrections of pelvic and lower extremity dysfunctions and/or imbalances, movement

modification, massage, stretching and strengthening of the calf muscle, ice, transverse friction massage, heel lift, selective rest (avoidance of painful movement), and ultrasound. The dancer is advised in the use of the stretch box as a preventive measure (5,8,12).

Peroneal Tendinopathy. In this relatively uncommon disorder, the dancer complains of pain at the lateral aspect of the ankle, often at the posterior aspect of the external malleolus. Frequently there is tearing and laxity of the peroneal retinaculum. The history is usually significant for previous lateral ankle sprain with a tendency to turn the ankle. Peroneal muscle weakness is demonstrated by the dancer's inability to plantarflex, evert, and abduct the foot against resistance. Acupuncture, ultrasound, massage, and exercises to strengthen the peroneal muscle group are all of value in treating this problem. However, none will be effective without correction of proximal and distal tibiofibular, subtalar, talocrural, and midtarsal dysfunctions. Correction of inefficient movement habits and alignment defects is essential. These principles are critical to the management of ankle sprains, tendinopathies, and, in fact, all dance injuries.

It should be kept in mind that the most common cause of tendinitis is relative muscle weakness or inappropriate usage and consequent repetitive overload. Acute tendinitis is actually quite rare, and chronic eccentric loading of weak muscle and connective tissue is more likely to cause tendinopathy.

Dislocation or Recurrent Peroneal Tendon Subluxation. The dancer complains of pain on relev to demi-pointe and sometimes on deep pli. Pain is specifically localized to the posterior aspect of the external malleolus. There is usually a history of repeated minor episodes of similar pain, or symptoms may have progressively worsened following either an ankle sprain or repeated plantarflexion/inversion injuries. Diagnosis is confirmed by having the dancer forcibly flex the foot while the clinician exerts pressure with the thumb posteriorly on the peroneal tendons at the lateral malleolus, which restrains them from slipping out of their groove. The dancer is thus able to point the foot without pain. Upon releasing the tendon, an unstable tendon will sublux, and repeated plantarflexion is limited and painful.

Although this condition may respond to conservative management, including strengthening exercises, physical therapy modalities, manual therapy, elastic support or strapping, and prolonged selective rest, surgical intervention to reestablish a retinaculum is often necessary to allow the dancer to continue dancing. Ligament proliferative therapy (injection of a fibroproliferative agent into the retinacular entheses) may be effective as well.

Posterior Tibial Tendinopathy. The dancer experiences pain posterior and inferior to the medial malleolus, initially upon relev to demi-pointe. The tibial tendon inserts into the plantar aspect of the navicular and acts as a powerful plantarflexor and inverter of the forefoot. The tendon lies superficial and anterior to those of the flexor digitorum longus and flexor hallucis longus.

Asking the dancer to relev repeatedly to demi-pointe elicits pain and weakness, but not crepitus. This lesion is due primarily to forcing turnout at the foot and ankle to compensate for restricted external rotation at the hip with resultant foot eversion, hyperpronation, and increased stress on the posterior tibial mechanism (7,26). This rarely occurs in dancers because most elite dancers have cavus feet, and most dance activity takes place in equinus. Thus, the posterior tibial tendon is shortened, and the subtalar joint is in eversion. Stress is therefore shifted to the flexor hallucis longus tendon—"the Achilles tendon of the foot"—and the tibialis posterior is usually spared (12).

Flexor Hallucis Longus Tendinopathy. This is classically a ballet injury (27) and the most common dancer foot injury, yet it is unheard of in other sports. The dancer has pain behind the medial malleolus on pointing, associated with a sensation of grinding, often with a triggering. Initially, the dancer is able to warm the foot up, but as the problem progresses, it gets

worse with activity, specifically grande-plié and relevé.

The basic causes of this syndrome are again related to maladaptive movement patterns in the attempt to compensate for restricted turnout at the hip. To achieve the necessary and desired turnout, the dancer externally rotates the lower limb at the knee and ankle, thus everting the foot and pronating the longitudinal arch. As the dancer goes into demi- or full pointe, the muscle belly in contraction tends to be pulled into the fibro-osseous tunnel in the medial talus normally occupied by the tendon alone. With the ankle in dorsiflexion (plié), the flexor hallucis longus tendon is stretched. Pain is then elicited by further passive dorsiflexion of the hallux (as in relevé to demi-pointe), which increases the tension on the entrapped muscle belly and tendon, and fires the Golgi tendon receptors, leading to a functional hallux rigidus. As the syndrome progresses, pain occurs even in demi-plié.

Treatment consists of local measures, such as ice, ultrasound, transverse friction massage, counterstrain to tender points in the proximal muscle belly, and acupuncture. Most importantly, correction of foot mechanics and dance technique must be done. Nonsteroidal anti-inflammatory drugs may be useful. Selective rest, particularly during the early stages, is essential (7, 12). Taping may support the tendon enough to continue dancing. Surgical release is frequently necessary to permit continued dancing.

Plantar Fasciitis. Although this is a common problem with runners, it is rare in the dance population, other than when related to posterior tibial or flexor hallucis longus tendinopathy, or cuboid dysfunction. Management consists of correction of foot mechanics with the use of orthotics in everyday footwear, and manual medicine techniques to correct tarsometatarsal, midtarsal, subtalar, talocrural, proximal and distal tibiofibular, and cuboid dysfunctions. These techniques are discussed in Chapter 24.3.

Cuboid Dysfunction Syndrome. The cuboid is the keystone bone of the lateral foot. It articulates with the calcaneus, fourth and fifth metatarsals, lateral cuneiform, and navicular. Cuboid dysfunction is frequently seen as one of the sequelae to lateral ankle sprains, plantar fasciitis, and peroneal tendinopathy. In the presence of foot eversion and hyperpronation, the cuboid is depressed plantarward and becomes locked in that dysfunctional anatomic relationship. The peroneus longus tendon passes through a groove on the inferior aspect of the cuboid, and cuboid dysfunction may be either a cause or effect of peroneal muscle dysfunction. Manual medicine techniques should address both the proximal and distal tibiofibular joints, as well as the cuboid dysfunction itself using a combination of counterstrain, muscle energy, and HVLA techniques (28). HVLA is particularly effective in cuboid dysfunctions when used after counterstrain.

SUMMARY

The sine qua non of success in prevention and treatment of all lower extremity dance injuries is to correct the technique and alignment defect causing excessive rolling pronation at the foot and ankle. As stated earlier, this is usually a product of limited external rotation at the hip joint and/or an unrealistic conception of what the individual's turnout can/should be. Turnout is primarily a genetic gift, and once the biomechanics and muscle balance are improved, there is little that can increase turnout safely. Manual medicine is extremely effective in diagnosing and treating lumbosacral and pelvic restrictions that could limit turnout and other motions.

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30

FOOTBALL

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OVERVIEW

The popularity of football has grown tremendously since its inception. The first game played in America is believed to have been played in 1869 between Princeton and Rutgers, following soccer rules. The first official rules for American football were written in 1876. Over the next 15 years, rugby rules seeped into the game design, and the sport gradually took a new shape, while its progenitor in America went from football to soccer, despite being called football by the rest of the world (1).

Injuries and deaths occurred near the turn of the twentieth century at alarming rates, necessitating a governing body to be created by President Theodore Roosevelt. First called the Intercollegiate Athletic Association, it was renamed the National Collegiate Athletic Association (NCAA) in 1910. Over many years the NCAA changed football, instilling safety measures such as helmets, pads, and rule changes designed to reduce catastrophic injury.

Today, football is arguably the most popular sport in the United States, with an estimated 1.8 million male participants and millions more people who are spectators. The sport spans Pop Warner and Pee Wee youth leagues through high school and collegiate football. Elite professional football has been played in the National Football League, while lesser professional leagues have come and gone throughout the years. The Canadian Football League, Arena football, and NFL Europe are three present-day manifestations that still play and compete.

The National Center for Catastrophic Sports Injury Research states there were approximately 1,800,000 participants in football during the 2000 season on all levels of participation in the United States. Associated with an increase in participation, however, is a natural increase in injury rates. In 1931, the American Football Coaches Association initiated the first annual survey of football fatalities. The surveys were started with the intention of making the sport a safer and more enjoyable activity for its participants. This monitoring of injury rates has led to the development of improvements in equipment, medical care, and coaching techniques. As a direct result of injury monitoring, in 1976 the tackling or blocking technique known as spearing (making first contact with another player with the head) was made illegal.

The year 1990 marked a record year in injury surveillance. It was the first year since the beginning of surveillance that no fatalities on any level of the sport were reported. From the years 1931 to 1965 there were 608 fatalities reported on all levels of competitive football. In 1968, 36 total deaths were reported, and that number was reduced to 5 total in 2002: 3 of those 5 were reported in high school athletes, which is the level at which most fatalities occur. The National Electronic Injury Surveillance System estimated there were 355,247 total injuries related to football reported from emergency rooms in the United States in 1998, an increase from 334,420 in 1997 (2).

By virtue of its combative nature, speed, and high-energy collisions, football participation results in a high incidence of traumatic injury. However, there are many nontraumatic, gradual-onset injuries that occur in football athletes which can negatively affect their playing ability, even to the point of missing participation (3). The application of manual medicine techniques

and exercise therapy in football players can minimize and even prevent injury as well as optimize performance on the field.

APPROACH TO THE ATHLETE

Football, the same as many other sports, requires both aerobic and anaerobic fitness in order for participants to be competitive on all levels. Football is a collision sport with medium static and dynamic demands (4). Plays typically last from 3 to 10 seconds, with a rest period in between. Weight training is required to build strength and size, particularly in players required to block other players, such as offensive and defensive linemen, fullbacks, tight ends, and linebackers. Agility, flexibility, and speed are qualities important to all players, but particularly to those athletes required to handle the ball and those sprinting on virtually every play such as halfbacks, receivers, and defensive backs.

The majority of injuries are traumatic and acute, so initial evaluation should take this into consideration. Football elucidates the issue of playing hurt versus playing injured, since players will play through pain as long as they feel confident they are not necessarily injured. How these terms are defined and how far these definitions go in the eyes of the clinician and player is the challenge. Most trauma and injuries are straightforward, such as simple fractures, sprains, and contusions. Many of these injuries can be protected, padded, splinted, or wrapped, and the player will compete while playing hurt.

Most complex straightforward in initial management once a preliminary diagnosis is made; apply the RICE protocol (rest, ice, compression, and elevation), make the decision whether the athlete will return to play immediately or sit out, and hold him out of practice until he is physically able to perform. Prognosticating return to play can be more difficult. Teams base personnel decisions on the eventual availability or inactivity of a player, and most of their decisions are based upon what the physician and trainer report to the head coach. A conservative approach in the general population usually works fine, but in the competitive football world, few games are played, and consequently, their individual importance is magnified. Therefore, players are less tolerant of missing one game than in a longer-season sport like baseball. Players usually have 7 days of preparation in-between games, so the challenge for the medical staff is getting them ready for the game. The gladiator mentality allows many college and professional players to play through injuries and pain, which is a blessing and a curse when administering treatment.

THROWING MECHANICS VERSUS BASEBALL

The quarterback is the only position in football whose main purpose is to throw the ball to another player (save for the occasional trick play by a teammate). Accuracy and velocity are just as important for the quarterback as for the baseball pitcher. However, there are several important differences in mechanics, described well by Meister (Fig. 30.1) (5):

1. The mechanics of a football pass are closer to a baseball catcher than a pitcher. Instead of the full windup and follow-through that a pitcher needs, the cocking phase of the quarterback is less dramatic, usually coming just behind the ear, and the elbow extends fully during the follow-through. This method allows the quarterback to throw quick passes from various positions and situations, even while on the run. The pitcher has no such requirements, and their target at home plate never moves during the game. This also unloads the elbow from the compressive rotational forces of the pitcher (5,7).
2. The heavier weight of the football affects shoulder position and stresses in all phases (6). Quarterbacks rotate their shoulders sooner and achieve maximum external rotation earlier in the throwing cycle than do pitchers, probably allowing more time for acceleration during internal rotation. However, even with the increased time afforded internal rotation, internal rotation velocities are significantly less when throwing a football,

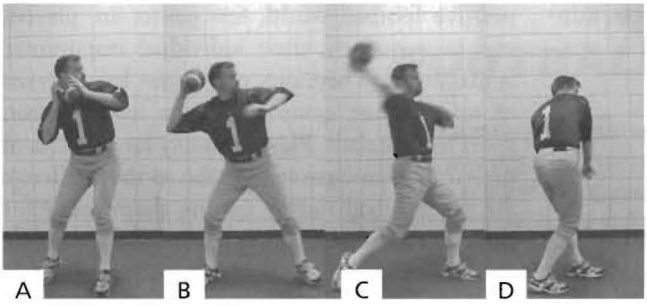

FIGURE 30.1. The phases of the football throw. **A,** Early cocking. **B,** Late cocking. **C,** Acceleration. **D,** Follow-through. (From Kelly BT, Backus SI, Warren RF, et al. Electromyographic analysis and phase definition of the overhead football throw. *Am J Sports Med* 2002;30:837–844.

7,600 degrees per second in baseball versus 5,000 degrees per second in football (5).

3. The heavier football also forces the quarterback to lead more with the elbow, which is undesirable in baseball pitching mechanics. Increased shoulder horizontal adduction coupled with increased elbow flexion is needed in the late cocking phase to decrease the impact of the heavier football by shortening the lever arm, lessening the potential load on the shoulder (5).
4. Quarterbacks throw in a more erect position, while pitchers fall forward with the torso at delivery. This decreases the contribution of the hip and legs in the throw, but it also results in decreased arm velocity. The erect finish of the quarterback also keeps him out of a more vulnerable position, that is, bent over and unable to escape impact from an oncoming defensive rush. The overall lower torques and forces generated on the throwing shoulder of the football player may also account for the lower incidence of shoulder injuries in football (5).
5. The workload on a quarterback is typically less than for a pitcher. The quarterback rarely throws more than 30 or 40 passes per game, and velocity can vary greatly, depending on whether a short lob screen, medium-range bullet, or a deep lofting bomb is needed. Humeral internal rotation speeds are 3 to 4.5 times faster in baseball than in football, and elbow extension velocity is 2 to 3 times faster in baseball as well.

   Plus, the starting pitcher throws to near maximum velocity or arm speed during every pitch for 100 to 120 pitches. Football games are played only once a week, whereas starting pitchers pitch twice and a reliever may pitch three or four times in a week. Pitchers also stress their arms with numerous types of spin put on the ball as it is delivered in order to create extra movement during the trajectory. On the other hand, the quarterback tries to throw a tight spiral pass with minimal or no extra movement, and variety comes from the trajectory and velocity, neither of which is disguised for the other team before release.
6. Medial collateral ligament (MCL) sprains of the elbow have a different impact in football than in baseball. They are merely a nuisance to the football player, even a quarterback. A study of MCL injuries over 5 years in the National Football League showed 19 acute MCL sprains, including two quarterbacks, and none required surgery (7). In fact, only four even missed games. In baseball, MCL injuries are severe, season-ending injuries. Complete tears require reconstruction, 12 to 15 months of rehabilitation before returning to the sport, and a 70% to 90% chance of returning to the previous level of competition, while partial MCL tears had a 42% failure rate of nonoperative treatment in returning to the mound, according to one study (8). This further proves the lower load that the shoulder and elbow carry in football throwing mechanics.

The mechanics are important to understand because two of the most common overuse in-

juries in football are rotator cuff tendinitis and elbow valgus overload (9), although they are far less common than traumatic injuries. Therefore, training for the quarterback position requires shoulder girdle exercises to ensure full, painless range of motion, rotator cuff muscle strength, and coordinated scapulothoracic function. Shoulder and elbow injuries should be evaluated and treated promptly.

INJURY PATTERNS

All football players are exposed to a multitude of possible injuries; however, certain positions pose a higher risk for certain types of injuries. The majority of quarterback injuries are caused by trauma. The most common shoulder injury in a quarterback is an acromioclavicular separation, not a rotator cuff injury (11). Also, the sudden cutting and explosive bursts of speed that running backs possess predispose them to groin pathologies, such as adductor/hip flexor strains. According to Prager et al., the tackle (i.e., lineman) has the highest rate of injury on the offense and the linebacker has the highest rate of injury on the defense (10). The three most common types of football injuries are sprains, strains, and contusions (3).

The difference in duties of each position on the field warrants more detailed investigation during the pre-participation examination for those areas predisposed to injury. An in-depth assessment and shoulder examination should be conducted on quarterbacks, assessing for optimum range of motion, rotator cuff strength, and scapulothoracic motion. Eighty percent of all knee injuries occur to the weaker of the two legs, and 88% of injuries occur in athletes with leg-length inequalities (12). Therefore, an assessment for lower extremity muscle imbalance and leg-length discrepancies should also be conducted in the pre-participation examination on all players. Detailed assessment of the lumbosacral area is critical in linemen and linebackers because of the higher incidence of spondylosis and spondylolisthesis in these positions due to explosive forces applied in extension (13).

SPECIFIC INJURIES

Concussion

Applicable to all football players is the risk of concussion during play due to high-impact collisions. It is estimated that in high school football alone as many as 250,000 concussions occur each year and that up to 20% of players sustain a concussion (14).

A concussion can be defined as any alteration in cerebral function caused by a direct or indirect (rotation) force transmitted to the head resulting in one or more of the following acute signs or symptoms: a brief loss of consciousness, lightheadedness, vertigo, cognitive and memory dysfunction, tinnitus, blurred vision, difficulty concentrating, amnesia, headache, nausea, vomiting, photophobia, or a balance disturbance (15). It may be important to rule out the possibility of an intracranial bleed using computed tomography or magnetic resonance imaging after following one of the many tiered injury protocols which change as clinical experience deepens. Playets who sustain a concussion should be held out to avoid a potentially lethal second-impact syndrome from a second head injury while postconcussive symptoms still linger. However, when a player presents with persistent vague symptoms following a head injury, such as a headache, it is clinically difficult at times to attribute the symptoms to concussion.

Manual Medicine Techniques

Occipital release and other cervical osteopathic manipulative treatments can reduce and possibly alleviate symptoms associated with somatic dysfunction in the occipitoatlantal region, such as headache. If a true concussion presents with symptoms including a headache, applying the suboccipital decompression technique can relieve some pain in the cervical area but will not completely alleviate the headache. However, many athletes who still feel residual symptoms from the concussion are actually feeling cervical dysfunction and soft tissue injury, thus being held out unnecessarily.

In a traumatic concussion, there will likely be dysfunction in the cervicothoracic region. Whiplash injury can occur with a concussion when a player is blind-sided, such as a quarterback passing downfield or a defensive player hit from the side while chasing another player. If whiplash is noted, the injury and sequelae should be addressed and treated as tolerated while the athlete recovers from the concussion.

It is important to ensure that the athlete is not exhibiting any significant signs or symptoms that would preclude the use of certain manual medicine techniques. When an athlete exhibits signs and symptoms such as loss of consciousness, nausea, vomiting or persistent post-traumatic amnesia, direct high-velocity, low-amplitude (HVLA) techniques are *not* recommended due to the risk of not only worsening symptoms but also possibly worsening such conditions as intracranial bleeding. *Thrust techniques should not be used in the acute setting of a concussion.*

Recommended Techniques

1. Occipital release.
2. Muscle energy of the occipitoatlantal and atlantoaxial joints.
3. Correction of cervicothotacic dysfunctions.
4. Myofascial release to trapezius, levator, and cervical soft tissue.

Rotator Cuff Tendinitis

Unique to rhe quarterback position is the need for an accurate and strong overhead throwing ability. Less than 15% of all shoulder injuries comes from overuse; over 80% come from blunt trauma (17). Intrinsic factors that may predispose the quarterback to this injury include impingement of the subacromial space due to poor scapulothoracic motion, weak or overused rotator cuff muscles, or subtle underlying instability (9). Extrinsic forces applied to the shoulder can also cause muscle strain and tendinitis, especially while the shoulder is in the forward-flexed and externally rotated position.

Rotator cuff tears are less frequent, but long-term impingement and shoulder trauma are the two most common causes. Offensive linemen and linebackers are most prone to injury, while quarterbacks do not get cuff tears as often as pitchers (18).

Recommended Techniques

1. Spencer techniques with muscle energy for restricted motion.
2. Scapular mobilization with muscle energy. The 19 muscles attaching to the scapula allow for its motion in many planes, including elevation, depression, upward and downward rotation, abduction, adduction, anterior and posterior tilt, ptotraction, and retraction.
3. HVLA of the thoracic spine and ribs. The goal of this technique is to optimize facet dynamics and scapulothoracic biomechanics, resulting in enhanced rotator cuff function and a coordinated throwing motion. This technique is used well during the pregame routine.

Lumbosacral Dysfunction

Lumbosacral somatic dysfunction can result from myofascial strain, facet syndrome, and disc disease. All player positions can encounter these problems. Part of the problem has been players playing on hard artificial turf surfaces. The prevalence of the newer turf technology with rubber and sand bases may prove to reduce these injuries, bur no current data exist.

There is some dispute as to what role football plays in athletes developing degenerative disc disease or facet degeneration. It is clear that spine trauma such as fractured vertebrae, ruptured discs, and spondylolysis is higher than in the normal population, and these factors lead to a higher risk of back pain and degenerative disease. Weightlifting and the violent hyperextension that occurs in the spine is also implicated, which is a natural part of football training but excessively loads the posterior spine (19).

As a lineman transitions from a three- or four-point stance to an upward and forward thrust against the resistance of the opposing player, a tremendous force is placed on the lumbosacral area, predisposing this area to spondylolysis and spondylolisthesis. In a review of 1,097 injuries

sustained at the professional level, 6% were back injuries. Approximately 50% of linemen report having a history of back pain (16). This makes more sense when one considers that linemen lift more weight more often in the weight room than anyone else on the team.

Restoration of full segmental motion and functional strength is paramount in returning an injured player to any position on the field. Facet injuries may require avoidance of extension for a short period of time, which may require the use of an extension brace. Core stabilization exercises are integrated as a daily routine for all players rehabilitating from injury, which is discussed later in this chapter.

Recommended Techniques for Players Experiencing Acute Pain or Muscle Spasm

1. Counterstrain of the lumbosacral tender points. The indirect nature of counterstrain alleviates the spasm sometimes associated with strains.
2. Muscle energy, for neutral or flexion-extension lumbar dysfunction.

Recommended Techniques for Players with Subacute or Chronic Dysfunctions

1. HVLA of the lumbar spine. This is also useful during the pregame routine.
2. Sacral mobilization. This technique can resolve most sacral somatic dysfunctions.

Hamstring Strain

Hamstring strains are one of the most common muscular injuries in football and have a high recurrence rate. Sprinters (running backs, receivers, and defensive backs) are the most likely group to experience hamstring muscle strains due to the excessive forces and sudden nature of muscle contraction generated during sprinting. These injuries are frequently observed when the players are fatigued (20).

Recommended Techniques

1. Muscle energy with reciprocal inhibition of the hamstrings. This technique uses the premise of reciprocal inhibition, that is, by contracting the quadriceps, the hamstring will intrinsically relax, while continuing to stretch the hamstrings.
2. Petrissage or vibration massage to the hamstrings to improve circulation, muscle elasticity, and clear subcutaneous bleeding.

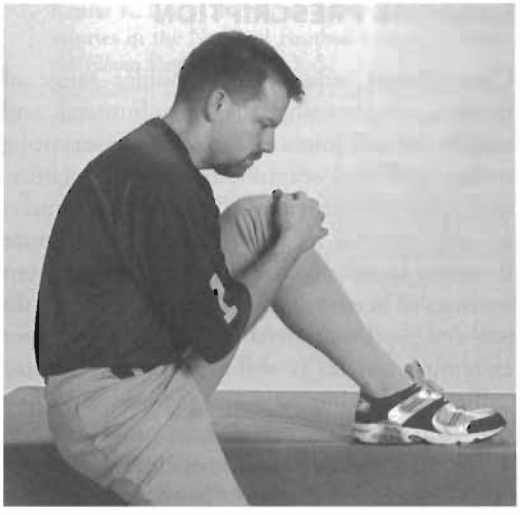

FIGURE 30.2. The Parker stretch.

Piriformis Syndrome

This syndrome can occur when spasm occurs in the piriformis muscle, often causing sciatic nerve irritation and radicular-type pain.

Recommended Techniques

1. Parker stretch (self-manipulation).
   a. The athlete sits on the table with the affected side on the table and the other leg on the ground.
   b. The leg on the table is flexed.
   c. With a straight back, the athlete leans in the direction of the flexed knee, feeling a stretch in the gluteal area.
   d. The athlete then pushes his leg into the table for 3 to 5 seconds, then releases (Fig. 30.2). This is repeated three times and followed by a passive stretch.
2. Muscle energy technique for piriformis spasm.

EXERCISE PRESCRIPTION

Quarterbacks require full shoulder range of motion, emphasizing the glenohumeral and scapulothoracic joints. They benefit from strong rotator cuff and scapulothoracic musculature, with adequate muscular balance between internal and external rotators in order to optimize throwing speed and accuracy as well as prevent overuse. Therefore, quarterbacks should do pre- and postgame stretching of the major upper extremity muscles as well as maintain a rotator cuff strengthening program. One exercise specific to the serratus anterior are push-ups with a plus, in which the athlete assumes the push-up position with the knees bent. At full extension of the push-up the athlete should protract the scapula to add the plus to the motion.

A lineman receives tremendous force on the lumbosacral area, predisposing this area to back pain, spondylolysis, and spondylolisthesis. Performing regular core stabilization exercises can prevent injury as well as protect a previously injured lumbosacral area.

The cervical spine is quite vulnerable to injury in football as the head is often the primary contact point during collisions. The ruling preventing spearing has considerably cut down on the number of catastrophic neck and back injuries, but the head and neck remain areas of high concern (21). Therefore, neck strengthening through isometric and specific resistance exercises is quite important.

In high school and college football, the most commonly injured joint is the knee (3). A study revealed that through a preseason conditioning program there was a 61% reduction in knee injuries in linemen in one season (21). Therefore, it is vitally important to maintain good leg strength in the hip girdle, quadriceps, hamstrings, and gastrocnemius-soleus muscles in order to optimize performance and minimize the risk of injury.

Core Stabilization Exercises

Core stability exercise can be defined loosely as the restoration or augmentation of the ability of the neuromuscular system to control and protect the spine from injury or reinjury (22). Core stabilization can be categorized into two basic principles, both of which are necessary for a good core stabilization exercise program:

1. Exercises to restore coordination and control of the lumbar spine.
2. Exercises to restore capacity (strength and endurance) of the lumbar musculature.
3. Treat lumbosacral dysfunctions.

The deep trunk muscles of the lumbar spine—transversus abdominis, multifidus, internal oblique, paraspinal, and pelvic floor muscles—are key stabilizers. The direct and indirect actions of these muscles via the thoracolumbar fascia and the so-called "intra-abdominal pressure mechanism" can coordinate and resist external forces applied to the lumbar spine.

Exercises designed to recruit the transversus abdominis and multifidus are based on the observation that these muscles contract in coordination with each other prior to limb movement. These muscles can be trained using the abdominal hollowing technique. While lying on his back in the neutral position, the athlete is instructed to relax the muscles of the abdomen, bend the knees, and while slowly breathing out, pull the lower abdomen inward as if trying to push the umbilicus toward the floor. This is a common exercise in Pilates classes, commonly known as the zipping-up maneuver.

The hollowing technique can be combined with multiple other exercises involving the lower extremities, such as straight leg lifts and knees-to-chest exercises, remembering to begin with the abdominal hollowing technique before adding limb movement exercises.

Abdominal exercises are an important part of any lumbar stabilization program in order to prevent muscle imbalance. There are many different abdominal strengthening exercises, and the athlete may wish to choose the ones he prefers and be instructed in proper technique to avoid other injury.

Coordination and control exercises are designed to help the body's proprioceptive systems and coordinate muscle control to prevent

injury during movements or when colliding into external forces. These exercises may include, but are not limited to, the McGill stability exercises, Pilates, Swiss ball programs, balance board, and shoe training.

CONCLUSION

The football athlete has a wide range of injuries, traumatic and nontraumatic. Throwing in football is fundamentally similar but biomechanically different from baseball, so knowledge of both throwing mechanisms aids in administering proper treatment. Manual medicine and exercise therapy when used effectively can reduce or prevent injuries, speed injury recovery, and enhance athletic performance. Core stabilization is a large part of preventing and treating injuries.

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31

GOLF

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KERRY GRAHAM

Golf is enjoyed worldwide by millions of people each year and is considered one of the most rapidly growing sports. In 2002, 32.3 million people in the United States played at least one round of golf. Of those golfers, 45% were between the ages of 18 and 39. Golfers 50 years and older represented 33%, and junior golfers make up 5% of the golf population. The National Golf Foundation (NGF) also noted that 20% of the golfing population plays over 25 rounds per year (avid golfers). There are 14,725 golf course facilities available in the United States, a 20% increase in the last 7 years. The golf economy in the United States accounted for over $62 billion worth of goods and services in the year 2000 (1).

THE ATHLETE

Much of golf's popularity stems from its low physical demand on the body, allowing the sport to be enjoyed by young and old alike. For those unfamiliar with the game, golf appears to be relaxed and unathletic. However, muscle endurance, strength, flexibility, and cardiovascular fitness are all required for the sport (2). Because golf is considered to be low intensity with low physical demand, it has not been considered a high injury risk sport (3). Unfortunately, though, injury is one of the most common reasons for players to leave the game each year. The types and frequency of injuries that occur while playing golf are common among men and women, amateurs and professionals alike, with some variations related to the low back, wrist, and hand (4). Common injury sites in golf players include the wrist, elbow, shoulder, knee, and most commonly the back (5). Excessive repetition, poor swing mechanics, lack of conditioning, poor fitness, and improperly fitted golf clubs are primary factors in the etiology of these injuries.

Fitness and strength training programs are now routinely part of the golf program at colleges and universities throughout the United States. Junior golfers and high school golf teams have more interest in year-round performance enhancement programs that include skill assessment, strength, conditioning, and fitness. This positive trend will promote the safety and well-being of these young athletes.

THE GAME

Significant advances in technology have improved golf clubs and golf balls enhancing the player's ability to hit longer and more accurate shots. New technology has also produced shafts and grips that decrease impact stress on the golfer's elbows, shoulders, and wrists. Video and computer motion-measuring technology has dramatically influenced the ability to analyze the components of the golf swing meticulously and with precision. When combined with the proper medical evaluation, clinicians and golf pros can detect swing flaws that may create pain syndromes and have an impact on performance.

Golf Swing Biomechanics

With new innovations in equipment and technology, the swing has evolved over the years, allowing shot making with increased precision and distance. Players using the classic swing (pre-1930) (Fig. 31.1A) and playing with hick-

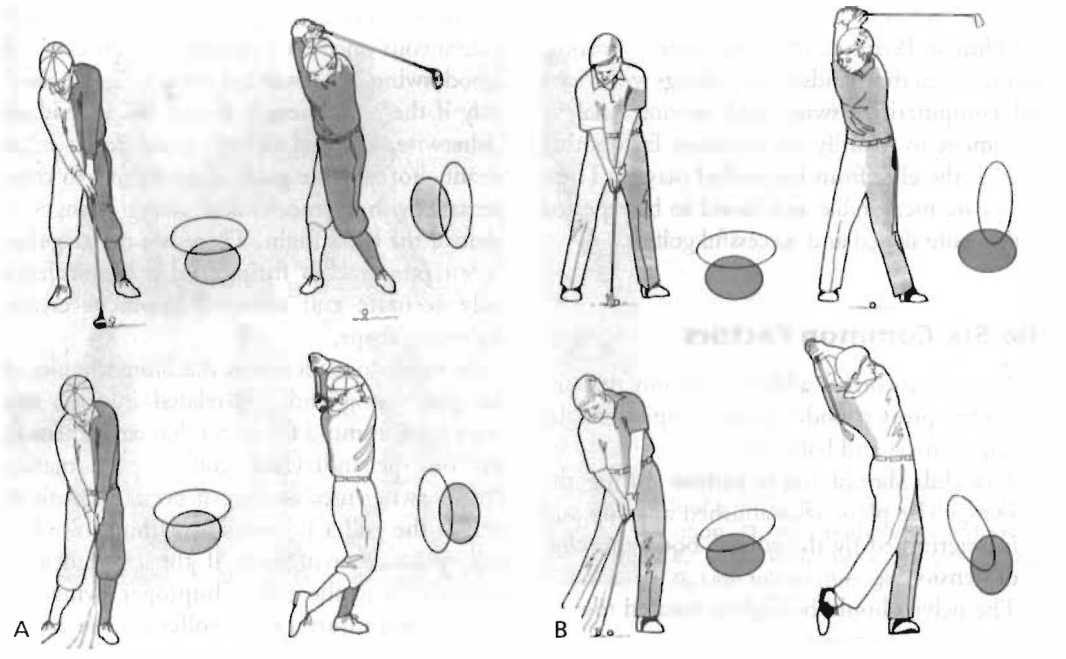

FIGURE 31.1. Evolution of the golf swing. **A,** Classic (pre-1930s). **B,** Modern (2000).

ory shafts and bulky clothing had a backswing that was on a more linear swing plane, allowing for concomitant hip and shoulder rotation with the player in an upright "I" position for follow-through. However, the modern swing (Fig. 31.1B) uses a more coiled body position in which there is a greater rotation of the shoulder with restriction of motion at the hips. As the swing has evolved, it has also created more stress on the body (6).

Evolution of the Golf Swing

1. Classic (pre-1930s)
   - Club shafts could not be stressed or they would break.
   - Players wore suit coats, which made the swing more restricted.
   - Hips and shoulders turned almost equally.
   - The player rose up on the lead toe in the backswing.
   - More upright "I" follow-through position created less stress on the low back.
2. Modern (2000)
   - Based on economy of motion and coil.
   - Coil and weight shift have become the main focus.
   - Large shoulder turn and minimal hip turn—the X-factor.
   - A back-bend, "C"-shaped follow-through torques the low back.

During the swing of an elite golfer, a repeatable sequence of muscle activation occurs. The abdominal muscles contract, transferring the forces into the torso and activating the pectoralis and latissimus dorsi muscles. Transfer of kinetic energy then moves into the shoulders and rotator cuff, especially the subscapularis and arms. A very important counteraction occurs when abdominal muscle contraction causes the spine to move forward, forcing the lumbar paraspinal muscles to contract, counteracting the abdominal muscles. Of additional significance, the spine receives forces with four different stress loads: (a) lateral flexion, (b) anterior posterior shearing, (c) rotation, and (d) compression (7).

The early scientific efforts by Jobe and Pink through their electromyographic analysis of the golf swing (8) set the stage for continued medical research that unfortunately has been fairly slow

and intermittent. Recently, SportExcel Health and Human Performance in Scottsdale, Arizona, has evaluated thousands of golf swings with video and computerized swing and motion analysis equipment to identify six common factors that separate the elite from less skilled players. These factors are measurable and found to be repeated by the more skilled and successful golfers.

The Six Common Factors

1. There is a correct address position that includes proper body posture, spine angle, grip, stance, and balance.
2. The club should stay on plane during the swing. The plane is established at setup and is determined by the golfer's body and club dimensions.
3. The pelvis should be slightly rotated toward the target at impact.
4. Balance and weight transfer. The golfer must remain in balance throughout the swing, and his or her weight transfers to the back foot in the backswing and to the forward foot during the forward swing and follow-through.
5. The back leg is "posted" in the backswing—i.e., it does not move during the swing—and provides resistance and stability as body weight is moved onto it (loads) in the backswing. The angle of flexion in the knee and hip is retained as the upper body rotates and weight is transferred.
6. Golf clubs should be properly fit to the individual.

The likelihood of achieving squareness of club face and centeredness of hit at impact markedly increases with greater recruitment of the six common factors during the swing; squaredness and centeredness are necessary to achieve maximum distance and accuracy. Inability by a golfer to accomplish factors 1 through 5 with excellence is often due to physical deficiency or injury. Increasing flexibility, improving conditioning, or resolving the injury often allows the golfer to increase excellence in the first five common factors.

The sixth common factor—proper club fitting—is vital to the performance of the first five factors. Feedback to the golfer's brain is instantaneous once the ball's flight is observed. A "good swing" is rewarded with a "good shot" only if the equipment is fit for the individual. Otherwise, a "good swing" could result in an errant shot and the golfer then begins to compensate with biomechanical swing changes to control the ball's flight. There is a marriage between proper club fitting and a biomechanically accurate golf swing. Equipment creates the swing shape.

In order to understand the biomechanics of the golf swing and golf-related injuries, one must keep in mind the effect that equipment fit has on the individual golfer's performance. Proper swing mechanics can occur repetitively only if the golfer is swinging without hitting a ball (practice swing), or if the equipment is properly fit to the golfer. Improper swing mechanics and injury to the golfer can be caused by compensation for improperly fit equipment.

THE SWING

There are six phases to a properly executed golf swing:

1. Setup/Address
2. Backswing
3. Top/Transition
4. Forward swing/Acceleration
5. Impact
6. Early Follow-through/Late follow-through

Setup and Address. The setup is where everything good or bad begins. By establishing the proper address position, the player has proper balance, both within his or her body and the golf club. Starting in a balanced position enhances the golfer's opportunity to remain balanced throughout the execution of the swing. If the club is properly fit to the individual's body dimensions and strength, the result of a balanced address and swing will give the desired outcome—an accurate shot in direction and distance.

At setup, the weight is evenly balanced both between the feet and toe-to-heel while maintaining proper spine angles (Fig. 31.2A). There is lateral spine flexion relative to the hips, but not

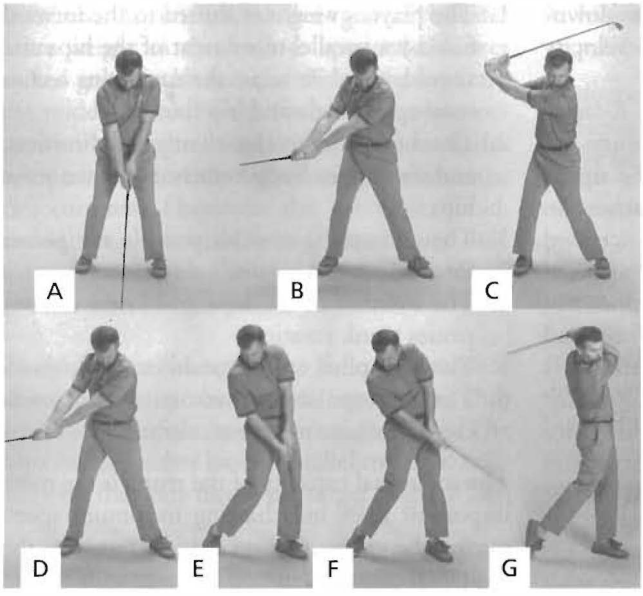

FIGURE 31.2. The golf swing. **A,** Setup and address. **B,** Backswing. **C,** Top/transition. **D,** Acceleration/forward swing. **E,** Impact. **F,** Early follow-through. **G,** Late follow-through.

flexion of the spine itself (9). The feet should be wide enough to support the swing motion without restricting pelvic rotation (about shoulder width apart), and the toes should be turned slightly outward. Commonly, professionals turn the forward toe out more than the back toe. If the toes are turned in too much, the knees may resist the turning motion which leads to excess stress on the knee joints. The knees and hips are in a game-ready position when the body is balanced and the spine angles and flexion of the hips and knees are proper. Proper clubs and equipment analysis relative to the individual golfer's body measurements, strength, and motion are critical as every sequence to follow is part of a domino effect, either positive or negative.

Backswing. Less than one fourth of all injuries occur during the backswing (10). It is a synchronous, linked progression of torque, rotation, and load transfer through the feet up through the knees, hips, torso, lumbar spine, and cervical spine (Fig. 31.2B). The player's weight transfers to the back foot, the left thumb hyperextends, the left wrist radially deviates, and the right wrist dorsiflexes and radially deviates (10). The head position remains fairly constant; however, individual preferences occur. While the head may move slightly away from the target during the backswing, it is improper for it to ever move toward the target during this phase of the swing. The key to a successful backswing is to allow each motion to flow and coordinate with the other motions; thus, flexibility is essential.

Golf swing motion theories often get validation from golf magazines and television instruction programs instead of scientific research or small sample groups. There has been little long-term scientific research conducted on the modern golf swing in relation to the effects on the body and parameters for success. For instance, research in 1996 was presented in popular golf magazines and on television golf instruction claiming that the difference between the hip and shoulder rotation at the top of the backswing is critical in generating power (11). The concept of creating torque by increasing shoulder rotation while keeping a more fixed hip rotation at the top of the backswing became known as the X-factor. McTeigue and Anderson noted that by creating a larger gap at the top of the backswing, a

more complete closure occurred on the downswing allowing for greater club head velocity at impact (11).

However, attempting to execute the X-factor has created an increased risk of injury for many golfers. Increased torsion of the upper torso with fixed hip placement causes an aggressive shoulder rotation with increased stress to the lower back. Cheetham and coworkers in 2001 noted that research does not prove that the X-factor increases club head speed or distance for professionals or amateurs (12). They claim instead that the X-factor stretch, which measures a separation distance at a point "between" the top of the backswing and impact, has a higher correlation to greater club head velocity at impact. Both of these theories invite additional research on a larger population.

Biomechanical inconsistencies during the backswing can lead to numerous injuries:

1. Left wrist flexor carpi ulnaris stretching, leading to tendinitis.
2. Neuropathy of the left wrist with ulnar nerve compression.
3. Dorsal carpal ligament syndrome and extensor tendinitis from right wrist dorsiflexion.
4. Impingement of the right shoulder during hyperabduction.
5. Low back strain from lumbar spine hyperrotation.
6. Medial epicondylitis from overstretching of the right elbow flexors.

Top/Transition. Differences in terminology exist for when the top of the backswing is completed (Fig. 31.2C) due to the fact that the body is beginning its forward motion while the club is still in a backswing mode, thus the resulting label of transition stage (13).

Acceleration/Forward Swing. During the forward swing, the club is returned to its original position to hit the ball at the correct angular trajectory and at maximum speed (Fig. 31.2D). Several events are occurring at this moment:

1. The player's weight is shifted to the forward foot by a parallel movement of the hips and shoulders, while he or she maintains a constant spine angle and hip flexion.
2. The hips initiate the change of direction, and the upper body rotates faster than the hips.
3. The abdominal muscles provide the power for the trunk to rotate.
4. The rotation of the hips and knees accompanies trunk rotation.
5. The paraspinal muscles stabilize the spine.
6. The subscapular and pectoralis major muscles contribute to arm acceleration.

The rotational capacity of the trunk is the most important lever in achieving maximum speed during the swing. Golfers that do not have the rotational capacity must compensate with greater muscular activity.

Biomechanical inconsistencies during the forward swing can lead to numerous injuries, such as the following:

1. Tendinitis as a result of left thumb hyperabduction.
2. Medial epicondylitis from right elbow forearm load of the flexors.
3. Ulnar-deviated wrist impaction leading to ulnar neuropathy, hamate fracture, or flexor carpi ulnaris tendinitis.
4. Right knee medial valgus strain, leading to tibial collateral ligament strain.
5. Lateral epicondylitis resulting from left elbow lateral extensor tendon lengthening with deceleration or impact with the ground.

Impact. When the club impacts the ball, the kinetic energy produced during the forward swing is transferred to the ball (Fig. 31.2E). At the point of impact, the acceleration that is created from the forward swing is coupled with acceleration from the wrist and hands. To maximize the distance that the ball will travel, acceleration does not stop until after the ball is struck. Weight transfer to the forward foot is critical to create proper balance and ball flight but also to avoid injury that could be caused by excess transfer of energy to the back and ex-

tremities. At impact, skilled golfers transfer more weight to the heel, as opposed to lesser skilled golfers who transfer their weight toward the middle or toe of the foot. Valgus stress occurs on the right knee, both wrists are under compression, and the left elbow extensor muscles contract. Therefore, the left wrist, hand, and elbow are at risk during impact, not only from contact with the golf ball but also from contact with the ground.

Early Follow-through. The follow-through is characterized by the deceleration of the club and rotation of the body around the spine. The hips and shoulders have a parallel rotation, ending with the body facing the target (Fig. 31.2F).

Late Follow-through. The follow-through should take as long as needed to stop the club and complete the swing in a balanced position (Fig. 31.2G). If the club is stopped too soon, the risk of injury increases. One fourth of all injuries occur during this stage (10). The muscular activity is primarily from the spine and shoulders, although the abdominal and pectoral muscles also play a role. The rotator cuff muscles are also activated during the follow-through, an area where injury may occur. Biomechanical inconsistencies during the follow-through can lead to numerous injuries:

1. Medial epicondylitis with left elbow supination strain.
2. Lateral epicondylitis with right elbow pronation strain.
3. Lumbar hyperextension and rotation leading to low back strain (reversed "C"-configuration).
4. Right knee valgus strain leading to tibial collateral ligament strain.
5. Left knee rotary strain leading to knee ligament strain.
6. Flexor carpi ulnaris tendinitis with ulnar wrist strain.
7. Right wrist volar flexion leading to medial epicondylitis, flexor carpi ulnaris tendinitis, or ulnar neuropathy.
8. Lateral epicondylitis, extensor tendinitis with left wrist dorsiflexion.
9. Impingement of the left shoulder with hyperabduction.

High-speed creative golf shots with abruptly terminated follow-throughs lead to injury if the golfer has poor muscle strength and endurance.

INJURIES

The primary causes of golf injury are overuse and mechanical errors related to technique, individual physical deficiencies, and/or the fit of the golf equipment. During the swing, injuries may occur due to physical fitness deficiencies such as poor flexibility and conditioning as well as the lack of proper stretching and pregame conditioning. Injury may also occur due to motions the golfer makes to compensate for poorly fit equipment. Occurrence of injury and the site of injury can be broken down for each phase of the swing. The majority of injuries occur during the forward swing followed by the follow-through and then backswing (10). The anatomic distribution of injuries is similar between professional and amateur golfers, with lower back injuries being the most commonly reported.

While injuries to the back and lead elbow are the most common, it seems that wrist injuries are increasing in incidence among low-handicap and professional players (14). Women professionals are more prone to wrist injuries than men (14% women vs. 6% men) (10).

Equipment Integration and Analysis

The many variables that make up the golf club must be considered when individually analyzing and selecting proper clubs for each golfer. Three examples of club-fitting variables and how, if misfit, they can contribute to injuries are explained as follows:

1. Length of the club. If the club is too long, repeated collisions with the ground (fat shots) stress the muscles of the upper body, which can lead to shoulder, elbow, and rib injury.

If the length of the club is too short, the golfer compensates for the deficiency by decreasing the flexion of the knees or changing the angle of the hip and spine during the backswing in order to remain balanced. Upon forward swing of the club prior to ball impact, the golfer may hit the ground misjudging the amount of flexion needed to get back down to hit the shot. The golfer's knee and hip angle can remain the same throughout the swing when he or she is using a proper length club, eliminating the need to stand up in the backswing and flex at impact and reducing the likelihood of hitting a fat shot.

2. Lie angle. The proper lie angle of the club head bend (junction of the adjoining shaft to the head) can vary many degrees between golfers. When the lie angle is incorrect, the club is improperly soled at impact causing ball flight direction and distance to be affected. To manipulate ball flight, the golfer often hyperextends the back into a pronounced "C" position during impact, causing increased lumbar strain. Providing the proper lie angle allows each golfer to keep his or her spine angle, and hip and knee flexion constant and in the position that was established at address. This causes much less stress on the lower back and other joints during the swing.
3. Shaft flexibility. The flexibility of the shaft, as well as its flex point, greatly influences the direction, distance, and height of the ball's flight. To create the desired ball flight with clubs of the wrong flex for his or her individual swing strength and rhythm, a golfer often appears to have swing flaws or errors leading to shoulder, elbow, wrist, and back injuries. If the golfer is properly fitted for shaft flex in his or her clubs, the swing flaw and aggravated injury often disappear.

Back Injuries

Most studies have shown the back to be the most commonly reported site of injury in both professional and amateur golfers (5). Back injury and pain show little respect for skill level, as many professionals have learned. In amateurs, faulty swing mechanics, poor conditioning, and ill-fitted clubs are the common culprits; overuse and conditioning issues are primary factors in elite golfers.

Injuries to the back can occur during all phases of the swing, most commonly at the top of the backswing, after impact, and during follow-through. The fitness level of the individual often dictates the risk for back pain (15). Individuals with a lower activity level tend to have reduced strength of the trunk muscles and an increase in back pain. Individuals who are active and fit may suffer from overuse injury from long practice sessions, extended playing time, or poor mechanics (15,16). The forces applied to the dorsolumbar spinal area during the golf swing act in four distinct directions: (a) lateral flexion, (b) anteroposterior shearing, (c) rotation, and (d) compression (Fig. 31.3) (7). The first three forces are more likely to affect amateur golfers more than professionals. Amateurs overenthusiastically drive the ball farther without having the technical skills of a professional. Disc degeneration limits the angular rotation of the trunk and reduces power, which results in overcompensation by the player.

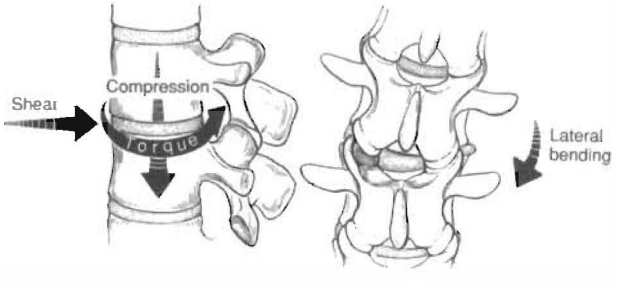

FIGURE 31.3. Forces applied to the dorsolumbar spine during the golf swing.

The golfer usually seeks medical care as a result of injury to the spine that occurs during a round or with a complaint of low back pain that has limited the ability to play golf. Low back pain can be placed in one of three categories:

1. *Acute.* Low back pain caused by a specific injury, e.g., an overaggressive swing.
2. *False acute.* This is really an acute exacerbation of a chronic back condition and is not due to a specific trauma, e.g., back pain as a result of picking up the ball.
3. *Chronic persistent.* Back pain over 90 days in duration or three or more past episodes of pain.

It is important to determine which category the golfer is in so that correct treatment and rehabilitation can be implemented. For decades, it was thought that chronic low back pain was a result of decreased abdominal muscle strength. Although still widely held, this theory has been discounted by a number of studies that have shown specific lumbar extensor strength deficits in people with chronic back pain compared with pain-free subjects (17). One study using isokinetic equipment showed weaker lumbar extensors than abdominals in patients with back pain. Another study compared myoelectric activity in the lumbar extensors to that of the abdominal muscles during lifting (18). Only the lumbar extensors correlated with increased resistance. Studies have also shown selective atrophy of the lumbar musculature, most active in lifting, visible on magnetic resonance scans as increased fatty infiltration (19,20).

Weakness of the lumbar extensors is widely regarded as the weak link in chronic back pain, and range of motion exercises, which have the effect of improving fluid exchange in the disc and other connective tissue, may not have much of an effect on improving spinal extensor strength. Maintaining an unsupported trunk in the prone position (Sorenson's test) can be an effective tool in predicting future low back pain (21).

Strengthening of the lumbar extensors is best achieved when the lumbar extensors are isolated by stabilization of the lower extremity and pelvis. One effective tool available for isolating the lumbar extensors are computerized testing and rehabilitation machines. Another device that has demonstrated some effectiveness in activating the extensor musculature is the classic roman chair. Its variable angles allow for increased demands as the individual becomes stronger.

Studies have indicated that people who suffer from back pain have a higher incidence of repeat episodes, so risk factors for chronicity must be addressed. These may include previous history of low back pain, total work loss in the past 12 months, heavy smoking, heavy physical occupation, radiating leg pain, reduced straight leg raising, signs of nerve root involvement, reduced trunk strength and endurance, poor physical fitness, and slower than normal response to treatment of spinal pathology such as spondylolisthesis (17).

For those golfers who present with a false acute injury, chronic back pain, or risk factors for chronicity, treatment should be aimed at reducing acute pain with the goal of facilitating a specific rehabilitation program.

Short-term and long-term treatment should focus on back stabilization and neutral spine protocols and techniques, including aggressive strengthening of the lumbar extensor, rectus, and external oblique musculature (22). Flexibility training and proper warm-up habits also decrease the occurrence of injury. Swing modifications to develop a safe backswing should be learned to reduce lumbar spine strain, such as the following:

1. Reduce shoulder rotation by shortening the backswing or increasing the hip rotation (decreasing the X-factor), thereby reducing the amount of difference between hip and shoulder rotation.
2. Train in neutral spine, while maintaining pain-free spine angles.
3. Reduce lateral flexion.
4. Reduce flexion and eliminate the "C"-configuration in the follow-through.

Elbow, Hand, and Wrist Injuries

The causes of tendon pain are more directly related to degeneration of the tendon (tendinosis) as opposed to an inflammatory etiology (ten-

dinitis). Lateral and medial epicondylitis (tendinosis) are the most common sites of injury of the elbow. Lateral epicondylitis occurs most commonly in the left arm, which is the lead arm in right-handed golfers (23). This injury results from sudden trauma or overuse of the extensor carpi radialis tendon inserting proximally into the lateral epicondyle (24). Medial epicondylitis, or golfer's elbow, affects the right arm in the right-handed golfer, and the left arm in the left-handed golfer; it commonly occurs when the flexor tendons of the flexor carpi radialis, flexor carpi ulnaris, and palmaris longus muscles become inflamed through overuse. Up to 20% of ulnar nerve symptoms with numbness on the ulnar side of the hand are associated with medial epicondylitis (25). Both medial and lateral epicondylitis occur with equal frequency in golf (26) and is similar in male and female golfers (27). There are differences between amateur and professional and male and female golfers in the incidence of upper extremity injuries (14).

1. Amateur female players have a higher incidence of elbow pain.
2. Professional female golfers have a one-third greater incidence of hand and wrist pain than their male professional counterparts. This has been attributed to women having less upper body strength than men.

The wrist is highly susceptible to injury. In 1996, a study of 226 golfers of the PGA and LPGA Tours reported 393 total injuries: 24% of the left wrist, 7% of the left hand, and 3% of the right wrist (27). The wrist often gets stressed beyond the normal wrist range of motion (28). Elongation beyond 4% of the tensile length of the connective tissue creates microtrauma as the molecular cross-linking of the collagen fibers ruptures (29). Cahalan et al. established that subjects with pathologic conditions of the forearm, wrist, and hand exhibited a greater arc of motion during the golf swing (30). Therefore, pain during the swing arose from the inability of the subject's weakened wrist muscles to resist counterforces causing excessive motion during the golf swing. Stover et al. found that the preimpact and impact swing phases place the right wrist in valgus positions, which maximally stresses the common flexor tendon and predisposes it to rupture (31). The most frequent overuse injuries ate the dorsal radiocarpal and distal radioulnar chronic sprain and the ulnocarpal chronic sprain (28).

De Quervain's tenosynovitis is a common golf injury that occurs when the tendon sheath of the abductor pollicis longus and extensor pollicis brevis become inflamed as they pass through the first dorsal compartment of the wrist (32). During the golf swing if the leading wrist takes an ulnarly deviated position at ball impact, the development of de Quervain's may ensue (33). Additionally, the premature uncocking of the wrists at the beginning of the downswing rather than during acceleration in an unsuccessful casting maneuver traps the leading thumb between the trailing hand and shaft and stresses the tendons in the first compartment, also predisposing the extensor carpi ulnaris to become inflamed. Repetitive dorsiflexion and cocking of the wrist during the backswing have also been implicated as a cause of acute, painful de Quervain's syndrome (27).

Golf injuries involving the elbow and wrists can be greatly reduced or avoided with the use of proper swing mechanics, preconditioning of the muscles through strength and flexibility training, and application of proper club fitting. Recognition is usually the rate-limiting step towatd recovery and prevention.

Shoulder Injuries

The shoulder is the fourth most frequently injured site in golfers, making up 10% of injuries in professional and recreational golfers (3). Shoulder pain is often related to overuse, but if the pain is in the lead shoulder, it may be caused by faulty swing mechanics. Right shoulder pain in the right-handed golfer is less likely to be related to golf swing mechanics (34). Additionally, injury can occur from excessive shoulder rotation and overuse and is influenced by the golfer's age and the power of the swing (28). Throughout the golf swing, the subscapularis is the most active of the rorator cuff muscles. A review of 412 patients at the Kerlan-

Jobe Orthopedic Clinic with injuries attributable to golf demonstrated 85 of these injuries at the shoulder and 79 involving the rotator cuff (35).

During the swing, the leading shoulder is subjected to a wide range of motions that increase the risk of injury. Anterior pain in the lead shoulder during the backswing may indicate degenerative changes in the acromioclavicular (AC) joint or impingement, whereas posterior capsular pain is due to capsule tightness. In comparing older and younger golfers, Jobe and Pink found that the swing of the older golfer (>35 years of age) stresses the AC joint more, where injury is first to occur (8). The repetitive positioning of the arm across the body can cause spur formation under the AC joint, impingement of the rotator cuff on spurs, and bursal-side partial cuff tears leading to a reduction of flexibility and resiliency of the tissue. By contrast, the younger golfer (<35 years of age) demonstrated hyperlaxity of the shoulder ligaments, leading to instability. To reduce stress on the AC joint without affecting the club's speed, the golfer should shorten the end point of the backswing. Fitness training of the rotator cuff and scapular muscles lessens the impact of fatigue and maintains improved upper extremity flexibility.

Knee Injuries

Injury to the knees accounts for approximately 9% of musculoskeletal golf injuries (16). However, knee joint forces generated during the golf swing are not large enough for golf to be considered an activity with high risk for traumatic knee injury (36). Most golf knee injuries are exacerbations of previous injuries that drove the player to golf from other sports. Being able to return to playing golf is often the most common functional goal for hip and knee replacement patients (37). Guten made general golf recommendations for the player with knee pain (34):

1. Use pain as a guide in reducing the frequency of play or practice. A golfer should play just up to the point where the knee hurts, but not through the pain. The golfer must be able to differentiate between good pain (that of a generalized muscle ache afrer exercise which does not persist or recur) and bad pain (localized pain of the joint or tendon with persisting recurrence and associated swelling). Unlike other sports, golf allows its players to patticipate by reducing the amount and intensity of play without penalty through the use of the handicap system.
2. Play or practice primarily with the shorter 7, 8, 9 irons and wedges. Less force and torque are required to make the swing. The golfer with a painful knee should be encouraged to gradually progress to longer clubs.
3. Take golf lessons. The use of modern video and computerized swing analysis by the teaching professionals may help in distinguishing improper form, leading to correction and alleviation of the injury-causing swing.
4. Strive for equal distribution of weight on both legs. Stabilizing the weight on both legs should equalize the stress on the knees.
5. Use graphite clubs. Graphite reduces the force load on the body by 30%.
6. Use shoes without spikes or have the spikes ground down by approximately 50%. With fewer fixations to the ground, the golfer's natural swing requires less intense force.
7. Incorporate an exercise regimen into a regular workout, focusing on flexibility, endurance, speed, and strength.
8. Wear a sports knee brace to reduce the pain and provide stability to the knee during golf. Usually a neoprene open patellar sleeve can be helpful. In more unstable knees, a hinged or joint unloading brace may be required.

MANUAL MEDICINE APPROACH TO THE GOLFER

The successful golfer has a fluid swing which minimizes variances on each attempt. Each golfer should be screened for areas of restriction and treated accordingly. Because the swing is unidirectional, crossed pelvic syndromes and sacral torsions can occur that can trigger pain and inhibition of the lumbar extensors. These lumbosacral dysfunctions need to be corrected, particularly sacral torsions, sacrotuberous and iliolumbar ligament dysfunction, and rotated

or sheared innominates. Posterior spine pathology, such as facet injury and spondylosis, can limit extension strength as well.

Foot and ankle pain due to various dysfunctions and injuries should not be dismissed because they can keep a golfer off balance from setup to follow-through. Weight is shifted toward the front, so any talar or subtalar dysfunctions should be diagnosed and treated to allow weight shift to happen without restriction.

The golfer should not be slumped over at setup, so if a golfer has difficulty stabilizing the spine, look for flexed thoracic dysfunctions and shortened iliopsoas muscles, which extend the lumbar spine and decrease its mobility. Treat its origin (T12-L2) with direct techniques and the muscle with counterstrain, myofascial release, or even direct muscle energy. Any golfer with back problems should also be on a core stabilization program to help keep the spine in alignment.

Upper body rotation generates club speed for distance, so shoulder pathology such as posterior capsule restrictions, rotator cuff impingement, and acromioclavicular osteoarthrosis can affect stroke fluidity and range. Any applied manual techniques should be matched with a stretching program.

Manual medicine techniques for overuse injuries of the upper extremity target several regions:

1. The *injury site* itself. Lateral and medial epicondylitis has been discussed previously. De Quervain's tenosynovitis can benefit from joint play treatments of the radiocarpal joint and the first carpometacarpal joint. Massage to the involved tendons and muscles helps decrease swelling, unless the injury is acutely inflamed. Stretches for the extensor and abductor pollicis tendons and muscles help as well.
2. *Proximal radioulnar joint.* This is usually restricted in epicondylitis and some wrist tendinopathies. Muscle energy and high-velocity, low-amplitude techniques are effective.
3. *Shoulder instability.* If a young golfer has loose shoulders that are leading to instability, a rotator cuff strengthening and proprioceptive training program should be part of the regimen as well. Older golfers with capsular restrictions and arthritic changes would benefit from Spencer techniques.
4. *Other joints proximal and distal to the injury.* Mobilizing the shoulder and cervical spine is important for any golfer to execute a swing smoothly and efficiently. Dysfunctions of the C7-T1-first rib complex, shoulder adduction, and the radial head should be evaluated and treated with myofascial, muscle energy, and/or counterstrain, depending on the exact diagnosis.
5. *Hip.* Mobilizations of the anterior capsule, muscle energy for external and internal rotation restriction, and correction of any pelvic imbalance allows for proper generation of energy through the core and into the upper body. If the hips cannot move properly, the upper extremities are forced to work harder, which may disrupt the swing.
6. *Lumbosacral spine.* Rotation needs to be fluid, and any lumbar dysfunctions that inhibit lumbar extensor strength affect the upper extremity and need to be treated.

SUMMARY

Golf injuries are often related to defects in the player's swing mechanics. The golfer should be evaluated and treated holistically, since the relationship among the lower extremities, trunk, and upper extremities must be working well in order to execute a swing. Golfers are athletes and need to train to improve conditioning, core strength, and flexibility. Manual medicine techniques are effectively applied in many chronic injuries, particularly in the lumbosacral and upper extremity regions.

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32

GYMNASTICS

LARRY NASSAR

Female artistic gymnastics is one of the most popular summer Olympic sports in the United States. Tickets are in high demand to attend the competition, and millions watch the television broadcast during the Olympics. Yet during the quadrennium between Olympics, it is almost forgotten by the general public. In the medical community it is thought of as a sport of young girls, but this has changed. The Federation of International Gymnastics has been increasing the age of participation at the Olympics. Currently, the minimum age to be eligible for the Olympics is 16, and in the United States there are several international elite gymnasts who are over 20 years old and one that is 30 years old.

Gymnastics is a sport in constant flux. Every 4 years the rules change. With the rule changes come more difficult requirements. The equipment has dramatically changed over the years and a completely new vault table has recently been introduced. These frequent changes make it difficult for the general public to understand the sport and challenge the sports medicine practitioner to understand the current injury epidemiology.

EPIDEMIOLOGY

The most common areas for injury in the club and collegiate female gymnast (1) in order of frequency are (a) the ankle, (b) the wrist, and (c) the spine. Brüggemann, the current President of the Scientific Commission of the Federation of International Gymnastics, recorded that at takeoff for gymnastic tumbling on a spring floor (roundoff to a back-tuck somersault), the resultant moments at the ankle joint were 310 N·m in the sagittal plane and 100 N·m in the frontal plane (2). This correlates to an Achilles tendon force of 7,500 N (~15 times the gymnast's body weight), a tibiotalar joint force of 11,000 N (~23 times body weight) and a talonavicular joint force of 8,000 N (~15 times body weight). These forces in the foot and ankle are almost doubled with improper foot eversion or pronation by decreasing the contact area of the joint surfaces. With these types of extreme forces, the likelihood of injury of the foot and ankle is high.

LOW BACK INJURIES

Current elite-level female gymnasts are reported to have an 11.1% incidence of spondylolysis and a 2.9% incidence of spondylolisthesis (2). These statistics do not significantly differ from the normal population, yet a 44.4% incidence of osteochondritic changes was found in female gymnasts. The majority of these changes were end-plate compression fractures, where the nucleus pulposus herniates into the vertebral body. In the immature spine, especially during the adolescent growth spurt, the disc is stronger than the cartilaginous end plate. Thus, the end plate becomes damaged before the disc when exposed to compressive forces. These fractures are mainly found in the thoracolumbar junction (T11-L3). This area of the spine is at high risk for fracture due to its relative neutral alignment and transition between the more rigid thoracic spine and more flexible lumbar spine (1).

Swärd et al. found that normal disc heights were present with first diagnosis of vertebral end-plate damage in the gymnast. However, subsequent follow-up revealed marked disc

degeneration 10 to 12 months after injury (3). Nassar and co-workers studied the 1999 USA Gymnastics Female Artistic National Team, and 11 of 19 gymnasts (58%) had lumbar disc degeneration on magnetic resonance imaging (MRI). Anterior apophyseal ring fractures were found in 7 of 19 gymnasts (37%) (4).

The skills and equipment in gymnastics have changed over the years, so findings in older studies on gymnasts are outdated and of little value. In the past, gymnasts performed many back walkovers, front walkovers, limbers, and other skills that created exceptional stress to the lumbothoracic spine. These skills are no longer performed with the same frequency as in the past. Gymnastic coaches have improved their technique with emphasis on proper use of the shoulders and thoracic spine to remove stress from the lower lumbar pars interarticularis. The addition of new training aids such as sting mats can reduce tumbling impact forces on the spine by 20% (2). Finally, reduction in the incidence of posterior column fractures may be attributed to coaches' and athletes' awareness to seek medical help early on for these injuries when only a stress reaction may be present and before a full stress fracture occurs.

The high-impact landing forces in gymnastics can be tolerated by the gymnast only by appropriate timing and balance of the muscular activity of the spine and lower extremity to distribute these forces most efficiently through the spine. If the gymnast performs a routine with improper timing or muscular firing sequence, the potential for injury on landing becomes great. Landing with a flexed trunk increases the pressure on the anterior end plates most significantly at the thoracolumbar junction. This leads to end-plate damage and marked disc degeneration noted 10 to 12 months after injury (3).

Hall states that the main cause of low back pain in the female artistic gymnast is uncontrollable hyperextension of the lumbar spine (5). Brady showed that a decrease in hip extension and poor thoracic extension motion contribute significantly to low back pain (6). Jull and Janda coined the term *pelvic crossed syndrome* (7) to explain how a gradual-onset nontraumatic overuse injury may develop in accordance with the explanations of gymnast lumbar mechanics given by Hall and Brady.

Pelvic crossed syndrome is an imbalance in the lumbar-pelvic-hip complex whereby the short and tight hip flexors and lumbar erector spinae muscles neurologically inhibit the proper firing and strength of the abdominal and gluteal muscles (7). This arrangement enhances an anterior pelvic tilt, increases lumbar lordosis, and shortens the hip flexors. The thoracolumbar erector spinae and hamstrings compensate by increasing activity, which creates hip extension.

The syndrome continues with gluteus medius weakness, which causes compensatory increased activity in the ipsilateral iliotibial band, tensor fasciae latae, and quadratus lumborum. The abdominal muscles display weakness with increased iliopsoas muscle activity, which increases trunk flexion. Sherrington's law of reciprocal innervation has been proposed to explain this muscle inhibition. This type of muscle imbalance is self-sustaining without appropriate therapeutic intervention (8). When the inhibited muscle is exercised to create a maximal contraction, the electromyographic activity of the muscle actually decreases. No matter how specific the method of strengthening is, the muscle imbalance pattern is only reinforced unless the hypertonic/shortened muscle is first stretched (8).

MANUAL MEDICINE APPROACH

Principles in Gymnastics

Effective manipulations on gymnasts are a customized combination of techniques. The clinician should prepare his or her manipulations as a master gourmet chef would prepare a special meal for his guests. The clinician should combine the manual medicine techniques together in just the right order to best fit the overall condition of the athlete.

In general, it is recommended that the clinician perform articulatory and joint play techniques as described by Mennell for the examination and treatment of joint dysfunction in the extremities of the gymnast (9). By ensuring that the joints of the extremities are functioning at their highest capacity, the clinician may add

improved quality of movement for the athlete. *Hypermobile joints in gymnasts should not be treated with articulatory, high-velocity, low-amplitude (HVLA) techniques mainly because they attempt stability with hyperlaxity.*

Regions

Two important body regions to focus on in gymnasts are the foot/ankle and the hand/wrist. The foot has chronic dysfunctions in the first metatarsophalangeal joint, cuboid, and tarsal navicular, while the ankle typically requires treatment in the subtalar joint and the proximal tibiofibular joint. For the wrist, close evaluation of wrist flexion-extension and supination-pronation motions of the forearm is recommended. The scapulothoracic articulation must be evaluated in conjunction with the foot and wrist, following along the kinetic chain principles.

Caution is warranted at the glenohumeral and scapholunate articulations, since these areas tend to be hypermobile. Research is needed to accurately detail the common areas of hyper- or hypomobility in the extremities of the gymnast.

Assessment and Application

The clinician who treats a gymnast during a competition needs to ascertain if the injury is acute or chronic, stable or unstable. It is not uncommon for a gymnast to arrive at a competition with a significant sprain, strain, or fracture for which she has not received any prior medical evaluation or treatment. The gymnast is generally a strong athlete with a very high pain threshold, and her gym performance is not always an accurate indicator of injury severity. An extreme case occurred at a junior Olympics national championship, when an adolescent gymnast sustained a fall onto her head 2 weeks before the competition. She continued to train and came to the meet hoping for manipulative treatment and clearance to compete. Her symptoms were severe enough that she did not compete and radiographs determined that she had a fracture-dislocation in her cervical spine requiring surgical stabilization.

Because of this loose correlation between injury and perceived pain, *articulatory or HVLA techniques should be avoided in acute injury* unless proper imaging studies are available. Attempting to mobilize an unstable hypermobile segment of the body is also contraindicated. A rigid, spasmodic area should be approached cautiously because it usually involves the athlete's splinting a fractured or strained area.

Length of treatment time also plays a factor in the selected techniques. During a competition, the gymnast competes and then rotates to the next event. In some cases the physician may have only 5 to 10 minutes to treat the gymnast between events. He or she may have only a chair and the floor to use as surfaces for treatment. The treatment may also be taking place in front of an audience and the media. Furthermore, the psychological makeup of the female gymnast needs to be understood. Is a doctor-patient relationship already established with the gymnast? Has the gymnast ever been manipulated before? Is she apprehensive to have someone treat her in a hands-on method? Thus, your selection of manipulative treatments must not only be appropriate for the injury, but also for the treatment setting and the psychological makeup of the athlete.

Myofascial Techniques

Soft tissue, articulatory, functional, muscle energy, myofascial release (MFR), HVLA, and craniosacral techniques are all appropriate treatments for the gymnast. These techniques should be used with the female gymnast as the clinician feels appropriate. The purpose of this chapter is to enlighten the reader on techniques employed frequently on the female artistic gymnast that may vary from typical treatments.

The obvious difference between gymnasts and other athletes is the need for extreme flexibility of the body while maintaining exceptional strength, power, and kinesthetic awareness. Manual medicine techniques are meant to maximize the gymnast's range of motion, restore normal physiologic function, and yet not enhance pathologic ligamentous instability.

The pelvis is the center of the body's link between the trunk, the upper extremity, and the lower extremity. It acts as an anchor for many vital tendons, ligaments, and fascial attach-

ments. Treatments to the pelvic girdle and its attachments can significantly benefit the gymnast by increasing mobility of the trunk, the lower extremity, and even the upper extremity without enhancing pathologic hypermobility. The pelvis and its attachments are used as the principal anatomic site treated in this chapter.

Myofascial Release–Combination Technique

Rationale: This is a combination of sustained longitudinal pressure MFR with longitudinal soft tissue stroking and digital ischemic pressure coupled with oscillating vibrations. This allows the connective tissue to elongate without exaggerating any ligamentous instability. Myofascial release spans the spectrum of manual medicine procedures and combines many of the principles of soft tissue, muscle energy, functional, and craniosacral techniques. MFR-combo techniques can be applied to the iliotibial band, paraspinal iliopsoas, ilioinguinal, and hamstring muscles, which are common sites of dysfunction in gymnasts.

Basic Technique

1. The treatment region is prepared with a small amount of massage lotion to prevent skin irritation.
2. The clinician applies perpendicular pressure to the contour of the area to be treated with his or her forearm. The amount of pressure is in general light to firm, about 10 to 15 lb. Do not push hard enough to bruise the tissue or cause undue pain.
3. The general path to follow is from distal to proximal; the speed of movement is about 1 in. per second.
4. The clinician repeats the process three to five times or until he or she feels the tissue tension resolve. If tender/trigger points or fascial restrictions are encountered, stop and apply mote direct, longitudinally oriented pressure to the area with the elbow.
5. In addition, the clinician can rotate his or her forearm briskly (supinate and pronate) to send an oscillating vibratory force into the area, which helps to release the area more quickly and with less discomfort than with direct pressure alone. This type of myofascial release variation can be performed with the hand, forearm, or elbow. With this technique, deep slow breathing is an effective myofascial enhancer.

Variation: Gluteal Myofascial Release Technique

1. The gymnast lies prone on the treatment table.
2. Using the MFR-combo technique, the gluteal region is prepared with a small amount of massage lotion, and the clinician applies perpendicular pressure to the contour of the gluteal muscle with the forearm (Fig. 32.1).
3. The clinician starts at the thigh-gluteal junction and progresses through the entire gluteal region, ensuring that all of the hip internal and external rotators are completely treated. The process is repeated three to five times or until he or she feels the tissue tension resolve.

The gluteus medius is the most common muscle with symptomatic tender points in the gymnast, far more than the piriformis. Travell considers the gluteus medius to be the lumbago muscle because it is so intimately involved with low back pain (11).

Tender Point Release for Gluteus Medius

Rationale: This technique uses ischemic digital pressure combined with a variation of strain–

FIGURE 32.1. Gluteal myofascial release technique.

counterstrain and myofascial release. The technique was developed to meet the specific needs of the gymnast. Travell's gluteus medius trigger points correlate closely to Jones's points at the lower pole of the fifth lumbar vertebra, high ilium–sacroiliac joint, and third and fourth lumbar vertebrae (11,12). On occasion, the hip is extended as described by Jones if the foldover alone does not relieve the pain completely. Releasing the sacrotuberous ligament is a vital part of treating the gymnast discussed later in the chapter. Rarely, trigger point injections are needed (11).

1. The gymnast is prone with the clinician sitting caudad to the region being treated.
2. The tender point is located, ischemic digital pressure is applied, then the clinician folds the gluteal muscle bulk over the tender point in a cephalad-lateral rotation-type motion. This foldover should help resolve or greatly reduce pain from the digital pressure (Fig. 32.2).
3. The position is held along with the digital pressure for 30 to 90 seconds or until the clinician feels the tissue release. Jones recommends removing the digital pressure while holding the counterstrain position; however, with this type of treatment the digital ischemic pressure is maintained.
4. The gymnast should be instructed to take deep, slow breaths to assist with the release.
5. The digital pressure over the tender point and the foldover of the gluteal muscle should be returned toward neutral slowly so as not to precipitate the dysfunction from returning, as Jones recommends.

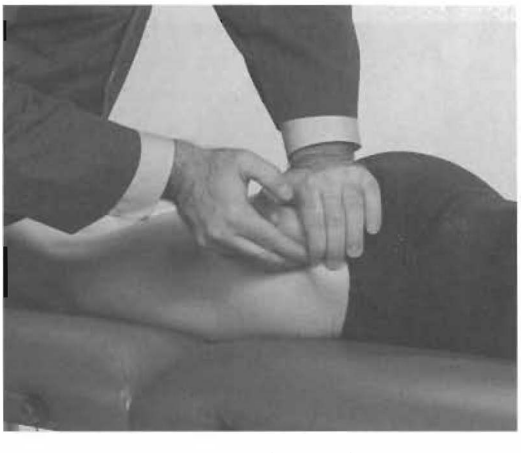


FIGURE 32.2. Gluteus medius tender point release.

INJURY ASSESSMENT

Optimal function of the pelvis is vital to allow for the extreme range of motion, power, and balance needed to perform artistic gymnastics. The pelvis is the major connection between the upper and lower extremities; therefore, the pelvis should be evaluated for dysfunction with a large variety of injuries, such as ankle, shin, quadriceps, hip flexor, hip adductor, iliotibial band, gluteal region, pelvic bones, abdominal musculature, spine, and shoulder complex.

Sacrotuberous Ligament

The sacrotuberous ligament forms a functional bridge between the ankle and the shoulder, extending from the ischial tuberosity to the coccyx, and fans out toward the posterior sacroiliac joint capsule and the posterior superior iliac spine. The proximal tendon of the long head of the biceps femoris frequently attaches to the sacrotuberous ligament, functionally connecting it to the ankle (14). The biceps femoris tendon distally attaches to the fibula and the investing fascia of the peroneus longus.

In addition, the gluteus maximus, piriformis, multifidus, and thoracolumbar fascia all have direct attachments to the sacrotuberous ligament (15). The sacrotuberous ligament is directly connected to the upper extremity and upper trunk by the following two pathways:

1. The erector spinae aponeurosis and the iliocostalis thoracis.
2. The gluteus maximus is connected to the lumbodorsal fascia and linked to the contralateral latissimus dorsi (10).

The peroneus longus muscle provides up to 18% of the stability of the sacrotuberous ligament through this kinetic chain (16). Since the peroneus longus provides lateral ankle stability, the forces that the gymnast endures through the ankles are empirically determined to affect the sacrotuberous ligament.

The sacrotuberous ligament may refer pain posteriorly down the gluteus muscle group, hamstring, calf, and lateral foot and all five toes. Sciatica is often due to referred pain from the sacrotuberous ligament as opposed to lumbar nerve root pathology (17). It is critical to perform a thorough physical examination to be confident in diagnosing the etiology of a radiculopathy for ptoper treatment.

Physical Examination

The sacrotuberous ligament is assessed (Fig. 32.3) with the gymnast in the prone position (10). The clinician's first and second finger web space is placed along the contour of the inferior gluteal folds with the thumbs at the inferior aspect of the ischial tuberosity. The clinician then reaches with the thumbs into the ischial rectal fossa area to position them inferior to the sacrotuberous ligament. The thumbs move further in a cephalic posterolateral motion. As the thumbs perform this hooking action, tension and tenderness of the sacrotuberous ligaments are assessed. Asymmetrical tension and tenderness are significant pathologic findings. Balance to this ligament complex needs to be restored.

Another way to palpate the sacrotuberous ligament is to start at the ischial tuberosity and proceed along the ramus of the ischium to the posterior margin of the pubic symphysis. This is the portion of the sacrotuberous ligament that has been called the falciform ligament, which follows the course of the ischiocavernosus muscle. In some cases, rectal examination may be necessary (18).

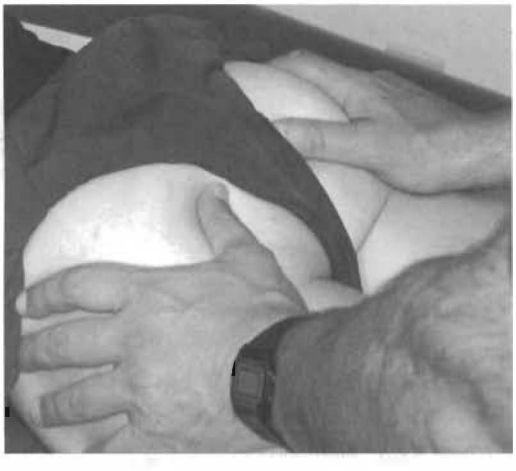


FIGURE 32.3. Sacrotuberous ligament assessment.

Hip Flexion/Adduction Test. First described by Bachrach, this test stresses the sacroiliac joint, and the iliolumbar and sacrotuberous ligaments (19).

1. The gymnast is supine as the examiner fully flexes the hip.
2. The examiner then adducts the hip and applies compression along the long axis of the femur (Fig. 32.4). The hip can be flexed at 90 degrees, 65 degrees, or 45 degrees to elicit a response.

Positive test: Pain at the sacroiliac joint on the tested side.

Indicates: Sacroiliac dysfunction or injury, sacrotuberous ligament injury.

Pain that is elicited in the anterior hip with the performance of this same test can often indicate anterior medial hip impingement, which is a result of iliopsoas tendinitis, iliacus bursitis, or restriction in the ilioinguinal ligament. Avoidance of hip adduction with this test is described in the sacroiliac posterior shear/thigh thrust test due to the anterior hip pain that may be present with the addition of hip adduction (20).

Maitland's Slump Test. Maitland developed this test to enable the clinician to detect adverse nerve root tension (21), although research offers only minimal support. Turl and George studied the slump test in relationship to repetitive grade I hamstring strains and determined that adverse neural tension is an important clinical feature of hamstring strains; however, the exact relationship between neural tension and hamstring strains requires further research (22). The slump position lengthens the vertebral canal, which strerches the spinal cord, lumbosacral nerve root sleeves, and nerve roots. This position also stresses the sacrotuberous ligament due to its connections with the erector spinae, lumbar intermuscular aponeurosis, and biceps femoris. Therefore, the slump test is

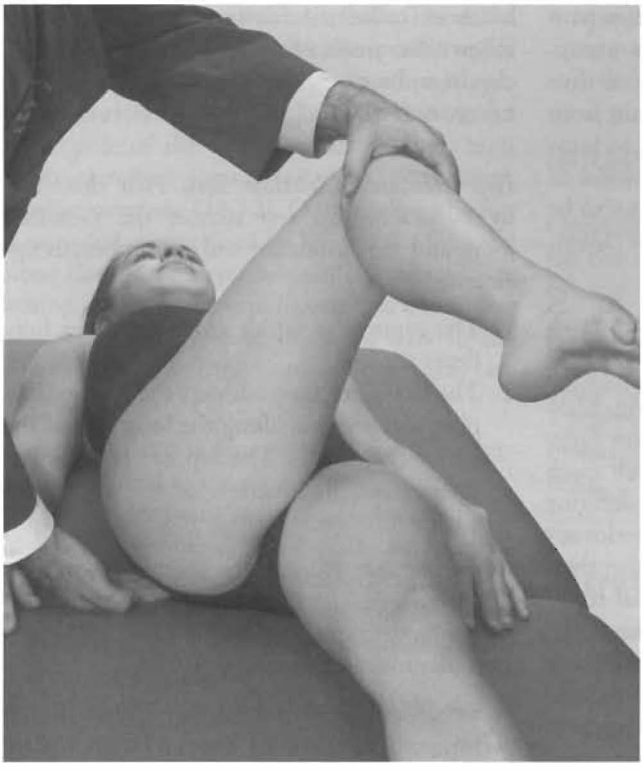


FIGURE 32.4. Hip flexion-adduction/ sacroiliac joint test.

more than a neural tension test; it is also a myofascial tension test. This brings together all of the above-stated information on the sacrotuberous ligament into one clinical problem, as detailed subsequently.

1. The gymnast is seated with her hands resting together just behind her back.
2. The gymnast slumps her trunk, rounds her shoulders forward, and flexes her neck so her chin rests on her chest.
3. The examiner applies gentle pressure to the upper trunk and neck to increase this slumped posture.
4. The gymnast then extends one knee and dorsiflexes the ankle.
5. The examiner applies pressure to the foot to increase the dorsiflexion (Fig. 32.5).
6. If the test is positive, the examiner then has the gymnast extend the neck; the symptoms should reduce or be eliminated if the test is positive. Plantarflexion of the ankle reduces symptoms as well (23).

FIGURE 32.5. Maitland's slump test.

Positive test: Reproduction of symptoms or hamstring pain in the low back and buttocks. Pain located in the popliteal fossa should not be considered a positive finding since this may be due only to tight hamstrings.

Indicates: Nerve root impingement or irritation, chronic hamstring strain, sacrotuberous ligament dysfunction.

Sacrotuberous Modification of Maitland's Test

Rationale: To isolate the sacrotuberous ligament in a positive slump test.

1. Immediately repeat the slump test with the gymnast sitting and the examiner's palm under the ischial tuberosity.
2. The examiner applies pressure with the fingers cephalad into the ischiorectal fossa (explain this maneuver to the gymnast before performing it).
3. As the examiner's fingers reach past the ischial tuberosity, apply lateral pressure into the obturator foramen (Fig. 32.6). When this pressure is applied, the symptoms from the slump test are either greatly reduced or eliminated.

Positive test: If the sacrotuberous ligament is involved, symptoms from the slump test will reduce or resolve. If there is no significant relief, then the sacrotuberous ligament may not be involved.

FIGURE 32.6. Sacrotuberous ligament isolation and counterpressure with Maitland's slump test.

Sacroiliac Joint and Gluteal Region

Gymnasts frequently land on their buttocks with great force, usually unintentionally. This places stress on the coccyx, pelvic diaphragm, and sacrotuberous and other sacroiliac ligaments. The ischial tuberosities bear enough weight in the sitting position to force the ilia apart, thereby placing increased strain on the sacroiliac joint capsule (24). These repetitive hard landings on the buttocks lead to significant dysfunction in the pelvis. If the pelvic diaphragm, coccyx, and sacroiliac ligaments are not examined, the gymnast may have persistent or recurrent symptoms.

This area of the body in these young females is often not fully examined due to its proximity to the genitalia and buttocks. This is referred to as the "no-fly zone" because of the many cultural stigmas in touching this area. However, it is only with thorough examination of the pelvic structures of the gymnast that dysfunction is found and proper treatment can be applied to resolve the symptoms. Take special measures to explain any examinations and techniques applied in this region, including appropriate draping, presence of a chaperone or another clinician, and warning in advance what you are planning to do.

One of the most useful treatments for the sacrotuberous ligament is a variation of Greenman's hand placement in the evaluation of the sacrotuberous ligament and his treatment of the pelvic diaphragm. Several treatment variations for the sacrotuberous ligament can be applied based on the physical findings discovered during the evaluation. Occasionally, all of the techniques are needed to help the gymnast. When the gymnast has sustained a hamstring strain, the area of the posterior superior iliac spine (PSIS) and the sacroiliac joint may be tender to palpate. If tenderness is present, monitor this area while treating the sacrotuberous ligament.

Sacrotuberous Ligament Release with Sacroiliac Joint Monitoring (Treatment Variation 1)

1. The gymnast lies prone with her affected leg abducted 30 degrees.

2. The clinician stands next to the gymnast's affected side.
3. The clinician places one hand with the fingers extended along the side of the affected ischial tuberosity.
4. The clinician's hand is then directed cephalad along the ischium until the obturator foramen is felt, then he or she applies a posterior-medial force to hook underneath the sacrotuberous ligament while releasing the pelvic diaphragm at the same time. This area is usually quite tender if pathology is present. The gymnast is instructed to take deep, slow breaths.
5. While the clinician is performing this procedure with one hand, the opposite hand monitors the PSIS and sacroiliac joint with direct pressure (Fig. 32.7).
6. Reassess after three to five deep, slow breaths. If tenderness over the sacroiliac joint/PSIS and sacrotuberous ligament treatment areas remains, inner hand placement should be altered.

Note: The main cephalad pressure can be focused with the hand placed anywhere in the pelvic floor muscles between the coccyx and the pubis. The location of the hand placement is dictated by the fascial restrictions found and the ability to relieve the pain under the sacroiliac joint monitoring hand.

Sacrotuberous Ligament Release with Gluteus Medius Monitoring (Treatment Variation 2). If tender points are found in the gluteus medius, then these points should be monitored with digital pressure while treating the sacrotuberous ligament, as described above.

Sacrotuberous Ligament Counterpressure with Straight Leg Raise (Treatment Variation 3). After treating the sacrotuberous ligament as described in Variations 1 and 2, reassess the gymnast's hamstring flexibility with the supine straight leg hip flexion test. If there is still discomfort and restriction, then retreat the area, but with the following variation:

1. The gymnast lies supine and the affected leg is kept straight while the clinician flexes the hip passively until discomfort is felt.
2. The clinician then slightly decreases the hip flexion and applies pressure with his or her extended fingers as described in Variation 1 (Fig. 32.8).
3. The clinician then further flexes the hip. There should be an immediate increase in pain-free range of motion. If the range of motion is not increased, then the clinician's hand should be repositioned, as described previously.
4. The new increased hip flexion position is held for 20 to 30 seconds before the hip is allowed to return toward neutral. The gymnast should breathe deeply and slowly while the stretch is maintained.

FIGURE 32.7. Sacrotuberous ligament counterpressure with straight leg raise.

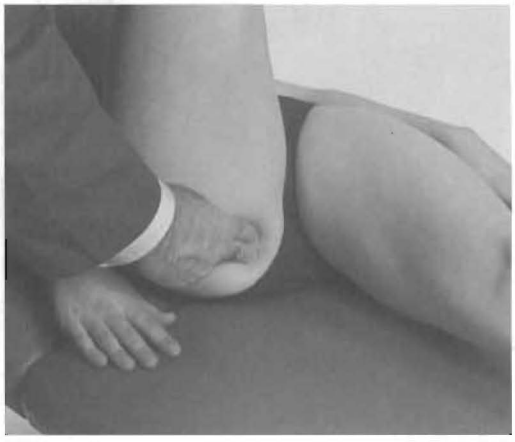


FIGURE 32.8. Triplane stretch with self–sacrotuberous ligament counterpressure.

Similar to the slump test with sacrotuberous ligament counterpressure (Fig. 32.6), this is another example of how treatment of the sacrotuberous ligament increases the injured gymnast's hamstring flexibility by providing improved pelvic function.

If this treatment is beneficial, then the gymnast may be taught to self-treat this area with a home exercise (Fig. 32.9).

Triplane Stretch with Self-Applied Counterpressure to the Sacrotuberous Ligament

1. The gymnast lies in the supine position and performs a straight leg raise to stretch her hamstring. If the gymnast cannot reach her own leg, then a towel may be used to assist with the stretch. She should use the contralateral arm for this assistance.
2. Once the leg is raised to the point of discomfort, the gymnast then reaches into the affected side's ischial rectal fossa with the ipsilateral hand (Fig. 32.9, showing the pressure point where the gymnast applies the counterpressure). The gymnast applies the same pressure that the clinician performed (see above exercises), holding this pressure while taking three to five slow, deep breaths.
3. The gymnast then pulls the leg further into hip flexion. This process is repeated until no further gain in pain-free motion is achieved.
4. Repeat this self-treatment three to four times a day.

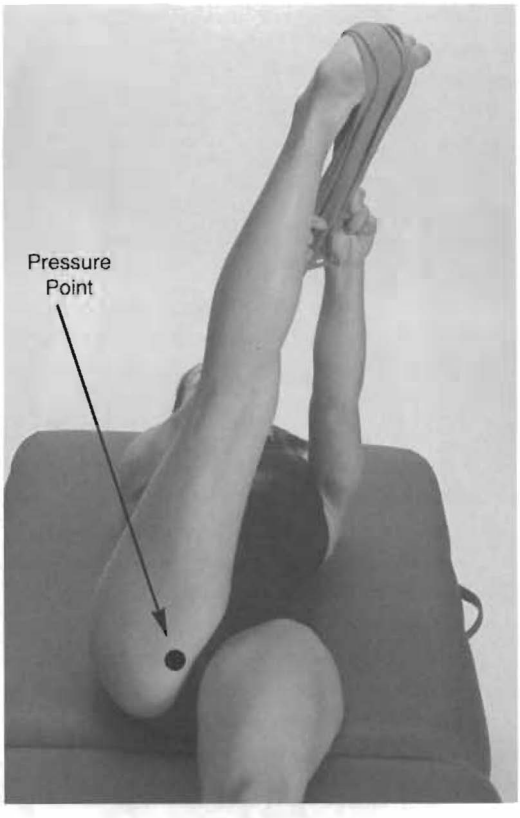


FIGURE 32.9. Triplane stretch with self–sacrotuberous ligament counterpressure.

Sacrotuberous Ligament Release, Standing (Treatment Variation 4). After treatment in the prone and supine positions, the gymnast may have pain standing even though she has significantly improved while lying on the treatment table. To assess if the treatment was successful, ask the gymnast to stand and then pike down at the trunk, keeping the knees straight, and touch her hands to the floor. If she has discomfort with this activity and if the range of motion is not significantly improved, then perform a standing sacrotuberous ligament treatment.

1. The gymnast stands with legs hip width apart, while the clinician sits along the side of the gymnast (Fig. 32.10).
2. The clinician palpates in the ischial rectal fossa, as in the above-mentioned manipulations. The gymnast may experience tenderness in this area once she is weight bearing even though it had resolved while on the treatment table.
3. While maintaining this pressure with one hand, the clinician then grasps the iliac crest with the other hand anteriorly. The clinician compresses the iliac crests together between his or her hand and chest area, bringing both anterior superior iliac spines closer toward midline.
4. The gymnast is then asked to repeat the trunk flexion. The gymnast may have pain in the ischiorectal fossa, but the painful restriction should be reduced or resolved.
5. The gymnast returns to the upright posture and the clinician maintains the anterior com-

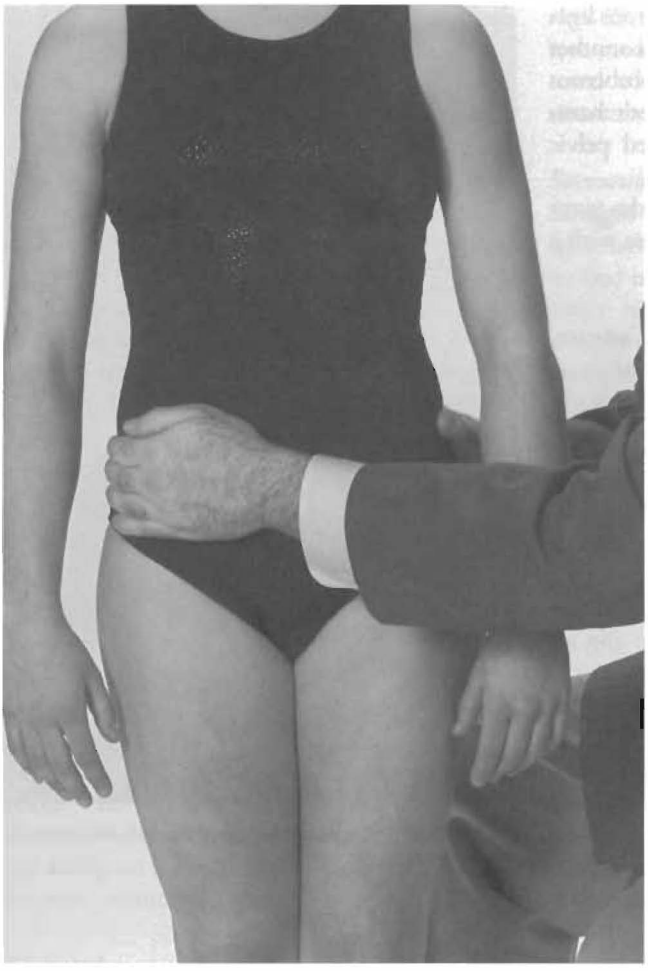


FIGURE 32.10. Standing sacrotuberous ligament release, anterior view.

pression and posterior pressure. The gymnast takes three to five deep, slow breaths.

6. The clinician removes all pressure and has the gymnast bend toward the floor again. The discomfort in the ischiorectal fossa should reduce or resolve, and the clinician should feel the pelvis return to a normal balance. The motion should now be increased with less or no discomfort. If some discomfort is still present, the procedure may be repeated.

In addition to the sacrotuberous ligament, the iliolumbar ligament, sacrospinous ligament, and posterior sacroiliac ligament should also be fully evaluated when examining the gymnast. The focus has been on the sacrotuberous ligament because of its intimate relationship with the biceps femoris and the need for extreme range of hip flexion.

Hamstrings

Gymnasts frequently strain their hamstring muscles because of the need for extreme range of hip flexion. The majority of strains heal without incident. However, if the ischial tuberosity is damaged in the process of the hamstring strain, the recovery from the injury becomes far more complex and prolonged. The previously mentioned anatomic relationships of the sacrotuberous ligament can be practically applied in hamstring injury. Few proximal hamstring strains at the ischium do not involve the sacrotuberous ligament. Treatment of

somatic dysfunctions associated with the sacrotuberous ligament has greatly improved the gymnast's hip flexion range of motion (at least 30 degrees with the first treatment) and thus accelerated her return-to-sport activity.

The examination of the gymnast with an injured hamstring should also include a thorough examination of the entire spine, pelvis, and gait. The gymnast may present with upper hamstring discomfort and tightness, buttocks or "tailbone" pain, or low back pain. If significant tenderness is present over the ischial tuberosity, imaging studies should be done to rule out an avulsion or bony edema. If an avulsion fracture is present, the gymnast should be placed on crutches until she can bear weight completely pain-free with a normal gait. This may take 1 to 3 weeks depending on the severity of the fracture. As with any other fracture, the site should be healed before performing manipulations that may adversely affect the anatomy.

Treatment. If the modified slump test (Fig. 32.6) demonstrates a hamstring injury, three to four treatments using the following approach can restore complete preinjury pain-free range of motion and function to as much as 30 degrees.

1. MFR-combo technique (see under Myofascial Techniques, earlier in the chapter) from the calf/peroneals to the upper thoracic spine. This includes the peroneals, gastrocnemius, hamstrings, iliotibial band, tensor fasciae latae, gluteal muscle group, lumbar and thoracic paraspinal muscles, and the latissimus dorsi.
2. Treat any spinal or pelvic malalignments with muscle energy and/or HVLA techniques.
3. Treat the sacrotuberous ligament.
4. The slump test is then repeated and the symptoms should be greatly reduced or eliminated.

REHABILITATION TIPS

The gymnast needs to be placed on a specific exercise program individualized for her problem after treatment with the techniques presented earlier. Manual medicine treatment failure may be related to lack of appropriate rehabilitation exercises. In general, follow these tips:

1. If a muscle imbalance is present in which tight muscles are inhibiting the strength of other muscles, the gymnast should be instructed to perform stretches first, before doing any core strength training/spinal stabilization exercises (8,10). This relates to the "pelvic crossed syndrome" described earlier in this chapter (7). The tight postural muscle groups are activated first in this muscle imbalance, but the weak/inhibited muscles are not strengthened (8). Spinal stabilization exercises can actually *reinforce the muscle imbalance pattern* and cause increased symptoms. Fortunately, by stretching the tight muscles, and restoring their normal tone and length, the inhibited muscles are returned to their normal function. Spine rehabilitation should consist first of flexibility exercises, then strengthening exercises.
2. Because gymnasts need excellent control of their spine and pelvis, rehabilitation should begin with mat spinal stabilization exercises. After learning basic control, the gymnast can move to advanced Swiss ball type of exercises or advanced Pilates exercises may be instituted.
3. Gymnasts should perform rehabilitation exercises not only with the trunk in neutral and flexion, but also in extension. Many rehabilitation programs neglect this aspect. A progression from basic to advanced exercises incorporating all positions of the trunk is needed to properly prepare the gymnast to return to her sport.
4. The extremities need to be addressed. Open- and closed-chain exercises should be incorporated for both the upper and lower extremity, as well as static and dynamic proprioceptive exercises. Rhythmic stabilization exercises for the upper extremity are usually of great benefit for the gymnast.

STRETCHES FOR GYMNASTS

Static stretches should be done at the end of practice, but dynamic stretches may be done before and during practice. McNeil has documented that stretching for 30 seconds with three different static stretches that are repeated twice in a circuit fashion significantly reduces

the power production of female gymnasts for approximately 1 hour (25), due to reducing the muscle activation and inhibiting the contractility at the cellular level (26).

Triplane Lower Extremity Fascial Stretches

Single-plane stretches are helpful (Fig. 32.11), but for optimal flexibility and function the extremity should be stretched in all three planes. Figure 32.12 shows a triplane stretch of the lower extremity that incorporates hip flexion, hip adduction, hip internal rotation, and ankle dorsiflexion. To add a stretch to the lateral leg, the same stretch is performed with the ankle inverted. Figure 32.13 incorporates hip flexion, hip adduction, hip external rotation, and ankle dorsiflexion. These same stretches can be performed with the use of a towel for the gymnast to do the stretches herself (Fig. 32.14).

FIGURE 32.12. Triplane straight leg raise stretch with hip adduction, hip flexion, hip internal rotation, ankle inversion, and ankle dorsiflexion.

Dynamic Stretches

Dynamic stretches are done after the gymnast has completed a warm-up. These stretches can

FIGURE 32.11. Single-plane straight leg raise stretch.

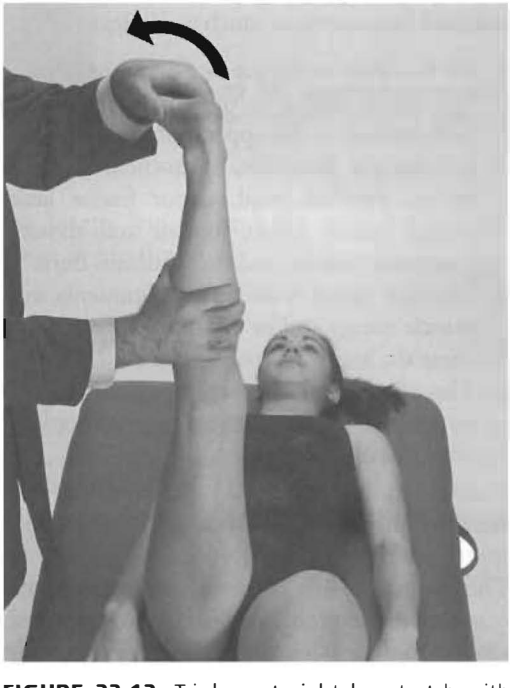


FIGURE 32.13. Triplane straight leg stretch with hip external rotation.

FIGURE 32.14. Self triplane stretch with use of a towel.

FIGURE 32.15. Dynamic passive straight leg stretch of the lower extremity.

be done with a partner or with rubber tubing. As part of a rehabilitation program for the hamstrings, I encourage the use of dynamic stretches after the MFR-combo treatment has been completed, especially when the treatment is performed during practice or competition to prepare the muscles for gymnastic activity.

Partner dynamic passive stretches are performed first by finding the available range of motion of the area to be stretched. For example, the partner performs a passive straight leg raise on the gymnast to find the end point of motion. If the gymnast is recovering from an injury, the area should be stretched in a pain-free range of motion. Once the end range is found, then the area is rapidly stretched through the available pain-free range of motion. The stretch is stopped at least 20 to 30 degrees before the end of the predetermined range. The velocity of the arc of motion performed starts off slow then increases to the fastest that the partner can safely move the extremity through the pain-free arc.

For the straight leg raise, add a quick dorsiflexion of the ankle at the end range of hip flexion (Fig. 32.15). This simulates landing on the floor in a piked position. This rapid, dynamic stretch is continued for 20 to 30 seconds followed immediately by a static stretch at the end range of motion. If the dynamic stretch is successful, there should be an increase in pain-free range of motion.

Sands and McNeal have shown that a gymnast can use a black Theraband to enhance functional active hip range of motion (27). Using 5.5 ft of a black Theraband with each end tied firmly (so it does not slide) to the ankles of the gymnast, she is instructed to perform five skills: forward kicks, side kicks, back kicks, straddle jumps, and split leaps. Three sets of five repetitions are performed and increased over time to three sets of 15 repetitions. The gymnast should kick, jump, and leap as high as possible while maintaining good form and alignment (Fig. 32.16). The strength of the Theraband is determined based on the strength of the gymnast.

FIGURE 32.16. Dynamic active stretch with black Theraband.

FIGURE 32.18. Crossed-leg pike down stretch.

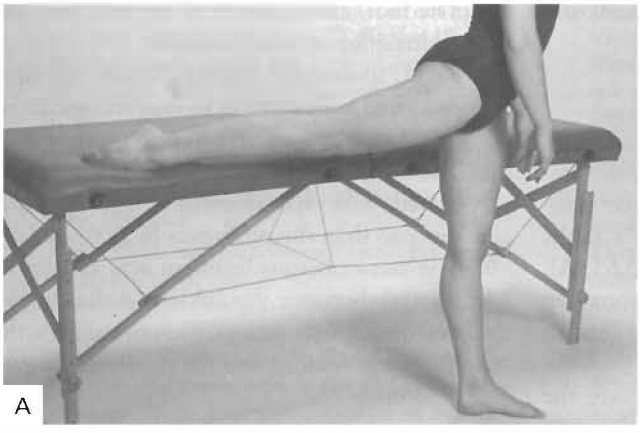
Self Iliotibial Band Stretch Combined with Quadriceps Stretch

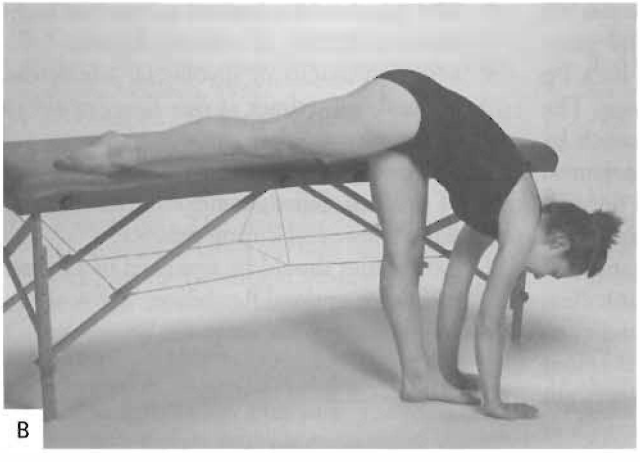
1. The gymnast lies on the unaffected side and fully flexes the affected knee with her ipsilateral hand.
2. The affected hip is then adducted toward the table using assistance from her unaffected ankle (Fig. 32.16).
3. The stretch is held for 20 to 30 seconds and repeated three to five times.

Crossed-leg Pike Down Hamstring, Iliotibial Band, Peroneal Stretch

1. The gymnast sits in a piked position and crosses the unaffected leg over the affected leg.
2. The affected leg is adducted, keeping the knee straight.
3. The ankle is inverted and dorsiflexed while the gymnast pikes down (trunk flexion) toward the feet. A towel is used to enhance the dorsiflexion/inversion of the ankle (Fig. 32.17).

FIGURE 32.17. Self iliotibial band stretch combined with quadriceps stretch.





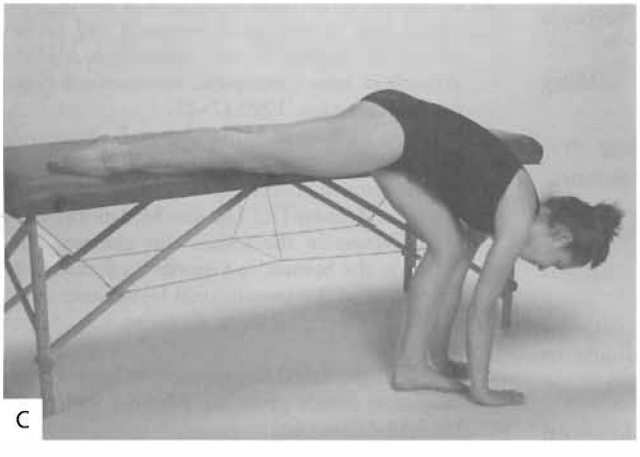


FIGURE 32.19. A, Closed-chain standing pike down hamstring and hip flexor stretch. **B,** Closed-chain standing pike down stretch. **C,** Closed-chain standing pike down stretch.

4. The stretch is held for 20 to 30 seconds and repeated three to five times.

Closed-chain Standing Pike Down Hamstring and Hip Flexor Stretch

1. The gymnast stands in an arabesque position with the back leg resting on a table, spotting block, or ballet barré (Fig. 32.19A).
2. The gymnast tries to push the support leg into the floor to engage the gluteus medius and support the pelvis.
3. The gymnast then attempts to square the hips as much as possible by pushing the anterior superior iliac spine (ASIS) of the back leg forward. This is an attempt to have the left and right ASIS aligned in the frontal plane while stretching the hip flexor of the back leg to 100 to 110 degrees of hip extension. The gymnast may assist this hip flexor stretch by placing her ipsilateral hand on the gluteal muscle posterior to the hip joint and pressing anteriorly to encourage a square hip alignment. The position is held for 20 seconds.
4. Next the gymnast pikes down (trunk flexion) in a slow, controlled manner and then supports herself with her hands on the floor (Fig. 32.19B), keeping both legs straight and creating a closed kinetic chain stretch of the hamstring, and holds for 20 seconds.
5. Finally, she flexes the support knee 30 to 40 degrees and holds for 20 seconds (Fig. 32.19C).
6. The gymnast straightens her knee, holding for 20 seconds.
7. The gymnast returns to the standing arabesque position in a slow, controlled manner.

SUMMARY

Key points in using manipulative medicine in gymnasts:

1. Avoid articulatory or HVLA techniques on gymnasts if hypermobility is noted.
2. Avoid articulatory or HVLA techniques in acute injury unless proper imaging is readily available.
3. The foot, ankle, and wrist are key areas to evaluate for manual medicine.
4. Rigid spasmodic regions should be approached with caution in case the spasm is splinting a more severe injury.
5. The gluteus medius is most commonly involved with symptomatic trigger points, more so than the piriformis in gymnasts.
6. The sacrotuberous ligament acts as a joining link between the ankle and the shoulder through its intimate relationship with the biceps femoris and the peroneus longus tendons.
7. Releasing the sacrotuberous ligament is key to restoring extreme ranges of hip flexion.
8. The myofascial release–combination technique is effective in multiple regions.
9. Many variations of myofascial release can be used, sometimes at one time, to aid in treatment.
10. Static stretches should be done at the end of practice, but dynamic stretches may be done before and during practice.
11. Extremities should be stretched in all three planes for optimal flexibility.

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33

ICE HOCKEY

JENNIFER GILMORE
STEVEN J. KARAGEANES

Ice hockey is a unique sport that combines speed, physical contact, skill, and dexterity on an unstable surface to create exciting competition that rivals few others. This combination also makes for many high-risk situations for injury. The top hockey players may have a higher level of multiple skills than in any other sport.

Most historians place the roots of hockey in the colder northern European regions, specifically Great Britain and France. When winter froze the numerous lakes, many forms of the sport were played on the ice. Various historical accounts of the nineteenth century describe versions of hockey played throughout various parts of Great Britain.

Modern hockey grew primarily out of Canada, and the first organized league was said to have been launched in Kingston, Ontario, in 1885, consisting of four teams. A famous portion of modern hockey tradition started in 1892, when the English Governor General of Canada, Lord Stanley of Preston, bought a silver bowl with an interior gold finish and decreed that it be given each year to the best amateur team in Canada. Over a century later, that trophy, the Stanley Cup, is awarded today to the franchise that wins the National Hockey League playoffs.

Professional hockey in the United States started in 1904 in Michigan's Upper Peninsula. After several leagues started and folded before and around World War I, the National Hockey League was formed in 1917 and took control of Lord Stanley's cup. Ever since then, the sport has continued to grow in popularity at all levels. Over a half million men and boys compete in the United States and Canada yearly (1), while over 43,000 Canadian women played in the 1999–2000 season alone (2).

THE GAME

Ice hockey is played on an enclosed sheet of ice approximately 200 ft by 85 ft, although international rinks are larger. The ice sheet is enclosed by boards, which are permanent in nature. Teams are made up of 20 players. Six players are on the ice at one time. Five of these players are referred to as skaters and the sixth is a goaltender. The goaltender defends a 4-ft by 6-ft goal. The players use sticks to pass, shoot, and carry a small vulcanized rubber disk, referred to as a puck, around the ice and try to put it past the opposing team's goaltender into the net. There is physical contact—body to body, stick to body, and puck to body. Players are usually on the ice for shifts of 40 to 60 seconds. The game is played at a very high speed and involves quick decisions by all five of the skaters. The goaltending position is unique to the game, and the goaltender relies greatly on quickness and positioning to keep the puck out of the net. An official hockey game consists of three 20-minute periods making for a 60-minute game. High school hockey runs for 15-minute periods, while youth hockey runs 12 to 15 minutes per period.

Competition Levels

Ice hockey is played internationally at the Olympic level, and professional leagues are worldwide, primarily in Canada, Europe, and the United States. The elite league in the world is the National Hockey League, perhaps the most diverse professional league with respect to the number and proportions of countries represented. The National Collegiate Athletic Association (NCAA) oversees college hockey, and USA Hockey administers the sport at the youth level.

Players begin playing as early as age 4, and there is recreational hockey up to the age of 70. Historically a men's game, the game now has women's collegiate and Olympic competition worldwide.

APPROACH TO THE ATHLETE

Required Skills

A hockey player must possess the ability to skate, handle the puck, shoot the puck, and pass the puck. As in any sport, ice hockey demands knowledge of the game, which requires players to make very quick read and react decisions at high speed to perform the necessary skills to be successful in the game. Players who succeed at hockey possess great speed, great mental awareness of where players on their team and the opposing team are on the ice, and the knowledge of how to maneuver themselves and the puck around the opposition to score goals.

Physically, the elite hockey player requires exceptional skating skills. Speed, power, and balance are all aspects that are crucial to everyone on the ice. A goalrender may require somewhat less power than position players, but better reflexes, flexibility, and balance. Many times the goaltender is the team's best skater.

Stick handling is an acquired skill in which a player carries the puck on the blade of the stick, controlling its movement so as to advance it without opposing players taking it away. This skill has to be executed while watching the playing surface for teammates and opponents and while maneuvering with skates on the ice.

Another skill is checking, the act of hitting an opponent player with the body or arms to impede the motion or action of the opponent. Most checks occur against the boards that surround the rink. The speed of the game combined with the boards and ice allows for high-energy collisions that contribute to the excitement of the game; consequently, it is another high injury risk situation. However, checking is an intrinsic aspect of the sport that requires skill and stamina to execute repetitively. Women's competition and most youth hockey do not allow checking.

Training Description

Hockey players train specifically with skating, passing, shooting, and puck handling drills on the ice. These are done at top speed and involve many people to simulate gamelike conditions. The goaltender trains specifically in stopping shots that are coming at him or her. Many of the drills simulate a shift (an individual player's time on the ice) in hockey, and the players train to exert themselves for 40-second to 1-minute shifts. Most of the training is done at a 1:3 or 1:4 work-rest ratio. Training areas of emphasis include speed, endurance, srrength, flexibility, and agility. These areas of training are done both on and off the ice.

Aerobic and anaerobic training is done during conditioning, but the majority of the sport is spent on anaerobic metabolism. The defensemen skate longer shifts and with less speed than the forwards, so the former group requires more aerobic capacity. Goaltenders require sudden bursts of energy and quick burst activity, so anaerobic metabolism is predominant (1). Conditioning should be consistent with the demands of the sport.

Preventive Examination

Ice hockey is a contact sport, so traumatic injuries comprise the majority of cases seen. However, with proper physical and mental preparedness these injuries can be minimized. Chronic overuse-type injuries are greatly reduced with programs to maintain proper function. Examination of the athlete needs to first evaluate any injuries that are not fully treated or healed. Because hockey players pride themselves in stoicism and playing through injury, the medical staff needs to be comprehensive and thorough in the history and physical, particularly with previous injuries.

Flexibility and balance in muscle groups are important, not just between agonist and antagonists, but in all muscle groups (3). The trunk needs to be stable due to the unstable nature of the playing surface and requirements for balance when skating and shooting. Stretching to maintain the proper length of particular muscle

groups is very important in the off-ice training regimen. It is of particular importance to focus on the proper length of the iliopsoas and quadriceps groups not only to improve the function of skating but also to prevent low back pain.

Scapulothoracic stabilization is also important to avoid chronic overuse problems in the upper thoracic, cervical, and shoulder region. Scapulothoracic stability helps to protect the player in the event of a traumatic event as well. Strengthening of the lower trapezius and rhomboid muscle groups along with stretching the levator scapulae, scalenes, sternocleidomastoid, and upper trapezius helps to achieve scapulothoracic stability.

Adductor strains are very common in hockey players and have increased in frequency. Most blame an imbalance in strength and flexibility between the adductors and the abdominal muscles. Core stabilization programs and plyometric training can help prevent or minimize adductor strains.

Anatomic short legs can cause associated sacral base unleveling, which underlies many chronic recurrent problems, especially adductor strains, hip flexor dysfunction, and recurring low back pain. Athletes with persistent or recurring problems should be assessed for the short leg/pelvic tilt syndrome and corrected appropriately.

INJURIES

Ice hockey injuries are of both acute (traumatic) and chronic (overuse) type. The vast majority of hockey injuries (80%) are acute. A study of Finnish hockey players' injuries reported in 1996 showed a composite rate of 14.7 for male and female players per 1,000 athlete-exposures; 70% of the 760 injuries occurred during a game (1). In the 1998–99 season, Canadian men's collegiate hockey players had 9.19 injuries per 1,000 athlete-exposures, while females had 7.77, which was not statistically significant (4).

In a study on Canadian female players in the 1997–98 season, risk factors significantly related to injury were (a) prior injury in the past year, (b) more than 5 years of hockey experience, and (c) high exposure level.

Acute injuries can occur almost anywhere on the body, in part due to the physical contact inherent in the game. Some of the more common acute injuries are the acromioclaviclar sprain, cervical sprain, temporomandibular sprain, sprain of the medial collateral ligament of the knee joint, and ankle sprain.

Chronic injuries include adductor strains, hip flexor strain, low back pain, cervicothoracic pain, and elbow and wrist pain.

Groin Pain

Groin pain is a very common complaint among hockey players, and the incidence of groin and abdominal strain has increased in the NHL from 1995 to 2001 (3). The differential diagnosis of groin pain can be vast, including adductor strain, hip flexor strain, inguinal hernia, athletic pubalgia, hip capsular restriction, and lumbar, sacral, innominate, and pubic dysfunctions.

Adductor strains are the most common groin injury, accounting for 43% of all muscle strains (20 of 47) in elite Finnish ice hockey players (4) and 10% of all injuries in elite Swedish players (5). A player was 17 times more likely to sustain an adductor muscle strain if his adductor strength was less than 80% of his abductor strength (6). This imbalance is felt to be the most important risk factor in groin injuries, as muscle strengthening programs have shown success in limiting groin injury rates (7).

The player who presents to the athletic trainer or team physician should first be evaluated for an inguinal hernia. If a hernia is not present, then a biomechanical examination should take place. The examination should focus on the symmetry of the pubic symphysis, the innominates, and sacral and lumbar mechanics. Discovered dysfunctions are treated effectively with muscle energy techniques.

Athletic pubalgia, or sportsman's hernia, is an injury thought to occur on the anterior pelvic floor, where in most cases the cause is tearing of the rectus abdominis tendon from the pubis. Females rarely suffer this injury (8). The mechanism is postulated to be a combination of abdominal hyperextension and thigh hyperabduction, with the pivot point being the pubic symphysis (9).

The fluid motion of the hip joint should also be examined in all of its planes of motion. If the hip has motion restriction, a tight hip capsule may be the cause, particularly the posterior hip capsule. If tightness is found, a hip capsule technique can be employed.

Proper function of the pelvic girdle is important to prevent adductor and hip flexor muscle strains. Recurrent or persistent groin pain may be related to an anatomic short leg and associated sacral base unleveling. The biomechanical examination plays a key role in the diagnosis. The definitive test is a standing postural radiograph that looks at the level of the sacral base. A measurement is done by comparing the two sides. If there is sacral base unleveling, a heel lift is placed on the short side. After the heel lift has been placed, associated muscle imbalances can be more effectively treated. Lifts can be made for the skate, and I recommend using a less moisture-absorbent material for the lift. If sacral base unleveling, mechanical dysfunctions, and muscle imbalances can be corrected, the physician can minimize loss of practice and game time.

Prevention. Tyler et al. found that functional strengthening of the adductor muscle groups significantly decreases the incidence of groin injuries (7). The target was improving the abductor/adductor strength ratio. Overall fitness is clearly an important preventive factor, and poor fitness off-season going into training camp has been shown to be a risk factor itself for groin injuries (3). Due to the nature of the sport, anaerobic and aerobic training should be a part of the off-season training program. Muscle endurance must be high enough to avoid significant fatigue near the end of games, particularly if the player is a forward or goaltender.

Acromioclavicular Sprain

The acromioclavicular sprain is a traumatic injury that is created by a lateral force into the boards, contact with another player, or contact with the ice surface. The injury varies in severity, depending on the extent of the injury to the stabilizing ligaments and capsule. Due to checking and falling on the ice, acromioclavicular sprains are commonly seen but rarely require surgery.

The overall goal in treating the acromioclavicular sprain is to restore the motion and the strength of the shoulder complex. Once the motion is restored, then strength work can begin. The player can return to play after these goals have been accomplished. The standard treatment for an acute acromioclavicular sprain includes ice, anti-inflammatory medications, and immobilization.

As a player attempts to regain loss of motion of the shoulder, manual medicine techniques are very useful. The loss of motion that is associated with an acromioclavicular sprain can be in the glenohumeral joint, the acromioclavicular joint, or the sternoclavicular joint. These joints are assessed individually and treated proximally to distally.

Sternoclavicular joint motion affects shoulder function and needs assessment for motion in adduction and horizontal flexion. Trauma to the acromioclavicular joint can be transmitted along the clavicle and disrupt sternoclavicular function, which can be missed if the examination focuses solely on the acromioclavicular joint. If motion loss is found, then a muscle energy technique is used to restore the motion loss.

Brumm's Technique for Sternoclavicular Dysfunction

1. The athlete is in the supine position.
2. Sternoclavicular motion is assessed, and the position of the clavicle in relation to the sternum is also assessed and compared with the contralateral side.
3. The clinician stands on the ipsilateral side of the dysfunction, placing his or her thenar eminence over the sternoclavicular joint (superiorly for an abduction dysfunction and anteriorly for a horizontal flexion dysfuncrion).
4. The clinician then grasps the upper arm of the athlete and alternates abduction and adducrion while maintaining pressure on the sternoclavicular joint (Fig. 33.1), repeating this maneuver several times. Reassess.

The acromioclavicular joint motion is assessed in 90 degrees of abduction and 30 degrees of horizontal flexion, then internal and external

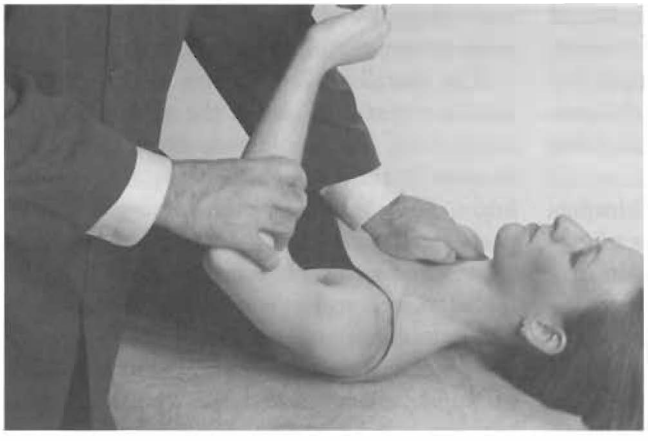

FIGURE 33.1. Brumm's technique for sternoclavicular dysfunction.

rotation is added. If there is restriction in internal or external rotation, the clinician can use muscle energy techniques to resolve it.

The acromioclavicular joint can also be evaluated using a positional approach. The position of the acromion is assessed and compared with the position of the contralateral acromion. If a difference in position is noted and motion loss has occurred a joint play maneuver can be employed in combination with muscle energy for restoration of motion and symmetry.

Brumm's Articulatory Technique for Acromioclavicular Dysfunction

1. The athlete is in the supine position, while the clinician stands on the ipsilateral side of the dysfunction.
2. The clinician grasps the acromion between two fingers of one hand, then uses the other hand to grasp the athlete's upper arm and alternates between abduction and adduction while mobilizing the acromioclavicular joint (Fig. 33.2).

If there is restriction in any plane of motion in the glenohumeral joint, then Spencer techniques and muscle energy can be used to restore the motion. The other areas that need to be assessed with acromioclavicular sprains are the cervicothoracic spine, rib cage, and levator scapulae muscle. The ipsilateral levator is a restrictor of motion of the acromioclavicular joint. If it is restricted or shortened, then muscle energy techniques and an appropriate stretching program are implemented.

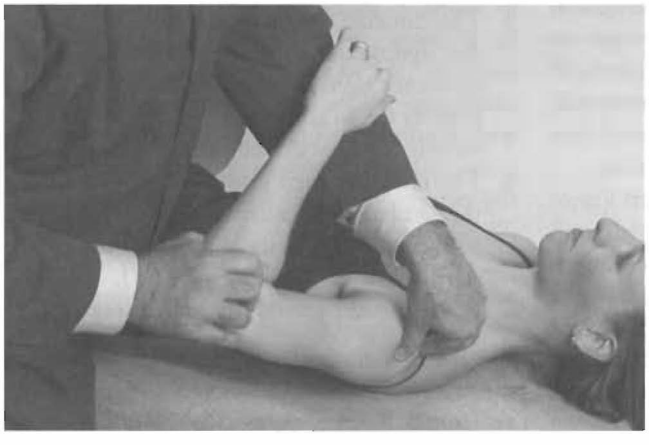

FIGURE 33.2. Brumm's articulatory technique for acromioclavicular dysfunction.

Cervical Sprain

Due to the speed and physical nature of hockey, cervical spine injuries occur frequently (1). Fortunately, most of the injuries that occur are relatively minor and involve some type of motion restriction at the facet joint and surrounding musculature. Hockey players do not have neck support built into their equipment like football players do (neck roll bars), and the speed that players generate on the ice surpasses any other sport. However, unlike football, the neck is not used as a weapon or a primary means to impede another player. Most neck injuries occur from direct trauma to the neck by a player (slash or cross-check with the stick) or in a collision with the ice or boards. Sudden death in hockey players has been reported from trauma to the neck (10).

When these injuries occur, a **complete evaluation is mandatory to assess for fractures, instability, and neurologic compromise prior to the use of manual techniques.** If there is any question, always err on the side of caution. Once these problems have been ruled out, manual medicine is an extremely useful tool for treating hypomobility and pain.

Cervical spine injuries occur when there is contact with another player's body or stick, the ice surface, or the boards. The player's neck and head can be in backward bending, side bending, or rotation as these forces are encountered. The player complains of pain lateral to the spinous processes. He or she also may complain of pain with motion. Usually there are no complaints of radiating pain; however, this can occur with facet immobility.

Assess the upper thoracic segment mobility and the upper rib cage as well. The first and second ribs on either side are often restricted when cervical hypomobility occurs. If there is motion restriction in the thoracic segments, muscle energy and high-velocity, low-amplitude (HVLA) techniques are applicable. Painful segments may respond better to functional technique or counterstrain.

Muscle Energy: Second Rib. The dysfunctional second rib usually is either posterior or in external torsion.

1. The athlete is sitting with the clinician standing behind.
2. The clinician contacts the rib just lateral to the rib angle with the clinician's thumb.
3. The athlete crosses the ipsilateral arm with the hand on the opposite shoulder.
4. The athlete takes a deep breath. With the breath held in, the athlete pushes the elbow to the ipsilateral side, while the clinician applies anteromedial pressure on the rib against the athlete's outward efforts for 3 to 5 seconds (Fig. 33.3).
5. Relax, reposition, repeat, and reassess.

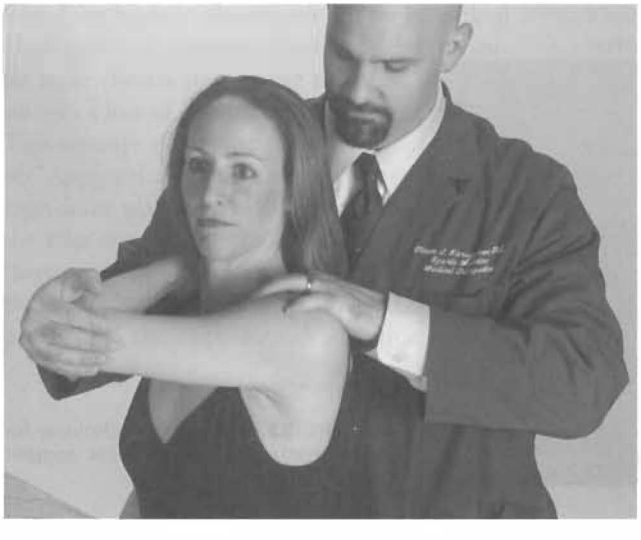

FIGURE 33.3. Muscle energy technique to the second rib.

Scapulothoracic stability is important in treating these injuries. An athlete who has acute or chronic cervical hypomobility should have scapulothoracic stability assessed. If deficiencies are found, specific exercises are given to the athlete to stabilize it. Soft tissue techniques and massage are helpful adjuncts in scapulothoracic dysfunction by treating surrounding tender points, reducing restrictions in the three planes of scapular motion, and reducing muscular tension in this region.

Prevention. Rules that govern checking have been in place to avoid checking from behind, which has helped in decreasing neck injuries (11). Mouthguards disperse shock absorbed to the jaw and mouth, a common occurrence in the game (11). Helmets with face masks are worn in essentially all competitive levels, except the NHL, where face masks are only on the goaltender's helmets. Stronger cervical stabilizing muscles help to absorb impact and limit whiplash effect. However, the most important preventive step for hockey players is discipline. Players who skate out of control and play wildly are at higher risk for injury to themselves and others. Practicing proper checking, improving foot speed, knowing positioning and footwork, and improving stamina are all indirect ways of limiting injuries in general. The less-conditioned, undisciplined player who gets angry from a hard check and skates out of control to compensate for lack of skill will be in position to get hurt more often than not.

Temporomandibular Sprains

The temporomandibular joint (TMJ) sprain occurs in hockey when external force is applied to the jaw. Mouthpiece pain while opening or closing the mouth may indicate fracture or sprain to the TMJ. Once a fracture is ruled out, manual techniques are very useful in the TMJ sprain.

Brumm's Technique for Temporomandibular Joint Somatic Dysfunction

1. The athlete is in the supine position with the clinician at the athlete's head.
2. The athlete opens and closes his or her mouth while the clinician looks for symmetry in the motion. A deviation of the jaw in either direction is considered a somatic dysfunction.
3. The clinician places his or her hand over the mandible on the side toward which the jaw deviates.
4. The clinician applies gentle pressure on the mandible in a medial direction while the athlete slowly opens and closes his or her mouth (Fig. 33.4).
5. Repeat three to five times, then reassess.

FIGURE 33.4. Brumm's technique for temporomandibular joint for somatic dysfunction.

Medial Collateral Ligament (Knee) Sprain

This is the most common knee injury involving the ligaments in the sport of hockey (1). The injury occurs as a result of a medial force on the lateral side of the athlete's knee while the skate is planted. The treatment in the acute stage is based on the symptoms, with the primary goal to reduce pain and swelling. Range of motion and a normal gait should be restored as soon as possible.

Manual techniques can be used to help restore range of motion and a normal gait. Fibular head and tibial torsional dysfunctions should be evaluated and treated. If gait has been affected, the clinician must restore propet function to the lumbar and pelvic mechanics.

Foot and Ankle Pain

Foot and ankle pain occurs in hockey from overuse. If a player has dysfunction in the foot or ankle, it inhibits his or her ability to push off, which reduces the power and speed of skating. If a player complains of foot or ankle pain which did not occur from a traumatic injury, a thorough orthopedic evaluation is done along with a biomechanical examination. Once fracture and instability are ruled out, each joint within the foot and ankle is evaluated using the joint play model. If restriction is found, it is treated and reevaluated.

The two most common restrictions that are found in hockey players are in the talotibial joint and with internal rotation of the navicular bone. Restriction at the talotibial joint results in a loss of dorsiflexion at the ankle joint. This severely affects the player's ability to push off. Appropriate treatments include muscle energy, joint play, and HVLA mobilization with the talar tug. An internally rotated navicular is diagnosed by locating the medial tubercle of the naviculat. Tissue tension is noted and compared with the opposite side. Motion is then tested by performing a wringing-type maneuver to assess range of motion and symmetry compared with the opposite side. If increased tension is noted, a muscle energy technique is effective in treating the rotation by engaging the barrier in external totation and then asking the athlete to internally rotate the foot against resistance for 3 to 5 seconds.

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LACROSSE

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INTRODUCTION

Lacrosse is a high-speed contact sport played by men and women at different levels, including interscholastic, intercollegiate, professional, and international. At one time lacrosse was an Olympic sport, but was dropped due to a lack of participating countries. So popular is lacrosse in Canada that it is their national sport.

Lacrosse has its beginnings with the Native Americans of North America, and it is the oldest of American games. They used lacrosse as training for warfare. At times, entire villages would participate in the events that often lasted hours or days, and covered many miles. While it is only a game to us now, it was actually used to settle disputes between various tribes. However, this was not always effective since there were cases when the losing team was so enraged with the defeat they resorted to warfare.

The object of the game is to score points by throwing a ball into a goal, while using the crosse, a stick with a net atop it, to carry, throw, or hit the ball. The ball remains in play while rolling, or if kicked.

There are three variations of lacrosse: men's field lacrosse, men's box lacrosse, and women's field lacrosse.

Men's Field Lacrosse

The most popular version is field lacrosse. The playing field has goals that are 80 yards apart, with playing areas of 15 yards behind each goal. The width of the field is 60 yards. Each team has ten members: one goalie, three defensemen (who stay near the goal area), three midfielders (who cover the entire field), and three attackmen (who cover the area closest the opponent's goal). The players use crosses with different length shafts and different width heads, but this is beyond the scope of this chapter. The hard rubber ball must be from $7^3/_4$ to 8 in., and weigh from 5 to $5^1/_4$ oz. The goal is 6 ft by 6 ft. Play is organized into four periods, each period lasting 15 minutes. If there is a tie at the end of regular play, the game continues into sudden victory. During sudden victory, 4-minute periods are played until one team finally scores. High school games are 48 minutes long, divided into 12-minute periods.

While attempting to prevent the opposing team from scoring, it is legal to apply body checks. Play is supervised by no less than two officials. A player is permitted to check in the following circumstances:

1. If the opposing player has possession of the ball, or is within 5 yards of a loose ball.
2. Contact must be made from the front or side, and must be above the waist but below the neck.
3. A player is allowed to check his opponent's stick with his stick if the opponent has possession of the ball or is within 5 yards of a loose ball.
4. The gloved hand of the opponent is considered part of the stick when on the stick, and can be legally checked.

No other checks are legal, and a partial (most certainly not thorough) list of fouls should be discussed so as to better understand possible mechanisms of injury.

1. Cross-check—checking an opponent with one's crosse using the part of the crosse between the player's hands.

2. Slashing—swinging a crosse at an opponent in a reckless manner: striking an opponent in the face, neck, chest, back, shoulders, groin, or head with the crosse, unless done by a player during the act of passing, shooting, or attempting to retrieve the ball.
3. Tripping—players may not trip opponents with any part of their body or crosse.
4. Unnecessary roughness—deliberate and excessively violent contact.

To help protect the players, certain protective equipment is mandatory, and some is optional. Required equipment for all players includes a helmet with a face mask and chin strap, mouthguard, shoulder pads, arm pads, gloves, shoes, and jerseys. Goalies are not required to wear shoulder pads or arm pads, but may choose to wear shin guards, chest protectors, and throat protectors. Optional equipment for goalies includes football pants with or without pads. Field players may wear shoulder pads and/or football helmets (as long as the face mask is replaced with a NOCSAE [National Operating Committee on Standards for Athletic Equipment] approved face mask). Mouthpieces must be brightly colored to allow for easy observation. Standardized gloves may not be altered. The helmet is designed primarily to protect from impact of the ball or stick, but not from player impact.

Increasingly, lacrosse is being played on artificial turf fields. This has led to an increase in certain injuries, since natural turf is prone to give way under forces while artificial turf stays in place. The newer synthetic turfs have a rubber base and artificial grass blades so there is more give when pushing off and cushion upon impact. These new products, called Fieldturf and Nexturf, have not had enough research to establish any improved safety data.

Box Lacrosse

Box lacrosse differs from field lacrosse. This form of the game is played inside, with six members per team, in a playing area roughly the size of a hockey rink. This is a smaller field, and players use slightly different equipment. Playing time consists of three 20-minute periods, and games may end in a tie, with no sudden-victory play (unless it is a playoff game). Also, there is a 30-second shot clock, the goal is 4 ft by 4 ft, and there is no restriction on the area the players may cover (all players may move forward). Penalties are basically similar to those in field lacrosse, attempting to provide a safe playing field for all players involved.

Women's Field Lacrosse

The women's playing field is larger than the men's (120 × 70 yards). Women do not employ the protective equipment used by men. All players must wear mouthguards, and optional equipment includes close-fitting gloves, soft head gear, noseguards, and eyeguards. The goalie is required to wear a face mask, helmet, throat protector, and chest protector. Optional for the goalie is padding on the hands, arms, legs, shoulders, and chest.

A team has 12 players: one goalkeeper, six defenders, and five attackers. Games consist of two 30-minute halves (except with high school games, which are 50 minutes long, each half being 25 minutes). The most influential difference in injury rates between men's and women's lacrosse is that women's lacrosse is noncontact. The only situation in which a check is legal is if the defending player is a step in front of her opponent, and she taps her crosse against her opponent's crosse in an attempt to free the ball.

APPROACH TO THE ATHLETES

Required Skills

To succeed at lacrosse, one must have endurance, speed, agility, and strength. The games are long, and consist of almost nonstop action during playing time. Furthermore, the players need to be able to accelerate quickly and change direction instantly. Increased strength allows for harder shots, harder checks, and more resiliency against player impacts. To sum up the requirements of lacrosse, participants must have full function of all their faculties, both physical and mental.

Training Description

Training regimens vary in relation to the sports season. Off-season preparatory conditioning is very different than in-season maintenance training. In the off-season, endurance is often emphasized and improved by distance running up to 25 miles per week. Plyometric training may also be used to develop endurance and enhance agility. Of all the skills needed to play goalie, agility may be the most important. Hand-eye coordination is almost as important, which it can be improved by participating in other racquet sports in the off-season, such as racquetball. To develop lower extremity agility, jumping rope and running tire courses are useful. Weight training should consist of 3 to 4 days of resistance training per week, but not so frequent that there is not ample time for recovery of muscle fibers. Areas of importance include the shoulder and lower extremities. Isotonic resistance is preferred to isometric contractions, since it permits strengthening throughout the entire range of motion. One example of an exercise to increase upper extremity strength is placing a weighted ball (i.e., a low-weight shot put) into the head of the crosse while cradling and performing running drills. Female athletes emphasize strength training less because there is less physical contact than in the male game.

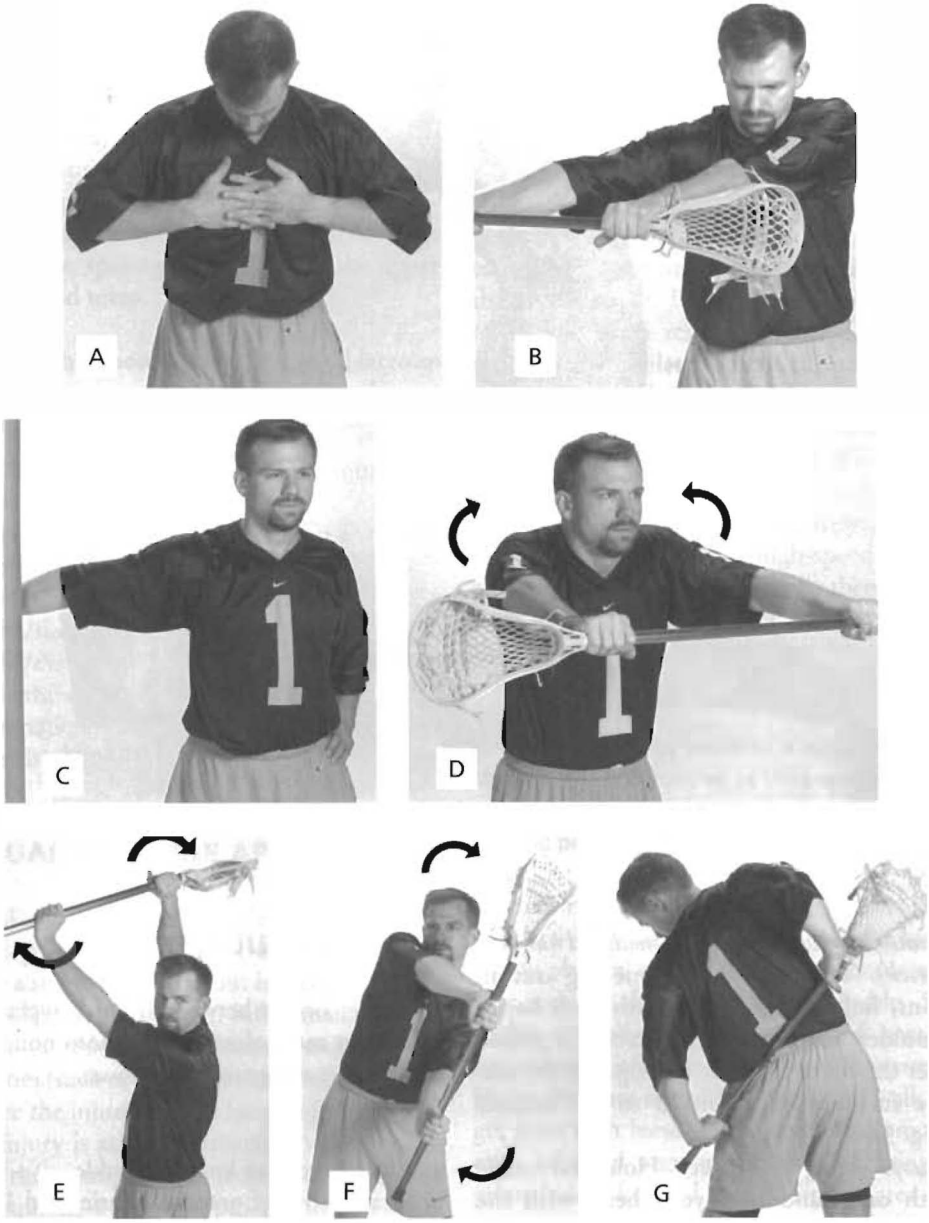
Anaerobic conditioning is crucial since lacrosse involves quick bursts of power and speed. To adequately improve anaerobic performance athletes must use high-intensity–short-duration (HISD) activities in addition to high-intensity–medium-duration (HIMD) activities. HISD includes sprint training at distances from roughly 50 to 400 meters, plyometrics, and interval training. HIMD involves sprint training of 100 to 800 meters using short recovery periods of 30 to 60 seconds.

Training during the season focuses on continued endurance. This is maintained and improved by frequent practices and scrimmages. Strength training should be reduced to two days per week, to allow for the recovery needed by the increased intensity of practice and scrimmages in addition to the weight exercises. Speed should be improved by using sprints and interval training.

Flexibility is the last component of conditioning and is equally important to men and women, being directly responsible for preventing or allowing certain injuries. Flexibility is a basic requirement, but it is frequently lost in an attempt to improve the previous mentioned qualities. Lower extremity flexibility is of paramount importance, and shoulder/trunk mobility is also a very important area to be addressed.

LACROSSE-SPECIFIC STRETCHES

1. *Cervical stretch.* Stand and clasp your hands together at chest level. Flex your neck and hold for 6 seconds. Reach forward with your hands and hold for 6 seconds (Fig. 34.1A).
2. *Horizontal adduction (using the crosse).* This stretches the posterior shoulder girdle. Hold the crosse in front of you, shoulder width apart at chest level. Pull your right arm across your body. Keep your hips facing forward. Repeat with the left arm. Hold for 6 seconds (Fig. 34.1B).
3. *Pectorals (without the crosse).* Stand next to a door jamb. Place your left hand behind your head. Place your left arm (elbow to axilla) along the door jamb. Turn your torso toward the right. Hold for 6 seconds. Repeat on the right side (Fig. 34.1C).
4. *Scapula (with the crosse).* Hold the crosse in front of you with your hands shoulder width apart. Perform shoulder circles but hold each position for 3 seconds; then do shrugs: squeeze your scapula together and pull down the scapula (Fig. 34.1D).
5. *Torso trunk stretch (with the crosse).* Hold the crosse above your head with the hands shoulder width apart (Fig. 34.1E). Keep hips forward and rotate the crosse from side to side. Repeat with the crosse in front with the arms forward flexed at shoulder height (Fig. 34.1F). Repeat with the crosse behind your back at waist level. Follow this by side bending to each side (Fig. 34.1G).
6. *Rhomboid (without the crosse).* Stand next to a door jamb. Lift your left arm straight out in front of you and grasp the door jamb. Lean back keeping your body straight. Hold for 6 seconds (Fig. 34.1H).



(using the cross)

FIGURE 34.1. A, Cervical stretch. **B,** Horizontal adduction stretch. **C,** Pectoral stretch (without the crosse). **D,** Scapula stretch (with the crosse). **E,** Torso trunk stretch (using the crosse), with the arms overhead. **F,** Torso trunk stretch (using the crosse), with the arms in front. **G,** Torso trunk stretch (using the crosse), with the arms behind the back. **H,** Rhomboid stretch (without the crosse). **I,** Hamstrings stretch (with the crosse)—modified hurdler's stretch. **J,** Triceps stretch (with the crosse).

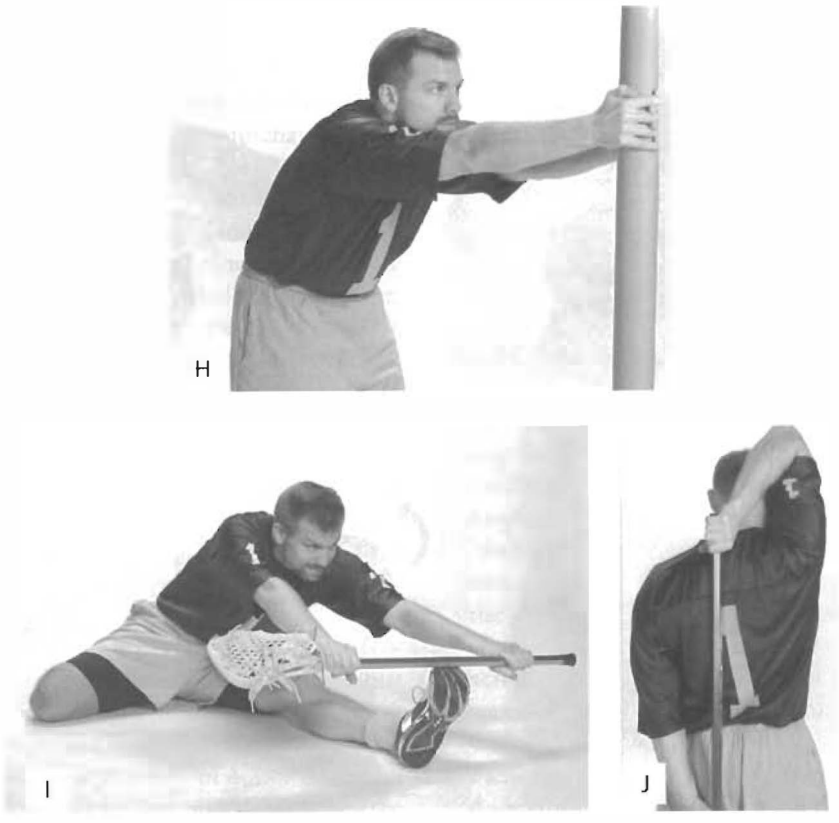


FIGURE 34.1. (*continued*)

7. *Hamstrings (with the crosse, modified hurdler's stretch).* The athlete has one leg out in front, holding the crosse with both hands shoulder width apart. The crosse is placed over the tip of the shoe to help flex the athlete further. Hold for 20 to 30 seconds (Fig. 34.1I).
8. *Triceps (using the crosse).* Hold the crosse with one hand over your head with the shaft of the crosse down your back. Grab the bottom of the crosse with your other hand. Bend the upper arm at the elbow and pull with the lower arm. Hold for 6 seconds. Reverse position of your arms on the crosse (Fig. 34.1J).
9. *Calf stretch.* Use a wall, but it may also be performed seated. With a towel or other item, pull your foot into dorsiflexion with the knee extended (see Chapter 24.3, foot and ankle stretches [Fig. 24.3.11]).

COMMON INJURIES

Crossover occurs between the two types of injuries, but most often they fall into noncontact and contact categories, as follows:

Noncontact

a. Ligamentous to knee and ankle.
b. Strain; 26% of practice injuries and 13% of game injuries.
c. Sprain; 25% of practice injuries and 34% of game injuries.
d. Contusion; 16% of practice injuries and 25% of game injuries.
e. Stress fractures.
f. Shin splints.
g. Turf burns/abrasions, with risk of secondary infection.
h. Wrist fractures/elbow dislocation/glenohumeral dislocation (secondary to fall).

i. Back strain, especially prevalent with midfielders and attackmen.

Noteworthy is the absence of upper extremity overuse injuries, such as epicondylitis, rotator cuff tendinitis, and other inflammatory injuries. The predominant reason for this is because the lacrosse stick provides great leverage to the athlete, sparing excess forces to the upper extremity and torso.

Contact. These account for 49% of all lacrosse injuries, and are more frequent in men than in women.

a. Concussion (most common head injury).
b. Clavicular fracture.
c. Acromioclavicular joint separation.
d. Anterior cruciate ligament tear.
e. Rib fracture, single or multiple.
f. Hand/finger fracture.
g. Facial/eye injuries—lacerations, contusions, and orbital fractures.
h. Traumatic prepatellar bursitis and olecranon bursitis.

MANUAL MEDICINE APPROACH

Manual medicine can complement the treatment regimen of athletes. It is effective when used as a single treatment, but is even more beneficial when used with anti-inflammatory medications to reduce inflammation and thermal modalities (such as cold packs and heating pads).

Once the injured area is localized, determine if the injury is acute or subacute, which determines the manipulation technique used. For acute injuries, it is best to proceed in a stepwise fashion and perform counterstrain or myofascial release first, and then muscle energy when the athlete can tolerate it, in order to augment the release of the strained muscle. It may not benefit the athlete to perform muscle energy on the first visit if the injury is acute; having the athlete return in 1 or 2 days after the initial treatment may be more beneficial. Generally, acute injuries benefit from more frequent manipulation, whereas subacute or chronic lesions might be adequately treated with one or two manipulations in conjunction with an appropriate stabilization program.

SPECIFIC CONDITIONS

Muscle Strains

Far and away, the most common injury that can be helped by manual medicine in lacrosse is the muscle strain. This overuse or overextension of muscle often responds quickly to manual modalities. If athletes do not adequately stretch before sports activity, or if they overexert themselves, they are at increased risk of straining a muscle. The increased incidence of back strains in midfielders and attackmen is a function of their role and the maneuvers they often perform (such as frequent high-speed rotations and changes in direction). While these are common injuries, it is important to make sure that the athlete presenting with pain does not have a more serious cause that may account for the complaints.

Diagnosis often involves a complete history that elicits a mechanism of injury similar to that stated above. If athletes do not remember a specific point when pain began, such as a particular turn of the torso followed by pain and restricted motion, they often relate a time shortly after activity when they started to feel achy.

On physical examination, tenderness and specific somatic dysfunctions are palpable. Spinal strains typically involve an area of paraspinal musculature, where it may feel tight, warm, and boggy. Evaluate the athlete prone and walk a finger from each hand up the transverse process of each vertebral segment, noting any single or group lesions found. Upon finding an area of decreased motion or pain, continue checking the rest of the athlete for compensatory changes.

Techniques beneficial to muscles are myofascial release to counterstrain with acute or severe injuries, and muscle energy when tolerated. Prompt use of modalities are effective in limiting pain and spasm.

Cervical Strains

Diagnosis can be made as described above and in Chapter 16.3. These strains tend to decrease

translation of the articular pillars of the cervical vertebrae. For the acute strain, follow this approach:

1. Perform counterstrain or facilitated positional release first, because they do not exacerbate the pain.
2. Myofascial release should be performed to stretch the muscles and tendons, both perpendicularly to their long axis and parallel with their long axis.
3. Performing direct muscle energy technique increases the range of motion and begins to restore normal function at a faster rate, as well as assisting the athlete in conditioning to prevent future recurrences of similar injuries because it improves flexibility.

For the subacute or chronic condition, direct high-velocity, low-amplitude (HVLA) techniques can be performed to resolve stubborn dysfunctions. However, there are more relative contraindications to this form of treatment than to the subtle, gentle techniques previously mentioned.

Thoracic and Lumbar Strains

Treatment of thoracic and lumbar paraspinal muscle strains is performed as mentioned previously, with one exception. Instead of palpating articular pillars, the examiner palpates the transverse process of the vertebral segments.

In addition to the aforementioned points, there are other contributing factors to think of when considering the thoracic region. Thoracic lesions often involve rib subluxation and rhomboid or serratus anterior muscular spasm. HVLA mobilization techniques are effective and appropriate, but performing a myofascial scapular lift or rotation to the affected side is beneficial in relaxing any muscular spasm. Often this allows for an easier HVLA correction.

Lumbar Strains

Sideline treatments may be done in the absence of neurologic symptoms and acute lumbar muscle spasm for lumbar strains and dysfunction. One effective sideline maneuver is the low-velocity lumbar articulatory technique described in Section 21.3. If the sacrum is involved, position the athlete supine but side-bent toward the unaffected side (the athlete is in a "C"-configuration position with the shoulders and feet side-bent in the same direction.)

Extremity Strains

Strains of the large flexors and extensors of the upper and lower extremities should be treated with myofascial release, and then indirect muscle energy technique. Injuries commonly occur in the extremities due to the sudden acceleration and change in direction needed in the sport.

Rotator cuff strains and anterior capsule strains are not very common injuries for lacrosse players, but they do occur. Manual medicine should be directed at freeing up restrictions and improving the range of motion by using the Spencer technique and unwinding of the involved upper extremity. Using these manual techniques allows the athlete to respond more quickly to the rehabilitation exercises.

Acromioclavicular separation and clavicular fractures secondary to falls and rough checking are also common in lacrosse. If the athlete is not recovering at the rate expected, examine the sternoclavicular (SC) joint for any possible displacement.

Sideline Sternoclavicular Test and Treatment

1. The athlete is in a sitting position while the examiner monitors the SC joint from behind.
2. The examiner instructs the athlete to perform push-ups against the air.
3. The manual technique chosen should be geared at correcting the dysfunction of the SC joint.

If the Sternoclavicular Joint Is Locked Downward (Inferiorly)

1. The athlete sits on the the examining table while the examiner stands behind the athlete, places his or her left hand on the ath-

lete's SC joint and holds the athlete's right arm with his or her right hand.
2. Taking the athlete's arm, circle it back, upward, and down across the athlete's chest and toward the opposite hip with a quick motion. Repeat this process as needed.

If the Sternoclavicular Joint Is Locked up (Superiorly)

1. The examiner holds the athlete's right arm with his or her right hand.
2. The examiner circles the athlete's arm forward, upward, backward, and down behind the athlete's back toward the opposite hip.
3. Repeat this process as needed.

Ankle Sprains

Ankle sprains are very common in lacrosse, as with all running sports. But because of the quick changes in direction and the many sudden stop-and-go motions, this injury is one of the predominant injuries the lacrosse athlete faces. If an athlete is not progressing as quickly as expected in rehabilitation, then reexamine the ipsilateral fibular head and the foot. The manual medicine approach is geared toward correction of the joint above and the joint below the injured ankle.

Posterior fibular head dysfunction is the most common direction of the fibular head with an ankle sprain. This can be reduced with an HVLA technique. Anterior dysfunctions should be treated in a similar fashion (see Chapter 24.3).

In an ankle sprain with a cuboid displacement, the medial border of the cuboid usually glides toward the plantar surface. The HVLA cuboid release technique would work in this scenario.

Ankle Rehabilitation Exercises. Rehabilitation exercises of the ankle are extremely important for the further prevention of repeated sprains. Proprioception training is of paramount importance. The crosse is a handy tool for the athlete to use. Have the athlete perform the ankle exercises with the crosse in his or her hands. For example, have the athlete perform toe raises while cradling the crosse. Also, have the athlete perform one-leg balances while cradling the crosse.

Contusions

While there are no immediate relief techniques for contusions, lymphatic drainage via effleurage and petrissage massage techniques can assist with moving the blood out of the soft tissue and speed up recovery. The sooner the blood resolves from the soft tissue, the less pain and muscle inhibition and the faster the athlete will be able to reach full function.

Stress Fractures

Caused by relatively low loads with high frequency, stress fractures can be considered when athletes report increasing pain during activity without other obvious causes. The onset is insidious, and often the athlete does not report pain, but rather discomfort. A correct diagnosis is made with a high index of suspicion and unremitting, persistent pain. Also, an in-depth history will likely expose a change in the training program. This may be a variation in the intensity, duration, or frequency, or it may be a change in surface or footwear. During the examination, point tenderness and edema are likely to be evident. Radiographs show late response, but scintigraphy, computed tomography, and magnetic resonance imaging are much more sensitive.

Initial standard treatment is aimed at pain control. This is accomplished with rest, ice, and anti-inflammatory drugs. If weight-bearing activity causes pain, the athlete should use crutches. Activity that does not promote weight bearing is allowable, such as swimming, water running (with flotation vest), and cycling. To assist the healing process, lymphatic pump techniques will increase the circulation and inflammatory drainage from the affected area. Evaluate the training program of the athlete to prevent recurrence.

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35

MARTIAL ARTS

LEONARD A. WILKERSON

The term *martial arts* means those arts concerned with the waging of war, but in the twenty-first century, martial arts no longer have a military role. Many feel that the study of the martial arts develops character, patience, or higher moral standards. As a result of this change, the martial arts came to mean "the way." An estimated 1.5 to 2 million Americans (of whom 20% are children) participate in the martial arts with an estimated male to female ratio of 5:1 (1). With such a wide cross-section of Americans participating in an activity about which little is known of the involved physical forces, little thought is given to potential morbidity and mortality. Information generated from a nationwide computer surveillance of emergency departments and surveys mailed to martial arts instructors showed that no deaths and no serious weapon injuries were reported (1). From this information the assertion is made that "all forms of the martial arts are safe." A review of some of the injuries sustained in practicing the martial arts will find that this information may not necessarily be accurate.

THE SPORT

The two most common martial arts practiced in America are karate (meaning the way of the empty hand), and Tae Kwon Do (Korean, meaning *foot, hand, way*). Tae Kwon Do became an official Olympic sport in the year 2000. Other martial arts are aikido (the way of harmony), Jiujitsu (compliance techniques), judo (compliant way), and kung fu (skill or art, a form of Chinese self-defense art, like karate). Hapkido is a Korean martial art very similar to aikido (Japanese) that includes kicks and hand strikes.

The study of Tai Chi Chuan is unique in the sense that it marks the historical meeting of many centuries of Taoist study known as Chi Kung (*excellence of energy*), which was primarily dedicated to physical health and spiritual growth, with the need of the time (approximately 1000 A.D.) for monks to defend themselves against bandits and warlords. The result was, and is, an unusual blend of the healing, martial, and meditative arts, which has been referred to as the internal practice of Tai Chi Chuan.

To understand better the injury trends in Tae Kwon Do tournaments, consider that each round is a 2-minute round. The fighter might fight a new opponent in a single or double elimination, or he or she might fight three 2-minute rounds in a single elimination. In karate, it is usually 3-minute rounds, and like Tae Kwon Do, can be either single elimination, or three 3-minute rounds. In both tournaments kicks to the head are allowed, as well as to the trunk, but not to the back. Punches to the head are prohibited in Tae Kwon Do, although in some karate tournaments, contact to the head with the hands is allowed, but it is supposed to be controlled. Tournaments are otherwise full contact to the body.

Equipment use varies among the different disciplines and tournaments. In Tae Kwon Do, chest protectors are worn, shin and full-arm pads, foot pads, and a groin guard, as well as a head protector. Optional is a mouthguard and hand covers, except at the international level where mouth protection is compulsory. Conversely, in karate, there usually is no chest protector, but shin and forearm pads are worn. Optional are foot pads, and a groin protector is

TABLE 35.1. INJURY RATE PER 100,000 PARTICIPANTS IN VARIOUS SPORTS

Basketball	188.0	Wrestling	26.0
Football	167.0	Sledding	24.6
Aquatic activities	46.0	Dancing	18.8
Lacrosse	39.5	Martial arts	16.9

From Birrer RB, Halbrook SP: Martial arts injuries. *Am J Sports Med* 1988;16:408–410, with permission.

worn. In some tournaments gloves are prohibited. It has been found that wearing protective gear decreases morbidity. Protective gear is available for the forearms, hands, chest, shins, and feet. The athlete is strongly encouraged to wear a mouthpiece and groin protector, and in Tae Kwon Do head protection is worn, which is discussed later in the chapter.

INJURIES

Incidence of injury is higher in the tournament situation. The injury rate per 100,000 participants in various sports is noted in Table 35.1 (2). McLatchie and co-workers reported that some form of injury occurs once in every contest (2), with incapacitating injury severe enough to cause withdrawal from competition once in every ten contests (3). Birrer and Birrer surveyed 6,347 athletes, finding that 59% of injuries were sustained in tournament, as opposed to 41% in nontournament, settings (4,7). They also found that the injury rate and experience were inversely related (4,7). Stricevic et al. confirmed this and also reported that punches have a higher injury rate than kicks, and that protective gear for hands, head, chest, and limbs decreased the morbidity (6).

Injuries are classified into three groups: (1) injuries to the head and face, (2) injuries to the trunk, and (3) injuries to the limbs. In all studies, the most common injuries were contusions, bruises, sprains, and strains (1,2,4,7–10). Orthopedic injuries result from direct impact, repetitive action, or ballistic and torsional maneuvers. Serious injuries seen are concussions, paralysis, and visceral rupture (6). Table 35.2 is a summary of the injuries by anatomic site reported as percentages from two Tae Kwon Do tournaments.

Zetatuk et al. looked at karate injuries for the 1995–1996 season and found that risk of injury increased with the number of years of training per week and rank, specifically brown belts versus lower belts (11,12). Critchley et al. reported that in 1,770 bouts in three national competitions in 1996, 1997, and 1998, in which light or touch contact was allowed and protective padding for the head, hands, or feet was prohibited, 160 injuries were recorded (12). The overall rate of injury was 0.09 per bout and

TABLE 35.2. SUMMARY OF INJURIES AT TWO TAE KWON DO TOURNAMENTS BY ANATOMIC SITE

	Adult Presentations (%) (n = 47)	Junior Presentations (%) (n = 91)	Combined Presentations (%) (n = 138)
Head and neck	49	54	52
Upper extremity	21	14	17
Lower extremity	23	13	17
Groin	4	8	6
Torso	2	7	5
Other (systemic)	4	3	
	—	—	—
Totals	99 (rounding error)	100	100

From Oler M, Tomson W, Pepe H, et al: Morbidity and mortality in the martial arts: a warning. *J Trauma* 1991;31(2):251–253, with permission.

0.13 per competitor; 91 injuries (57%) were recorded. They feel that the absence of protective padding does not result in a higher injury rate, but do state that strict refereeing is essential to maintain control and minimize contact.

COMMON INJURIES

Head and Neck

Common head and neck injuries from martial arts include lacerations, epistaxis from a nose blow, and periorbital hematoma, which can occur from an accidental fist or foot from a high kick. There have been cases of corneal abrasion as a result of a scratch from a toenail or brushing by the eye with the toes. Concussion is seen more commonly from a high kick or a spinning kick. If the athlete is knocked out, always consider a possible cervical spine injury.

Oler et al. report a fatality from a spinning hook kick to the face (1). Postmortem examination revealed an occipital skull fracture, bilateral acute subdural hematomas, contusions of the frontal and temporal lobes, and hemorrhage and herniation of the brain stem. A back-spinning kick or back-hook kick cannot necessarily be thrown with control; too slow a kick allows the opponent a chance to kick back, possibly in the back. If a kick is thrown with proper speed, it cannot be controlled to decrease the impact. Because of the high injury risk, the author convinced the martial arts masters sponsoring tournaments to take out the back-hook and back-spinning kicks to the head and have only front leg kicks to the head, such as a round kick, in controlled fashion. This change markedly decreased the chance of serious injury. At state and national levels of competition, officials allow these back kicks because they are considered potent weapons of the competitors, practiced by the individual for the purpose of winning the match, and used in international competition.

Head gear prevents most soft tissue injuries to the face but is not as protective to the brain as many believe in the martial arts. There is now a movement in the United States toward mandatory head gear. Rhulen Insurance Company of New York, one of the largest insurers of the martial arts in the United States, has informed its policyholders that head gear is now required if insurance is to be in force during free sparring. Mandatory head gear has now reached the tournament circles, as well as many schools and associations. Unfortunately, instructors and students believe that they are protected from serious head injuries, but this is not the case.

Studies from the neurosurgical literature have compared peak acceleration of blows to the head, with and without head gear, using punches both to the front and the side of the head (1,15–17). Safety equipment failed to soften or lessen peak accelerations compared to subjects without head gear. The subjects in the study thought that safety equipment for the hands and feet was protection for the wearer, rather than for the opponent. Head gear and protective padding to the hands and feet may lessen the force of brain acceleration, which seems to decrease the chance of a fatal bleed, but increases nerve fiber shearing. Thus, *extra padding may reduce the chances of death, but it will not prevent brain damage due to tearing of brain substance.* Repeated blows to the head over time may lead to permanent neurologic impairment, such as dementia pugilistica, often seen in boxers (13).

Extremities

Injuries to the extremities are numerous, with strains, sprains, fractures, dislocations, and tendon avulsions being common. Hematomas to the forearm, thigh, shin, calf, and dorsum of the foot are the most common injury. In tournament situations where kicking is below the belt, hematoma to the quadriceps often causes the competitor to retire from competition. Dislocation of the proximal interphalangeal (PIP) joints occurred when punches were poorly executed. Lacetation of the fingers occurred from scratches, if gloves were not worn (2,6).

There was no evidence that karate predisposed to the early onset of chronic tenosynovitis or osteoarthritis, after looking at 22 artists in Great Britain over 5 years (5). Four activities

seemed to have the greatest potential for arthritic damage (5):

1. Doing push-ups on the knuckles.
2. Repeated punching of a firm target.
3. Sparring.
4. Breaking objects.

The metacarpophalangeal joint may have chronic synovitis from wood-breaking techniques. Avulsion of the extensor tendons of the distal fingers or dislocation of the PIP joints can also occur from hitting hard objects using a spear finger technique (5). Karate kid finger, named by Chiu, is injury to the ulnar dorsal digital nerve of the fifth finger, which overlies the prominence of the ulnar hemicondyle of the middle phalanx at the PIP joint (18). This nerve is vulnerable to contusion when the hand is used to perform karate chops. Localized perineural and interfascicular fibrosis may ensue. If acute treatment fails to help, neurolysis is the next option.

Elbow dislocations have been seen, mostly posterior. Supracondylar and intracondylar fractures are rare, but can occur. Tendinitis of the medial and lateral epicondyles is common. In the shoulder, 95% of dislocations are anterior, with 5% posterior. Acromioclavicular sprain is seen if a fall or roll is inaccurately executed.

The knee is vulnerable in the martial arts, especially in karate and Tae Kwon Do, where the ballistic and twisting moves are the rule. Injury is usually from hyperextension, rotation, flexion, or valgus or varus clipping. Injuries vary from meniscus, ligament, patella tendinitis, patella subluxation or dislocation, and epiphyseal fracture, to Osgood-Schlatter disease. Knee dislocation is very rare, and a true emergency. Hematoma can occur in any of the extremities, and is the most common injury, as previously stated, with the quadriceps the most common site. In all studies, contestants had to stop fighting secondary to the pain (3,6–8). A late complication is myositis ossificans. Ankle sprain and fractures are seen just as in any contact or collision sport. Injuries to the great toe, second toe, and fifth toe are common, and may develop osteoarthritis in later years.

Trunk

Injuries of the trunk are common in the martial arts. Many of the martial artists are aiming at the region of the solar plexus surrounding the celiac ganglion. This blow causes the classic winding of the opponent, leaving them vulnerable for further attack. Damage to the chest is usually by direct kick or punches. Costochondritis, rib fracture, or even pneumothorax has been seen. There have been three reported cases of death from anterior chest trauma from these types of blows. The roundhouse kick, also called the round kick, to the trunk usually can damage the vulnerable organs, that is, liver, spleen, kidney, and pancreas. One case report in the medical literature described renal vein thrombosis following a traumatic blow (19).

Testicular injury usually occurs from an uncontrolled kick, and often causes forced retirement of the competitor. Groin guards markedly decrease this risk. Although there can be some pain, usually the competitor can continue to fight after a time-out.

Some deaths have been reported from the martial arts. One was from a spinning kick hitting an opponent in the face, knocking him out, and on hitting the floor he expired. Only three cases of deaths from anterior chest trauma are described in the medical literature (3).

COMMON MEDICAL DISORDERS

Athletes with Seizure Disorders

Studies have shown that a regular exercise program may have a beneficial effect on seizure control. There are no reports of status epilepticus triggered by exercise. It is a difficult decision for physicians, parents, and martial arts trainers to give permission to the participant. Reservations are based on the following concerns (20):

1. Would a seizure during practice or a tournament predispose the athlete to a serious injury, particularly the brain or spinal cord? Most data to date do not support this.
2. Would a single blow to the head or cumulative head blows adversely affect seizure

control or cause an immediate or early posttraumatic seizure? To date, reports suggest that this should not be a concern; however, because of inherent dangers, kicking or punching to the head should be excluded.

3. The Committee on Children with Handicaps and Sports Medicine of the American Medical Association recommends that children who have seizure disorder should be allowed to participate in physical education and interscholastic activities, including contact and collision sports, provided there is (a) proper medical management, good seizure control, and proper supervision, and (b) avoidance of situations in which a dangerous fall could occur.

Based on the aforementioned studies and data, athletes with a seizure disorder can participate, but when they are ready to participate in sparring, head contact should not be allowed, and contact should be limited to the trunk (chest and abdomen). Martial artists should wear a chest protector as well.

Asthma

Asthma and exercise-induced asthma can usually be controlled with beta-agonists alone, or in combination with corticosteroids cromolyn sodium or nedocromil sodium (Tilade). If breakthrough occurs, the athlete should be reevaluated. Sometimes ipratropium bromide inhalers help prevent breakthrough, or a sustained-release theophylline can be added. Combination disk inhalers are effective in simplifying administration of the medicine. A good warm-up before activity induces bronchodilation and refractoriness to exercise-induced asthma.

CONTRAINDICATIONS TO PARTICIPATION

Athletes with the medical disorders listed in Table 35.3 should be disqualified from participation in the martial arts, including competitive sparring. Acute illnesses need individual evaluation so as nor to worsen the illness or put others at risk at being in contact with a contagious individual. Two brief articles in *AIDS Policy Law* cite the case of a human immunodeficiency virus–positive individual being barred from karate lessons (21,22).

TABLE 35.3. MEDICAL DISORDERS THAT DISQUALIFY AN ATHLETE FROM PARTICIPATION IN THE MARTIAL ARTS

Carditis
Severe uncontrolled hypertension
Severe congenital heart disease
Absence or loss of function in one eye
Absence of one kidney
Hepatomegaly
Splenomegaly
Poorly controlled seizure disorder
Pulmonary insufficiency
Atlantoaxial instability
Skin infections and conditions that are contagious (boils, herpes, impetigo, and scabies)

If the participant wants to do the martial arts for self-defense, fitness, or flexibility, without ever participating in full contact sparring, then some of the above conditions will need individual assessment, allowing some persons to train only in a noncontact environment.

PHYSICIAN RESPONSIBILITY AT A KARATE OR TAE KWON DO COMPETITION

Among the physician's duties at a martial arts competition is the examination of the competitors before the competition on request by officials. The physician is to administer first aid, while circulating on the floor duting simultaneous sparring matches, which could be in any multiplication of rings going on at the same time. The fighting area should be inspected to ascertain that adequate flooring is used. The physician treats any minor injuries received, such as lacerations, strains, and sprains and facet restrictions, primarily of the cervical, thoracic, and lumbar regions. In serious injuries, it is best to refer the athlete to a hospital emergency room. When requested, the physician advises the referees as to the fitness of a competitor to continue in a competition.

Injuries that would exclude further participation are the following:

1. Fractures.
2. Concussion with resultant disorientation, amnesia, or loss of consciousness.
3. Ocular injuries with visual impairment, including periorbital injuries, i.e., hematoma and lacerations.
4. Testicular injury with slow recovery or presence of scrotal hematoma (3,16).

MANUAL MEDICINE

Manipulative treatment provides an excellent model for recognizing and integrating the full range of manual medicine techniques into clinical practice because of the wide range of techniques employed. Manipulation is commonly used with all levels of martial art practitioners from the most experienced to beginners. The holistic principles behind medical disciplines such as osteopathic medicine are similar to those of most martial arts, particularly Tai Chi Chuan and Qi Jong. The most common symptomatic areas where manipulative medicine is best applied are facet restrictions in the spine. First and second rib facet restrictions are also troublesome in a martial artist needing quick cervical rotation.

Martial artists complain of pain from sharp to dull aching, with muscle tightness and/or spasm, reducing range of motion and hindering performance in practice or competition. Mobilization of the facet restriction(s) is usually followed with immediate relief and return to practice or competition. The high-velocity, low-amplitude (HVLA) thrust technique is one of the oldest and most widely used forms of osteopathic manipulation. Muscle energy technique is used more with the wrist or foot areas or to relieve spasm of the spinal and lower extremity areas.

In the middle of a competitive match, there is minimal to no opportunity for manipulative therapy. Injury time may only be 2 minutes or 5 minutes depending on the tournament rules or officials. The other fighter and referee can object to excessive medical aid in the middle of a single round or multiround fight, which discourages use of manual medicine during competition.

Between matches, manual medicine can mean the difference between success and failure. Muscle energy techniques are employed mostly, commonly to the extremities, with the lower extremity the most frequent. HVLA and muscle energy techniques are frequently used to mobilize spinal facet restrictions and relieve spasm, primarily of the spine and lower extremity. Foot injuries such as cuboid and navicular dysfunctions can occur due to the absence of shoe support and the amount of direct contact during kicks. This can also affect one-legged stands which are imperative in performing most kicks in karate. High-velocity techniques described in Chapter 24 are most helpful, while joint play techniques should be applied as soon as possible to *healed* foot fractures, as stiffness and fibrosis from immobilization can limit intrinsic motion and make return to competition difficult.

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36

ROWING

PATRICK F. LEARY

The sport of rowing dates back to the early Egyptians. They documented the sport circa 3300 B.C. Ancient Greek galleys had a total of 170 oars on three decks, 85 oars per side. The forefather of the modern-day coxswain/coach, rhe stroke master, was also the disciplinarian. Early on he must have observed the need for symmetrical corporal punishment, less the ship would endlessly circle.

Paddle sports include rafting, rowing, kayaking, and canoeing. Each presents with different injury patterns both on and off the water. Varied training cycles, diverse equipment, many participants, during differenr seasons, make for a sports medicine challenge. The scope of this discussion is limited to sweep rowing, sculling, and crew.

From ancient Athenian mariners to the Head of the Charles, rowing a boat has been an essential function for thousands of years. For work or play, for war or sport, the object has always been to arrive first. Club, college, amateur or professional, rowing popularity in the United States came with the urbanization of the cities. The Detroit Yacht Club was established in 1839, and was represented by singles, pairs, and sixes. The first intercollegiate sport in the United States was a row between Harvard and Yale in 1852. This race featured the classic eight shell, which is still used today, where the coxswain sits in the back of the boat (the stern), that is, the last portion of the boat to cross the finish line. The rowers sit on slides with their feet laced in the foot stretchers, facing the coxswain (Fig. 36.1), who acts as the on-board coach and maintains maneuverability. The rower closest to the cox is called the stroke, the quintessential rower with the best technique to be emulated by the rest of the crew.

In 1873 the Boating Almanac and Boat Club Directory boasted 289 boat clubs in 25 states (1). The clubs raced sixes without a cox, racing for 3 miles with a 180-degree turn in the middle. This created confusion and chaos and on occasion, disastrous results.

The Ivy League schools met in 1875 in front of 25,000 spectators at Saratoga, New York, for a 3-mile straight eight shell with a coxswain. Historically, the shells were shorter, wider, and heavier, as were the rowers themselves.

At the turn of the century, rowing challenged baseball as America's favorite pastime. Regional communication was improving with urbanization. Industrialization was linked to water supplies.

Consequently, rowing popularity followed the plight of the big ciry. Gambling caught on from Maine to Milwaukee, and Boston to San Francisco. Thousands turned out for the popular regattas, which shared a festival quality and filled an entertainment void. For the rowers, a sense of health and character complemented a feeling of pure pleasure. Rowing catered to a growing number of participants and spectators with great affluence and free time.

Head races are usually held in the fall season; races are 4,000 to 6,000 meters (2.5 to 3.75 miles) with a running stagger start and they are usually held on local rivers and lakes. The official National Collegiate Athletic Association (NCAA) sanctioned races in the spring are in eight shells with a cox for 2,000 meters (1.25 miles), or approximately 120 strokes. The shell is 60 ft long and weighs approximately 200 lb. The races last anywhere from 5 to 9 minutes.

Sculling by contrast is usually singles, pairs, and fours. Each rower has two smaller oars.

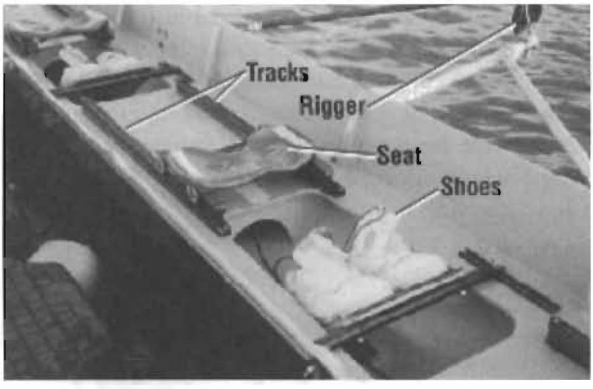

FIGURE 36.1. A typical rowing boat includes a rigger, seat, tracks, and shoes, which are fixed to the boat. (Courtesy of *Physician and Sports Medicine*, Vol 28, No. 4, 2000.)

A single shell is 27 ft long, 10 in. wide, and weighs 23 lb (1).

Summer Olympics rowing is second only in number of participants to track and field. Dr. Benjamin Spock, the famous pediatrician, rowed the eight shell to a gold medal in the summer of 1924. However, the expense of the equipment and the large number of rowers needed have limited the popularity of crew rowing. Available waters and weather conditions and hectic school schedules have limited the development of broad-based intercollegiate participation.

Women have participated in rowing for 90 years, but since the inception of the Title IX Amendment to the Equal Opportunity Act of 1972, rowing has afforded women great opportunities to participate in collegiate competition. Universities have been able to offer literally tens of thousands of young women a chance to participate in college athletics as a result of rowing alone. New technologies in equipment and training have contributed to the increased popularity of the sport. Unfortunately, technology has also sparked a whole new variety of sport-specific injuries.

Classifications can include juniors, who are under 18 years of age, and masters, who are older than 27 years of age. Flyweight classifications are for women under 100 lb and for men under 135 lb. Midweight women are under 135 lb and men under 165 lb. All others are included in the open or heavyweight classification. Other classifications emphasize experience without regard to weight. They include the novice-varsity designation.

Race distances vary in the fall depending on the venue. A dash is 500 meters long, while head races are 4,000 to 6,000 meters. The annual Head of the Charles race in Boston attracts 3,000 rowers, 700 crews, and 50,000 spectators crowding the banks of the Charles River.

Rowing is one of the oldest and most strenuous of all sports of the modern Olympics. A successful rower needs well-developed aerobic and anaerobic capacity, strength and stamina year round, and competitive anthropometric characteristics. These include height, arm span, girth, body mass, and skin fold measurements (2). Distribution of slow twitch and fast twitch muscle fibers in a canoeist demonstrated 71% slow fibers and 29% fast fibers (3). A tall athlete has the ideal physique for a sweep rower. Champion rowers tend to be taller and heavier than their counterparts (2). Greater height has been reported to enhance the sweep rower's leverage; however, this feature may cause greater injury to the spine. Upper body strength is no more important than strong legs and a flexible lumbosacral spine. A 2,000-meter race combines a unique balance of early aerobic and later anaerobic exertion. Maximal oxygen uptake for 20- to 35-year-old rowers is 60 to 72 mL/kg/min for men and 58 to 65 mL/kg/min for women. Ranges of relative body fat for rowers are 6% to 14% for men and 8% to 16% for women (3).

PREHABILITATION

Sports medicine physicians must look for a broad scope of possible injuries in a rower. Rowing is an endurance sport which includes the fall head races, winter dry-land conditioning, spring NCAA competitions, and summer regattas. Injuries common to weightlifters, swimmers, cyclists, and distance runners are also seen in the rower.

Disc herniations in a sweep rower frequently have a different presentation in a sculler. The symmetrical motion of the sculler tends to extrude the disc centrally and posteriorly, avoiding the classic symptoms of sciatica. Coxswains need to be screened for signs of the the athletic triad (amenorrhea, osteoporosis, disordered eating) (6). The one-legged squat maneuver can eliminate concern over ankles, knees, hips, and sacroiliac joints along with some indication of hamstring–quadriceps strength and balance. The Watkins protocol can help screen for low back pain, inflexibility, and strength deficits. This protocol employs core stabilization maneuvers in a ramped sequence which screens for low back deficits.

COMMON INJURIES

Most rowing injuries can occur as a result of training or equipment incompatibility. Injuries are more likely to occur during practice than in the shell, and are more frequently a result of running, weightlifting, and erging than racing. Rarely, injuries are sustained as a result of weather conditions or even frank trauma.

Rowers develop injuries at various sites (Fig. 36.2). Hosea et al. reported on 180 rowers over a 3-year period. The two most common injuries were of the knee (29%) and the back (22%). Back pain ended careers in 16% (4). Back pain has been reportedly caused by sacroiliac dysfunction, spondylolysis, and disc herniation. Costovertebral joint strains are common with sweep rowers. Training injuries include rib stress fractures, and all other stress fractures associated with overuse including the sacrum (7). Medial tibia stress syndrome, patellofemoral syndrome, plantar fasciitis, forearm tendinitis, and sculler's thumb have all been reported in rowers. Equipment injuries include callus formation of the hands, blisters, track bites, and ischial tuberosity bursitis or rower's butt. Several different injuries have been reported by a fatigued crew awkwardly lifting a wet shell out of the water and carrying it into the wind. Rarely, traumatic injuries result from catching a crab, capsizing, or collisions.

A recent assessment of back pain in rowers by Teitz suggested that back pain was associated with a younger age, greater weight, and increased volume of erging (14). Rowers younger than 16 years and weight greater than 178 lb in men and 149 lb in women and erging greater than 30 minutes promote back pain and injury.

Injuries on land have a tenfold increase in risk than water-related injuries in college rowers (4). Training appears to take a greater toll on the rower than racing. Summer, fall, and winter are usually spent cycling, distance running, and erging to develop strength and stamina. Timing and coordination are also essential. Crew has been compared to eighr athletes running full speed, in step, for 6 minutes (5). Balance and posture keep the shell in a neutral and stable position. Strength and speed generate the power to propel the shell through the water. The mental discipline required for year-round training, travel, and academics limits the participation of many.

Decreasing intensity in the weight room, decreasing roadwork, and limiting time and resistance on the erg machines can prevent overuse training injuries. Observing and modifying stroke technique can be helpful. Shortening oars and modifying grips have helped eliminate elbow and wrist injuries. Changing sides from port to starboard is controversial but may benefit a painful back. Adjusting the foot stretchers can eliminate knee and back injuries. The hatchet versus tulip oar is yet another optional equipment modification that can be attempted to reduce stress on the back and ribs (4).

SPECIFIC INJURIES

Back Pain

Stallard first reported back pain in a study of 29 rowers in 1980 (12). Back pain appears to be one

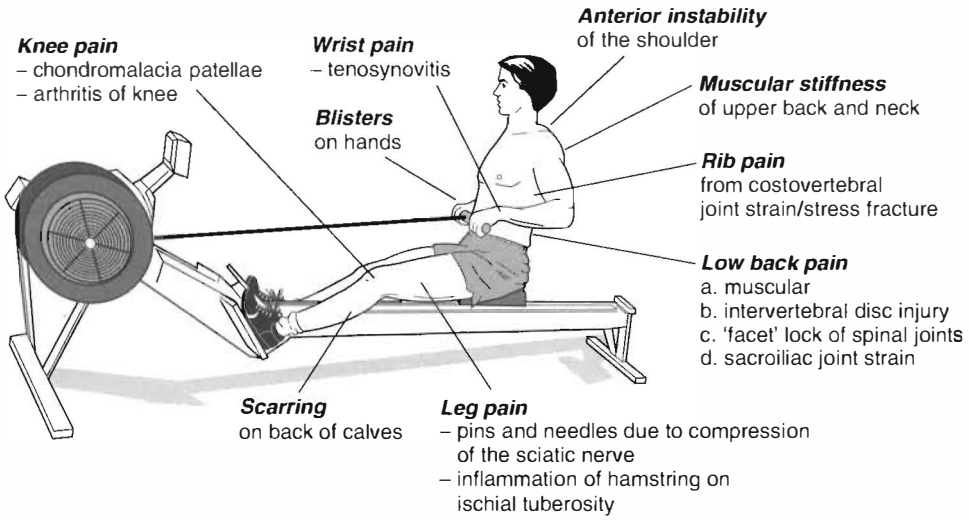

FIGURE 36.2. Various sites of rowing injury and the conditions that can occur.

of the most prevalent injuries noted in the rowing literature. Outdoor rowing techniques lead the list of mechanisms of injury followed by weightlifting, ergometer training, indoor rowing, and boat lifting. An increase in back pain with outdoor rowing may be related to the greater instability of the boat on the water, thus creating an asymmetrical loading (wind and rigging). When compared to the stable erg machine, the greater variety of techniques used for training correlated with greater incidences of back injury. In this regard, more was less.

Most injuries on the water were noted to be soft tissue injuries. The "too much, too soon, too often, too fast" pattern of training results in a number of overuse injuries. Spondylolytic defects were found to be quite common.

Posterior facet insufficiency can lead to slippage of the vertebral body inferiorly resulting in spondylolysis and spondylolisthesis. Rowing is a sport that requires loaded hyperextension of the fragile spine at the finish. As with rib stress fractures, in 1992 the transition from the tulip-shaped oar blade to the fully submerged hatchet blade has caused an increase in back injuries. Tulip blade slippage in the water is a reported mechanism of back injury.

Disc herniation and sciatica can be the result of a loaded, flexed spine at the catch. Central herniations, sometimes seen in scullers, result from the symmetrical flexed load and do not present with the typical pain of disc rupture and nerve root entrapment. The added torque of a sweep rower in the flexed and loaded (catch) and hyperextended and loaded (finish) position often creates an environment for posterior lateral disc herniation (Fig. 36.3). Persistent back discomfort, after a trial of relative rest, modalities, and

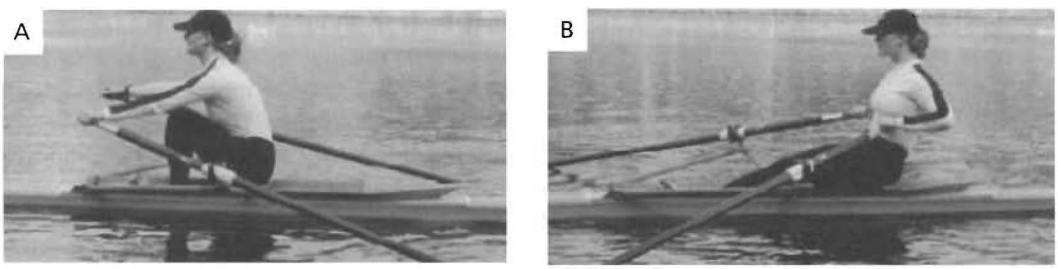

FIGURE 36.3. The catch **(A)** and finish **(B)** of the stroke. Between these phases, the back is loaded while in flexion, which puts extraordinary pressure on the lower back. (Courtesy of *Physician and Sports Medicine,* Vol 28, No. 4, 2000.)

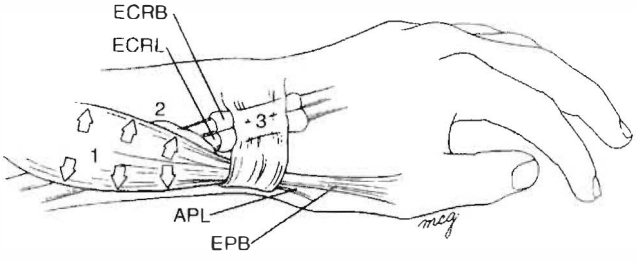

FIGURE 36.4. The wrist extensor tendons. ECRB, extensor carpi radialis brevis; ECRL, extensor carpi radialis longus; APL, abductor pollicis longus; EPB, extensor pollicis brevis. Note the proximity of the tendons, which can lead to an intersection syndrome.

nonsteroidal anti-inflammatory drugs (NSAIDs), should be further evaluated with plain films, bone scans, and possibly magnetic resonance scans.

To prevent such injuries, core stabilization is imperative with trunk stretching and strengthening, with emphasis on the hamstrings. Training cycles should minimize the volume, intensity, and types of training. Ergometer sessions should be less than 30 minutes. Much of the literature points to excessive erging as a causative factor in low back pain in rowers (9). The level of erging resistance (drag factor) is dialed from 1 to 10 levels on the Model C ergometer. It has been proposed that cardiovascular fitness is best accomplished with long duration and minimal resistance (<3). Development of strength is better accomplished in the weight training cycles than with more intense resistance erging (5 to 10). Other preventive measures relate to coaching techniques. Rowers are instructed to avoid "falling into the catch" or "shooting the slide." These two errors contribute to many lumbosacral strains and time away from training.

Manual Medicine

Sacroiliac joint counterstrain techniques and lumbar spine muscle energy and high-velocity low-amplitude techniques have proved to be great adjuncts to reduce stiffness and pain and return the athlete to a pain-free range of motion. Core stabilization exercises such as the bird dog, bridging, and crunches have been successfully used in rowing training rooms. McKenzie back extensions, Williams exercises, and Swiss ball workouts are often recommended for back strengthening and rehabilitation.

Rehabilitation consists of relative rest, icing, and use of NSAIDs, with other modalities to reduce pain and inflammation. Osteopathic manipulation techniques can be employed to promote early mobilization and pain-free range of motion. Success of rehabilitation is gauged by completion of the Watkins exercise protocol (15). Cross-training includes swimming rather than running at first. Sculling and erging can resume when pain-free motion has been achieved. Changing rowers from port to starboard has been shown to be of benefit. Eventual return to sport-specific function (sweep rowing) is allowed but curtailed if pain returns. Reevaluation and further diagnostics may become necessary.

Knee Injuries

Twenty-nine percent of the rowers in Hosea and co-workers' study suffered knee injuries (6). The rowing maneuver takes the knee through a full range of motion, and the knee is loaded during flexion at the catch. Women have a propensity for overuse knee injuries, partly due to increased Q-angles greater than 17 degrees, ligamentous laxity, and a wide gynecoid pelvis. These factors are implicated in patellofemoral syndrome and iliotibial band syndrome. Genu valgum deformity predis-

poses to patellofemoral syndrome, while varus deformity predisposes to iliotibial band syndrome. Training in winter, off the water, including weightlifting, hill running, and erging, all contribute to overuse knee injuries. Meniscal tears and collateral ligament damage have also been reported in rowers.

Foot stretchers can be manipulated for toe in and toe out, height, width, dorsiflexion, and plantarflexion. As a result, the rower's mechanics can be modified to reduce tension and subsequent pain and dysfunction. Modifying training schedules with decreased stair climbing, squatting, and leg presses might help limit knee pain. Isolated strengthening of the vastus medialis muscle and patellar taping are of benefit.

Manual Medicine

Counterstrain techniques to stabilize and strengthen the medial and lateral complexes of the knee have been employed successfully. Modalities of icing and phonophoresis and use of emollient NSAID gels have been effective in reducing pain, restoring motion and function, and facilitating a quicker return to sport.

Thorax

Thoracic injuries are common in rowers. Both arms are loaded and acting in unison in front of the body. The sternum, ribs, scapula, and vertebral bodies act to transmit forces through the contracted serratus anterior, placing excessive pressure on the middle ribs. Costochondritis, stress fractures, and costotransverse rib subluxations all can result from the rowing stroke's catch and pull mechanism.

Lower rib stress fractures in elite oarsmen bear resemblance to the dynamics of the cough mechanism (7). The serratus anterior produces a superior load on the ribs, while the external oblique produces inferior and anterior loads promoting a shear force. It is estimated that 10% to 15% of all elite rowers will sustain a rib stress fracture at some point in their competitive careers (8) (10). These fatigue fractures are more commonly seen as a result of excessive loads on normal ribs. A case series of ten elite oarsmen reported by Karlson suggested that a technique modification would reduce the stress (8).

A sweep maneuver with less reach, pull-through, and layback might eliminate the forces that promote stress fractures (15). Three other factors that reportedly have precipitated these stress fractures are (a) long-distance training with dialed up ergometer resistance (>5), (b) multiple training techniques, and (c) the new hatchet oar with a wider blade, which replaced the tulip oar in 1992. Novice rowers should work predominantly with tulip oar blades until they develop the strength to appropriately handle the hatchet oars (7).

Portside sweep rowers have greater stress on the inside thorax. As a result, the examination should focus on the lateral aspects of ribs 5 through 9. Often a portside rower complains of pain on the right lateral ribs at the finish of the stroke.

Acute treatment for these overuse injuries is less rowing and more training, that is, more swimming, running, and cycling. Reduce pain with modalities and NSAIDs when indicated. Improving hand position from catch to finish has been successful in preventing rib cage injuries. An exercise prescription of push-ups, upper extremity step-ups, and serratus rhythmic stabilization has proved effective. Maintaining good posture in and out of the shell has contributed to rib stability.

Manual Medicine

A rower with thoracic pain emanating from the posterior middle four ribs can benefit from rib mobilization and release techniques. Subluxation of the costotransverse joints responds to the classic posterior thoracic high-velocity, low-amplitude (HVLA) mobilization (10). Myofascial release and counterstrain can be used on tender points around the scapula and near the cervicothoracic junction. Trapezius trigger points can be handled the same way, or the clinician can opt for spray and stretch technique. Lower cervical dysfunctions must be cleared up. Lumbar flexion should be evaluated and rreated if somatic dysfunction restricts motion.

Wrist and Elbow

Intensive outdoor rowing training can lead to tenosynovitis or inflammation of the extensor tendon sheath as it courses through the distal forearm and wrist due to gripping the oar too tightly (16). This occurs more commonly on the outside hand that grips an oar handle that is too large. The most common tendinopathy is de-Quervain's tenosynovitis, or sculler's thumb. Intersection syndrome can result from inappropriate feathering, a common novice error. The intersection of the first and third dorsal compartments of the wrist becomes irritated and swollen and may create a feeling of crepitus on examination (Fig. 36.4). Finklestein's test is usually positive, eliciting pain along the abductor pollicis longus and extensor pollicis brevis.

Medial epicondylitis can occur in scullers during the drive phase by overloading a hyperflexed wrist. Lateral epicondylitis can occur in the inside hand of a sweep rower. Treatments are usually effective if early training errors are corrected and modalities of ice, phonophoresis, and NSAIDs are employed. Equipment changes, adequate grip strength, correct feathering techniques, and proper splinting and bracing can be helpful. Corticosteroid injections into the tendon sheaths are often effective. Counterstrain and muscle energy techniques have been employed successfully at the elbow and wrist.

CONCLUSION

Injuries from numerous repetitive training techniques occur frequently. The clinician must be well schooled in the many different maneuvers used in rowing. Rowers are engaged in a dynamic and sequential effort using strength, stamina, power, and posture. Crew incorporates teamwork, individual achievement, character building, and rules of fair play. A comprehensive osteopathic approach to rowing injuries, one based on a thorough musculoskeletal examination with an emphasis on symmetrical structure and closed-chain function, is essential for speedy recovery. In the words of T. C. Mendenhall: "Commercialization has not degraded crew from play to work and has not yet subordinated the athlete's pleasure to the spectator's" (11).

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37

RUNNING

GREG COPPOLA

"I run to improve my perception of life."[46]

—GEORGE SHEEHAN, MD

One of the basic principles of osteopathic medicine is *how structure alters function*. Perhaps nowhere else is this more evident than in the sport of running. When the human body is placed into motion, structural weaknesses or postural imbalances are often discovered. As the late George Sheehan wrote in a *Runner's World* article entitled *Detection and Correction* in October 1988, running itself does not cause injuries (1). Runners are subject to a variety of somatic dysfunctions which are defined as an altered function of related components of the somatic system: skeletal, arthrodial, and myofascial structures; include also are vascular, lymphatic, and neural elements. The three classic diagnostic criteria for somatic dysfunction include asymmetry, tissue texture changes, and restricted range of motion.

The runner's body is subjected to significant ground-reactive forces which, when coupled with improper joint movement and restricted muscular support, can lead to venous and lymphatic congestion. This cycle can affect proprioceptive input, as trophic flow is impaired and rhere is irritation of free nerve endings. The visceral afferent impulses bombard the dorsal root ganglion with nociceptive information. This can result in pain patterns that can keep a runner from training.

These concepts help form the framework to evaluate structure and function and apply manual medicine techniques to correct undetected deficiencies within the kinetic chain. This chapter focuses in part on how running can contribute to somatic dysfunctions and ways in which they may be effectively treated.

HISTORY

Running history credits the legendary Greek Philippides in 490 B.C. for completing the first marathon. On a hot summer's day he ran 26 hilly miles from Marathon to Athens to deliver the news that the Athenian army defeated the Persians. Exhausted, the legend claims he died after the news reached the city. When the modern Olympic Games were inaugurated in 1896 in Greece, the legend of Philippides was revived by a 24.85 mile (40,000 meters) run from Marathon Bridge to the Olympic stadium in Athens. The first organized marathon on April 10, 1896 was especially important to the Greeks as the host nation. Twenty-five runners gathered on Marathon Bridge, and 2 hours, 58 minutes, 50 seconds later (7 minures/11 seconds [7.11] per mile pace), the Greek postal worker Spiridon Louis finished 7 minutes ahead of the pack. The host nation was ecstatic, and the marathon was born (3).

Running has grown in popularity, and numerous running surveys point to its cost and time-efficient nature as reasons why many people choose the sport. Most clinicians agree that physical fitness is the best predictor of longevity. Individuals who run consistently have a reduced risk of cancer and cardiovascular disease, although the incidence of early arthritis and degenerative joint disease, long considered a side effect of running, is no higher than in sedentary controls.

Fields and Reece estimate that 40 to 50 million Americans run for firness two to three times a week (4). This works out to be nearly one in seven people in the United States who are running several times a week. Recent studies indicate that 10 million people run on more than 100 days per year, and about 1 million compete in local races per year. Prospective studies reveal that 35% to 60% of runners have a significant injury each year. Running injuries are dependent on the age, experience, and size of the runners, as well as the type of running

(cross-country vs. track). There are different energy and physiologic demands, depending on the event in which the runner competes: sprints (100, 200 and 400 meters), middle distance (800 and 1,500 meters) and distance (3,000, 5,000, and 10,000 meters: a marathon) (4).

TRAINING PRINCIPLES

A runner must gradually, but consistently, stress his or her body to improve performance. Training overload leads to improved performance and adaptation of the musculoskeletal system (4). However, there is a fine line between maximal training and overtraining. Runners are often subject to overuse injuries. Improper training can be linked to most running injuries. Doing too much mileage, too fast, too soon is a common part of a runner's history. A careful review of an individual's training log often reveals the reason for an overuse injury. Clearly, runners who run 7 days a week with too-frequent interval workouts, tempo runs, or long distance runs are at risk for overtraining injuries. Tempo runs are designed to help runners run at race pace. The benefit of specific speed workouts appears to be the physiologic changes within the cardiopulmonary and musculoskeletal systems. These changes appear secondary to circulatory and enzymatic enhancements within muscle groups along with adaptations within the nervous system. It is evident that increasing distance must be done reasonably slowly in order to prevent injuries.

A common training principle is to increase weekly mileage by no more than 10% at a time. Long runs should be no more than 30% of the cumulative weekly miles. For example, if a runner is logging 20 miles a week, long runs should not exceed 6 miles. This is a reasonable and conservative measure by which to counsel runners about training programs, although many training ptogram abound which may vary.

Another training principle for runners returning from injury is to return at 50% of the runner's normal training mileage at a subtempo pace (4). The severity of the runner's injury may create a need for less mileage upon return. This is also the opportune time to discuss the use of recovery runs. Understanding how much weekly rest a runner has built into his or her schedule and how much sleep is actually needed is a significant part of evaluating and treating an injured runner.

There is a progression involved for a successful return to running and training that is designed to help the runner reach a goal. This plan involves scheduled rest days. Referring to Fields and Reece again, it has been found that runners that train 7 days a week on a regular basis are injured more than those that take intermittent rest days (4). Rest days should be part of all training plans. The recreational runner usually does not run every day, so concern about injury prevention is centered more on footwear, gait problems, chronic injury, and sudden changes in distance or terrain. For the competitive runner, these are considerations, but attention must be given to the type of workouts incorporated into the training schedule as well.

FOOTWEAR

Selecting Proper Footwear

The runner should know some general principles for the selection of appropriate footwear. This can only come from the practitioner educating the athlete on the foot type he or she possesses. The three categories of shoe type discussed are shoes for the low arch foot (pes planus), high arch foot (pes cavus), and neutral foot.

1. **Motion control.** Motion control shoes provide medial support and stability, with the purpose of decreasing excessive pronation. Strong heel counters in the shoes help to control calcaneal valgus positioning. This is important to stabilize and limit motion from heel strike to midstance, and finally to toe-off. Many motion control shoes have a straight last that gives maximum support to the foot. The sole is firm with a dual-density quality that has the most firm portion along the inner edge providing support that resists pronation (5). Motion control shoes are best for the low to flat arch (pes planus) foot.

2. **Cushion.** Cushion shoes are the opposite of motion control shoes. A shoe for the pes cavus foot needs to allow pronation, not restrict it, because at heel strike the high-arched foot contacts the surface in a varus or inverted rearfoot position. Cushion shoes are designed to allow the foot to pronate, while discouraging supination. They have a medium-density outer sole, made of a soft material. This makes them less supportive allowing them to work with the foot throughout the gait cycle, not to control it (5).
3. **Stability.** These shoes are for the neutral to mild pronator. Stability shoes have a strong heel counter and a lightly curved shape (semicurved last) offering more support than cushion shoes. Stability shoes should be advised for the runner who needs the support of a motion control shoe and the shock absorbance of a cushion shoe.

Wearing the proper footwear as a runner is crucial to prevent overuse injuries. Motion control shoes are designed to support the foot and limit excessive pronation. Cushion shoes are designed to allow the shoe to work with the foot allowing more motion throughout heel strike, midstance and toe-off. A stability shoe, designed to support the neutral foot, is a balance between the cushion and motion control shoe.

Shoe Wear Patterns

1. **Normal heel strike.** The normal heel strike should be just slightly lateral of the center of the heel, 8 to 15 degrees (6). The axis considered normal for gait and wear is a line drawn lateral to the center of the heel, bisecting the sole, and slightly medial of center at the toe box. Any deviation of wear is determined to be abnormal.
2. **Supination.** This wear pattern appears on the lateral side of the heel and sole. Visual inspection of feet will determine a high-arch varus or a tibial varum heel strike.
3. **Pronation.** The wear pattern is on the medial side of the heel and sole of the shoe. Visual inspection of the shoes may demonstrate that the heel counter has shifted medially, while the front quarter has shifted laterally.

RUNNING WORKOUTS AND INJURY

A schedule for the competitive runner should be designed focusing on the specific events the runner will participate in. It is common for an elite athlete to design a cyclical schedule that incorporates not only rest days but also increases and decreases in intensity of training. This is as beneficial psychologically as much as it is physiologically. Getting into this habit is just as important for the competitive runner as it is for the elite-level runner. A healthy competitive drive exists at all levels, not just at the elite level. Because training error is the leading contributor to running injury, runners must have a healthy and safe running program that is tailored to their needs but controls and monitors total mileage (2).

The practitioner treating running injuries should be familiar with some of the standard running workouts. Additionally, a good history often provides the information necessary to determine the cause of most running injuries. Along with a complete history, a thorough examination of the entire spine and lower quarter is essential if the practitioner is to accurately determine the ultimate cause of the injury. The foot, leg, thigh, pelvis, and lumbar spine respond to isolated deviant motions often leading to a change in gait. Just as overtraining can result in tissue and joint damage, faulty mechanics can also lead to debilitating injuries.

Recreational runners are only one type of runner to consider. Competitive runners sustain injuries at a level higher than recreational runners, with total mileage being the primary factor. With regard to running injury management, the same general principles apply to both the recreational and the competitive (elite) runner.

MANUAL MEDICINE

The kinetic chain theory of running refers to how the body must absorb tremendous

ground-reactive forces throughout the musculoskeletal system. The foot and ankle mechanism is the first link in the kinetic chain. Twenty-six bones articulating at 30 synovial joints, and supported by over 100 ligaments and 30 muscles, help to dissipate these ground-reactive forces (8). If the musculoskeletal system has a structural problem, it can alter a runner's function and can overload certain anatomic areas. A runner returning from an injury may have lingering changes in the range of motion of the previously injured site. Theoretically, this may change the biomechanics of the runner. In addition, weakness of a muscle that had prior inflammation may change its energy absorption potential and the protection that it could provide the area.

A blocked or dysfunctional joint complex creates a need for nearby or adjacent structures to compensate for the structural problem. This altered function may be a contributing factor for a tendinitis, bursitis, or stress fracture. Most injuries are related to training errors, structural defects, or intrinsic running errors (leg-length discrepancies, excessive pronation or supination). Foot and ankle dysfunctions can lead to dysfunctions at the knee, which can alter pelvic mechanics, which in turn can affect the back and neck region. This type of musculoskeletal chain reaction is referred to as the kinetic chain theory of running.

Foot and Ankle Mechanics

Assessing and treating somatic dysfunction of the feet is an integral part of the osteopathic evaluation. These dysfunctions may occur within the forefoot, midfoot, or rearfoot, but are most common in the midfoot. In middle and long distance runners, the midtarsal bones may even sublux. Somatic dysfunction of the cuboid involves the medial edge of the bone. The cuboid moves in a plantar direction and rotates medially around its anteroposterior axis (2). Cuneiform somatic dysfunction often involves the intermediate (second) cuneiform gliding directly in a plantar direction. Navicular somatic dysfunction also involves the medial edge of the bone gliding toward the plantar surface and rotating medially around its AP axis.

The principal movements of the feet are coupled motions, which allow the feet to absorb and disperse ground-reactive forces. Ground strike occurs 750 to 2,000 times per mile for the average runner (14). Ground-reactive forces are 1.5 to 5 times body weight (7). The shear forces through the joint complexes are 50 times that of walking (7). A runner's foot strike at a slower pace (long distance running) occurs with the calcaneus, but at higher speeds (sprinting) foot strike occurs with the forefoot.

Understanding the coupled motions of pronation and supination is important when evaluating runners. Pronation includes dorsiflexion of the subtalar and midtarsal joints, abduction of the forefoot, and calcaneal eversion of the foot. Supination includes plantarflexion at the subtalar and midtarsal joints, adduction of the forefoot, and calcaneal inversion. When these movements become excessive, they can create stress upon adjacent anatomic areas. Because structure and function are intimately related, these changes in foot structure produce effects within the musculoskeletal system at distant sites.

Initially, at rearfoot contact, the foot is in a supinated position. This is a rigid, close-packed position, which loads the tarsal bones. The foot then pronates during midsrance with the tarsal joints opening up and becoming less rigid. This transition allows for better shock absorption of ground-reactive forces. Internal rotation of the tibia on the talus also occurs with pronation. The runner then progresses to toe-off, as the subtalar joint supinates and the tibia externally rotates (2).

MUSCLE IMBALANCES

Muscles absorb approximately 80% of the impact of running, with the rest of impact forces going to bone and adjacent tissues (4). One rehabilitative principle that has widespread applicability to runners is the concept of muscle imbalances. The goal of treating muscle

TABLE 37.1. APPLICATION OF JANDA'S PRINCIPLES IN THE EVALUATION AND TREATMENT OF THE LOWER QUARTER

Postural Muscles Responding by Facilitation, Hypertonicity, and Shortening	Dynamic Muscles Responding by Inhibition, Hypotonicity, and Weakness
Iliopsoas	Gluteus maximus
Rectus femoris	Gluteus medius and minimus
Tensor fasciae latae	Rectus abdominis
Quadratus lumborum	External and internal obliques
Short thigh adductors	Peroneals
Piriformis	Vasti, especially the vastus medialis oblique
Hamstrings	Tibialis anterior
Lumbar erector spinae	

Adapted from Greenman PE. *Principles of manual medicine,* 2nd ed. Philadelphia: Williams & Wilkins, 1996, 453–456.

imbalances is the protection of the osteoarticular system. The repetitive and often excessive nature of running creates a host of musculoskeletal injuries and dysfunctions. Janda determined that muscle dysfunction is not a random occurrence but that muscles respond in characteristic patterns (9). These patterns manifest themselves in different ways, even in sports. The concept focuses on the aspect of dynamic and postural muscles (Table 37.1). Runners characteristically spend the majority of their time in hip flexion (iliopsoas) and little of the running stride in hip extension (gluteus maximus).

As a result, a pattern of dysfunction can occur within the iliopsoas mechanism, such that the muscle becomes facilitated, hypertonic, and shortened. This physiologic change, in turn, affects its antagonist muscle group, the gluteus maximus. Therefore, the hip extensors respond by inhibition, hypotonicity, and weakness. Janda describes the weakness as a pseudoparesis, due to inhibition rather than being intrinsically weak. Once this pattern develops, it can alter the arthrokinetics of the lumbopelvic region and lower quarter. This reciprocal inhibition of the tight and dysfunctional iliopsoas affects the antagonist gluteal mechanism (9).

A runner who exhibits such imbalance will also reveal signs of a tight anterior hip capsule. The conventional modified Thomas test reveals muscle tightness of the iliopsoas, rectus femoris, and tensor fasciae latae. Hip tightness may also be detected by assessing symmetry using the pelvic rock test (see Chapter 22.2), Hip and Pelvis: Physical Examination (Fig. 22.2.9). The side of restriction can be further evaluated by placing the athlete in a prone position and observing the motion of the hip and thigh with extension. A restricted anterior hip capsule resists extension of the hip (9).

A tight anterior hip capsule can be treated with a joint play technique for anterior hip mobilization (Fig. 37.1A) or muscle energy into the hip extension barrier. In both techniques, the athlete is positioned in the same prone position, and a sequential 1/8-in. impulse is directed in a posterior to anterior glide. To do muscle energy, bring the femur into extension while the athlete gently flexes the hip against the clinician's resistance (Fig. 37.1B).

The goal of treatment, as noted previously, is to restore reciprocal interplay between the agonist and antagonist muscles for improved shock absorption to prevent impact loading on the joint surfaces. Correcting these muscle imbalances can restore normal lines of stress across the articular surfaces. Clinical evaluations have indicated that disturbances of function within the locomotor system appear earlier than do degenerative morphologic changes. Correcting muscle imbalances teduces strain on joint capsules and ligaments.

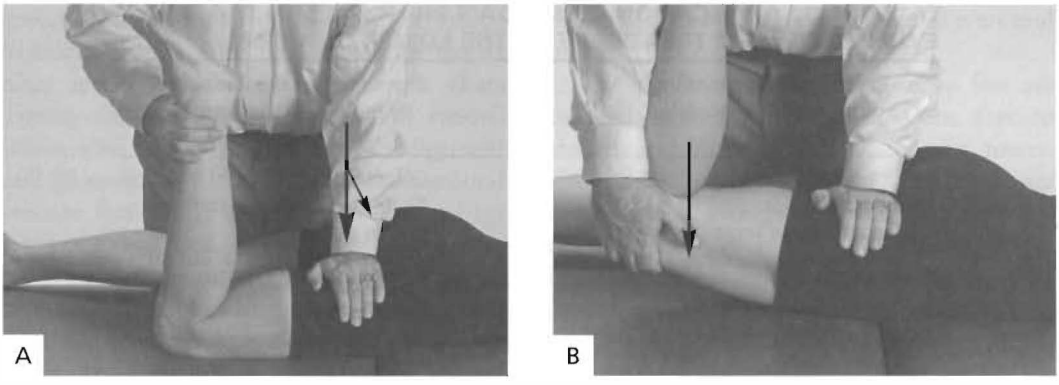

FIGURE 37.1. Anterior hip mobilization. **A,** Joint play. **B,** muscle energy.

COMMON INJURIES

Achilles Tendinitis

Achilles tendon injuries are the most common contributors to overuse injuries of the lower extremity in runners (10). These tendon injuries account for up to 5% to 18% of the total number of running injuries (10). Athletes may present with many different forms of Achilles tendon injuries, including paratenonitis, tendinosis, paratenonitis with Achilles tendinitis (tendinosis), insertional tendinitis, retrocalcaneal bursitis, Haglund's deformity, partial ruptures, and complete ruptures (11), but in this section the focus is on Achilles tendinitis (tendinosis).

Achilles tendinitis is usually a degenerative process (tendinosis) occurring within the tendon. This common misnomer is a separate entity from the actual inflammatory process that occurs with insertional tendinitis.

Biomechanics

As with any physical activity, running greatly amplifies any and all of the demands and stresses that are placed on the body. Many studies have been devised to evaluate and quantify these forces. One such study found that the force on the Achilles tendon during running approaches that of six to eight times body weight, which approximates the maximal strength of the Achilles tendon itself. Another interesting point regarding excessive forces being applied to the Achilles tendon deals with an individual's foot type and gait pattern. Because the tendon inserts onto the calcaneus, the tendon is exposed to stresses that occur secondary to subtalar motion (11).

Three popular hypotheses explain the occurrence of Achilles tendinitis in runners:

1. *Hyperpronation, or pes cavus feet* subjects the tendon to excessive stress that is amplified during the running motion. The normal individual makes initial foot contact or heel strike in a supinated position, followed by pronation during the midstance phase, then supination again as toe-off approaches (10). This repeated supination-pronation cycle becomes more rapid and frequent during running and produces a whipping or bowing action within the Achilles tendon (10), stressing the tendon and contributing to tendinitis.
2. *Contradictory rotational or wringing forces* applied to the Achilles tendon. During midstance, the foot normally pronates, which subsequently causes the tibia to internally rotate (11). With extension of the knee, an opposite external rotation force is applied to the tibia. Many experts now believe that this occurrence during midstance generates excessive force through the tendon (10).
3. *Eccentric loading* of the calf muscles during the normal gait cycle from impact to

propulsion. At impact, the gastrocnemius-soleus complex shortens rapidly (10) then lengthens as the tibia rotates anteriorly over the foot. The complex suddenly reshortens at the forward propulsion phase (29), and these quick muscle action alterations may cause microtears within the Achilles tendon.

Etiology

Multiple intrinsic factors are related to Achilles tendinitis, including age, sex, previous injury, aerobic fitness, body size, limb dominance, flexibility, muscle strength or imbalance, anatomic alignment, and the aforementioned foot morphology. The most common intrinsic factors are excessive rearfoot motion and gastrocnemius-soleus insufficiency. Another cause that is frequently missed is functional hallux limitus, in which the first metatarsophalangeal joint has restricted dorsiflexion. This causes a functional blockade of the gait cycle, and instead of the joint dorsiflexing fully to disperse force and motion, the next level up has to disperse more than its usual share. When the ankle makes up the difference, the Achilles tendon receives more tension and eccentric load, which can eventually lead to overload.

The extrinsic factors are similar to most degenerative tendinopathies, such as level of competition, years running, skill level, shoe type, ankle bracing, running speed, frequency of stretching, cumulative mileage, and running surface (7,10).

Prevention

As a common runner's injury, treating Achilles tendinitis should begin with a proper training program. Prevention starts with educating the athlete about principles such as adequate stretching and warm-up, running shoe selection, custom-made orthotics to correct any malalignment in the foot, and proper training techniques. Athletes must be cautious of the too much, too soon error when beginning their training. Because the Achilles tendon has a particularly poor blood supply, especially the more distal aspects toward its insertion into the calcaneus, injuries to this segment tend to heal relatively slowly. By preventing overtraining, the chance of getting Achilles tendinitis decreases.

An important step in preventing Achilles tendinitis is correcting common training errors before an injury is sustained. Some of these errors include increasing mileage too quickly; interval training; running on sloping, hard, or slippery roads; indoor track running; and inadequate warm-up and stretching (11,12).

Runners should increase their weekly miles by no more than 10% at a time. For example, if a runner is averaging 30 miles per week, and wants to increase the mileage, he or she should not add more than 3 miles the following week. Also, long runs should be no more than 30% of the cumulative weekly miles.

Standard Treatment

Acute treatment includes nonsteroidal anti-inflammatory drugs (NSAIDs) ice, relative rest (including partial or non–weight bearing), and bilateral heel lifts. Steroid injections are discouraged because they weaken the tendon, and histologic studies have shown no inflammatory cells in the paratenon or the muscle/tendon itself. Rest is especially important because of the poor vascularity of the tendon (12).

The runner should be cross-training to maintain endurance and stamina. When the pain is at its worst, the upper body ergometer and swimming are good choices, as they provide low resistance to the lower extremity. Typical progression in the healing process allows the runner to use the stationary bike next, while progressing to pool running. Before progressing to flat running, the athlete can also use the elliptical machine to prepare the body for beginning stages of running.

Subacute treatment includes continued icing, ultrasound, and cross-friction massage to break down adhesions that may have developed during the injury and healing process. Underlying problems, such as a tight gastrocnemius-soleus complex, overpronation, or pes cavus should be identified and treated. The overpronating foot should be fitted with a stability or motion

control shoe, while the supinating foot should be fitted with a cushion shoe. By stabilizing midfoot and forefoot pronation and supination, a great deal of stress is taken off the Achilles tendon (13).

As the runner becomes pain-free and improves ankle dorsiflexion, he or she can begin open kinetic chain strengthening exercises with rubber tubing or ankle weights. Progression extends to closed kinetic chain strengthening using concentric, then eccentric exercises. Strength progression should be closely monitored to avoid overshortening the musculotendinous complex and predisposing the Achilles tendon to reinjury (12).

Functional training includes proprioceptive training, such as balancing exercises (one-leg stands on a minitrampoline or pillow, slide boards, and Boppy boards). Once the tendon is pain-free and rehab is adequately completed, the runner can progress to a walk-jogging routine on a flat and forgiving surface. Depending on the pre-existing level of cardiovascular fitness, this walk-jogging program will probably not be adequate for the athlete to maintain his/her aerobic capabilities. Therefore, the athlete should be encouraged to continue and progress with the cross training already discussed (13).

Manual Medicine Treatment

The main purpose of manual medicine in runners with Achilles tendinitis is to promote relaxation and elongation of the musculotendinous complex and eliminate other causative factors. Tight hamstrings contribute to increased tension on the lower leg, predisposing the runner to tendinitis, as well as fibular head dysfunctions.

Muscle Energy

Most runners demonstrate restricted range of motion, especially in dorsiflexion. Dysfunctions effectively treated with muscle energy techniques include anterior or posterior fibular head dysfunction, soleus, gastrocnemius, hamstrings, and hip flexors.

Counterstrain

The runner with Achilles tendinitis often complains of point tenderness anywhere throughout the lower leg, both anteriorly and posteriorly. By treating these tender points with counterstrain, the entire lower leg, including the Achilles tendon, will relax and become less painful. Classic tender points of the lower leg include points in the muscle belly of the gastrocnemius and soleus, as well as the point at which the two combine into the common Achilles tendon, known as the musculotendinous junction. Common anterior points include those in the muscle belly of the tibialis anterior and peroneal muscles (Table 37.2).

Counterstrain on runners may require subtle fine-tuning of each position with movements in all three planes of motion in order to completely eliminate the tenderness.

Do not only palpate for tender points in these suggested locations; every athlete is unique in his or her pain patterns. Also, these positions should be modified for each individual to allow optimal tissue relaxation and pain relief.

Myofascial Release

Myofascial release (MFR) techniques and other forms of soft tissue therapy are effective in improving relaxation and elongation of the lower leg musculature. (See Chapter 6 on myofascial release for a thorough discussion.)

TABLE 37.2. COUNTERSTRAIN FOR GASTROCNEMIUS-SOLEUS AND ANTERIOR TIBIALIS MUSCLES

Tender Point Location	Treatment Position
Gastrocnemius-soleus	Athlete is prone; knee is flexed to 90 degrees; ankle is plantarflexed; fine-tune with internal/external rotation of tibia.
Tibialis anterior	Athlete is prone; knee is flexed to 90 degrees; ankle is everted and dorsiflexed; fine-tune with internal/external rotation of tibia.

Treating the posterior lower leg with MFR begins once an area of tension is palpated. The clinician can go either into the barrier (direct MFR) or away from the barrier (indirect MFR). The preference is to go into the barrier to attain the most effective release possible. If the athlete is in severe acute pain, the clinician should use only indirect MFR, in pain-free motions (2).

If the clinician uses direct MFR, then the tissue is taken into the restrictive barrier. Holding firmly into that myofascial barrier, the tissues are allowed to tighten momentarily, just prior to the release. This release occurs rather quickly and is palpable. Often the barrier only needs to be engaged for a few seconds, sometimes a bit longer, and the manipulation can be repeated until the myofascial tissue palpably relaxes. Often this treatment technique allows the clinician to conveniently progress into other treatment modalities, such as muscle energy or counterstrain.

When indirect MFR is determined to be the best treatment option, the clinician slides the tissue surface away from the restrictive barrier. For runners in acute pain from tendinitis, this takes the pressure off the area of irritation immediately and creates more movement within the tense tissues. As with direct MFR, palpate the entire posterior leg for areas of restriction and treat each of the positive findings. For areas not as painful, a combination of direct and indirect MFR can be used.

Runner's Knee

Runner's knee, also known as patellofemoral stress syndrome (PFSS), is the leading cause of chronic knee pain in young adults, women, and athletes (14,15). Its multifactorial nature can make diagnosis and treatment challenging.

Biomechanics and Etiology

Running greatly increases the biomechanical forces applied to all areas of the body. Any slight deviations from normal mechanics will be greatly amplified with running, in turn increasing the athlete's chances of sustaining an injury.

Immature runners may present with pain due to Osgood-Schlatter disease or Sindig-Larsen-Johansson osteochondrosis, which mimics PFSS (7). Pediatric athletes need to have their mileage monitored more closely than others because of their skeletal immaturity, particularly those with open physes.

The basic etiology associated with runner's knee is some degree of lateral patellar tracking. Various intrinsic factors are discussed in Chapter 23.3 on conditions of the knee.

Prevention

As with most other types of overuse injuries, the treatment of runner's knee should always begin with the concept of prevention. There are many different predisposing risk factors that exist with runner's knee and eliminating as many of these risk factors as possible is the best preventive approach. Some of the risk factors to modify include

- Training schedule (making sure to follow the 10% to 30% rule previously mentioned).
- Training surface (avoiding crowned surfaces and training on more flat surfaces).
- Proper footwear (making sure to track a shoe's mileage while trying not to exceed 300 to 400 miles per pair of shoes).
- Proper foot support (selecting proper shoes along with the possibility of custom orthotics if biomechanically necessary).
- Adopting an adequate stretching/strengthening protocol, particularly with the hip flexors and hamstrings.
- Avoiding excessive stair-climbing and/or squatting.

Cross-training during recovery is extremely important to an in-season runner with PFSS. Runners should avoid as much excessive knee flexion as possible, due to increases in tension within the patellofemoral joint, which exacerbates the already present inflammatory process. Athletes may participate in low-impact, limited flexion, less weight bearing activities in order to elevate the heart rate and maintain stamina and endurance. Some examples of these exercises include

- Elliptical machines.
- Stationary bikes (with the seat in a higher position to avoid excessive knee flexion).

- Swimming (pool, underwater treadmills, countercurrent exercise).
- Ski machines.

Manual Medicine Treatment

The first area to address is a restricted fibular head. This can be either an anterior or posterior restriction and is checked by grasping the proximal shaft below the head of the fibula and moving it anteriorly and posteriorly in a direction 30 degrees from neutral when facing the athlete. Always compare with the alternate side. Accepted techniques include high-velocity, low-amplitude (HVLA) and muscle energy techniques (see Chapter 23.3 for techniques).

After the knee examination, perform a standing and seated flexion test. The muscle groups around the hip (extensors and flexors) can significantly affect the position of the innominates, which may lead to multiple lower limb dysfunctions. The flexion test lets the physician determine iliac involvement, as it is not unusual to find the innominate on the side of the PFSS complaint posteriorly rotated. A check of the hamstrings can reveal tightness contributing to the tension bringing about the unilateral hip rotation. The muscle imbalance between the hamstrings may also cause reflexive inhibition of the quadriceps mechanism adding to the vastus medialis obliquus dysfunction. Iliopsoas restriction limits hip extension, shortens the stride, and increases load on the patella. The lumbar spine should be screened to address any lumbar restrictions as well. Treat any dysfunctions diagnosed in the hip and pelvis region.

If the standing and seated flexion tests have determined that the problem is of iliosacral origin, treatment involves using the quadriceps muscles to reset the innominate to its normal position. Muscle energy techniques can be used to treat an anterior or posterior innominate. Counterstrain techniques should target anterior, lateral, and posterior tender points, as described in Table 37.2. Extension ankle points are treated at lower margins of the popliteal space on either side of the split of the gastrocnemius while plantarflexing the foot to hyperextend the ankle.

Myofascial release techniques are effective in treating lateral retinacular tightness, iliotibial band restrictions, and hip capsule restrictions along with many other fascial and connective tissue dysfunctions. Both perpendicular and parallel myofascial release techniques can provide significant relief of somatic dysfunctions and often are a good way of releasing any restrictions prior to performing other osteopathic treatments (13).

Plantar Fasciitis

Plantar fasciitis is the most common hindfoot problem in runners (13). This is a very debilitating condition that plagues many individuals on a day-to-day basis. This again is attributable to the fact that during running, the body is exposed to amplified degrees of forces and stresses (see the plantar fasciitis section in Chapter 24.3).

Biomechanics and Etiology

Many of the instigating factors are biomechanical in nature. The most common factor is repetitive stretching of the plantar fascia that leads to microtears and inflammation of the plantar fascia, usually at its attachment into the calcaneus (16). Long distance runners, with high weekly mileage, and shorr-distance sprinters, with repetitive but explosive eccentric load, are both at risk.

The repeated stretching and eccentric loading of the plantar fascia can be exaggerated by a number of structural abnormalities, including pes planus (flat feet), a rigid cavus foot (high arch), and a tight Achilles tendon (16). All of these conditions compromise an individual's ability to absorb shock. A tight gasrrocnemius-soleus complex causes the foot to spend more time in a plantarflexed position (particularly during sleep), which ultimately shortens the plantar fascia. This shortening makes the plantar fascia more susceptible to the repetitive overstretching, as described previously.

The force load on the body during running increases tremendously. The biomechanical faults of an individual's gait are greatly amplified during running, particularly overpronation. One

study reported that 81% to 86% of athletes with symptoms consistent with plantar fasciitis had been classified as excessive pronators on examination (17). On standing and walking, there is an obvious and measurable tension increase along the plantar fascia (17). During pronation there is also a compensatory inward rotation of the tibia, previously discussed in the Achilles tendinitis section of this chapter. This internal rotation itself can increase the stress that is placed on the plantar fascia and the Achilles tendon (16).

Several other etiologies associated with plantar fasciitis are much more specific to runners:

Extrinsic

- Exceeding the 10% to 30% rule for increasing weekly mileage.
- Wearing improper or worn-out shoes.
- Running excessively on hills or running on crowned roads.
- Not having proper foot stabilization (i.e., custom orthotics).
- Not scheduling adequate rest and recovery time every week.
- Not implementing a proper stretching and strengthening protocol.

Intrinsic

- Heavier runners place more stress on the fascia (Newton's second law).
- Leg-length discrepancies; anatomic or functional causes.

Prevention

Plantar fasciitis is best managed from a preventive standpoint. Runners should correct modifiable risk facrors. Clinicians need to educate runners about the injury and emphasize the importance of therapeutic compliance.

Some of these risk factors may include, but are not limited to (2)

- Obesity.
- Past history of plantar fasciitis.
- Pes planus or rigid pes cavus feet.
- Tight Achilles tendon.
- Excessive pronation with walking or running.
- Training errors.

Manual Medicine Treatment

Few recent studies have been conducted to specifically assess the success of manual medicine when treating plantar fasciitis. This, however, does not diminish the importance of somatic dysfunctions associated with plantar fasciitis and only stresses the value of continued research.

Direct myofascial release with plantar fasciitis was studied using trigger band and continuum techniques, showing 77% improvement in pain. The average number of treatments that were necessary for satisfactory resolution of pain was 2.7 (18).

The techniques benefit the arhlete by relaxing and stretching the dorsal foot structures, enhancing circulation and nutrition, improving reflex activity, and increasing immune response (2).

Myofascial Release: Trigger Band Technique. This is similar to the cross-frictional deep massage technique for plantar fasciitis (17):

1. The athlete is supine with the clinician sitting at the foot of the table.
2. The clinician uses a closed fist to contact the sole of rhe athlete's involved foot just proximal to the metatarsal heads.
3. While applying pressure to the dorsal aspect of the athlete's foot, the clinician positions the foot into dorsiflexion and toe extension. The clinician then drags his or her fist over the plantar fascia toward its calcaneal insertion (Fig. 37.2). Repeat as necessary.

Myofascial Release: Continuum Technique. This is a more static rechnique than trigger band (18).

1. The athlete is supine with the clinician placing overlapping thumbs over the origin of the plantar fascia at the medial tubercle of the calcaneus.
2. The clinician should then apply adequate pressure over this region until he or she feels

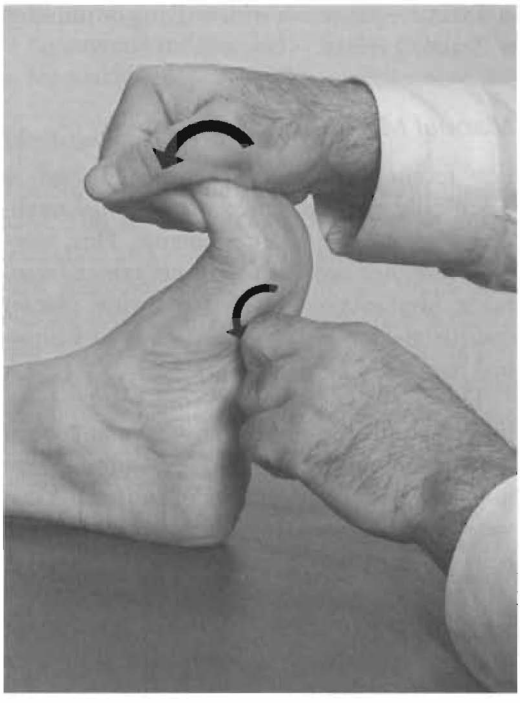

FIGURE 37.2. Myofascial release: trigger band technique.

a release of the plantar fascia. Discomfort may be felt (Fig. 37.3).

Some additional techniques may be beneficial:

1. Counterstrain to the plantar fascia is helpful in releasing the fascia (see Chapter 24.3).
2. HVLA for any dorsally displaced tarsal bones (see Chapter 24.3).
3. Muscle energy techniques targeted at releasing any gastrocnemius-soleus or dorsal foot musculature restrictions.
4. Corrections for any fibular head dysfunction (HVLA, muscle energy, or myofascial in nature).
5. Myofascial release of the plantar fascia using the modified technique for transtarsal somatic dysfunction (Fig. 37.4) (8).

Medial Tibial Stress Syndrome

As one of the most common injuries associated with running and general exercise-induced leg

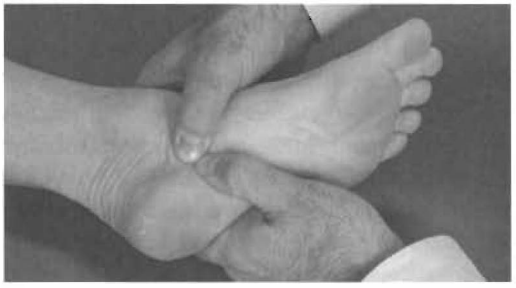

FIGURE 37.3. Myofasical release: continuum technique.

pain, medial tibial stress syndrome (MTSS) is not fully understood. For decades, any lower leg or tibial pain was referred to as shin splints. This term is neither descriptive nor accurate. The term MTSS was originally coined by Drez and reported by Mubarak et al. in 1982 (19). It implies both a specific location (medial tibial) and etiology (stress/overuse) for this common injury. While runners are most commonly afflicted, with one study suggesting a rate as high as 13%, MTSS is also common in jumping sports, such as volleyball and basketball (20).

In the beginning stages of the syndrome, there is usually significant point tenderness along the posterior medial edge of the distal tibia. The pain is diffuse along this edge, often extending 3 to 6 cm. Pain is increased by active plantarflexion and inversion. Characteristic of MTSS, pain is usually the worst at the beginning of the run, diminishes as the run progresses, and recurs hours after finishing the workout. Finally, all fracture tests, including percussion and radiologic, are negative in MTSS, as

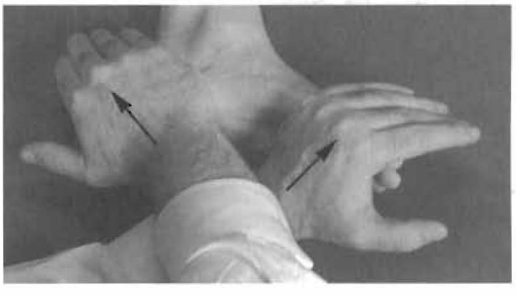

FIGURE 37.4. Myofascial release of the plantar fascia for transtarsal somatic dysfunction.

TABLE 37.3. COMPARISON OF MEDIAL TIBIAL STRESS SYNDROME (MTSS), STRESS FRACTURES, AND CHRONIC COMPARTMENT SYNDROME (CCS)

	MTSS	Stress Fracture	CCS
Palpatory tenderness	Diffuse	Point focal	Variable
Percussion pain	Negative	Variable	Negative
Neurovascular	Normal	Normal	Variable
Painful range of motion	Variable	Variable	Negative

Adapted from Kortebein PM, Kaufman KR, Basford JR, Stuart MJ. Medial tibial stress syndrome. *Med Sci Sports Exerc* 2000;32(2, Suppl):S27–S33.

is neurovascular testing. Table 37.3 compares MTSS with tibial stress fractures and chronic compartment syndrome (CCS).

Diagnosing MTSS is accomplished most importantly by clinical evaluation. Radiologic tests are used to rule out a stress fracture, when suspected MTSS is refractive to treatment. Plain radiographs are almost always normal in the athlete with MTSS (22). If any irregularities show up on a radiograph, a stress fracture should be ruled out with a bone scan, computed tomography scan, or magnetic resonance imaging. Considering the distinct symptoms of MTSS, it is not surprising that most cases are diagnosed and treated without the need for imaging.

Because of the inherent connection between structure and function, it is necessary to comment on ankle and foot functional anatomy when the topic of MTSS is discussed. Once again, the integrity of the medial longitudinal arch (MLA) is of the utmost importance in proper structure of the running foot. Understanding the distal attachments of the lower leg musculature helps to comprehend their connectedness to the MLA and overpronation. Remember that the tibialis posterior, after crossing the medial malleolus, runs on the plantar surface of the foot, attaching to the navicular and first cuneiform. Also, the tibialis anterior runs on the dorsal aspect of the foot, attaching to the navicular and first metatarsal. These two muscles work synergistically to stabilize the midfoot and MLA. Another key structure to the support of the MLA is the plantar fascia and its fascial connections, through the calcaneus, to the Achilles tendon and calf muscles. As structure impacts function, so too will somatic dysfunction of any of these components affect the optimal outcome of the runner with MTSS.

Biomechanics and Etiology

In contrast with sprinting, where only the forefoot contacts the ground, long-distance running requires heel contact. As with walking, in running, the foot initially contacts the ground in a supinated position, on the lateral heel. As the foot advances to the stance phase, it progresses to a more pronated position. It is theorized that this change from supination to pronation is when the most stress is applied to the medial tibia, through the eccentric contraction of the medial soleus muscle (26). The primary plantarflexor and invertor of the foot, the soleus contracts and lengthens, to help prevent any excessive dorsiflexion and eversion, as the foot progresses to pronation. Therefore, it makes sense that overpronation would be a cause for increased stress on the soleus, and ultimately MTSS (21).

While some research points to the medial attachment of the soleus as the main cause of MTSS, many other theories exist, including tibialis posterior tendinitis, fasciitis, and periostitis. The differential diagnosis includes stress reaction, stress fracture, tendinitis, muscle strain, and CCS (23). Several training practices can promote the development of all of these conditions:

- Running too much (intensity, duration), without proper rest days.

- Poor running shoes and running surface.
- Prior history of MTSS.
- Inadequate warm-up and stretching.
- Running on crowned surfaces and the same side of the road.

Prevention

Physicians and runners should be aware of methods to prevent medial tibial stress syndrome. These include a proper flexibility and strengthening program, isolating the Achilles tendon (gastrocnemius-soleus complex), Tom Dick Harry complex, and tibialis anterior muscles. Also, correcting poor foot mechanics, such as overpronation, and inadequate running mechanics should be addressed. Ensuring proper footwear entails keeping a running log of miles run, in order to change shoes every 300 to 400 miles. These details, along with avoiding the too much, too soon training error should help to decrease overuse injuries in the lower extremity. Runners should be cognizant of this training regimen on a daily basis, with the intent to prevent MTSS before it starts.

Standard Treatment

Initially, the treatment of MTSS involves pain control and relative rest to reduce lower leg stress. This may require decreased mileage, walking, cross-training, immobilization, or even no weight bearing. Additional modalities, such as ultrasound, phonophoresis, cortisone injections, and electrical stimulation, are also used. The level of weight bearing is correlated with the duration and severity of the symptoms.

Once the pain and inflammation are controlled, runners can be counseled on cross-training exercises, to maintain cardiovascular fitness. Exercises should be pain-free, with minimal ankle plantarflexion and dorsiflexion. Some of the better choices are water-running, upper-body ergometer, and rowing machines. As injury rehabilitation progresses, one can add the elliptical machine, stationary bike, and flat surface walking/jogging.

Once pain-free, the athlete should progress to gradual incteases in flexibility and strengthening, as delineated previously. Exercising the deep compartment muscles, such as the soleus and tibialis posterior, helps to strengthen the exact fascial–bone interface that caused the injury (21). Basic rehabilitation tenets should be followed, including the progression from isometric exercises to isotonic exercises. As the level of fitness increases, the athlete should extend to isokinetic exercises and more functional activities, such as the elliptical machine and balancing exercises. Finally, when adequately strengthened and functionally stressed, the athlete can return to a gradual running program. This should be completely pain-free. Beginning with flat, forgiving surfaces is imperative. Any pain experienced during these trial runs should alert the athlete to stop and visit the physician or athletic trainer for reevaluation. Conservative therapy is generally effective for most cases of MTSS, but for those unresponsive to treatment, surgical release of the deep fascia is an option (23). Surgery is rarely needed for an accurate MTSS diagnosis.

Manual Techniques

Correcting the biomechanical abnormalities of the runner with MTSS is paramount. This includes a thorough analysis of the walking and running gait by a sports medicine specialist, including physicians, physical therapists, or foot specialists. From this analysis individualized orthotics should be designed, with the purpose of correcting any midfoot pronation or excessive movements. While the orthotics are being produced, arch taping can be a helpful technique to stabilize excessive pronation. Also helpful for decreasing excessive foot motion is limiting any forefoot varus present with a medial forefoot post (21).

Because the expected etiology of MTSS pertains to the musculoskeletal system, it is extremely amenable to manual medicine. The goal of manual medicine therapy in the treatment of MTSS is to improve musculat contractility, relaxation, and remodeling, as well as decrease inflammation. These are accomplished by the improved vascularity and lymphatic flow facilitated through various techniques. Manual medicine

should not be used independently, but should be included in the comprehensive recovery plan for the runner. As previously discussed, muscle energy, myofascial release, and counterstrain are all viable options for the treating physician.

Several common somatic dysfunctions in MTSS have already been discussed. Anterior and posterior fibular head dysfunctions are common with MTSS and can be treated with HVLA techniques. Muscle energy treatment is effective for lengthening and relaxing the gastrocnemius-soleus complex. The treating physician can add ankle inversion and eversion to the technique, in order to apply different angles of stress to the complex.

Myofascial release should be concentrated on areas of warmth and restriction. When applying one's hands to the injured area, the physician must be certain to carry on an interactive conversation with the athlete. It is important to know how the therapy feels to the athlete, whether it is painful or soothing. Ask the athlete if he or she feels the tissues relaxing when you feel as though they are relaxing. This can make for a more effective treatment session if the physician helps facilitate the athlete's sense of self-perception.

With regard to MFR, remember to use indirect motions when the runner is in acute pain. Techniques that increase pain perpetuate the pain cycle and cause more injury. Use different techniques for the release. Both horizontal and vertical movements can be used, as well as a wringing motion of the lower leg. These help to affect all planes of motion. Soft tissue techniques to the plantar surface of the foot are effective in loosening any attachment adhesions built up from overuse injury.

A common somatic dysfunction associated with MTSS is anterior displacement of the distal tibia on the talus. This abnormality applies undue stress to the lower leg musculature. In relieving this dysfunction, one releases the restriction allowing normal biomechanics to occur. The HVLA technique for this dysfunction is explained in Chapter 25 on gait analysis (13).

Counterstrain tender points located throughout the lower leg should be treated to help relax the myofascial connections in the lower leg. By allowing the overstressed muscles to relax and regain their original structure, the body's homeostatic mechanisms for repair and healing can take over and facilitate a more efficient return to running.

Iliotibial Band Friction Syndrome

Iliotibial band friction syndrome (ITBFS) is the third most common running injury (7) and the second most common overuse injury (15). This syndrome describes a diffuse lateral knee pain just above the lateral joint line, sometimes extending down to the insertion of the iliotibial band into the lateral tibial tubercle (Gerdy's tubercle) (24). Among all reported overuse injuries involving runners, the incidence of ITBFS varies from 1.6% to 12% (25). (See Chaptet 23.3 for more information.) Hip abductor weakness is often related to symptoms (24).

Biomechanics and Etiology

Factors involved in developing ITBFS include such training variables as excessive mileage, sudden increases in training intensiry and duration, running on crowned surfaces, interval training, excessively worn or inappropriate running shoes, and cross-training with excessive cycling. (See the discussion in Chapter 23.3 for structural factors of ITBFS.)

Prevention

Preventing ITBFS is best achieved by controlling the etiologic factors. A training program with healthy habits and schedules should be implemented with no sudden increases in mileage and intensity. Smart planning and running journals assist in proper preparation for long runs and races. Adequate warm-up and stretching, particularly the tensor fasciae latae, hamstrings, quadriceps, and hip adductors, is helpful (although controversy exists as to whether or not stretching is inherently protective). Icing the lateral knee after running can be enough to head off the condition. The runner should note any subtle preliminary signs, such as palpable or audible popping of the tendon over

the lateral femoral condyle, accompanied by mild pain.

Manual Medicine Treatment

The primary goal is to relax the tissues and allow them to regain their preinjury status. Muscle energy, myofascial release, counterstrain, and deep tissue massage are effective in achieving this goal. Manual medicine should also address structural dysfunction, especially in the hip, that may predispose the runner to ITBFS.

Applicable techniques include

Counterstrain to the gastrocnemius and soleus.

Cross-friction massage or petrissage to the distal iliotibial band.

Muscle energy to the hip flexors.

Muscle energy to the hip abductors (see Chapter 22).

Myofascial release to the iliotibial band and tensor fasciae latae.

Myofascial release to the hip girdle.

HVLA or muscle energy for a fibular head dysfunction.

Treatment positions for myofascial release techniques and soft tissue releases are dependent on the severity of the syndrome. If the athlete can tolerate it, perform myofascial release on the iliotibial band with the athlete positioned in the lateral recumbent position on the unaffected side (Fig. 37.5). From this position, both myofascial release and alternate muscle energy techniques can be used (26).

Try to release the superficial tissues first, and in several different planes of motion. An alternative to the slide and hold technique is the kneading technique. This form of soft tissue treatment involves repeated kneading of the tissue, either into or away from the barrier. It can be especially effective in mobilizing fluid and edema.

Myofascial Release: Hip Circumduction with Stabilization at the Sacroiliac Joint

1. The athlete lies on the unaffected side, with his or her back toward the clinician.

FIGURE 37.5. Myofascial release to the iliotibial band.

2. The clinician grasps the popliteal fossa of the affected side with the caudad hand, while stabilizing posteriorly with the cephalad hand on the sacroiliac joint.
3. While supporting the cephalad hand's elbow on his or her thigh, the clinician's caudad hand brings the affected leg through the complete hip range of motion.
4. This motion is gently performed several times, each time expanding the circle of motion. Often, a release is palpated, allowing increased range and quality of motion (Fig. 37.6).

In the extremely acute and painful athlete, the best form of manual therapy could be counterstrain, which is completely passive and low stress. Typical counterstrain tender points are along the lateral surface of the thigh. There are three that have been publicized in the literature: posterolateral (PL) trochanteric, lateral trochanteric, and posteromedial trochanteric (8).

Since many runners with ITBFS develop tight and painful hip flexors, treating hip trigger points with counterstrain can be beneficial (Table 37.4). The lower leg should be treated as well (Table 37.5).

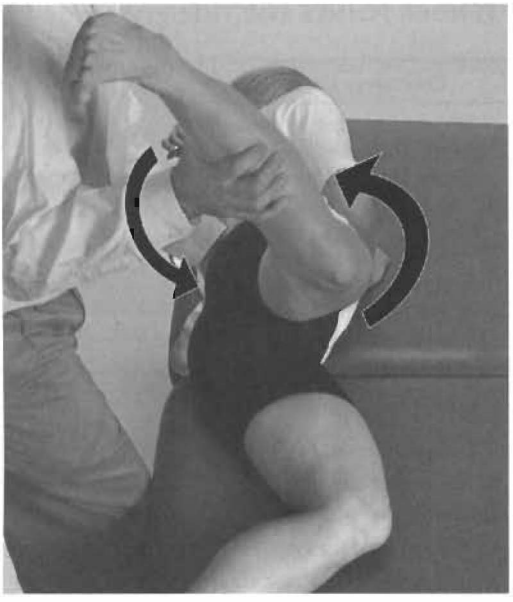

FIGURE 37.6 Myofascial release: hip circumduction with stabilization at the sacroiliac joint.

Metatarsalgia

Metatarsalgia is an overuse injury caused by excessive pressure on the metatarsal heads. It is often the result of improper biomechanics of the lower limb. The pain that a runner reports with metatarsalgia is most often felt at the base of the second metatarsal head. It is associated with a short, hypermobile first metatarsal (first ray syndrome), posteriorly displaced sesamoids, and a thickened second metatarsal shaft. Injury occurs due to altered biomechanics of weight bearing (i.e., hyperpronation), with repetitive stress shifted to the metatarsal heads. Pain in this syndrome can occur at any of the metatarsal heads, but is most commonly felt at the second head. A contributing factor to this condition can be a tight Achilles tendon, which increases forefoot load during the late stance phase (27).

Biomechanics

The normal gait cycle has the foot spending 62% in stance phase (heel strike, foot flat, heel rise, and toe-off) and 38% in swing phase. The point of maximum stress is late in stance phase during heel rise and toe-off. The center of pressure starts in the medial portion of the heel, moving quickly across the midfoot to the forefoot. In the forefoot, the main pressure is under the second metatarsal head. As the foot toes off, the high point of pressure is under the hallux. During examination, calluses or wear areas of footwear can suggest mechanics or alignment dysfunctions aiding in the diagnosis.

In a healthy foot, weight bearing is shared by the metatarsal heads and the calcaneus, with the location of increased force depending on the phase of gait. Metatarsalgia, on the other hand, occurs because one or more of the metatarsal heads is absorbing more force than its counterparts.

TABLE 37.4. COUNTERSTRAIN TENDER POINTS FOR THE HIP

	Tender Point Location	Counterstrain Position
Posterolateral (PL) trochanteric	PL surface of greater trochanter	Hip extended and abducted; fine-tuning with external rotation
Lateral trochanteric	5–6 in. below greater trochanter, laterally	Leg abduction; fine-tuning with flexion
Posteromedial trochanteric	2–3 in. below trochanter, along posterior shaft of femur, toward ischial tuberosity	Thigh extended, adducted, and externally rotated

Adapted from DiGiovanna EL, Schiowitz S. *An osteopathic approach to diagnosis and treatment*, 4th ed. Philadelphia: Lippincott Williams & Wilkins, 1997.

TABLE 37.5. COUNTERSTRAIN TENDER POINTS FOR THE LOWER LEG

	Tender Point Location	Counterstrain Position
Iliacus	Just superior to the midpoint between the ASIS and pubes	Athlete is supine; knees and hips are flexed; fine-tuning with rotation toward tender point
Psoas	Just inferior to the midpoint between the ASIS and pubes	Athlete is supine; knees and hips are flexed (slightly less than iliacus); fine-tuning with rotation toward tender point

ASIS, anteriorsuperior iliac spine. Adapted from DiGiovanna EL, Schiowitz S. *An osteopathic approach to diagnosis and treatment*, 4th ed. Philadelphia: Lippincott Williams & Wilkins, 1997.

Etiology

Metatarsalgia is another of the common overuse injuries found in runners. One metatarsal is commonly longer in comparison to its neighbors, which creates an imbalance in support that often ends with bruising of the tissues of the distal metatarsal head. Overuse compounds the problem.

A common differential to be considered is Morton's, or interdigital, neuroma. This is a mechanical entrapment neuropathy of the interdigital nerve (28), most often occurring between the distal portions of the third and fourth metatarsals. Lateral compression of the metatarsal heads usually provokes pain. Examination includes visual inspection of the foot and footwear, and motion testing of the joints including the ankle. There should be similar flexibility and play of the joints in both feet.

Excessive pronation and supination can contribute to metatarsal pain, as abnormal motion shifts the force angles throughout the foot. Overpronation often places increased stress on the medial metatarsals. In a foot with a long second metatarsal, this stress can be exaggerated. Likewise, in an oversupinated foot, a stress imbalance can be created in the fourth and fifth metatarsal heads.

Prevention

Prevention of metatarsalgia begins with careful attention to training methods. The 10% to 30% rule is a good place to start. Quality, proper-fitting footwear is just as important. Many runners buy two or three pair of running shoes and alternate them to avoid back-to-back days wearing the same shoes. Runners should change shoes with excessive wear and miles. Runners should watch for subtle signs of shoe failure, such as increased fatigue after a typical run over 1 to 2 weeks, heavier ground impact with the foot, and excessive ankle eversion. Gait and foot analysis may identify technique flaws, which may require orthotics or a specific type of running shoe (29).

Standard Treatment

After standard acute care (rest, ice, compression, and elevation—RICE protocol and NSAIDs), a metatarsal pad placed proximally to the offending metatarsal head may reduce injury time as well as prevent problems from arising (28). The pad spreads the metatarsal heads, dispersing the force of gait away from the injury site. This allows a more equal sharing of body weight. Many of the criteria for return to activity have been mentioned previously, but a review with focused application is useful. A thorough lower limb motion and function examination can provide information that may indicate secondary causes of the problem. A progressive return to running should be established for those with significant dysfunction.

Manual Medicine Treatment

Several areas should be evaluated and treated. Applicable techniques are as follows:

- Tarsal dysfunctions (cuboid, navicular, or cuneiform)—HVLA.
- Achilles tendon, gastrocnemius, and soleus tender points—counterstrain and muscle energy.
- Proximal and distal fibular head dysfunctions—HVLA and muscle energy.
- Pelvis—muscle energy, counterstrain, myofascial release, depending on the findings.

A common finding with metatarsalgia is plantar displacement of any of the tarsal bones. For instance, the cuboid medially rotates upon an anteroposterior axis and presents its medial border on the plantar surface of the midfoot. To correct this, use the cuboid release found in Chapter 24.3. Cuneiform and navicular dysfunctions are treated in a similar fashion.

CONCLUSION

Evaluation and treatment of the injuries presented in this chapter, as well as any running injury, demonstrate the interrelatedness of the person, the pattern, and the outcome. Structure and function are interrelated, and the body functions best when fluids flow unhindered by the effects of injury and disease. The body can repair itself, given the chance. This philosophy of whole body health is intrinsic to the philosophy of many runners.

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38

SOCCER

ZENOS VANGELOS

Soccer is a sport that attracts a diverse population of participants. Worldwide participation is certainly one example of this claim. However, diversity in soccer can be seen in a variety of other situations. Consider the fact that soccer is played by athletes of all ages. There are games played among children, whose shins are approximately the same length as the diameter of the ball. On the other hand, there are organized leagues, especially in Europe and South America, for seniors extending well into their 70s.

Soccer is also nondiscriminatory when it comes to body type. There are obvious height and weight requirements in sports such as football and basketball. This is not the case in soccer. A player's height may somewhat determine the position that he or she plays; however, both short and tall people have equal opportunity to excel professionally in this sport.

Gender also does not discriminate participation in soccer. Men and women have equal opportunity to participate. With the recent successes of both U.S. men's and women's soccer teams, it appears that men and women in the United States have had ample access to training and facilities.

Another factor that has made soccer accessible to a great population of people throughout the world is the fact that a team can be outfitted with fairly modest financial means. Compare this to a hockey team whose goalie pads probably cost as much as a full soccer team's set of uniforms and balls. This makes soccer truly accessible to both rich and poor, and has allowed people such as Pelé, who came from the Brazilian ghetto, to become a household name throughout the world.

The game itself speaks to diversity by virtue of the fact that it is played year-round. Spring, summer, and fall seasons are played outdoors. During the winter when fields are not accessible, it is played on an indoor field either in a gymnasium or on artificial turf.

THE GAME

The preferential use of the lower extremity in soccer leaves a very limited role for the upper extremity. There are only two situations in which a soccer player can touch the ball with his or her hands. One situation requires a player to throw a ball onto the field of play after it has been knocked out of bounds. This maneuver must be accomplished with two hands, requiring the player to hold the ball above his or her head and in some situations use the whole body in a whiplike action to propel the ball. A nuance to the throw-in includes using more of one hand than the other to put a spin on the ball. However, if there is too much use of one hand it can be deemed an illegal throw-in.

The other situation involves the goalie, who is the only player on the field allowed to touch the ball with his hands. This is extremely advantageous to the goalie since his or her primary way of stopping goals is either deflecting or catching the soccer ball. Catching the ball is preferred, so that no deflections can be kicked in for a goal. The goalie is also allowed to advance the ball by throwing it. This is typically done in an overhand bowling motion as opposed to the motion used by baseball and football players.

The upper extremity is also used in contact with opposing players in the struggle for ball control, field position, and jumping for head balls. These uncommon injuries to the upper

extremity are very amenable to myofascial release and a variety of osteopathic manipulative therapy. Goalies are particularly vulnerable to hand and finger injuries including contusions, dislocations, and lacerations from being stepped on by opposing players. Mobilization procedures are effective in reducing edema and improving range of motion with these injuries. It is important to also assess the cervical spine and specifically the scapulothoracic joint to look for related somatic dysfunction.

It is no surprise that soccer is thought of as a predominantly lower extremity sport. Certainly, the most efficient and often-used way to advance the ball and score a goal is by use of the foot. The rules of the game rarely permit players to use their hands other than the goalie. However, soccer is like many other sports in which the total body must work in a coordinated fashion.

A good example is the player who is attempting to shield the ball from an opposing player. He or she must first use superior foot skills and coordination of the lower extremity to maintain the ball in his or her possession. The player also must use the pelvis and thorax in an effective manner to shield the ball from the opposing player. His or her upper extremity is also being used in the shielding process for balance, and for obstructing the opposing player. The player then uses his or her lower extremity to pass the ball to a teammate and uses the whole body to avoid and separate from a defender. Once this is accomplished, he or she runs toward the goal, jumps in the air, and hits the ball with his or her head, a ball that is traveling at speeds of up to 80 and 90 mph. He or she twists and contorts the entire spine in ordet to powerfully deflect the ball into the net. This is but one scenario in soccer, which requires a coordinated effort of the entire body.

This example is reminiscent of the holistic approach that is central to osteopathic medicine. It also shows that soccer players require total body fitness to play the sport. This full level of body involvement means that injuries can occur in almost any part of the body. Manual therapy has been beneficial in treating soccer injuries and works as a powerful adjunct to physical therapy and rehabilitation. A holistic approach remains critical in treating these athletes.

ATHLETES

A soccer player must show athletic diversity. Most individuals who watch soccer would consider it an aerobic sport. However, exercise physiologists who have studied soccer suggest that soccer consists of a more equal division between aerobic and anaerobic capacity. Though a soccer player does run close to 6 miles during a game, much of this running is done in bursts. Therefore, one needs both aerobic capacity to endure the 6 miles of running, and also the tremendous anaerobic capacity to perform the sprints and cuts that are key to the game.

Diversity also applies when considering the skills involved in participating in soccer, and the injury patterns that occur. Most people look at soccer and think that the only concern soccer players have is injury to their legs. Contrary to this belief, soccer is a sport in which the whole body is working to propel either the player and/or soccer ball to a specific point with varying levels of speed and strength. With this in mind, the pelvis becomes a good starting point for examining skills specific to soccer.

COMMON INJURIES

Epidemiology

Although the whole body needs to be functioning properly to be playing soccer at peak performance, the majority of the work—and the injuries—involve the lower extremities. According to some studies, as many as 68% to 88% of all soccer injuries involve the lower extremity (1). The knee and ankle are responsible for the vast majority of injuries. One retrospective study of 150 female soccer players found that injuries to the ankle and knee accounted for 39.5% and 16.9% of the 248 injuries, respectively (2). Another study found that 49% of the 78 injuries in female soccer players occurred in the knee and ankle (3), and a third study found 89% of 79 injuries in the lower extremity (4). Female

soccer players have a higher risk of injury, in some cases twice as high as males. Factors include poor hamstring to quadriceps strength ratio and hyperextension of the knee (5).

Elias looked at 90,000 adolescent international soccer players and evaluated 3,840 new, play-related injuries during 290,344 player-hours of competition from 1988 through 1997 (6). He noted that 65% of the injuries occurred in the lower extremity, with ankle sprains being the most common injury. Head and neck injuries occurred in 13.6% of injured sites. Upper extremity and trunk injuries made up 12.3% and 8.6%, respectively. Female injury rates were higher than males in this study: the female rate was 14.15 per 1,000 player-hours, while the rate for males was close at 12.69 per 1,000 player-hours. In other youth tournaments, the disparity can be small (8.1 vs. 7.6 in the 1993 Norway Cup) or large (17.6 vs. 9.9 in the 1984 Norway Cup), but through the years, the difference has diminished (6). Despite the high numbers, most injuries are minor.

Elias did note in his study that the injury rates over the years have decreased in both the Norway Cup (1975 to 1997) and the USA Cup (1988 to 1997), while the occurrence of catastrophic injuries is relatively rare (6). This is reassuring to those trying to improve injury rates thtough equipment and training.

Pelvis

The pelvis acts as a base from which all of the other body parts work. Soccer coaches understand this, since they teach players to watch a player's hips rather than his or her legs when pursuing the ball. A player can move the ball in many directions without moving but cannot propel himself or herself in a certain direction without moving the pelvis. This results in a tremendous amount of torsional stress throughout the pelvic girdle into the sacroiliac joints and also to the articulations between the spine and the lower extremity. There is significant energy transference during running and stop-and-go movements, which are common in soccer. This is accentuated in the indoor game where the floor is less forgiving.

These overloads can be acute, for example, the player who is driven to the ground landing on some part of his or her pelvis, or of a chronic nature due to repetitive running, placing torsional forces on the pelvic girdle. Common somatic dysfunctions of the pelvis include upslip innominates, sacral torsions, and pubic shears. Typically they are seen in combination, rather than one particular area being affected. High-velocity, low-amplitude (HVLA) manipulation has been very effective in treating these areas. Other forms of manual therapy including, but not limited to, sacral release are also very effective. It is very important to treat the pelvis as a whole. There should not be a correction of a sacral torsion without correction of a pubic shear and any other associated dysfunction that has been discovered. The pelvis is basically a ring, so that any manipulation that is done anteriorly affects manipulation posteriorly, and vice versa. It is important to remember all basic tenets of osteopathic manipulative therapy when assessing and treating somatic dysfunctions in the pelvis.

An interesting athletic injury of the pelvis first noted in soccer is the "sports hernia," or athletic pubalgia. This involves a vague pain in the inguinal region adjacent to the superior pubic ramus. Injury occurs in the connective tissue and abdominal wall due to the shearing forces across this area where abdominal and adductor musculature attaches.

The best theory is that an imbalance exists between the strong adductor muscles and the relatively weak lower abdomen, which leads to attenuation, avulsion, or tearing of structures in the pelvic floor. Studies have implicated several structures intimately involved in the region, including the transversalis fascia, internal oblique muscle, external oblique muscle and aponeurosis, and even the genital branches of the ilioinguinal or genitofemoral nerves (7). However, more research is needed to better isolate the culprit and the cause in order to facilitate timely recovery.

Clinical features include a vague pain in the region that resembles the pain of a hernia; however, hernia is not found on examination. Typically, this is a diagnosis of exclusion ruling

out such injuries as adductor strains and true inguinal hernias. In its early and mild stages this injury is very amenable to manual medicine, especially since physical therapy and surgery are the only other alternatives. The goal of treatment is to correct the imbalances in pelvic range of motion and strength. Muscle energy and hip mobilizations are effective in reestablishing proper hip extension and rotation. Innominate rotation, sacral torsions, and lumbar dysfunctions should be identified and corrected using whichever techniques the clinician feels comfortable with. Once the pelvis has symmetry, core strength needs to be developed to link the upper body and lower extremities and defend against the twisting oblique forces that soccer generates.

Lower Extremity

The only area of the body used to propel the soccer ball more frequently than the head and neck is the lower extremity. Lower extremity control and coordination are paramount to the game of soccer. These attributes allow a soccer player to acquire the foot skills necessary to protect the ball from opponents, trap a ball, pass, dribble, and shoot with power and accuracy. Soccer skills require excellent muscle strength as well as gross and fine motor coordination. There also needs ro be a balanced level of aerobic and anaerobic capacity so that the soccer player can both endure the length of the game and accomplish the constant starting and stopping involved in the attacking and defending portions of the game. This level of involvement puts the lower extremity at high risk for injury.

Contusions, sprains, and muscle strains of the lower extremity are inevitable in soccer players. They occur in contact situations when two players are vying for a ball, in collisions, and in falls. An uncommon contusion that can be devastating to soccer players is myositis ossificans. This injury typically occurs in the anterior quadriceps after the player is kicked. Because relative rest is extremely important in the treatment of myositis ossificans, a soccer player may be lost for several weeks. Adjunct treatment for this problem includes oral medications and rehabilitation. Manipulation typically in the form of indirect techniques as well as myofascial release are excellent techniques for improving muscle elasticity in this type of injury and in any type of contusion.

Muscle strains can be found throughout the lower extremity. Strains of the hip are very amenable to myofascial release and many other direct and indirect techniques. It is important to assess the pelvis in treating hip strains since it can also be involved. Quadriceps strains are very common in soccer players because the quadriceps muscle is a major muscle used in kicking, passing, blocking, and dribbling the soccer ball. This dominance of the quadriceps muscle often causes imbalance in the quadriceps–hamstrings strength ratio, thus causing injuries to the hamstring muscle. These strains as well as strains to the gastrocnemius musculature can all be improved with manipulative therapy that concentrates on improving muscular elasticity and joint mobility.

Ligament injuries of the lower extremity are typically more serious and difficult to treat. They include anterior cruciate ligament tears, medial collateral ligament tears, meniscal tears, and various levels of ankle and foot sprains. Obviously, osteopathic manipulative therapy is not the treatment of choice in these issues. However, manipulation can be an excellent adjunct for improving range of motion and decreasing edema. Techniques including myofascial release and lymphatic pump can be readily used. Fortunately, simple ankle sprains make up the majority of injuries in this category. When assessing and treating the ankle, it is important to remember that there can be dysfunctions in the fibular head. HVLA mobilization of the fibular head can correct subluxations in the region and speed ankle sprain recovery.

Contusions, sprains, and strains can accumulate placing the soccer player at greater risk for arthritis. Subsequently, degenerative changes can be seen at any joint in the lower extremity. One joint in particular, the first metatarsophalangeal (MTP) joint, is very commonly

involved. The first MTP joint is being constantly used by soccer players. It is stressed while running, stopping, and cutting. It is also involved in virtually every touch the soccer player puts on the ball. Depending on the level of arthritis, mobilization techniques can be very effective in reducing pain and maintaining motion in the first MTP joint as well as any affected joints in the lower extremity.

MANUAL TECHNIQUES

Manipulation in the pelvis is closely associated with manipulation in the spine due to their intimate anatomic location. The lumbar spine sits atop the sacral base, which forms the posterior aspect of the pelvis. Movement in the lumbar spine is key to soccer, since it helps in trying to outmaneuver an opponent by shifting the torso side to side. There is also quite a bit of torsional force through the lumbar spine when the ball is kicked. The spine also plays a role in positioning when a player jumps to strike the ball with his or her head. These types of maneuvers, as well as associated trauma from falls, tend to cause a variety of somatic dysfunctions in the lumbar spine, most of which are found in the L4-S1 region. In particular, players who fall on their pelvis can traumatically cause innominate rotations, shears, and sacral torsions that may not resolve with stretching alone. When evaluating and treating the spine, the pelvis has to be included.

Spinal manipulation is generally very successful in the lumbar spine. When one or two sessions of manipulative therapy have not helped, a thorough workup including radiologic evaluation should be considered. Soccer ranks high among contact sports in the development of active spondylolysis. This injury typically occurs from repeated extension stress to the lumbar spine, and most commonly is noted in the L5-S1 region. Some have theorized that soccer players are more prone to this injury because of the amount of extension that they do while kicking a soccer ball; however, this theory has never been proved. No one has been able to identify the factors that make soccer players more prone to spondylolysis. When spondylolysis is detected, rest, bracing, and rehabilitation may all be useful. Unfortunately, manipulation, although it may give some temporary relief, does not play a major role in a true spondylolysis.

Just as the pelvis is interconnected to the lumbar spine, the thorax connects to the lumbar spine; thus, there is interplay between the thoracic spine, chest and ribs, and the lumbar spine. Because soccer players are not allowed to use their hands, the chest plays an important role with balls that are played in the air. It can be used to block a ball, deflect a ball to one's feet, as well as redirect the ball to another player. Accomplishing this requires synchronized movement among the thoracic spine, ribs, and anterior chest muscles. Playing the ball with the chest requires scapular retraction and thoracic extension, as well as associated movement within the costovertebral joints. Repetitive torsional movements can cause chronic somatic dysfunctions in these areas as well.

There are also acute injuries that occur in the thoracic spine and ribs. Some occur due to falls, which are typically more injurious in the indoor game. Still others occur as the upper body and torso are used to block opponents from the ball and jostle for position. This leaves a player susceptible to somatic dysfunctions of the ribs, thoracic spine, and acute injuries including contusions and rib fractures. In the case of a rib subluxation, the athlete may complain of pain with deep inspiration radiating around from the back.

Once physical examination and diagnostic testing have ruled out acute bony pathology such as rib fractures, manual medicine can be applied to the ribs and thoracic spine in much the same way as described previously for the lumbar spine and pelvis. The use of Jones's tender points has been particularly helpful in both anterior and posterior rib lesions that have been unsuccessfully treated with HVLA manipulation. Somatic dysfunction in the lumbar spine may deter or prohibit correction of dysfunction in the thoracic spine.

During competition, HVLA techniques can be applied to a soccer player with an acute costothoracic dysfunction, as long as the musculature is not in spasm and there is no direct blunt trauma that could cause other pathology, such as a rib fracture or pneumothorax. If there is shortness of breath after an injury, as opposed to pain with deep inspiration, then HVLA should be withheld. Again, indirect techniques can always be applied based on the athlete's tolerance, but they tend to be minimally effective in the middle of competition.

The thoracic spine is also affected by issues concerning the cervical spine. The head and neck play a unique role in the game of soccer in that they are used to propel the ball both to pass and score goals. The head is used to apply a force to the ball, while the neck is used to direct the ball in a certain direction. The neck is also used in a whipping motion to apply a vector force to the ball. This is quite an intricate process, since the neck sometimes has to redirect the ball which is coming toward a player's head at speeds of up to 80 and 90 mph. It requires the neck to position the head correctly with intricate movement, and also redirect the tremendous amount of force of the ball as it hits the head. These activities put the cervical spine and its surrounding musculature in a very vulnerable position.

Consequently, cervical somatic dysfunction is readily seen in soccer players. The whipping motion just described is also discussed in Chapter 16.3 as a mechanism for whiplash syndrome. Keep this in mind as you evaluate the soccer player with a heading injury. These dysfunctions can be found at almost any cervical segment and also in the anterior cervical muscles. Occipitoatlantal and atlantoaxial dysfunctions are seen more commonly in soccer players than in the normal population. Because these segments provide the greatest amount of rotation in the cervical spine, they tend to become more vulnerable when they are stressed to their limits while having a shearing force applied to them. This occurs when a player is rotating his or her neck while hitting the ball at the same time.

Another common problematic area is the cervicothoracic junction. The whiplike action that is used to strike the ball can put tremendous shearing forces across this region. This occurs when force is moving through mobile segments of the cervical spine and then is stopped at a stable first thoracic segment.

HVLA manipulation of the cervical spine typically provides very effective relief for these dysfunctions. Unfortunately, many people express concern over manipulation in the neck. Fortunately, muscle energy and craniosacral techniques, such as an occipital release, are also extremely effective in correcting these somatic dysfunctions. Review Chapter 16.3 for details on these techniques.

PREVENTION

Any contact sport will have injuries as part of the game. However, soccer players can train their bodies to avoid injuries. Extrinsic factors need to be controlled as much as possible. The two most common factors are playing surface and shoewear. Many men's soccer teams in high school share their field with the football teams, who tend to damage natural turf, causing irregularities and defects that increase the risk of injury. The new artificial turf fields have more give and cushion, yet maintain a flat surface that wears little. The technology is relatively new, so injury data are limited.

Junge et al. discussed the success of their program in preventing soccer injuries. Their recommendations are warm-up, regular cool-down, taping of unstable ankles, adequate rehabilitation, and promotion of the spirit of fair play as well as specially designed F-MARC Bricks. F-MARC Bricks are ten sets of exercises designed to improve the stability of ankle and knee joints, the flexibility and strength of the trunk, hip, and leg muscles, as well as to improve coordination, reaction time, and endurance (8).

Preseason conditioning is a part of most teams' preparation and has been shown to decrease injuries in adolescent female athletes (1). Shin guards have been shown to prevent tibia fractures and contusions, although different types of guards yield varying levels of protection (9,10).

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39

VOLLEYBALL

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Since its invention in 1895 in Massachusetts by William G. Morgan, volleyball has been one of the most popular team sports. According to its international governing body, the Federation Internationale de Volleyball (FIVB), volleyball is one of the big three international sports. With its 218 affiliated national organizations, the FIVB is the largest international sporting federation in the world. Men and women of all ages and skill levels participate on both indoor hard and outdoor sand courts. The first world championships took place in Prague, Czechoslovakia, in 1949. Volleyball made its Olympic debut in 1964 in Tokyo, and beach volleyball made its Olympic debut in 1996 in Atlanta. All levels of sport participation continue to grow exponentially today.

DESCRIPTION

Volleyball is a game played by two teams consisting of six players per team on a court measuring 60 ft in length by 30 ft in width and separated by a net. Beach volleyball consists of two teams with two players each. The net height measures 7 ft 4 in. in women's play and 8 ft 0 in. in men's play. Volleyball is purely a rebound sport (one cannot hold the ball). The game consists of passing (bumping), setting, spiking, digging, blocking, and serving the ball. Players each have starting positions and rotate each time a sideout occurs. A sideout occurs when a team fails to serve the ball over the net, hits the ball out of bounds, lets the ball touch the ground, or fails to return the ball back over the net after touching it three times.

Traditionally, the six positions are back right, back center, back left, front right, front center, and front left, with each player having a special assignment. One team initiates play by serving the ball over the net. The opposing players in the back row pass (dig/bump) the ball to the setters. The setters set the ball up to the spikers. Spikers (hitters/attackers) hit the ball over the net into the opponent's court. This is called a bump-set-spike pattern. Blockers try to block the spiked ball as it crosses the net. If the block fails, then the opposing team initiates the bump-set-spike pattern, and so on. Depending on the level of play, a point is awarded to the serving team each time the opponent makes a sideout. During rally scoring, a new type of scoring adopted by colleges and international play, a point is awarded with every sideout regardless of which team actually served the ball.

The sport is played at many different levels including grade school, high school, college, professional, and Olympics. Scoring differs among different levels of competition. Currently, the best three out of five games are played at the high school level, and the first team to score 15 points wins the game. In college, club volleyball, professional, and Olympic competitions, the match is also played for best three out of five games, but the first team to score 25 or 30 points wins. Usually, a team has to win the set by two points.

REQUIRED SKILLS

1. **Pass.** This is also known as *bump*. The player passes or bumps a serve or a free-ball. If a ball comes over the net via a spike, the return is normally called a *dig*. In order to pass a ball, the player's elbows are extended

out in front of him or her with hands clasped together below the waist to form a platform to pass the ball to the desired target. The player is also in a ready position characterized by feet shoulder width apart and knees bent at approximately 30 degrees. This allows the player to be able to move quickly to the ball and get in the desired position to pass the ball as accurately as possible to the setter.

2. **Set.** To set the ball, a player's hands must be open and relaxed. The hands are placed about 6 in. from the player's forehead with index fingers and thumbs forming a triangle. In addition, the elbows are flexed at about 90 degrees. When the ball approaches the hands, the ball falls into the player's hands and then the wrists flex and the elbows extend to send the ball to the desired target, the hitter (attacker/spiker).
3. **Spike.** First, the player must approach the net to get momentum to jump and attack the ball. The approach consists of either a three-step or four-step approach taken from the 10-ft line. During the last two steps, the player flexes his or her knees, plants the heels, swings the arms back, and then explodes upward and swings at the ball at the top of his or her jump. The player uses a forward overhead arm swing to spike the ball over the net and into the opponent's court.
4. **Block.** The player faces the net approximately one step back from the net with his or her body in ready position (elbows and shoulders at 90 degrees of flexion and knees slightly flexed). This allows the player to be able to move side to side and then to jump with the arms straight overhead to attempt to block the opponent's spike from reaching his or her side of the court.
5. **Serve.** Serves can be either overhead or underhand. A player can perform an overhead serve from either a stationary position or perform a jump serve. From a stationary position, the player tosses the ball up into the air with the nondominant hand so that upon impact the ball will be able to clear the net on a fly. The player can either serve a floater or a top-spin serve, both of which use and stress different muscle groups. The jump serve is performed exactly like a spike approach except that it is served from the back line of the court.
6. **Dig.** A dig is very similar to a pass or bump, but the player must be able to get very low to the ground. This allows the athlete to reach the arms out as far or as low as possible to dig up the hard-hit ball by the opponent spiker. The athlete may even have to dive out onto the floor or sand to attempt to get the ball.

By its nature, volleyball is a quick sport that demands both power and finesse. As such, many qualities allow the athlete to excel in volleyball (Table 39.1).

TRAINING

A comprehensive training program that emphasizes maximizing the aforementioned qualities is essential for athletes to excel in

TABLE 39.1. REQUIRED QUALITIES FOR EXCELLING IN VOLLEYBALL

Quick coordinated movements (start/stop/change direction)	Endurance (for long matches/ tournament play)
Good vertical jump (powerful legs)	Hand-eye coordination
Accuracy	Soft touch (for setting)
Flexibility	Strong rotator cuff (for serving/spiking)
Good core strength/balance	Strong latissimi (for blocking)
Strong triceps/pectorals (for setting)	
Good lateral speed	

volleyball and minimize injury (1). Practices should include a combination of weight training, stretching, aerobic fitness, plyometrics, court/agility drills, scrimmaging, and psychological preparation. Scrimmaging and psychological preparation help develop accuracy and hand-eye coordination. Aerobic training provides good endurance to play in 8-hour tournaments. Weight training, stretching, plyometrics, and court drills enhance power, flexibility, and agility. In particular, plyometrics has become a staple of female conditioning programs because the exercises reduce the risk of anterior cruciate ligament (ACL) injuty (1,2). Recent evidence suggests that teaching players to land on both feet with the knees slightly flexed and the ankles plantarflexed helps to minimize knee injuries (especially ACL tears) by dissipating the forces more evenly. Indeed, most takeoffs occur using both feet, and as a result very few acute knee injuries occur during the jumping phase. On the other hand, fewer than half of the landings occur with both feet, which causes the vast majority of acute knee injuries (3).

PREVENTIVE EXAMINATION

The pre-participation examination provides an excellent opportunity to address specific areas of the body that require proper function, strength, and range of motion prior to engaging in any activity. Ankle stability, strength, and range of motion are imperative in volleyball due to the quick starts and stops, lateral movement, and jumping/landing. Likewise, shoulder range of motion (especially external rotation), stability, and rotator cuff strength are important for serving and spiking. Normal wrist flexion and extension are essential for setting, as are strong triceps and pectorals. Strong latissimi are required to withstand the force sustained from the ball during a block. Obviously, jumping requires proper strength and flexibility of the quadriceps, the hamstrings, and the ankle complex of the gastrocnemius, soleus, and Achilles tendon. The ten-step osteopathic screening examination, as described by Greenman, helps to identify treatable somatic dysfunction lesions in all parts of the body (4).

Abdominal strength and thoracolumbar strength and mobility are essential for overall performance and endurance. Volleyballers with chronic shoulder problems may be suffering from a weak core, especially if the shoulder pain is worse when serving. The player leaps with the back extended, then flexes and torques the core to aid the shoulder in generating the arm speed and angle to drive the ball with velocity. Always evaluate the volleyball player for core abdominal strength when evaluating subacute and repetitive injuries.

TYPES OF INJURIES

Volleyball injuries can be characterized as either acute or chronic (overuse), with overuse injuries being more common than acute injuries. The majority of injuries are associated with blocking and spiking, presumably due to jumping and landing. Defense, serving, passing, and setting are associated with fewer injuries, but since all players rotate through each position they are all exposed to approximately the same rate of injury. Data from competitions show that the injury rate may be 1 in every 25 to 50 player-hours. One report showed that 53% of players suffered an injury during the course of 1 year; however, few were season-ending injuries. In addition, the type of court surface has an influence on the rate and type of injuries. For example, patellar tendinitis is more prevalent in those who play on concrete or linoleum than in those who play on softer-impact wood courts. The injury rate was five times as great when elite collegiate players played on hard indoor courts rather than soft sand courts (3,6–10).

The type and approximate prevalence (if available) of acute and overuse injuries are presented in Tables 39.2 and 39.3, respectively (1–5).

Knee

Volleyball players are prone to acute knee injuries. The most common are meniscal, medial

TABLE 39.2. COMMON ACUTE INJURIES IN VOLLEYBALL PLAYERS

Injury	Approximate % of Total Acute Injuries
Lateral ankle sprain	15–60
Thumb or finger sprain	10
Knee sprain or meniscus tear	15
Acromioclavicular tear	Unknown
Sand toe (plantarflexion injury to metatarsophalangeal joint of great toe)	Unknown
Thoracic facet joint sprain	Unknown
Sacroiliac joint sprain	Unknown

collateral ligament (MCL), and anterior cruciate ligament (ACL) injuries. Meniscus tears are often caused by a loaded, twisting mechanism. There is often a sudden pop and pain, with subsequent locking as well. The volleyballer diving for a dig may twist and flex the knee while falling, which can set up this injury. MCL injuries are caused by valgus and varus stresses to the knee, respectively. Jumping and landing awkwardly after a spike or serve can traumatically overload the MCL, causing sudden pain in the area of the ligament. Surgery is usually not necessary in isolated injuries, and a prophylactic knee brace to prevent varus/valgus stress aids in recovery.

In volleyball, ACL tears are relatively common, usually due to asymmetrical landing following a jump. Like basketball, women are more prone to ACL injuries than their male counterparts due to various extrinsic and intrinsic factors (3). Intrinsic factors include joint laxity, limb alignment, intercondylar notch size and shape, ligament size, and hormonal influences. Extrinsic factors include muscular strength, level of conditioning, shoes, and motivation. Other mechanisms of ACL injury include twisting with a valgus force or an anterior-lateral blow to the knee. Both of these acute injuries cause immediate pain and effusion, a sensation of the knee giving way, and instability.

Chronic overuse injuries make up a large portion of volleyball complaints. Iliotibial band friction syndrome, patellar tendinitis, and patellofemoral maltracking are commonly seen. These are related to the constant eccentric load from jumping and landing in both practice and game situations. The patella is designed to add a mechanical advantage to the knee when

TABLE 39.3. COMMON OVERUSE INJURIES IN VOLLEYBALL PLAYERS

Injury	Approximate % of Total Overuse Injuries
Patellar tendinitis	Up to 80
Rotator cuff tendinitis/tear/impingement	8–20
Thoracic, lumbar, and sacral back pain	10–14
Iliotibial band syndrome	Unknown
Metatarsal stress fracture	Unknown
Suprascapular nerve injury	12–33
Achilles tendinitis	Unknown
Plantar fasciitis	Unknown

flexed, but since volleyball stresses absorb the body's full load when landing from a jump, any patellar instability in the trochlear groove can cause pain when the knee is loaded. The patella needs to fit appropriately in the trochlear groove to disperse the force from the jump, since anywhere from 10 to 20 times the athlete's body weight is absorbed by the knee. (See Chapter 23.3 for a discussion of specific treatments for these conditions.)

Hand

Blocking can cause a myriad of common hand injuries, including collateral ligament sprains, mallet finger, and fractures of the metacarpals and phalanges. Rare hand injuries in volleyball include pisiform fractures and antebrachial-palmar hammer syndrome. Sprains and closed fractures of the fingers can be managed with the RICE protocol (rest, ice, compression, and elevation), nonsteroidal anti-inflammatory drugs (NSAIDs), and buddy taping/splinting. The most common sprain, resulting from blocking, is of the radial collateral ligament of the metacarpophalangeal (MCP) joint. This sprain is treated with the aforementioned measures as well as thumb spica taping (5). In addition, joint play techniques are useful in the treatment of collateral ligament sprains of the MCP and interphalangeal joints. They usually consist of anterior/posterior translation, lateral side bending, longitudinal traction, and rotation.

Foot

Sand toe is a plantarflexion injury to the metatarsophalangeal joint of the great toe in beach volleyball players. This injury is rare in other sports, but it is seen in beach volleyball because athletes play barefoot on an uneven terrain. Volleyballers also spend a lot of time looking up at the ball, so they cannot anticipate and compensate for the uneven surfaces. Sand toe occurs when an athlete's foot strikes a hard spot in the sand that causes the toes to buckle or roll under the foot. Then, the weight of the athlete's body drives the rolled or buckled toes into the sand. This results in a very painful sprain to the toe's joint capsule, ligaments, and tendons. It is most commonly seen during a running spike or an approach to the net after a jump serve. It often results in significant functional impairment, especially for running and jumping motions because of the needed push-off from the great toe. Treatment consists of RICE, NSAIDs, physical therapy, taping, shoe modification, and joint play techniques (if tolerated). Recovery can take up to 6 to 12 months and is often accompanied by loss of dorsiflexion (1).

Metatarsal stress fractures are a result of the constant jumping in volleyball. The second metatarsal is most commonly affected followed by the third and fourth metatarsals. The history elicits dull, achy pain in the forefoot. Return to activity is usually within 4 to 6 weeks (1). Manual medicine should focus on correcting pes planus and leg-length inequalities in addition to optimizing cuboid and subtalar motion. Joint play should be evaluated and treated, especially at the fifth metatarsal-cuboid and intermetatarsal articulations. Direct high-velocity, low-amplitude (HVLA) techniques should be applied to cuboid or navicular dysfunctions that are not acute.

Achilles tendinitis and plantar fasciitis are two other conditions seen often in volleyball (11). (See Chapter 24.3 for a discussion of their treatment.)

SPECIFIC INJURIES

Ankle Sprains

Sprains of the lateral ligaments of the ankle are very common in volleyball. The vast majority of these injuries occur in the front court during the landing of a spike or block. The most common mechanism occurs when one player's foot lands on another player's foot underneath the net. The landing foot is forced into supination, and an inversion stress is placed on the lateral ankle ligaments (5).

Treatment should first focus on addressing the pain and swelling with RICE, NSAIDs, and possibly an air cast. Early physical therapy is essential to restore range of motion, strength, and flexibility. This is accomplished with Theraband

exercises, closed kinetic chain stabilization exercises on even and uneven surfaces, and proprioception exercises using the biomechanical ankle platform system (BAPS). Because lateral movement and jumping are more necessary to compete than straight-line running or sprinting, a lateral stabilizing brace should be used during the functional phase of rehabilitation and in the immediate return-to-play period. However, long-term use of an ankle brace may not always be beneficial because the athlete tends to rely on the brace for support and the surrounding muscles become weaker.

Prevention includes teaching players to avoid the center line and to avoid jumping forward during a spike. Spikers should take longer final steps in approaching their jump and then jump straight up rather than forward (5).

Applicable manual techniques include talar and subtalar release, muscle energy, and HVLA for cuboid dysfunctions and joint play of the fifth metatarsal base and lateral malleolus. Fibular head dysfunctions should be treated as well (4). In addition, leg-length inequalities and pes planus should be evaluated and addressed.

Patellar Tendinitis

Patellar tendinitis is also called jumper's knee (see discussion in Chapter 27) and is the most common chronic ailment of volleyball players. Again, this is due to the repetitive jumping involved with the sport. Those athletes who generate the greatest power and have the highest vertical jump are at increased risk (12). Those who have increased external tibial torsion and deeper knee flexion at takeoff may also be at increased risk (5).

Athletes usually present with anterior knee pain at the inferior pole of the patella that worsens with activity. Tenderness at the inferior pole of the patella is evident on clinical examination. Radiographs are generally not necessary.

Treatment consists of RICE, NSAIDs, and physical therapy. Common physical therapy modalities to treat patellar tendinitis include pulsed ultrasound, ice massage, and cross-friction massage. Exercises include closed kinetic chain strengthening (lunges and wall slides) and stretching of the quadriceps and hamstrings with eventual progression to plyometrics (13). Cross-training is essential. Patellar straps (Cho-pat) help to change the stress point of the tendon during jumping and are beneficial. Prevention includes the teaching of proper jumping and landing techniques.

Manual medicine treatment of patellar tendinitis is somewhat limited when pertaining to the patellofemoral articulation. Dysfunctions around the knee can influence recovery and therefore should be evaluated and treated. Identification and correction of leg-length differences and pes planus are imperative. Muscle energy to the quadriceps is helpful to improve muscle length and flexibility. Identify any pelvic dysfunctions, particularly rotated innominates and sacral torsions, both of which influence dynamic leg-length discrepancies.

Rotator Cuff and Biceps Tendinitis

Shoulder injuries are common in volleyball and generally involve overuse tendinitis and/or impingement of the supraspinatus or biceps tendons. This is a result of repetitive microtrauma that occurs with overhead motions such as blocking, spiking, and serving (13). Predisposing factors that may lead to impingement include acromioclavicular arthritis, acromial spurring, glenohumeral laxity/instability, and weak rotator cuff musculature (1,5). In addition, impingement forces are increased during a spike, as contact with the ball occurs at maximal shoulder abduction (5). Athletes generally complain of anterior/lateral shoulder pain that is worse with overhead motion.

Competitive volleyball players occasionally use aggressive serving techniques that can lead to this problem. In this mechanism, the server typically takes a couple of steps toward the serve line, jumps up with the lumbar spine extended and the back arm cocked, and then propels his or her torso and arm forward to generate the velocity to hit the ball, exploding into the ball. This violent motion requires a strong trunk to allow the torso to come forward yet keep the axial skeleton stable for the scapula and glenohumeral joint.

If the core is weak, the upper body has to work harder to bring the arm forward, and without the force transferred through the kinetic chain from the lower extremities and trunk, the arm velocity decreases. A dynamic instability develops in which the anterior capsule and rotator cuff are stressed from the excessive load, fatigue develops, and the humeral head is not seated in the glenoid cavity during the transmission of force. Because the majority of competitive volleyball servers have higher than normal joint laxity, the rotator cuff, biceps tendon, and scapular stabilizers are already overdepended on for stability. Excessive load strains the tissue, leading to tendinitis, bursitis, and impingement.

For players who spike the ball at close range, the mechanism is similar. The main difference is that the serve technique and position can be similar each time the athlete performs it, but spiking the ball is more spontaneous and responsive to the flight of the ball, so the windup and follow-through are not consistently as violent.

Weak abdominals not only destabilize the shoulder but allow excessive lumbar extension, which increases stress on the posterior spine elements as well. In fact, a back injury that weakens the core can easily lead to shoulder instability and pain. If extension is limited, velocity decreases due to limited cocking, so the athlete may try to push harder with the upper extremity. If flexion is limited, follow-through is cut short, depriving the upper extremity a smooth dispersion of energy through the elbow and hand, and the sudden deceleration can stress the rotator cuff and biceps (14).

Rehabilitation is geared toward dynamically stabilizing the humeral head better in the glenoid cavity. This incorporates the rotator cuff, larger power muscles, scapular stabilizers, and cervicothoracic spine. These muscles need strength, coordination, and endurance to handle the eccentric stress of overhead sports like volleyball. The scapula needs to be mobile in elevation and depression, and it must reach a retracted stable base against the thoracic spine in the cocking phase of spiking and serving (3).

Acute treatment consists of ultrasound to the supraspinatus to increase blood flow and improve healing, cross-friction massage, and ice massage. Pain-free strengthening exercises to strengthen the rotator cuff muscles are important, but functional retraining of glenohumeral and scapulothoracic motion is more often needed. *Core stabilization is absolutely essential for volleyball players*, for stabilizing the humeral head without core stabilization leads to chasing one's own tail and never accomplishing treatment goals.

Subacromial corticosteroid injections are often helpful. Surgery is often reserved for those who fail conservative treatment (1,5). A subacromial decompression procedure is done for impingement with or without rotator cuff tearing. A stabilization procedure to repair either labral pathology or excessive capsular laxity can be done when the symptomatology is secondary to underlying instability.

Manual medicine treatment first consists of correcting any lower cervical, upper thoracic, and upper rib dysfunctions. Trigger point release and cross-friction release can be done to the rotator cuff muscles to improve firing and restore length. Sternoclavicular and acromioclavicular dysfunctions can be treated with muscle energy and articulatory techniques. Muscle energy is extremely useful in treating glenohumeral restrictions, integrated with Spencer techniques (4). See Chapter 17 for more detail on shoulder tendinitis treatments.

Suprascapular Nerve Injury

An injury relatively unique to volleyball players is suprascapular nerve injury. A terminal branch of the nerve, which has pure motor function and supplies the infraspinatus muscle, may be compressed by ganglion cysts at the spinoglenoid notch. Another mechanism is traction neuropathy caused by floater serves. These serves require the player to stop the overhead follow-through as soon as the hand strikes the ball. This results in little spin on the ball, making it difficult to pass. Unfortunately, floater serves require a forceful eccentric contraction of the infraspinatus muscle in order to decelerate the arm, which results in traction of the nerve.

Some players develop shoulder pain. Most, however, are asymptomatic, but weakness and atrophy of the infraspinatus may be found during the pre-participation examination. Electromyography and magnetic resonance imaging are helpful in establishing the diagnosis and identifying ganglion cysts, respectively. Treatment consists of relative rest and physical therapy. Surgical decompression of the nerve is an option if conservative measures fail (1,5).

Manual medicine treatment includes correcting any somatic dysfunctions in the lower cervical and upper thoracic spine, and the upper ribs. Once this is addressed, the scapula should be treated for myofascial trigger points and scapular restriction. Scapular lift and Spencer techniques should be used in this situation, while muscle energy should be used on restricted shoulder girdle muscles such as the trapezius, levator scapulae, and anterior scalenes.

Back Pain

Back injuries are common in volleyball and sprain/dysfunction can range from thoracic facet joint to lumbar to sacroiliac. Common somatic dysfunctions in volleyball include single and group dysfunctions in the thoracic and lumbar vertebrae, sacral torsions, and innominate shears and rotations. The mechanisms of injury are multifactorial, including landing forces from jumping, diving, or bending forward with extended knees, and lateral movements with concomitant trunk twisting and extension of the arms (1).

As previously described, weak abdominals can lead to injury to the posterior elements, such as facet syndrome, stress fracture, and spondylolysis. This can be seen in those who are serving or spiking frequently. Digging for shots requires more lumbar flexion, which can lead to sprains, strains, and lumbosacral dysfunctions, particularly since most digs are quick reactions. Herniated discs can occur in this mechanism, but they are not as frequent.

Players with back pain complain of sharp, achy, or spastic pain that is generally worse with activity and better with rest. Because these injuries generally involve the muscles, ligaments, and joints of the back, athletes usually do not complain of radicular symptoms (radiation, numbness, tingling, or weakness). Radiologic imaging is generally not necessary unless there is evidence of neurologic involvement or if the athlete does not respond to conservative treatment.

Rehabilitation should focus on abdominal and back stabilization exercises (pelvic tilt and physioball), particularly for athletes with hypermobility of the vertebrae. This includes stretching exercises for tight quadriceps, iliopsoas, piriformis, and hamstrings. Correcting leg-length differences and pes planus can balance the pelvis. Athletes need to adopt proper jumping and landing techniques and maintain the ready position with knees flexed and the lumbar spine in neutral position.

The lumbar spine needs to be able to extend when serving and setting, yet stable when blocking at the net. Tight iliopsoas, group lumbar dysfunctions, sacral torsions, and facet injuries can limit extension. The direct etiology of back pain can be elusive, especially in volleyball players who stress their back daily.

Manual medicine treatment is abundant for back injuries. Longitudinal and lateral soft tissue stretching of the paraspinal muscles provides an excellent warm-up for further treatment. Myofascial release can follow the soft tissue stretching and is an indirect technique in which the clinician attempts to place the thoracolumbar fascia in a position of ease and maintains that position until release is felt. Next, use muscle energy, followed by HVLA, on the thoracic, rib, lumbar, sacral, and pelvic dysfunctions to complete the treatment (4).

PREVENTION

Plyometrics are excellent exercises for jumping athletes, as they increase lower extremity power and decrease the risk of ACL tears. However, a high level of strength, endurance, and flexibility must be achieved before starting a program; otherwise, bad compensatory habits are rein-

forced and injuries from training can occur. A gradual increase in skill training should happen to allow the athlete to build body awareness. Plyometrics should not be started until the athlete can lift two times his or her body weight in a hip-sled squat (15). As with all competitive athletes, year-round conditioning is done and should be sport-specific.

Proprioceptive training can reduce injury in volleyball, because so much of the sport is spent looking up away from the playing surface. Every player tracks the motion of the ball, which diverts focus from the body. Lower extremity awareness must be high to land properly from jumps, while hand-eye coordination couples with upper extremity awareness to perform digs and dives with minimal reaction time. Coupled with the quick reaction needed to return serves and spikes and the amount of jumping involved, the volleyball player needs increased awareness and coordination to succeed and avoid injury (15).

CONCLUSION

Volleyball is one of the most popular sports today and continues to grow. With this continued growth comes, unfortunately, more injuries. Sports medicine professionals have the unique opportunity to help these athletes recover and return to play quicker by combining traditional treatment with manual medicine techniques. Clinicians need to teach proper biomechanics to players and use manual medicine and holistic principles to optimize the neuromusculoskeletal system of the athlete prior to engaging in activity. Proprioceptive training and core stability are crucial for players to stay healthy and competitive.

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SECTION

V

SPECIFIC POPULATIONS

40

THE INDUSTRIAL ATHLETE

PHILLIP C. ZINNI III

Industrial or occupational injuries are a staggering economic cost to employees and employers alike. In 2000 (the last year of complete data), there were 5.7 million Americans injured on the job according to the Occupational Safety and Health Administration (OSHA), resulting in 1.7 million lost-time injuries (1). This cost employers in the United States $56 billion dollars in direct medical costs and $45.9 billion dollars in indemnity costs (includes cash wage replacement payments) for a total of nearly $102 billion dollars. These figures come from the National Institute of Occupational Safety and Health (NIOSH) (2) and the National Academy of Social Insurance (NASI) (3). On average it costs employers $805 dollars for each injury. These injuries and illnesses cost the employees and employers slightly less to treat than the direct and indirect costs of all cancers ($107 billion dollars) or a little over one third of the costs attributable to heatt disease and strokes (4) ($286 billion dollars). The average cost of health care per person in the United States is about $3,925 dollars per year.

However, about 70% of illnesses are preventable, and it stands to reason that a majority of occupational injuries are also preventable (5). Of these lost-time injuries, according to the Bureau of Labor and Statistics (BLS), 25% were back/lumbar sprains/strains; 10% were arm, wrist, and hand sptains/strains; 8% were knee sprains/strains; 6% were shoulder sprains/strains; 4% were ankle sprains/strains; 2% were cervical sprains/strains; and 2% were carpal tunnel syndrome.

A newer approach to industrial injuries is the sports medicine paradigm. In this concept, the *industrial athlete* is cared for using a team concept similar to the organization of collegiate and professional sports medicine establishments. Because industrial workers perform physical labor and skills
not too dissimilar from the professional athlete, who performs physical labor and skills for money as well. This paradigm has been successful, and organizations such as The Industrial Athlete have developed on-site fitness programs and facilities at industrial settings to allow industrial athletes to train in-season. There are training rooms, on-site doctors and nurses, and even general manager types whose primary responsibility is to oversee the medical management of these industrial athletes.

JOB REQUIREMENTS

The tasks that industrial athletes perform include lifting, pushing, pulling, carrying, gripping, grasping, reaching, bending, stooping, and squatting in a time frame labeled as continuous, frequent, occasional, ot seldom. The job task analysis, akin to a position description in sports, describes the essential functions required. The essential functions or tasks are those which the industrial athlete must perform in order to successfully complete the job, similar to the sports athlete who fulfills the designated position on the field of play in a sports arena.

An *ergonomist,* who may or may not have a physical therapy background or degree, usually performs the job task analysis. The ergonomist, akin to the football team equipment manager, is responsible for matching the player to the equipment. Similarly, the physical therapist, akin to the defensive coordinator and strength and conditioning coach, is

responsible for preventing injuries by showing players how to lift and how to maintain their strength, dexterity, and stamina.

With the job task analysis in mind, the staff physician can perform an examination on the industrial athlete tailored to the task that the individual must perform. In this perspective, the staff physician is a position coach. He or she can effectively place the industrial athlete in the correct and appropriate job task to maximize the individual's natural talent and capabilities, and also match him or her to the essential job functions. One would not expect a wide receiver like Jerry Rice to be able to lift or push 300 pounds and play an interior lineman; the same rule applies to industrial athletes.

PREVENTION

The first moment of intervention is usually during the initial pre-employment physical examination. This is performed similar to an athletic pre-participation examination. Screening for functional axial spinal and appendicular range of motion, adequate ligamentous stability, and baseline grip strength is performed. The emphasis is on the cervical and lumbar spine, shoulder, wrist, hip, and knee range of motion. The industrial athlete screening is a quick examination.

As with most competitive athletes, the industrial athlete wants the best care so he or she can get back on the playing field. However, the workplace does not always foster a trusting relationship; the environment is often adversarial or even hostile. It falls to the physician to deftly handle these situations and act in the best interest of all parties involved.

This basic philosophy is anchored by an accurate diagnosis and grounded in appropriate treatment. The *P-R-I-C-E-M-M-M-Well* protocol is used to ensure that no matter who treats the industrial athlete's injury in the clinic, the same thought process is used. The protocol ensures that each industrial athlete is evaluated for his or her need of appropriate *P*rotection, *R*est, *I*ce, *C*ompression, *E*levation, *M*anipulation, *M*edicine, *M*odalities (physical therapy), and *Well*ness. The protocol is discussed in detail throughout this chapter.

PROTECTION AND REST

If the industrial athlete is injured but capable of protecting himself or herself from further injury, then the person is asked to perform only those functions allowed by his or her capabilities. Work capacity is determined primarily by the person's progress with progressive resistive exercises comfortably performed in physical therapy. The first evaluation bases the assessment of capabilities on the activities of daily living that the industrial athlete comfortably performs.

Additionally, conventional braces, gloves, splints, and wraps seen in the athletic training room or on the sidelines are also implemented in the industrial setting. These protective devices not only protect the industrial athlete from further injury but also allow the affected part to be supported and rested. Usually the industrial athlete can still use the body part to some degree. An example is the postoperative industrial athlete recovering from an ulnar nerve transposition procedure who can adequately protect himself or herself but is restricted to one-hand duty with the unaffected arm.

WELLNESS

The wellness component of the protocol is the summary of each industrial athlete's visit to remind the clinician to engage the athlete and/or plan for the next visit. At this point in the visit, the clinician should review any contributory effects of body mechanics or workstation setup and inspect the workstation personally or have the company ergonomist do it. Additionally, the clinician or company physical therapist should review the industrial athlete's biomechanics. Exacerbating factors such as deconditioning, poor fitness, obesity, and smoking should be addressed and treated. If the initial

visit is the pre-employment physical examination, then the clinician should plan a Prehab program, which is somewhat of a preseason conditioning program, jointly determined and administered by the physical therapist and physician. Additionally, the physical therapist can implement a back program to teach and promote proper lifting techniques.

The clinician should always teach and promote self-care as the greatest preventive tool. This reinforces the fundamentals of warm-up, stretching adequately and at appropriate times, icing if needed, aerobic and strength conditioning, and lifestyle with behavioral modifications when applicable.

TREATMENT PHASES

Most athletic injuries are treated through a series of phases to get the athlete back to preinjury status. This is a broad generalization that can apply to any musculoskeletal injury. For more precise prognostication, the Occupational Disability Guidelines (ODG) (5) or the Medical Disability Advisor (MDA) (6) can give the clinician an estimated time of return to work for any injury (International Classification of Diseases [ICD]-9/10 code) relative to Department of Labor classifications (7) of the type of work (sedentary/clerical to heavy manual) the industrial athlete is going back to perform. For example, an industrial athlete with a herniated disc, ICD-9 code 722.1, treated conservatively at first, would be expected to return to work within 3 days if the person had a clerical position or within 28 days if he or she performed manual labor.

Phase 1: Pain Control. This period lasts about 1 week for large muscle group injuries or injuries that involve weight bearing. These injuries commonly entail all aspects of the *P-R-I-C-E-M-M-M-Well* protocol with the most emphasis on the initial *P-R-M-M-M* portion. In shift workers, somnolent medications can be used to induce sleep, because the industrial athlete with a disrupted sleep-wake cycle is handicapped by difficulties in recovery. In addition, this aids in developing a positive attitude toward recovery, as it applies a multimodel holistic approach toward an injury. For small muscle group and non–weight-bearing injuries, phase I may run concurrently with phase II, again with emphasis on the initial *P-R-I-C-E-M-M-M* portion.

Phase 2: Stabilization. This period lasts about 2 to 4 weeks for large muscle group injuries or injuries that involve weight bearing. The industrial athlete at this stage of rehabilitation generally has full passive range of motion of the injured body part or limb, needs less medication, and can control symptoms more with self-care taught by the physical therapist or physician.

Phase 3: Functional Restoration. This period lasts about 4 to 12 weeks for large muscle group injuries or injuries that involve weight bearing. The industrial athlete at this stage of rehabilitation generally has full, pain-free, active range of motion of the injured body part or limb. He or she commonly needs minimal protection and medication, but relies heavily on modalities in the form of physical therapy rehabilitation. For small muscle group and non–weight-bearing injuries, the athlete usually progresses much more rapidly through the first three phases.

Phase 4: Maintenance. Phase 4 lasts 12 weeks for large muscle group injuries or injuries that involve weight bearing, and ideally continues indefinitely. At this stage of rehabilitation, the industrial athlete is recovered, implying that he or she is at optimal preinjury health and functioning status or at maximum medical improvement (MMI), and that the person has no disability and is capable of performing any and all activities of daily living (ADL) and their usual and customary (U&C) job tasks at work with their previously injured body part or limb. This phase relies almost solely on a physical therapy–directed home exercise program (HEP). A HEP is a collection of resistance exercises and stretches that the industrial athlete can successfully perform at home or in a community gym/health club setting after adequate explanation,

guidance, and verification of the athlete's compliance by the physical therapist. For small muscle groups or non–weight-bearing injuries, the maintenance phase lasts 8 weeks.

MANUAL MEDICINE TREATMENT OF COMMON INDUSTRIAL INJURIES

Lumbar Spine

The most commonly seen injuries in industrial athletes are lumbar sprain and strain injuries. Low back pain is the leading cause of disability under age 45, the third leading cause in those over age 45, and the second leading cause of visits to primary care providers for all ages. The majority of industrial athletes are treated conservatively, with less than 5% requiring trigger point injections or proliferative treatment, less than 5% requiring invasive epidural space steroid injections, and less than 2% going on to surgery. Lumbar spine injuries occur most commonly during an unexpected or unprotected delivery of force, such as lifting, pulling, twisting, bending at the waist, or during a slip and fall.

Statisticians have unsuccessfully tried to link the occurrence of or predilection to low back pain to a multitude of factors such as hypertension, diabetes, obesity, smoking, or body mass index (BMI); however, the only predictor validated in studies is fitness level (8). This is borne out in the industrial setting; however, the individual who is unfit usually is obese to some degree and often has comorbidity, as all three factors are intertwined. Because of these observations, when evaluating an industrial athlete for back pain, the clinician should screen for certain red flags indicating potentially serious disease. History should rule out conditions such as acute fractures or dislocations, infection, tumor, progressive neurologic deficit or nerve root compression, progressive vascular event, acute renal disease, or progressive intestinal inflammation (9).

Lumbar spine injuries could also be associated with adjacent dysfunction in the thoracic and sacral spine and associated sacroiliac joint, so the examination should be inclusive. The structural examination should review the general appearance, build, condition, comfort, posture, leg length, and pelvic symmetry of the industrial athlete. The most common findings are muscle spasm and flattened lumbar lordosis. Presenting as soft tissue firmness, muscle spasm feels cooler to the touch with little to no skin resistance on sliding-finger palpation; tenderness to palpation and resistance to passive movement are noted as well. The initial goal is breaking the pain-spasm-dysfunction cycle.

Manipulative treatment for industrial athletes should be approached like an office procedure. Treatment documentation should also be similar to office procedures. An example of such documentation is as follows:

> I talked about the pros/cons; risks/benefits; advantages/disadvantages of HVLA *(type of technique such as high-velocity, low-amplitude)*. After I had informed verbal/written consent, s/he was taken through soft tissue treatment, muscle energy, HVLA *(list all techniques for billing documentation and appropriate reimbursement)* of the CT spine and rib cage *(list all areas treated for billing documentation and appropriate reimbursement)* with adequate mobilization which s/he tolerated well. This was explained to and understood by the industrial athlete and all questions were answered and understood by the athlete. S/he was discharged from the clinic in stable condition without complaints or untoward reactions.

Document the status of the industrial athlete at the end of the note, again as you would after any other procedure.

Group Lateral Curve. The individual with a pure lumbar strain presents with an antalgic posture, listing into the area of greatest dysfunction as the muscular spasm attempts to splint the spine against motion to the opposite side. This represents a group lateral curve, and it leads to pelvic and core instability as well as compensatory changes in multiple areas. A preferred approach for treatment would be functional and indirect techniques rather than direct techniques.

Non-neutral Dysfunction. The individual with a non-neutral dysfunction presents with more localized discomfort, they may have an antalgic

posture, listing into the area of greatest dysfunction as the muscular spasm attempts to splint the spine against motion. For example, an industrial athlete with a forward-flexed, left-rotated, and side-bent segment at L4 would present with local tenderness to palpation and fullness at the transverse process on the left. The person may list to the left and would exhibit decreased rotation and side bending to the right with increased discomfort. This represents a non-neutral dysfunction. Direct techniques such as low-amplitude (HVLA) thrust and muscle energy are effective in these cases.

Cervical Injuries

Cervical sprain and strain injuries are the second most common type of industrial injury. Upper cervical spine injuries are less frequently associated with thoracic spine injuries than lower cervical spine injuries; however, the thoracic spine should always be suspected and evaluated for dysfunction. The majority of cervical injuries are treated conservatively, with less than 5% requiring trigger point injections, 1% requiring invasive epidural space steroid injections, and less than 1% going on to surgery.

Cervical spine injuries usually occur during an unexpected or unprotected delivery of force, such as lifting, pulling, reaching overhead, looking up and extending, rotating, looking down and flexing the neck, after a slip and fall, or after a blow to the head.

Thoracic Spine

Thoracic sprain and strain injuries are most commonly seen in conjunction with either cervical (more frequently) or lumbar (far less frequently) spine injuries, although some are associated with shoulder injuries, most commonly involving the rotator cuff. All thoracic spine injuries have been treated conservatively, with less than 5% requiring trigger point injections and none requiring invasive epidural space steroid injections or surgery.

The rare, isolated thoracic spine injury most commonly occurs during an unexpected or unprotected delivery of force, such as lifting, pulling, pushing, activities that require prolonged flexion or extension of thoracic spine posture, or carrying heavy items that suddenly shift while in the industrial athlete's arms. Again, all have been treated conservatively with none requiring trigger point injections, epidural space steroid injections, or surgery.

Non-neutral Dysfunction. The individual with a non-neutral dysfunction presents more commonly with localized discomfort; rarely is an antalgic posture seen. Direct techniques are effective in this region, but soft tissue techniques such as massage or myofascial release may be needed in areas of spasm. Because each thoracic vertebra has a costal attachment, overall motion is less than that of cervical and lumbar spine segments, and muscle spasm limits motion even further. Performing direct techniques first may irritate the muscle spasm and cause more dysfunction. HVLA thoracic mobilization should be used with care on extended segments. If the segment above the dysfunction is not flexed during the technique setup, that segment can be forced into further extension, worsening the condition. This can be avoided by diagnosing the proper dysfunction, using proper hand placement, and flexing the segment before the thrust is delivered.

Upper Extremity

Shoulder, elbow, and wrist sprain and strain injuries are common in industrial athletes (1,3). While the majority of these injuries are treated conservatively, 10% of supraspinatus strains require injections, and 25% of lateral epicondylitis require injections. Furthermore, 50% of significant grade II supraspinatus strains and all grade III strains require surgical repairing. Less than 1% of lateral epicondylitis require surgical repair.

Shoulder Complex

The most commonly injured muscle in the industrial athlete is the supraspinatus. Injuries occur with or without glenohumeral joint involvement, and most commonly occur during an unexpected delivery of force, such as lifting, pulling, pushing, or during an activity that

TABLE 40.1. SUCCESS OF STANDARD TREATMENT OF CARPAL TUNNEL SYNDROME

Disease Severity	% Requiring Injection	% Requiring Surgery
Mild	Less than 10%	Less than 10%
Moderate	Less than 50%	25%
Severe	Greater than 90%	75%

requires repeated overhead or above the shoulder reaching, as in shoulder flexion or abduction. This injury sometimes results from an unprotected impact, such as from a fall.

In prolonged rotator cuff pathology, dysfunctional glenohumeral joint motion can cause thoracic spine dysfunction, particularly if the levator scapulae and trapezius are overcompensating for the loss of shoulder abduction. If the industrial athlete continues to perform overhead or repetitive labor without occasional shift rotation for recovery, the scapulothoracic region becomes dysfunctional, distal compensation occurs, and muscle firing patterns change. Dysfunction then becomes difficult to rehabilitate.

The individual with glenohumeral motion restriction presents most commonly with localized discomfort on motion testing. Adhesive capsulitis can occur in the industrial athlete who has minor shoulder pain that he or she works through but does not get treatment for. The underlying inflammation and irritation are thought to provoke a synovial inflammatory reaction, which leads to restriction from adhesions, capsular thickening, and pain. The joint usually exhibits near-normal internal rotation but significant loss of external rotation.

Treatments for the Shoulder Region

1. Spencer techniques are effective in this area because they treat inherent glenohumeral joint motion (11).
2. Myofascial trigger points should be treated around the shoulder girdle and in the rotator cuff muscle bellies.
3. Stretching can help prevent any further range of motion loss and improve muscle function around the glenohumeral joint.
4. Joint play techniques have a place as well, but their effectiveness depends on the stage of adhesive capsulitis.

Wrist

Carpel tunnel injuries are seen disproportionately more often in the industrial athlete than in the general population. Treatment results, which are similar to supraspinatus strains, are shown in Table 40.1. Median nerve injury most commonly occurs during an activity that requires repeated flexion of the wrist or pinching and/or grasping. This injury could result from an unprotected impact, such as forced flexion, or a fall; however, the majority of wrist problems in the industrial athlete are not acute.

Motion Restriction Dysfunction

The industrial athlete with a motion restriction dysfunction presents most commonly with localized discomfort on motion testing as the wrist is brought to the barrier of the restricted motion, usually in extension. The person exhibits normal flexion and decreased extension. Muscle energy and other techniques discussed in Chapter 19 can improve the motion restriction dysfunction, as they are easily adapted to the natural motion of the wrist.

Sucher Myofascial Release Technique

Sucher has eloquently researched and developed a simple technique for myofascial release of carpal tunnel syndrome (12–14). The industrial athlete should receive manipulative treatments one to two times per week for 8 to 12 sessions. Additionally, and probably of equal if

not greater importance, the industrial athlete should self-stretch three to five times daily initially, then as symptoms abate, scale back to a maintenance stretch of one to two times daily and stretches as needed if symptoms increase. The procedures are found in Chapter 19.3.

Lower Extremity

Knee, ankle, and foot sprain and strain injuries are seen disproportionately less often in the industrial athlete than in the general population. While the vast majority of these injuries are treated conservatively, 50% of meniscus sprains require injection and/or surgical repair, and 90% of anterior cruciate ligament tears require surgical repair. Various types of injuries occur from acute or chronic mechanisms, depending on the job requirements. Common acute injuries in the industrial athlete include ankle sprains, foot fractures, meniscal tears, and knee sprains. Chronic injuries seen most often are patellofemoral syndrome, knee osteoarthritis, and plantar fasciitis.

SUMMARY

The principles of sports medicine can be similarly applied to the industrial athlete with significant success. The mechanisms of injury are similar, and the criteria for "return to work" are similar to return to play. The added advantage comes in prevention, so that industrial athletes have a better ability to adequately protect themselves because the environment is usually predictable and constant from day to day. Sports have elements of randomness and spontaneity that make prevention more difficult than for an industrial athlete in a plant plugging parts together or welding doors each day. For jobs that require repetitive hand use, wrist splints may be beneficial, while floormen who walk miles each day inspecting plant operations and equipment may have orthotics or insoles inserted into their shoes to prevent plantar fasciitis.

These principles applied to an industrial medicine practice aid in returning industrial athletes to work after injury promptly and in restored health. Practice methods should be consistently evidence-based over time with little variance, and based on clinical objective assessment. When implementation has been achieved and the process has matured, the full benefits can be realized. The culture of the company is often reflected in the attitude between the workers and management, or between union and management in a unionized organization. The principles of sports medicine, applied completely in light of the *P-R-I-C-E-M-M-M-Well* protocol, connect all aspects of care. This paradigm is driven by premium quality service at an equitable and fair cost that appeals to management, the employee, and unions.

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THE DISABLED ATHLETE

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Clinicians have a much larger mandate than just diagnosing and treating diseases and their consequences. Patients need care oriented toward their person, not just their disease (1). Nowhere does this apply more than for people with disability. The maximization of life is much more than just the minimization of disease. It is more than just the prevention of secondary, unnecessary disability; it is the reaching for life's potential and the expansion of one's options, the effects of which reach far beyond the individual to all of society (2). Recreation and sport are vital and necessary for an individual to accomplish his or her goals.

HISTORY OF SPORTS FOR PEOPLE WITH DISABILITIES

The history of sports for people with disabilities can be reviewed from two perspectives: the medical and the sports model. Sports activities today exhibit influences from both models. (3). Viewed from the medical standpoint, sports for people with disabilities had its origins in the related activities of gymnastics, and in the concept of exercise being therapeutic and contributing to fitness and health (4). These beginnings are well described by Guttman in *Textbook of Sport for the Disabled* (5). Later, therapeutic exercises, including group activities, became recognized as a fundamental part of the orthopedic management of fractures (6). The use of sports as a further motivation to enhance the physiologic and anatomic aspects of musculoskeletal healing and strengthening was a later natural development. The step from group gymnastics, sports, and exercises to more integrated rehabilitation sports took place when Guttman incorporated sports activities into the total program of rehabilitation for patients with spinal cord injury (7).

Individuals who learn to play disability sports in rehabilitation centers value this experience and practice enough to become highly skilled, thereby generating opportunities to compete at national and international levels. Separate organizations govern Paralympics, Deaf Sport, and Special Olympics and hold events specific to the disabilities they serve. Paralympics, first offered in 1960, provides competition for elite athletes with spinal paralysis, amputations, cerebral palsy (including stroke and traumatic brain injury) and other locomotor conditions (including dwarfism) called *les autres*, and blindness. Deaf sport, begun in 1924, primarily serves athletes who use sign language. Special Olympics, begun in 1968, serves only individuals whose primary disability is mental retardation. Since 1996, a few of the best athletes with mental retardation have been integrated into the Paralympics. Paralympics, Deaf Sport, and Special Olympics each are modeled after the Olympic Games, with elaborare opening and closing ceremonies, rules that demand excellence, and scheduling of summer or winter games every two years.

Official competitive sports at the international level vary by disability. Some sports, like track and field, equestrian, sailing, and swimming, are functional for all disabilities. Others are specific to one disability. For example, individuals who are blind compete in goalball, judo, and tandem cycling. Both ambulatory and wheelchair sports are offered for individuals with amputations, les autres conditions, and cerebral palsy. For individuals with C6 spinal paralysis or equivalent function, all competition is in

specially designed manual sport wheelchairs. Individuals so disabled that they must use power wheelchairs (i.e., C5 and above or equivalent function with cerebral palsy or other conditions) typically engage in boccia ball (an outdoor yard game with a premise similar to lawn darts), a specially designed swimming track, or equestrian events (8,9).

Organized Sports—National and International

Guttman's efforts led directly to the creation of an international sports movement for the spinal cord injured. This movement was identified for many years as the International Stoke Mandeville Games Federation (ISMGF), which sponsored the first international games for people with disabilities in 1952. Its name changed more recently to International Stoke Mandeville Wheelchair Sports Federation (ISMWSF).

Other organizations developed their identity with specific disability groups, such as blind, deaf, cerebral palsy, amputee, and intellectually impaired. Within these organizations, control of the direction, staffing, and goal setting was made by persons who, for the most part, came from the medical or educational systems.

The wheelchair sports movement in the United States was sparked by Ben Lipton, director of the Bulova School of Watchmaking, in New York. He established the U.S. National Wheelchair Association (NWAA) in 1957, which sponsored the annual national wheelchair games. For many years these games were held on the grounds of the Bulova School of Watchmaking in Queens, New York. In 1994 the NWAA was renamed Wheelchair Sports USA, a name it has retained.

There was another quite separate and distinct influence that led to a change in the character and direction of sports for people with disabilities. This was based on the concepts of the value of sports in its own right, and the perceived need to identify the participants as true sportsmen and sportswomen, rather than part of a rehabilitation process. This effort has led to the development of an international governing body, the International Paralympic Committee (IPC), which is associated with the Olympics movement. Through this development, the Paralympic Games have gained stature and have served as the showcase for elite performers in disabled sports. In the 2000 Paralympic Games held in Sydney, Australia, 3,842 competitors (967 women and 2,867 men) from 122 countries participated before crowds that included 340,000 schoolchildren, 1.16 million paid spectators, and 300 million television viewers (10). The 2002 Salt Lake City Winter Paralympics totaled 416 competitors (88 women and 328 men) from 36 countries. This development has led to a close link between national governing bodies for Paralympic sports and the Olympics sports movement. It is estimated that over 500,000 athletes worldwide compete in Paralympic Sports (Table 41.1) (11).

Disability Group Sports Versus Integrated Disability Sports

Sports development at all levels, until recently, has been mostly identified with specific disability groups. Wheelchair sports is primarily for people with spinal cord injuries and bilateral lower extremity amputations, and it developed through an organizational structure at international, national, and local levels, from the Stoke Mandeville organization down to local community groups.

Cerebral palsy sports, both wheelchair and ambulatory, achieved its own American identity in 1978, when a U.S. National Sports organization, the National Association of Sport for Cerebral Palsy, was formed, facilitating regional and national competitions. This in turn led to the creation of a national team, which competed internationally for the first time in 1978. In 1987, cerebral palsy sports were reorganized as the U.S. Cerebral Palsy Athletic Association (USCPAA). More recently, in 2001, this organization broadened its mission to include other disability groups, and has been renamed the National Disability Sports Alliance (NDSA).

Amputee sports developed through several separate pathways. Winter sports offered the opportunity for unilateral amputees to ski, using some simple pole modifications, usually

TABLE 41.1. SPORTS IN WHICH ATHLETES WITH DISABILITIES FREQUENTLY ENGAGE

All-terrain vehicles	Golf	Skydiving
Alpine skiing	Gymnastics	Slalom
Aquatics	Hunting	Sledge hockey
Archery	Ice skating	Snowmobiling
Athletics	Ice sledding	Soccer
Basketball	Ice sledge hockey	Softball
Beep baseball	Lawn bowling	Table tennis
Biathlon	Martial arts	Team handball
Boating	Nordic skiing	Tennis
Boccia	Orienteering	Track
Bowling	Powerlifting	Volleyball
Cross-country	Power soccer	Water skiing
Cycling	Quad rugby	Weight training
Equestrian	Racquetball	Wheelchair dance sport
Fencing	Road racing	Wheelchair rugby
Field events	Roller skating	Wilderness experiences
Fishing	Rowing	Wrestling
Fitness programs	Rugby	
Floor hockey	Sailing	
Football	Scuba diving	
Goalball	Shooting	

Modified from Sherill C. *Adapted physical activity, recreation, and sport*, 5th ed. Dubuque, IA: WCB/McGraw-Hill, 1998.

not using a prosthesis. In more recent years, technical developments led to the participation of bilateral lower limb amputees in sled, and mono-skiing downhill events (12). Summer sports for amputees developed differently in Europe than in the United States. In Europe the emphasis was on the use of prostheses in sports as much as possible in field and track events, whereas in the United States, initially, amputees participating in sports favored the wheelchair toute to competition. The development of technically more sophisticated prostheses, such as energy storage foot and ankle systems, has resulted in significantly more amputees participating in sports with their prostheses (e.g., in track events). Bilateral above-knee amputees, who represent a higher degree of impairment, usually continue to compete in wheelchair sports such as track and field and basketball. Disabled Sports USA, formerly National Handicap Sports U.S.A., is the U.S. organization that represents amputees in competitive sports activities.

The most recent trend in organizing sports at the international level involves an attempt to integrate all types of locomotor disability into one system. This effort is being driven by the IPC in an attempt to reduce the multiplicity of athlete classes and events and ease the problem for sports organizers. Initial enthusiasm for the idea has subsequently been tempered by the realization that both the quality of performance and the actual level of performance are so variable that it may be meaningless to attempt to compare athletes with such diverse types of impairment in one competition.

In winter sports a classification system has been devised that includes classes for standing competitors in both alpine and Nordic skiing, while the use of so-called sit-skis and ice sledges ice hockey in Paralympic terms accommodates disabled athletes with spinal injury and high bilateral lower limb amputations. As a result, competitors with a wide range of impairments have access to alpine and Nordic skiing and ice hockey.

Despite these concerns, the integrated approach does appear to be the dominant trend. An often overlooked theoretical basis to integrated classification is whether it is impairment or disability based (13,14). Although the last

word has yet to be written, an impairment approach is gaining more support.

Involvement of Individuals with Disabilities in Able-bodied Sports

Some athletes with disabilities compete in able-bodied sports despite significant impairment. They have achieved this integration by succeeding in overcoming the effects of the impairment. Interestingly, in 1960, the year of the first Paralympics, a boxer with a below-knee amputation was denied entrance into the Olympic trials because it was felt that his lightweight prosthesis would give him an unfair advantage over able-bodied boxers by dropping him into a lighter weight class. More recently Marla Runyan, a runner with macular degeneration, placed eighth in the 1,500-meter race of the 2000 Olympics and subsequently, against sighted competition, shattered the American indoor record for the 500-meter race (15). Such success reflects the individual's drive and motivation, which enable him or her to break this particular barrier, and it also demonstrates the resiliency and adaptability of the human neuromuscular and musculoskeletal systems.

IMPACT OF EXERCISE IN SPORTS FOR INDIVIDUALS WITH DISABILITIES

The impact of vigorous physical activity on athletes with disabilities and their families is the same as for able-bodied people, except perhaps more significant because the need is greater. The impact can be positive or negative, depending on the goals of the individual, the respect accorded the body, and the personal meaning of activity outcomes. Several professions exist to maximize positive outcomes and minimize negative outcomes. This goal is typically achieved by teaching individuals with disabilities the importance of physical exercise and sport involvement, helping them develop the skills and fitness for success, supporting them in finding and/or initiating sport activity that meets personal needs and interests, and helping them to achieve goals and stay involved during all seasons of the year at a level equal to or exceeding the recommendations of the Surgeon General (16). Two of the key professions in this endeavor are adapted physical education and therapeutic recreation. As rehabilitationists, it is important that we become familiar with these professionals and what they have to offer.

Participation

There must be recognition of the breadth of ability that exists within the population who have a disability and the specific challenges faced by individuals at different stages in their lives. For those with congenital impairments, the school has a major role to play in physical education. For those who have an acquired impairment, the challenge may be about breaking down perceived and real barriers and accessing suitable activities and tuition.

Good communication skills are required by all those who work with individuals with a disability and in particular for those who have a sensory or learning disability. Teachers and coaches are challenged in so many ways and so very often have to go back to the drawing board to achieve the best results. Many teachers who have moved into special education, or mainstream coaches now working with disabled athletes, admit the transfer has greatly enhanced their ability to deliver in their chosen field of expertise.

There are individuals who view their disability as restrictive and believe it affects so many different aspects of their life. Participation in physical activity may be viewed as inappropriate or too inconvenient because of care issues, physical barriers, or lack of confidence (17). International growth of the sport of boccia ball, which requires minimal backup for effective participation, is a clear indication of what can be achieved when a situation is less threatening and offers an instant return.

Children are more likely to participate in physical activity than teenagers (in particular, teenage girls). Embarrassment, inappropriate activities, environmental factors, and fear of failure are just some of the reasons identified by teenagers (18) who have a negative view of physical activity. Service providers must find out if their

ideas on what is appropriate concur with those of the young people themselves. A community-based aerobics program for teenage girls with a learning disability with backup from mainstream peers proved highly popular because of the quality of the leadership and the girls' own interest in music and dance type movements.

Within the population with physical, sensory, and learning disabilities, the range of abilities is significant and has a major bearing on program planning. Individuals with severe and complex needs offer the greatest challenge for service providers, and although the costs may be high, they are necessary in our efforts to create an inclusive community (19). Swimming offers a freedom of movement to many individuals with severe mobility difficulties that is not available on dry land. Swimming pools that offer space in changing rooms, variable-height changing trolleys, poolside and dressing room hoists, and shower chairs are making a quite significant contribution to creating a barrier-free leisure environment.

Quality physical activity experiences should be available to individuals of differing abilities in a variety of settings at every stage of their lives. Active people should be directed toward appropriate sports programs, and health professionals are strongly encouraged to play a part in providing support and information to relevant agencies. Nonactive individuals should be encouraged to become active through the intervention of a multidisciplinary team of professionals.

Physical Activity Leading to Sport

From active play within the family through preschool- and school-based physical education, children and young people with a disability are well prepared for involvement in structured community activity including sport. Adults with acquired disabilities will hopefully experience a similar pathway and thereafter gain access to similar opportunities.

There are many models of best practice and some examples are as follows:

1. *Multiactivity Junior Groups.* These community-based groups are located in recreation centers and offer a range of activities appropriate to the interest of the group. If the session specifically attracts young people with physical disabilities, then opportunities are geared toward those with mobility difficulties, perhaps more individual than team, and have an end product of sports such as boccia, archery, soccer, wheelchair basketball, table tennis, and so on. If the group has a strong representation of blind young people, then goalball and other similar activities for the blind could be included. If the group is predominantly for juniors with a learning disability, then a full range of team and individual activities will make up the program. The emphasis is on variety and of course fun and enjoyment. Young people may attend out of interest, but only quality leadership will sustain that interest.

2. *Sport-specific Groups.* Swimming is an ideal sport for the development of this model. Essential to success is support information for children and young people often with parent or caregiver help. For adults, similar provision is provided, and in each instance essential support information is made available by health professionals. Quality leadership and an appropriate pool environment are critical to success. A parallel mainstream learn-to-swim program is a bonus and offers children, young people, and adults the additional attraction of inclusive tuition. Community recreation services normally deliver this model and then offer a transfer to sport-specific swimming clubs or groups specifically for disabled swimmers. The more options the better, and when the voluntary and statutory sector work together, the better the end product. Training, coaching, and competition then follow at a level appropriate to individual abilities and interests. This model with appropriate pathways can be repeated for all sports and is based on quality leadership, appropriate facilities, and multiagency cooperation.

3. *Adult Multiactivity Sessions.* Evening and weekend multiactivity opportunities for adults in a community recreation center offer much to promote the notion of sport for all and not simply for the selected few. Progressive service providers who offer transport backup and concessionary charging schemes for the vulnerable

and traditionally excluded populations are rewarded with high levels of participation. Sessions are held at popular times, and leadership and volunteer backup are of a high quality. Great strides have been made in raising awareness, overcoming prejudices, and accessing regular physical activity. Groups of adults with learning disabilities have benefited greatly from this model.

4. *Governing Body Talent Identification.* Operating on a sport-specific basis, governing bodies can play their part in ensuring that sports people with a disability are afforded every level of support as they progress along the competitive pathway. The "Flying Start" program offered by Scottish Swimming encourages young swimmers with a disability to enroll for a program of workshops that offer advice on everything from stroke technique to warm-up, from mental preparation to fair play and competitive structures (20). Parents and local coaches are encouraged to become involved, and they are invited to participate in specific workshops. The importance of getting off to the right start is a key outcome of this particular program.

5. *The Role of Pandisability/Disability-specific National Organizations.* National associations have a major role to play in ensuring that the single individual who has demonstrated competency and interest in sport is offered every opportunity to progress to a level appropriate to his or her ability and commitment. Any worthwhile national organization should have a network that addresses local needs at times when it is most needed by the athletes who are considering active participation in competitive sport.

In Scotland, the system adopted is that of a branch network that covers the whole country. The national association has a direct involvement with governing bodies of sport in the selection of individuals for the Area and Scottish Institutes of Sport. This model is based on partnership and cooperation between the many agencies that have a role to play in nurturing sports talent. The more progressive areas have appointed officers to promote and coordinate physical activity programs for people with a disability of all ages and abilities. Athletes and parents play an important role in the running of local branches, and issues addressed include club development, coach education, transport, funding, recruitment, disability awareness, and volunteer development.

Local organizations have the responsibility of providing programs of competitive sport that meet the needs of local people and mirror than provided by national associations. They should be familiar with the range of opportunities than exist at the national level and prepare teams accordingly. Local area programs organized on a pandisability (covering all disabilities) basis are the most successful and tend to eliminate the rivalries that often hinder initiatives organized on a sport/disability-specific basis.

Every local area group should have the facility of feeding into a national framework that offers individuals and groups the opportunities to realize their full potential in sport. Efficient national associations offer and resource a full range of competitive sports events. Sport-specific national coordinators have a direct involvement in sports development and, in particular, oversee national squad training, and team selection and management.

6. *The International Structure.* The International Paralympic Committee (IPC) and the International Governing Bodies of Sport (IGBS) lead the development in sports internationally. In disability sport, some sports have a closer relationship with their IGBS counterpart than others, and all sports have the additional complication of a classification system.

At the national level, countries sometimes promote their own classification systems for specific sports to encourage participation and widen opportunities. The most popular are the time/distance-banded systems for swimming and athletics, which are easy to understand and can be implemented by the coach, teacher, administrator, or volunteer. The stopwatch or tape measure determines the class of an individual, and those administering these schemes do not require the specialist knowledge normally associated with the international systems. Athlete profiling as a method of classifying has also been successfully introduced nationally for a range of sports with great success.

The classification systems that apply internationally are either disability- or sport-specific. The most widely used disability-specific systems are those promoted by the International Blind Sports Association (IBSA) (B1–B3) (21) and the Cerebral Palsy International Sports and Recreation Association CP-ISRA (CP1–CP8) (22). The former is based on the degree of sight loss, and the latter on neurologic function and how it impacts sports.

Sport-specific classification systems continue to be developed and refined. At one time every disability group had a unique classification system for their specific sport. The growth of the Paralympic movement and the massive increase in levels of participation have prompted a radical rethink by the International Paralympic Committee and International Organization of Sports for the Disabled on how classification is administered internationally. There has been a subsequent commitment to sport-specific systems and reducing the number of events in the Paralympic/world championship program and resultant medals.

IPC swimming has undergone a 10-year period of development and now offers a more settled functional system for the swimmers of the world. Wheelchair basketball operates a points system based on a player's ability to execute the skills associated with the sport. IPC athletics incorporates both the functional and disability-specific approach, but there are plans to develop a more integrated system in the future. All of these approaches present challenges to national organizations as they strive to involve medical and sports technical personnel in their organizations who can prepare their members for international competitive sport.

ATHLETES WITH DISABILITIES AND MANUAL MEDICINE

Sport is certainly a way of developing, using, and expressing one's neuromuscular system. As we have pointed out, there are many opportunities and benefits for participating in sport. Encouraging this participation is certainly very holistic. Stimulus to the neuromuscular system is essential to improving function.

When using manual techniques with athletes with disabilities, the clinician must evaluate any manual abnormalities with a skeptical eye, particularly in regard to potential clinical impact. Often these are asymptomatic, benign conditions that may not need intense treatment.

Osteopathic manipulative therapy offers benefits as well as potential risks in these athletes. The techniques used must be individualized to the athlete, just as manipulative treatments need to be individualized to any patient.

Techniques

There are special concerns with the use of *high-velocity, low-amplitude techniques*, such as the thrust technique. Athletes who have fragile bones, such as in osteogenesis imperfecta, or active synovitis, such as in inflammatory arthritis, are at risk of injury. There may well be a problem with using these techniques on athletes with spasticity. Spasticity is a velocity-dependent resistance to motion change; therefore, the use of a thrusting manipulative technique has the potential risk of increasing spasticity.

Muscle energy techniques are often useful. However, their use requires the athlete to have voluntary control of the musculature, thus negating the techniques in athletes with either immobilization to the joints, such as with spinal fusions, or those with spinal injuries below the level of the lesion. Muscle energy is also handy in that it can be applied effectively to almost any joint in the body. These athletes, in particular, have good responses to muscle energy techniques in the thoracic and cervical spine. For example, we have found that athletes with diplegic cerebral palsy often respond to muscle energy iliosacral and sacral techniques.

Articulatory and myofascial release techniques are often very helpful with disabled athletes, particularly as they develop stress from overuse and constraining for competitions. These work well with this population because they require no voluntary patient participation, and they have few contraindications.

The use of *indirect techniques* may be very helpful in cases of severe spasticity, as through reciprocal innervation. It can decrease the intra-

fusal muscle fiber tension of spasticity, thus allowing more mobility and function. Indirect techniques are effective when done first during a manipulation session to reduce spasticity and relax the patient. More direct techniques can then be applied to treat the somatic dysfunction.

Injury Issues

In an athlete with a spinal injury, the clinician has to be concerned about several issues. One, of course, is spinal stability. The body's dynamics certainly are thrown off when one has a fused spine or otherwise injured spine, and the key principle is to do no harm. Therefore, caution is advised with HVLA techniques. Lesions palpable below the spinal injury, if it is a complete injury, are usually of no significance.

Wheelchair athletes often develop shoulder dysfunctions, such as overuse impingement syndrome and rotator cuff tendinitis. Due to the excessive use of the upper torso required by wheelchair sports, carpal tunnel syndrome can also develop at the wrist, more so if an imbalance of the musculature develops. The use of appropriate exercise, as described by Burnham et al. (23), is often very helpful in treating these athletes in addition to traditional medicine. There may be associated painful myofascial restrictions that respond to myofascial release techniques, most notably in the forearms and transverse carpal ligament.

Athlete Interaction

Medical clinicians, such as doctors, trainers, and therapists, are in a position to effect tremendous positive changes in these athletes without medications or surgery. In many cases, the relationship between the athlete and clinician is the most therapeutic and motivating prescription that can be written. In treating these athletes, clinicians should pay attention to their attitude and discussions with the athlete. Athletes should be evaluated positively for what they have and can do and not assessed for what they cannot do.

For example, an athlete who has an amputation or spinal cord injury above the level of the lesion or injury should be treated like anyone else, with specific attention to his or her musculoskeletal status. By bringing attention to the skills that the athlete does have, the clinician allows the athlete to see what is possible. Framing the discussions in this light is more motivating and inspiring than overemphasizing the disability to the exclusion of all else. Therefore, the use of braces and prostheses can be framed in such a way that talk focuses on enhancing athletic skills instead of merely coping with what the athlete is unable to do.

Athletics can be a life-changing experience for many of these athletes on many levels. For some, it is an escape. Others learn to expand their abilities beyond what they or others believed they could do. It empowers some, granting them more control over their own lives and functions. Teamwork and camaraderie inspire a sense of belonging, value, and responsibility. Most of all, *sports are fun*, and that is healthy for everyone.

It is within this framework that manual medicine has such a strong influence and benefit. Physical contact, or laying on of the hands, forms a connection between clinician and athlete that is therapeutic and holistic. Much has been written about the healing power of touch and the activation of energy centers in the body, although research supporting it in this context is limited. Yet, manual medicine can build trust in the clinician-patient relationship, which is often difficult in some athletes with communicative disabilities. The athlete begins to trust the healing touch, whether the contact itself is meant to be therapeutic. Touch brings comfort, and comfort can soothe and relieve.

The body communicates its disease in many ways, and our hands can be our ears to what the body is saying. Clinicians can manually demonstrate improvement and progress in an athlete's function, which often speaks loudest to the disabled athlete. Improvement can be gauged easily throughout treatment, sometimes immediately, by applying the techniques and examinations discussed in earlier chapters of this text. Such

quick feedback maintains focus and motivation while allowing the clinician to target treatment to the most needed areas, even if the athlete cannot communicate them.

CONCLUSION

The bottom line on treating disabled athletes is to evaluate them as total people from a manual medicine viewpoint, while keeping an eye on certain unique situations, such as spasticity, lack of voluntary control, and hyper- and hypomobilities that may be associated with their condition. With these principles in mind, manual medicine can be a very active and beneficial adjuvant to the treatment of these athletes.

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THE GERIATRIC ATHLETE

KURT HEINKING

Growing numbers of senior athletes are exercising routinely and wanting to play competitive sports. Currently, the geriatric population comprises 12% of the total U.S. population. By the year 2030, 18% of our population will be over the age of 65, and by 2040 the mean life span will be in the 80s. The old-old, those over age 85, are the most rapidly growing segment of the population (1).

The United States Public Health Service declared that physical fitness and exercise is one of five priority areas in which improvement is expected to lead to substantial reduction in premature morbidity and mortality (2). Hundreds of studies delineating the beneficial effects of exercise have emerged. National recommendations advocating the development and maintenance of lifelong patterns of physical activity have been published. Many serious health problems in the elderly can be controlled, improved, or obliterated through moderate, consistent physical activity. There is considerable evidence that regular exercise, in conjunction with other risk-reducing behaviors, protects against an initial cardiac episode (primary prevention). It also aids in the recovery of patients with myocardial infarction, coronary bypass surgery, or angioplasty; and it reduces the risk of recurrent cardiac events (secondary prevention). Due to the beneficial effects of exercise and dietary modification, there has been a 30% decrease in coronary artery disease in the United States since 1960. Aerobic exercise and endurance training can also lead to numerous favorable metabolic effects. These include, but are not limited to, a more favorable lipid profile, control of obesity, decreased blood pressure, improved glucose tolerance, higher bone density, and an improved self-image.

However, this rise in physical activity has been accompanied by an increase in the number of sports-related injuries. It is estimated that over 17 million Americans seek medical care each year because of athletic and recreation-related problems (3–5). Considering that 12% of the population are over the age of 65, an enormous volume of elderly participants must be treated and educated on athletic-based injury. Regular exercise and appropriate training increase physical safety, reduce susceptibility to acute and chronic disease, and improve psychological outlook (6).

PHYSIOLOGIC CHANGES WITH AGING

The natural process of aging is based on the organism's genetic makeup and capacity to respond to changes in the external environment with internal homeostasis. Although there is much to learn about aging, three theories on the mechanism have been proposed (1):

- The immunologic theory.
- The transcription theory.
- The oxidative stress theory.

The immunologic theory states that changes in the thymus gland and immune system functions produce age-related changes. The transcription theory describes a defect in the repair of transcription errors and protein synthesis. The oxidative stress theory relates to the production of free radicals and their deleterious effects on the host's organs and tissues. It is important to note that aging is relatively linear, with a 70-year-old aging at the same rate as a 40-year-old. Exercise improves a patient's physiologic age; thus, although

a person may be chronologically 75 years old, he or she is physiologically only 55 years old. Exercise improves immune function and helps protect against the deleterious effects of free radicals. Aging affects all body systems; however, for the scope of this chapter, we concentrate on the musculoskeletal, cardiovascular, and neuroendocrine systems.

Aging athletes have losses in height, lean muscle mass, body water, and bone mineral density. Their connective tissue becomes less elastic and they lose flexibility. Their body fat increases; however, subcutaneous fat decreases. Systolic and, to a lesser extent, diastolic blood pressure increases, heart valves become thickened and sclerotic, and systemic peripheral resistance increases. There is decreased baroreceptor reflex activity, and altered electric neuronal conduction. Although resting cardiac output is unchanged, heart rate decreases and renal blood flow diminishes. There is a general increase in peripheral insulin resistance and impaired glucose tolerance. Neurologic function is also affected, along with postural instability, altered gait, and decreased reaction time. The sports medicine practitioner needs to understand these factors in injury diagnosis, management, and manual treatment. Because of the medical complexities of this age group, multidisciplinary care and a multidimensional treatment approach are necessary and beneficial (1).

RISK OF PARTICIPATION

The American College of Sports Medicine (ACSM) has developed a system to classify risk factors for individuals *based on age and underlying cardiac condition* (7). The three categories are as follows:

1. Apparently healthy individuals without known cardiac risk factors or evidence of underlying disease.
2. Higher risk individuals with one or more coronary risk factors.
3. Individuals with disease who have known active cardiopulmonary or metabolic diseases or who have symptoms suggestive of underlying disease that have not yet been evaluated.

Chisholm et al. (8) developed a self-administered set of seven questions known as the Physical Activity Readiness Questionnaire. On this questionnaire, individuals responding *yes* to any question should consult a physician before implementing an exercise program. Individuals classified as higher risk or individuals with disease in the sports medicine categories should be evaluated by a physician before beginning exercise.

PRE-PARTICIPATION

Treating geriatric athletes is challenging for the sports medicine specialist because of the involvement of one or several comorbid health conditions and their treatments, all of which is in addition to any sports injury. For instance, exercising can be difficult if an athlete has to take a beta-blocker and diuretic to control blood pressure, and rehabilitation can be even worse. Therefore, a modified pre-participation physical in elderly athletes is crucial in order to properly issue an exercise prescription. The pre-participation history and physical examination have numerous functions, with screening for life-threatening conditions topping the list. Elderly athletes may have atypical presentations of diseases and underreported symptoms. It is also difficult for the practitioner to determine if their symptoms are due to the natural process of aging or due to an undiagnosed medical condition. Their pain tolerance is usually increased, and they do not typically bother seeing a doctor for nuisance-type problems. They may be in denial over their visual or hearing loss, urinary incontinence, or diminished sexual functioning. Some of their symptoms may be due to drug interactions or medication side effects. A functional approach to their history is important to address their activities of daily living (ADL) and instrumental activities of daily living (IADL).

In 1996 the American Heart Association with the 26th Bethesda Conference on Cardiovascular

Health produced a document entitled *Cardiovascular Screening of Competitive Athletes* (9,10). Although these guidelines are clinically useful, there is no nationally accepted form for completing the pre-participation physical examination. However, the following are goals and objectives that should be considered during the pre-participation examination:

- Establish rapport with the athlete.
- Perform a screening musculoskeletal examination with emphasis on evaluation for the presence of somatic dysfunction (most often done by an osteopathic physician).
- Screen for life-threatening conditions.
- Decide eligibility or restriction of play (based on classification of sport: contact, limited contact, noncontact).
- Determine disqualifying medical conditions for sport participation.
- Evaluate the function of the musculoskeletal system and prior injuries.
- Record a baseline mini–mental status examination.
- Check for communicable diseases.
- Counsel/educate on athletic injury prevention and cancer screening examinations based on the population.
- Evaluate for injury potential and areas of performance enhancement.
- Educate the athlete on closed head injury, use of ergogenic aids, and proper protective equipment.

The physical examination is a screening examination only, but it should include a thorough cardiac evaluation in at least two positions, carried out with maneuvers. In the older population, the physician should focus on these four systems:

a. Cardiopulmonary.
b. Peripheral vascular.
c. Musculoskeletal.
d. Metabolic and endocrine.

Cardiopulmonary

Baseline resting electrocardiograms, echocardiography, and cardiac stress testing are valuable in this population. Each individual's cardiac risk factors, lifestyle, and physical examination should be taken into consideration. The athlete's safety comes first. Athletes with strong family histories or prior events should be evaluated by a cardiologist. Protocols for the evaluation of athletes at risk are beyond the scope of this chapter but should not be ignored. The reader is referred to the ACSM guidelines for exercise testing and prescription (9,10).

Peripheral Vascular

A history of smoking, claudication, and especially rest pain is important to elucidate. Palpation reveals a cool extremity in arterial problems, a swollen tender extremity in venous problems, and a tense, woody feel in compartment syndromes. Older athletes may have chronic deep venous thromboses, phlebitis, and varicosities. A hand-held Doppler ultrasound is useful in the office. If arterial compromise is suspected, the ankle/brachial index and segmental pressures are valuable initial examinations. An angiogram, although somewhat invasive, is the gold standard. Duplex ultrasonography is useful for determining patency of the arterial and deep venous systems. Often in an athlete, an exercise-induced compartment syndrome is confused with a vascular obstruction. If this is a concern, intracompartmental pressures can be measured before and after exercising.

Musculoskeletal

The musculoskeletal screening should include not only evaluation of symptomatic joints, but a focused palpatory examination, and motion testing of body areas that contain significant tissue texture abnormality. Osteopathic findings should not be separated out from the orthopedic conditions that present. Is postural imbalance contributing to the athlete's problem or complaint? Is the painful exttemity on the concave or convex side of a spinal lateral curve? Is a flat thoracic kyphosis contributing to an extension dysfunction in the upper thoracic region? Is there a crossover pattern in the cervicothoracic junction or thoracolumbar region? Does an increased thoracic kyphosis contribute to

protracted shoulders and an anteriorly translated humeral head position?

Musculoskeletal problems may prevent the athlete from doing the aerobic exercise necessary to achieve the benefits. These factors may appear during the physical examination, or they may become apparent when the athlete starts exercising. The osteopathic physician is in a unique position to evaluate and treat musculoskeletal complications so that the athlete can realize the benefits of aerobic exercise.

Metabolic and Endocrine

Diabetic athletes have significant subcutaneous palpatory findings. These findings resemble a doughy feel similar to putty underneath the skin. The upper thoracic area (T1–T4) is a common place to find these changes. These changes typically are worse when their glucose is not well controlled. In the type 2, overweight diabetic, the upper thoracic kyphosis is usually increased, which may contribute to pain or muscular tension in the cervical spine. A combination of diabetic control, weight loss, manual treatment, and exercises to strengthen the rhomboids and lower trapezius is beneficial.

MANUAL MEDICINE APPROACH TO THE GERIATRIC ATHLETE

Many musculoskeletal injuries or athletic-based illnesses have a somatic component that is never diagnosed or addressed during treatment. Not only does treatment need to incorporate the original injury and associated somatic dysfunctions, it should take into account the chronic diseases and medications as well.

Elderly active athletes often demonstrate palpatory evidence of somatic dysfunction. The type, location, duration, and severity of the somatic dysfunction must be determined. The somatic dysfunction in this population may be related to their chief complaint, or to another illness, injury, or condition. The *art* of treating these patients lies within the determination of how much and what somatic dysfunction is clinically significant, and then what to do about it. Many manipulative techniques need to be modified or adjusted to be safely and effectively applied to the elderly athlete. Proficiency in indirect technique is an especially important skill for sports medicine clinicians to acquire, as it presents less stress to the elderly body.

Functional Screening Examination

The standing screening examination in the older athlete is performed in the same fashion as for the general population; however, it is not uncommon to find some differences. During the osteopathic standing structural examination, it is common to see various asymmetries of bony landmarks, muscular development, and soft tissues. The left and right halves of the body when viewed from a midsagittal plane are not typically bilaterally symmetrical.

Observation of these asymmetries begins as the athlete walks into the room. Viewing the athlete walking from more than one angle is also advantageous. Spinal asymmetries can also be palpated during active or passive motion. Static asymmetry is very different than functional asymmetry. The osteopathic standing structural examination looks for positional asymmetries of various anatomic landmarks and evaluates anterior-posterior and lateral spinal curvature. Standing asymmetry may or may not be clinically relevant unless it is a component of a TART (*T*issue texture change, *A*symmetry, *R*estriction of motion, *T*enderness); then it has structural and functional implications. Standing asymmetries are basically a snapshot in time of what form the body's structure is maintaining under the influence of gravity. Long-term adaptations to these asymmetries become "fixed," with functional implications.

Dynamic asymmetry occurs as an athlete walks or runs, steps up or down, balances or jumps, or performs a sit-up or other exercise. Mitchell described a motion cycle of walking in which various postural asymmetries and arthrodial patterns change based on the phases of gait (13). This type of evaluation is clinically useful for the geriatric athlete because the concept can be expanded into the athlete's daily activities or sport.

Have the athlete mimic motions and body positions that he or she repetitively uses throughout the day. Does the athlete frequently extend the neck? What happens to the anatomic landmarks as he or she does this? Does the athlete reach to the right from a sitting position? What happens to the scapula when he or she does this? Have the older athlete stand balancing on the left foot on a 2-in.-high beam, or stepping up and down. What happens to the levelness of the iliac crests as he or she does this? To lumbar lordosis? To the head position? Now repeat the evaluation on the right foot.

From a functional standpoint, these findings help the clinician to determine how the patient compensates through daily activities. The clinician can then determine why these movements are not symmetrical. Dynamic asymmetry evaluation allows the clinician to palpate and evaluate distant areas for somatic dysfunction. Following treatment, the process can be repeated. This determines if treating the somatic dysfunction improves the functional asymmetry and quality of motion used in day-to-day or athletic activities.

Palpation

Palpation can give a clinician significant information about disease process, injury, or dysfunction. Is there an axial component to the athlete's complaint? Palpation of muscles for activation or tone, connective tissues for tension or laxity, and articulations for barriers can be done during the exam, or as the athlete performs various activities and exercises. For example, palpate the lumbar spine or ilia as the athlete maintains a neutral spine (posterior pelvic tilt) during a supine heel slide. Try palpating the scalenes as the athlete practices abdominal breathing, or attempt to place the athlete in the position of injury and palpate the ease or bind of myofascial structures, comparing it to the static position. Does the athlete's upper thoracic dysfunction improve or worsen while standing on one foot? Is his or her breathing more labored or easier while holding a posterior pelvic tilt and walking down the beam?

Athletic patients have an overall firmness to their muscles and tissues not found in the general population. Healthy muscle feels smooth and homogeneous, with taut fascia and fewer subcutaneous findings. The palpatory examination can determine the presence and severity of tissue texture abnormality, as well as the size, shape, and tone of the muscles. The extent and location of muscle splinting or guarding (which is a common finding in athletic injury) needs to be determined using palpation; for example, muscle splinting of the hamstrings gives a false-negative anterior drawer test at the knee.

Another palpatory finding is subcutaneous bogginess or puffiness. In the upper thoracic region, these findings may be prominent over the rib angles or underneath the scapula. Motion testing the segmental area of the spine that has this type of tissue change reveals a rubbery, nondistinct end-feel to joint motion. These palpatory findings may relate to a viscerosomatic reflex. This is useful clinical information that may prompt the physician to investigate gastric acid reflux, asthma, or angina.

Palpation reveals injury to ligamentous structures, such as in the acute ankle sprain. Acutely torn ligaments are locally tender and may have a palpable defect. Rents (tears) in the fascia with or without muscle herniations can also be palpated. Increased tissue tension, muscle firmness, fullness, and pain to applied pressure are common palpatory findings in compartment syndromes of the extremities.

It is important to palpate an injured area (and sometimes distant areas) when the athlete is performing a particular motion, such as performing a sit-up. Tenderness, muscular findings, or even a hernia may be accentuated at this time. Tenderness of muscle during a concentric or eccentric contraction may indicate a muscular strain. Scar tissue does not actively contract and may be locally tender during this examination.

Beyond manual medicine, observation and/or palpation of muscle firing patterns can also be evaluated. A common example of this is the low back pain athlete who cannot fire his or her gluteus maximus while performing the commonly used prone leg extension. In lower back pain, the gluteus maximus becomes functionally

inhibited. The same inhibition is commonly seen when the patient does a step-up. Palpation at this time also allows the clinician to determine if a particular muscle is firing or is inhibited. Consider the vastus medialis muscle. It commonly becomes flaccid and atrophic following knee arthroscopy or injury. This is a neurologic inhibition, not a true weakness. This factor is important to determine and treat before prescribing more advanced therapeutic exercises.

There are particular patterns of muscular contraction seen in various movements. Athletes who have significant muscular imbalances, somatic dysfunction, or injury are prone to having these abnormalities. Consider low back pain athletes who fire their lumbar paraspinal instead of their gluteal muscles with each hip extension. This pattern becomes learned and will persist and lead to further injury or incomplete recovery if not corrected by appropriate neuromuscular retraining.

Motion Testing

Motion testing should include joints, soft tissues, fascia, and cranial motion. It is performed in areas that contain significant tissue texture abnormality. Before motion testing, it is important to understand that athletes may have very different qualities of motion, yet these are still normal findings. For example, motion testing an athlete's innominates may reveal a symmetrical yet firm compliance. A sedentary athlete is typically found to have a soft, mobile pelvis. Older athletes with significant osteoarthritis are very stiff and have a markedly diminished range of motion. Some athletes have significant flexibility of their ligaments and joints, yet this is not appreciated readily because of the generalized tone of the muscles. Clinically, it is more difficult to palpate areas of articular hypermobility than hypomobility. Adolescent females are commonly found to have ligamentous laxity or hypermobility. This is commonly seen at the knees and the shoulders. Sports that accentuate this include gymnastics and swimming. Older athletes appear to be stiffer and typically do not suffer from ligamentous laxity. These differences in flexibility may be genetically determined or age related. Symmetrical range of motion and an appropriate range of motion for a given activity are important factors to elucidate in motion testing and later on during rehabilitation.

Motion testing includes an evaluation of the quantity and quality of motion. Articular somatic dysfunction typically occurs in the joints minor motions. A significant variable during motion testing is the concept of end-feel, which is a qualitative finding at the end point of physiologic motion. End-feel may be appreciated as resistance or motion or laxity to motion. Microtraumatic injuries (repetitive overuse) may have a firm end-feel due to muscle tightness. Macrotraumatic injuries evaluated within the golden hour may produce a loose or sloppy end-feel, especially if ligamentous disruption occurred. After disruption, muscle splinting of the injured area occurs, and the end-feel may become firm. Viscerosomatic reflexes may produce a rubbery end-feel to the tissues. The type of restriction found during motion testing helps guide the selection of manipulative treatment.

GENERAL MANIPULATIVE TREATMENT GUIDELINES

Elderly athletes' tissues are less flexible, more fibrotic, and stiffer than younger athletes. This affects the way in which geriatric athletes are treated and examined. Seated manipulative techniques work well, especially for the thoracic and lumbar areas. Deep articulation is a reasonable approach to improve global and segmental spinal motion. For the shoulder, a side-lying or seated approach is important, as it is well tolerated by elderly athletes. The scapulothoracic articulation can be treated as well as the lumbar paraspinals. Elderly athletes typically need two or three pillows to support their neck when lying supine. This is in part due to the increased high thoracic kyphosis and also due to the fact that cervical extension causes a narrowing of the spinal canal. Osteoarthritis of the spine causes a restriction in segmental lateral translation. This is especially evident in the cervical spine.

Dosing of medication is definitely an issue in the geriatric athlete. Older athletes have a higher

pain threshold than younger patients; however, they still may be sore after a manipulative treatment. Dosage of treatment is a significant factor. A good rule of thumb is to treat no more than two regions at the initial visit so that the clinician can determine the patient's response. Elderly athletes have both acute and chronic musculoskeletal problems. They respond slower than younger athletes and generally need better instruction in performing home exercises or stretches.

The type of treatment applied to the geriatric athlete must be carefully individualized and performed after thorough evaluation. Indirect technique is usually effective for the geriatric athlete, as it is atraumatic and positions lesions away from the restrictive barrier; however, he or she may not be able to assume certain counterstrain positions. Direct techniques such as high-velocity, low-amplitude (HVLA) thrust ate relatively contraindicated in the osteoporotic patient, and muscle energy must take into account the athlete's strength and ability to relax. If the athlete is unable to relax or execute the procedure, muscle energy should not be used.

Lateral Curvature of the Spine

Lateral curvature of the spine is a common finding in the older adult population. Clinically, the challenge is to determine if the curve is the etiology of the problem, the effect of the problem, or totally unrelated to the chief complaint. Long-standing curves with anatomic adaptation resist change and over time these curves become the *new neutral* position of the spine. A lateral spinal curvature is a lateral deviation of the spine in a coronal plane, from a plumb line dropped, bisecting the body into right and left halves. A lateral curve is also known as a group curve, which is defined as a lateral deviation of the spine by three or more vertebral segments. The motion pattern of these curves follows Fryette's first principle of physiologic motion. Harrison Fryette described group curves in the 1920s. He described physiologic motion of the spine (12). When the spine is in neutral position without marked flexion or extension, and side bending is introduced, the vertebrae rotate opposite to the side bending, into the produced convexity. Group curves are characterized as a type I dysfunction.

Fryette felt that group curve (type I) mechanics occurred in the thoracic and lumbar spine. Functional group curves are named according to the side of the convexity as well as the level of the apex. The apex segment of the curve is the most rotated vertebral body. Curves can be *C*-shaped or *S*-shaped (double curve).

According to Mitchell, throughout the cycle of walking, as weight is shifted from one leg to the next, spinal group curve mechanics are in play (13). With each heel strike, an alternating lateral curve is produced. If heel strike occurs on the right, the left sacral base declines momentarily causing a left lumbar convexity. Since the eyes must be kept on the horizontal plane, a compensatory right thoracic convexity occurs. This scenario produces an *S*-shaped curve: the primary curve is lumbar, and the secondary curve is thoracic. With the next heel strike on the left, the right lumbar spine is the convex side, and the left thoracic compensation occurs. In 1983 *Postural Balance and Imbalance* was published by the American Academy of Osteopathy (14). It reviews the osteopathic profession's contribution to the clinical and theoretical significance of postural balance. Clinical approaches and radiographic methods of assessing postural balance are discussed.

There are numerous etiologies for lateral curvatures. The most common are the following:

Unilateral muscle spasm
Long-term anatomic adaptation
Leg-length inequality
Postural imbalance
Traumatic fascial or cranial strain patterns
Compensation from an injured or weakened area
Nociception
Unilateral changes in autonomic function
Viscerosomatic reflexes
Somatosomatic reflexes
Segmental somatic dysfunction (out of step)
Traumatic vertebral wedging
Myofascial trigger points

Anterior-posterior (A-P) curves can be determined when visually inspecting the patient from the side. Athletes demonstrate less lumbar lordosis and have a more military spine. Clinically, this may be seen if the athlete participates in exercise involving extensive military presses, overhead racquet sports, and repetitive spinal hyperextension activities. The athlete with a flattened lumbar lordosis typically has tight iliopsoas muscles and a sacrum that prefers to extend. The athlete with an increased lumbar lordosis typically has a sacrum that likes to flex, and often presents with a painfully hypermobile L5 vertebra.

Runners may have a variety of foot problems, including asymmetrical pes planus, which can present as a short leg on structural examination. In this situation, the iliac crest and greater trochanter may be low on the same side. Pelvic side shift is away from the short leg side, and a lumbar spinal convexity is commonly palpated on the short leg side. While lying supine, the leg may appear shorter and externally rotated, especially if a posterior innominate shear is present.

Asymmetrical hamstring tension, which is common in athletes, can affect posture and the standing flexion test. A tight hamstring muscle may hold the innominate down, giving a false-negative test on the same side. A tight iliopsoas affects the standing examination. For instance, it may produce pelvic side shift away from the psoas spasm. The athlete may appear bent over forward and to the side of the spasm, and the belt line may appear low on one side.

Extended Somatic Dysfunctions of the Spine

Of particular concern is an extended (Fryette's type II) dysfunction in the upper thoracic region. These lesions have a palpable pothole or a flattening of the thoracic kyphosis on palpation. Somatic dysfunction of the upper thoracic spine and ribs can affect scapulohumeral rhythm and scapular position. Smooth, efficient movement of the scapula and coordinated strength of the scapular stabilizing muscles are necessary to prevent rotator cuff impingement. Extended upper thoracic segments are not only painful and persistent; they also produce a wide array of cervical and upper extremity complaints and may produce somatovisceral responses in the cardiac or respiratory system. These responses must be noted carefully because geriatric athletes already have a higher likelihood of cardiac or pulmonary disease.

Extension exercises are a valuable modality for treatment of extended somatic dysfunction of the spine. The extended somatic dysfunction is maintained by flexed areas (extension restriction) above and below the extended dysfunction. Extension exercises address the flexed areas that maintain the segment with extended dysfunction. Hyperextension, however, is painful for these patienrs. If the upper thoracic interscapular area is involved and the patient must perform a prone spinal extension exercise, excess extension can be controlled by limiting extension of the head and neck. This is accomplished by keeping the athlete's chin toward the chest. These exercises need to be performed slowly, one vertebral segment at a time.

Cervical Spondylosis and Neck Pain

The upper thoracic spine is a crucial area ro palpate and diagnose in cervical spine complaints. The upper thoracic spine and ribs (especially T1-T4) supply sympathetic tone to the head, neck, and upper extremities. Afferent fibers from the cervical spine (and nerve roots) synapse in the upper thoracic cord (intermediolateral cell column) and can become a focus of hyperactivity of the sympathetics. Many neck problems have an identifiable upper thoracic component through this mechanism. Hyperactive upper thoracic sympathetics (often T2 and T3) can refer pain, tingling, or abnormal temperature sensations to the arm. It is not uncommon to find the upper extremity patient with a gastrointestinal viscerosomatic reflex pattern in the T5-T6 segments, although this is often due to the prolonged consumption of nonsteroidal anti-inflammatory drugs in their treatment that must be addressed as well. Palpation of these

findings may signify why symptoms persist despite treatment. Orthopedic disease of the neck (herniated disc, osteoarthritis, and stenosis) may refer pain to the upper thoracic region, shoulder, arm, and hand. Athletes with cervical root disease develop shoulder and/or arm pain when they lie flat on the table. Somatic dysfunction of the lower neck and cervicothoracic junction can produce arm symptoms through three mechanisms:

- Direct irritation of cervical spinal nerves.
- Neurovascular compression (thoracic outlet syndromes).
- Myofascial restrictions and lymphatic dysfunction of the thoracic inlet.

Neck pain is often associated with instability of the cervical joints and foraminal narrowing due to degenerative disease or osteoarthritis. Active exercises are necessary to strengthen and stabilize those weakened segments of the cervical spine. First, find a dose of sttengthening exercises that will work without causing pain. Mild isometric exercises with the neck in a comfortable neutral position are often a good starting point. Circumduction neck exercises (neck rolls) that were often recommended by clinicians in the past have been found to localize the forces to the unstable area and may exacerbate the instability.

Lumbar Spinal Stenosis and Sacroiliac and Lumbosacral Pain

In a patient with lumbar spondylosis and stenosis, identifying and treating existing psoas hypertonicity is a priority. A flexed Fryette's type II dysfunction is commonly found in the upper lumbar region. During the evaluation of gait, if the patient walks in a forward-bent position, he or she may have an iliopsoas spasm. The following specific findings on physical examination may indicate this somatic dysfunction:

- Restriction of hip extension.
- A belt line that is low on one side.
- A flexed dysfunction in the upper lumbar spine (L1-L2).
- Pelvic side shift that frequently occurs toward the side of the longer leg (or opposite to the side of the tighter psoas muscle).
- A recurrent anterior sacrum or forward sacral torsion.

A tight psoas puts the lumbar spine in a vise and adds compressive forces across the lumbar discs. It typically causes the upper lumbars to be flexed and the lower (L4-L5) lumbars to be extended. The L5 vertebra typically is the most painful to palpation and motion testing, largely because it is the most mobile of the lumbar segments and is often unstable. Athletes with backache and iliopsoas syndrome need extension exercises to strengthen paraspinal extensor muscles, yet because of the pattern of lumbar dysfunction, extension movement of L5 produces subjective pain.

Treatment

Treating the flexed lumbar component, followed by stretching the tight psoas muscle, releases the hypertonic psoas. Care must be taken not to apply excessive forces through the area of spondylosis and stenosis during treatment. A muscle energy or counterstrain technique would be preferable for the upper lumbar Fryette lesion, followed by static or dynamic stretching and/or myofascial release for the psoas muscle.

Extension exercises can be performed comfortably if the clinician positions the athlete prone on the table and places one or more pillows under the pelvis to flex the L5 vertebra. This positioning tends to restrict extreme hyperextension of L5 so that the athletes can tolerate the spinal extension exercises. These exercises also help stretch the iliopsoas.

Lumbar disc problems usually do not tolerate a flexion load of the lumbar spine. However, gentle flexion exercises are often indicated in these patients. If the athlete's head is elevated to reduce the gravitational load and a slant board is used for a sit-up or curl abdominal exercise, lumbar exercises are much easier for the patient to tolerate.

Sacroiliac dysfunction is also commonly present in athletes with iliopsoas hypertonicity and

lumbar stenosis. The type of sacroiliac dysfunction is variable and related to the dysfunction. Bilateral iliopsoas spasm usually presents with sacral extension. Unilateral iliopsoas spasm often presents with pelvic side shift away from the spasm and backward sacral torsion.

Thorough treatment of the iliopsoas hypertonicity typically helps sacroiliac dysfunction. If significant sacral dysfunction is still present after treating the iliopsoas, muscle energy, myofascial, or indirect techniques applied to the sacrum are useful. Dysfunction of the innominates or pubic symphysis should be evaluated and treated.

CONDITIONS SEEN IN THE GERIATRIC ATHLETE

Hip or Knee Joint Arthroplasty

Unwanted results from total hip or knee arthroplasty include short leg syndrome, postural imbalance, and lumbosacral pain. Often an ipsilateral hamstring contracture is present, confounding the situation. This also contributes to making the postoperative leg functionally shorter by keeping the knee flexed. Heel lift therapy in these patients and dynamic stretching of the hamstring work well. Occasionally, the arthroplasty makes the leg longer, and a heel lift is needed on the opposite side. Standing pelvic radiographs with assessment of sacral base declination is the standard method of diagnosing a true short leg syndrome. For best results, perform the radiographs after treating any sacroiliac and lumbar dysfunction. Once the short leg is established, treatment should level the sacral base.

Rotator Cuff Impingement Syndrome

Rotator cuff tendinitis and subacromial bursitis are common sports medicine diagnoses, particularly in athletes over age 45 (15). Impingement syndromes can occur due to multiple factors, many of which are discussed in Chapter 17. In the geriatric population, postural factors are more significant. These changes with aging are well documented, including the kyphotic cervicothoracic hump, the protracted scapulae, anterior cervical and head carriage, and flattened lumbar curvature. These changes limit scapulothoracic motion, resulting in more impingement.

Another factor is accumulation of wear and tear seen in geriatric athletes (15). Tendinosis, not to be confused with tendinitis, is a thickening and degeneration of a tendon that develops over time from use and overuse, and impingement increases due to the increased thickness of the tendinotic tissue or spurring from an arthritic acromioclavicular joint. Additionally, connective tissue loses its strength and flexibility over time as a natural part of aging, making the tendon less adaptable to stress and prone to breakdown.

Osteoarthritis

Osteoarthritis is difficult to deal with from a manual approach. Knees, spine, and shoulder typically suffer the most in osteoarthritis. Achiness, decreased range of motion with a stiff end point, crepitance with motion, and morning stiffness are common patient complaints in degenerative arthritis. Standard conservative treatment may be helpful, and arthroscopic débridement can be helpful in some cases.

In using manual medicine with osteoarthritis, treatment objectives include the following:

- Mobilize the glenohumeral and scapulothoracic joints as much as possible.
- Lower autonomic tone from the upper thoracic spine and ribs.
- Decrease overuse and tension of the neck muscles.
- Improve lymphatic drainage from the upper extremity.

EXERCISE PRESCRIPTION/ REHABILITATION

Any rehabilitation and exercise prescription must have the whole athlete in mind. The rehabilitation of the older athlete involves deter-

mining the extent of injury or illness, the body's functional compensations, and any intrinsic or extrinsic factors present. Functional compensations are produced in response to gravity, ground-reaction forces, and momentum. Define the extrinsic environment (weather, geographic location, equipment, personal competition) as well as intrinsic environment of the patient (structural malalignment, strength, endurance, flexibility, emotional state, disease state) when tailoring a rehabilitative program or treatment approach. Choose exercises that are safe, effective, and efficient. A logical progression chosen for the specific athlete is important. Choose goals at each level of rehabilitation, such as improved range of motion, decreased swelling, improved gait and posture, strengthened core muscles, improved balance and coordination, improved endurance, and decreased pain, with a graded return to activity. These variables should be evaluated based on what movements and activities are necessary for the athlete to compete or participate.

Because older athletes typically have concomitant medical conditions, some types of exercises are discouraged or contraindicated. Poor balance in an osteoporotic cyclist could lead to a fall and traumatic or pathologic fracture. A well-tolerated alternative for the athlete, whose musculoskeletal system is too compromised to deal with the effects of gravity, is to exercise in a warm-water therapy pool. A therapeutically directed Tai Chi program is also useful before proceeding to more aggressive exercises. Safe, controlled weight-bearing exercises along with adequate calcium and vitamin D help stimulate bone density. Another approach, with the benefit of loading the bony matrix, is for the elderly to perform controlled anaerobic weight training. When tolerated, a strengthening program with weights can be very beneficial in this population. There has also been information evaluating exercise methods used in cardiac rehabilitation. Research suggests that resistance exercises and circuit weight training are of value in these patients (16,17).

Exercise Preparation

The intrinsic and extrinsic factors affecting the athlete prior to competition should always be evaluated in a checklist fashion. This includes hydration, nutrition, medications, warm-up activities, equipment status, and safety issues. A period of warm-up exercises should precede any prescribed exercise program. Warm-up requires mild muscle contractions to promore circulation. These exercises warm up the muscles by stimulating arterial dilation. Warm-up prior to activity is not synonymous with stretching (see section on flexibility). Warm-up exercises for a runner would involve walking first and then running slowly. For a tennis player, walking, then slow running, followed by lobbing the ball warms up the muscles. Warm-up activities should include motions common to the sport being played. A tennis player does a few groundstrokes and serves, a bowler throws a few frames, and a runner jogs. The goal is to break a light swear. The older athlete has less hydration of their connective athlete and decreased elasticity, which means they generally need more time to warm up and cool down. They also need longer periods of time to acclimate to their geographic location. Many geriatric athletes take medications that could affect their performance. Diabetic athletes may hold their insulin prior to activity and have to be cautious in their fluid status, monitoring their glucose level frequently. They may need to consume frequent carbohydrates to ward off hypoglycemic episodes.

Flexibility

There are many types and methods of stretching. Ballistic stretching uses momentum and repetitive terminal bouncing to achieve muscle lengthening. In older athletes, dynamic stretching of a well warmed-up muscle group seems to work the best.

Another type of stretching involves an isometric muscle energy technique that probably uses the Golgi tendon organ reflex. The muscle to be stretched is lengthened to engage the restrictive barrier, held in that position, and then actively contracted against some isometric holding force. The hamstrings may be stretched using this muscle energy approach. The person stands on one leg, places the heel of the other leg (leg straight and knee locked) on a

chair or stool, and positions the body to load the hamstrings. While maintaining this position, the heel is pushed against the stool by actively contracting the tight hamstring. The muscle stretch is repositioned to engage a new restrictive barrier, and the process is repeated. Isometric muscle contraction away from the restrictive barrier not only stretches the muscle but also provides a circulatory benefit with movement of fluids. This type of muscle contraction is believed to reduce the gamma tone to the tight muscle.

In addition to muscle, collagen fibers are also subjected to the stretching force. Collagen has properties of both fluids and of solids, termed viscoelastic properties. The viscoelastic properties of collagen dictate that stretching requires time. The fluid component of collagen requires time for the collagen to flow as it is lengthened. Collagen must be stretched slowly. If stretching is overdone or done improperly, it can lead to adverse side effects. Collagen can be overstretched to a point at which it is permanently deformed and will not return to its original length, in the same way that a spring can be overstretched so that it is permanently deformed. Overstretching of muscles, tendons, ligaments, and collagen fibers is called plastic deformation. Joint instability is the usual consequence of plastic deformation of tissues. It is possible to overstretch a strong low back and produce a weak, unstable, painful low back. Static stretching provides no trophic stimulus and so does not strengthen muscles. However, dynamic range of motion produces increased muscle strength and flexibility.

Strengthening

Manual medicine techniques to relieve somatic dysfunction or fascial tension of the spine and/or extremities can be the key to a successful strength training program for the geriatric athlete. Injury requires rest time as well as strength training in between training sessions. However, prolonged rest can undo any strength gains, so in recovery from injury only a limited amount of rest is needed. Continued inactivity leads to a progressive cycle that includes weakness, reduced resistance, and increasingly severe clinical consequences resulting from minor stresses. Inactivity coupled with the gradual loss of strength and flexibility from aging demonstrates the need for strength and flexibility training.

According to the 1995–96 Surgeon General's report and the 1995 American Health Association report, weight training in the older athlete has been shown to do the following (16,17):

- Increases or improves bone mass, strength, flexibility, endurance, and balance.
- Decreases the incidence of coronary disease, cancer, and type 2 diabetes.
- Improves self-confidence and esteem.

Older adults are recommended to perform one to two sets of 8 to 12 repetitions in eight to ten body regions, approximately two times per week. This recommendation is for healthy sedentary adults. Athletes train significantly above this level; however, the guidelines set a minimum requirement for all older adults to participate in an active lifestyle.

CONCLUSION

Manual and manipulative treatment of the older athlete is worthwhile and beneficial for a variety of conditions. Clinicians need to perform a thorough musculoskeletal screening examination, a palpatory examination, and motion testing to determine if a somatic component is present. If somatic dysfunction is present and significant to the athlete's chief complaint, its treatment needs to be integrated into the total rehabilitative plan. There are many exercise programs for older athletic patients. Manipulative treatment sets the stage for proper healing to begin. Exercise programs should include concomitant manual treatment with progression based on objectives to be accomplished. The athlete's fitness level, other medical problems, and abilities need to be reviewed frequently. Patience is necessary on the part of clinician, trainer, and patient because older athletes tend to respond more slowly. Adjusting their therapy is frequently necessary. Handouts listing specific exercises for injury rehabilitation are appropriate after the athlete has

already learned the proper form for doing the exercises one on one. Categorizing and sequencing exercises with objectives in mind make it easier to prescribe appropriate exercises for the geriatric athlete.

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43

THE EMERGENCY ROOM ATHLETE

FRANK A. PAUL

Frequently, emergency medical patients and athletes have a musculoskeletal component to their presenting complaint, or it may be their primary concern or a finding during the physical examination. The treatment should conform to the standard of care. This care plan should include the use of manual medicine, which has been a part of the therapeutic health care armamentarium for over 2,000 years (1).

One of the goals in treatment of acute musculoskeletal injuries is to *decrease edema* in the injured area. Therapies that decrease motion restrictions and optimize physiologic range of motion help decrease swelling and improve recovery. Lymphatic drainage and venous return are facilitated through three mechanisms: (a) gravitational forces, (b) pressure changes generated through respiration within the thoracoabdominal-pelvic cylinder, and (c) musculoskeletal activity generating a pumping mechanism.

Application of manual medicine provides the patient with the potential for an improved structure–function relationship, which should facilitate lymphatic drainage and venous return. Table 43.1 contains a list of contraindications to the use of manipulation in the emergency department patient.

INITIAL EMERGENCY ROOM INTERVENTION

A clear, concise protocol for the diagnosis of sports injuries and concomitant musculoskeletal injuries must be established. First, the emergency room (ER) physician needs to rule out life-threatening and neurovascular emergencies quickly. A history of the injury and a relevant physical examination follow. The often-used Ottawa knee and ankle rules were devised to decrease the unnecessary use of standard radiographs in these common areas of injury.

Appendicular bone and joint computed tomography (CT) and magnetic resonance imaging (MRI) studies are typically outpatient procedures, whereas axial spine fractures may be detected only on CT and should be a part of the athlete's emergency evaluation if clinical suspicion warrants the study.

The reduction of closed dislocations and minor fracture deformities is typically delayed until the prereduction condition is established and documented with standard radiographs. Exceptions include the presence of any neurovascular compromise distal to the derangement or significant tenting of the overlying tissue. In these cases, prompt reduction to a more native anatomic position should be performed.

Salter-Harris type I fractures are difficult to rule out using standard radiographs in the setting of pain due to injury of a nonfused growth zone. Therefore, regardless of the findings using standard radiographs, potential injuries to epiphyseal plates should be treated as a fracture until proved otherwise with immobilization and serial examinations during follow-up evaluations. Ligamentous injury is common in the athletic patient. Passive range of motion and ligamentous testing should be deferred until ruling out significant structural damage or bony instability. MRI is the study of choice to rule out ligamentous instabilities of the axial spine. The ER physician should have a low threshold for ordering urgent studies of the axial spine.

TABLE 43.1. MANUAL MEDICINE IMPLEMENTATION IN THE EMERGENCY DEPARTMENT

General Contraindications
Time constraints
Other seriously ill patients that require undivided attention
Contraindications to Local Application Using Direct Action Techniques
Vascular insufficiency or infarction of the spine
Severe arthritides that cause instability of the spine
Direct techniques on sprains/strains exceeding first degree
Acute fracture, subluxation, dislocation
Severe osteoporosis
Neoplastic processes of/or adjacent to the dysfunctional segment
Congenital or acquired lesions causing spinal cord compression
Acute herniated nucleus pulposus within 72 hours of onset (the identified segment)
Spinal cord tumor
Uncontrolled coagulopathy
Dizziness without defined etiology
Any acute neural deficit
Osteomyelitis, discitis
Retinal tear, detachment
Ocular lens dislocation
Intracerebral bleed
Any trauma to an incompletely closed bone growth plate
Carotid stenosis
Vertebrobasilar insufficiency

Adapted from Koss RW. Quality assurance monitoring of osteopathic manipulative treatment. *J Am Osteopath Assoc* 1990;90(5):427–434.

Significant concomitant musculoskeletal injuries can be distracting, effectively masking visceral and spinal trauma. A history of the injury and a relevant physical examination to rule out concurrent injuries must be thorough and comprehensive.

Standard Treatment Approach

The key to treating the athlete in the emergency room is ruling out high morbidity conditions quickly. The sooner a diagnosis is made, the sooner proper treatment can begin. For the competitive athlete, each missed step along the way can delay return to sport by days or sometimes weeks. The average ER patient, by contrast, usually does not have such temporal restrictions.

The typical acute sports injury is initially treated with the PRICE protocol—*p*rotection, *r*est, *i*ce, *c*ompression, and *e*levation. Oral analgesics should be provided when there is no need for immediate surgical intervention. Otherwise, the use of parenteral pain management is indicated. Aftercare instructions should include prompt outpatient reevaluation to ensure the institution of aggressive physical or occupational therapy. This also prevents complications from prolonged splinting and immobilization while enhancing compliance with the treatment plan.

Manual Medicine Approach

The stable ER athlete examination should follow a comprehensive structural approach that includes the components listed in Table 43.2. Decisions about the utility of manual medicine can then be made. Because initial standard radiographs cannot always rule out a fracture, all areas that are potentially fractured should not be manipulated with direct techniques.

TABLE 43.2. COMPONENTS OF THE STRUCTURAL EXAMINATION

Postural asymmetry
Soft tissue/bony palpation
Contractures/deformity
Areas of tenderness
Regional/segmental dysfunction
Atrophy/hypertrophy
Amputations

Direct techniques should be used only in areas where the absence of fracture is certain and only by the skilled practitioner at this stage of treatment. These areas benefit from balanced membranous tension, indirect myofascial release, and craniosacral techniques. These techniques are usually well tolerated by the patient and reduce muscular spasm and vascular congestion, facilitating venous drainage and lymphatic return.

LOW BACK PAIN

The ER evaluation should include a complete review of symptoms, history of present illness, preexisting health problems, and an assessment of the patient's ability to function with these symptoms. Past medical, surgical, family, social history, and current medications all should be obtained. The differential diagnosis of low back pain is enormous, so do not assume the athlete's complaint is isolated to one body system or that the complaint is associated with age-related degenerative disc disease.

The physical examination is essential for sorting through the potential causes for low back pain. Acute sports injuries should have a mechanism of injury, so attempt to obtain it from the athlete, trainer, parents, ambulance driver, or coach. Abnormal vital signs should not be dismissed as being caused by musculoskeletal pain without considering other system pathologies. Temperature elevation may be as simple as a component of the flu with accompanying myalgias or as a consequence of physical activity. However, it may indicate urinary, peritoneal, retroperitoneal, inflammatory, or spinal infectious processes. The acute pain response can elevate the heart rate through autonomic mechanisms, but the differential diagnosis for any person with isolated tachycardia, including shock, should be factored into the clinical picture. Elevation of blood pressure can be secondary to pain but may be reflective of renal, aneurysmal, central nervous system, or circulatory origins of back pain. A rise of the diastolic component of blood pressure, causing a narrowed pulse pressure, may also be indicative of shock, as this component of blood pressure measurement rises with moderate volume loss, while the systolic pressure remains unchanged. Sustained tachypnea should not be overlooked as a pain-moderated physiologic response.

The structural examination can begin on arrival to the emergency room. Notice posture, method of arrival, positioning, and movement while in the ER. This can be accomplished with minimal additional time added to the physical examination and obtained almost entirely with the patient in the supine position. Pertinent laboratory and clinical radiologic studies should be performed to confirm the clinical impression. A high level of clinical suspicion for occult neoplastic or inflammatory processes should be maintained, particularly with a history of trauma (2).

The clinician should eliminate the emergent causes from the differential diagnosis before arriving at a musculoskeletal diagnosis or instituting manual medical treatments. Pain relief and muscle relaxation facilitate the movement of interstitial fluids and decrease the stresses on inflamed tissues. Care for significant musculoskeletal back pain should include use of skeletal muscle relaxants, nonsteroidal anti-inflammatory agents (NSAIDs), and narcotic analgesics. Early rest is recommended followed by an increase in level of activity as tolerated. The athlete should be given comprehensive aftercare instructions, with timely follow-up. Documentation should include the physical findings necessitating the intervention, modalities used, subjective treatment toleration, the degree of mobilization achieved with treatment, and the results of a post-treatment neurovascular examination.

Manual Medicine Treatment

Clinical experience has repeatedly demonstrated that manual medicine can provide pain relief for patients with retroperitoneal abscess, pelvic inflammatory disease, pulmonary embolus, appendicitis, and pelvic neoplasms. Treatment of dysfunctional areas that restrict fascia and hamper lymphatic flow can offer some symptomatic relief without manipulating the areas directly involved. These areas include diaphragmatic attachments (T9-L1), the thoracic inlet (T1), and the remainder of the thoracoabdominal-pelvic cylinder.

Manual medicine is also used in the management of patients with herniated nucleus pulposus (HNP) (3,4). However, since scientific literature has yet to clarify whether manipulation is indicated or contraindicated in caring for the patient with acute HNP, avoid manipulating segments that correlate with true radicular symptoms in the ER setting. These patients obtain some symptomatic relief by treating the areas above and below the involved spinal segment using indirect techniques.

CHEST PAIN

The clinician must rule out the emergent etiologies of chest pain before addressing the musculoskeletal component of the patient's condition. Do not assume your athlete started the activity in perfect cardiovascular and pulmonary health, particularly in the absence of thoracic trauma. A musculoskeletal diagnosis may accompany cardiovascular or pulmonary pathologies. Fifteen percent of patients with an acute myocardial infarction have reproducible chest wall pain with motion, palpation, or respiration (5). It is estimated that at least 21% of patients with pulmonary embolus have respirophasic, pleuritic, and reproducible chest pain (6). The clinician should rely on the history and physical examination to help differentiate the cause of the complaint. The differential diagnosis of chest pain is detailed further in Tintinalli's Emergency Medicine.

Chest trauma is common in sports medicine, yet has major structural and visceral considerations that may require acute diagnosis and management. Pulmonary contusion is reported to be present in 30% to 70% of patients with significant blunt chest trauma (7). Because the abdominal organs can be located as high as the level of T4 with deep exhalation, be sure to assess the upper abdominal organs for concurrent injury with any mid or lower chest trauma. The chest wall trauma evaluation should focus on the health of the underlying tissues. This is especially true in the pediatric population. Pediatric patients rarely fracture ribs but are more likely to sustain damage to the underlying tissues as a consequence of their bony elasticity.

Ancillary studies are used to establish baseline pulmonary health on chest trauma patients and to confirm the diagnosis. Chest radiographs and pulse oximetry are appropriate studies for patients with acute chest trauma. Standard rib radiographs may miss 10% to 50% of simple rib fractures (8). Their usefulness in the management of these patients is delegated as an initial study to prepare for medical clearance to resume contact sport activities as tolerated.

The musculoskeletal evaluation of the athlete includes lateral and anterior-posterior compression of the chest as well as flexion, extension, and rotation of the thoracic spine. Palpate the paraspinal tissues to assess for acute and chronic tissue texture changes. Viscerosomatic reflex patterns should also be identified. A list of these reflexes can be found in DiGiovanna and Schiowitz's. *An Osteopathic Approach to Diagnosis and Treatment.*

Manual Medicine Treatment

Visceral and neural pathology generates musculoskeletal pain through facilitation (9–11) and through direct local irritation. Musculoskeletal manipulation often improves the athlete's subjective pain complaints regardless of the cause of pain. Manipulative treatment of athletes with pathology such as an acute myocardial infarction, pneumonia, asthma, and pulmonary embolus may provide some degree of temporary symptomatic improvement. Do not use manual medicine results to determine whether the symptoms are visceral or somatic in origin.

Diagnostic decisions and patient management or disposition should not be altered based on changes in or resolution of the symptomatology following manual medicine.

The treatment plan for the acutely injured patient is consistent with that discussed for low back pain. The focus is on decreasing the athlete's discomfort, respiratory effort, and restoring normal chest wall motion. This aids in the prevention of pneumonic processes forming as a consequence of the mechanical and pain-mediated interference with respiratory function.

Treating somatic dysfunctions identified in the transitional spinal areas (T1, T12-L1, L5-S1) decreases the tension on the painful areas by diminishing the amplitude of the normal compensatory spinal curves, providing some symptomatic relief. This also allows for more range of motion before involving the injured, painful region of the spine. The clinician will have more success in mobilizing acute dysfunctional segments if these are addressed later in the process. High-velocity, low-amplitude (HVLA) thrust technique (in the absence of fracture potential), thoracic bowl technique to the upper thoracic spine, muscle energy technique, strain–counterstrain, and balanced membranous tension rib release techniques are time-efficient procedures. All identified viscerosomatic dysfunctional segments should be treated and monitored during follow-up care.

Adequate analgesia is imperative for managing acute chest wall trauma to prevent pneumonic processes from developing in otherwise healthy individuals. Some combination of NSAIDs narcotic analgesics, and muscle relaxants should be prescribed. Evaluate the athlete's respiratory effort, making sure it is appropriate for outpatient management. Incentive spirometry helps prevent problems caused by hypoventilatory breathing from voluntary patient splinting. Rib belts and therapeutic splints are contraindicated in the care of chest wall trauma. The athlete should receive aftercare instructions and timely outpatient follow-up. Documentation of the physical findings, segmental areas of dysfunction, and treatment modalities rendered to these areas should be included in the medical record.

CERVICAL SPINE TRAUMA

Cervical spine trauma sustained during athletic activities is a frequent reason for an ER evaluation. The history must include questions that would reveal any reason for the patient to have a preexisting condition predisposing the spine to injury. These conditions may include a history of intravenous drug abuse, cancer, tuberculosis, or prior injury. If the trauma is consistent with potential for ligamentous or bony injury, cervical spine stabilization precautions should be instituted including use of the supine position and a rigid, properly fitted cervical collar.

The review of symptoms should search for problems with autonomic dysfunction, numbness, or muscular weakness. Determine if the pain is radicular or radiating from a trigger point. Radiating pain in a dermatomal pattern suggests a radiculitis due to nerve root impingement. The physical examination should include evaluation of the reflexes, strength and sensation of the cervical nerve roots, and any other peripheral nerve complaints. Palpation of the cervical spinous processes and transverse processes as well as paraspinal tissue texture should be performed. Excessive motion with anterior-posterior or lateral translation, crepitus, and stepoff are often ominous. These findings should be communicated to all involved in the athlete's care. No range of motion testing should be performed until instability and injury are ruled out by physical examination and/or radiography. Keep in mind that distracting injuries may interfere with recognition of significant neck trauma.

Standard radiographs should be obtained with a history of significant trauma, especially if bony tenderness is present on physical examination. Some cervical spine osseous injuries may be detected only on CT and should be a part of the athlete's emergency evaluation if clinical suspicion or standard radiographs warrant further study. MRI is more sensitive than CT for ligamentous injury and hematoma, while CT is preferred for spine fractures. Any recognition of progressive radiculopathy tequires prompt MRI/CT and surgical evaluation.

Manual Medicine Treatment

Minor soft tissue cervical spine injuries benefit from indirect techniques, appropriate direct techniques, and craniosacral techniques. Treatment to reduce compensatory spinal curve amplitude with mobilization of the transitional segments (T1, T12-L1, L5-S1) decreases the tension on the acutely injured cervical spine and can be beneficial to the patient's recovery. Fascial release techniques applied to the acute segments are tolerated well.

Manipulation of the acutely injured cervical spine should be limited to prevent overtreatment of these regions. Using counterstrain is helpful in treating acute spasm and dysfunction without irritating injured tissue, but the clinician should be sensitive to how the athlete responds. HVLA is strongly discouraged acutely on ER admission.

TORTICOLLIS

Acute wryneck, or torticollis, is a common neuromusculoskeletal problem in the active individual. It often presents after a relatively minor trauma like a glancing blow to the forehead from a basketball, falling down, or it may follow strenuous physical activity. Abnormal neck posture from sleeping on an airplane or in a strange bed may even generate the problem. A thorough history and physical examination should be performed. Standard radiographs, if indicated, help rule out osseous or facet pathologies. They should be considered if one intends to use direct techniques to the acute dysfunctional areas. Providing muscle relaxants and NSAIDs prior to obtaining radiographs is helpful in disrupting the pain-spasm cycle and facilitates subsequent manipulative intervention. Encouraging the athlete to remain supine prior to treatment also helps decrease the tone of the paraspinal muscles.

Manual Medicine Treatment

Isolated trigger points causing radiating pain respond to ethyl chloride spray and stretch manipulation, as well as strain–counterstrain and direct inhibition techniques. Use of soft cervical collars is controversial as a form of splinting. Given the need for rest of the injured area, bed rest or use of a recliner should be encouraged to eliminate weight bearing of the cervical spine for 48 hours. The typical patient, if given a splinting brace, is inclined to overuse the neck by being more active and upright. Muscle relaxants, NSAIDs, and narcotic pain medications should be used to supplement the mobilization and neck rest regimen.

The neck is frequently a site of pain and disability not related to trauma or wryneck. Neck pain may be related to physical exertion, somatic dysfunction, or compensatory changes in other areas of the axial spine. Reducing compensatory spinal curves as well as increasing range of motion through mobilization of the transitional segments often relieves the athlete's symptoms without direct cervical spine mobilization. Mobilization of the occipitoatlantal junction using myofascial release, suboccipital tension release, and balanced membranous tension is beneficial. If time allows, treat the most acute paracervical or midline counterstrain tender point and any hyperacute tender points in the trapezius, levator scapulae, or supraspinatus muscles. Patients generally have some residual soreness but acquire significant increases in range of motion with less pain.

ASTHMA

Patients arriving in the emergency room with acute bronchospasm are usually anxious, tired, and overwhelmed due to the effects of air trapping on the body's compensatory mechanisms. The athlete often complains of chest pain or tightness and shortness of breath. The range of motion of the chest wall is decreased (12). These motion restrictions compound the sensation of shortness of breath and fatigue, while increasing the work of breathing. Initial episodes of difficulty in breathing should always be evaluated with a chest radiograph.

Treatment of the acute asthmatic episode should conform to the standard of care.

However, the use of manual medicine in the asthmatic patient can provide a substantial decrease in the level of anxiety and the subjective work of breathing. Bockenhauer et al. demonstrated a statistically significant increase in chest wall excursion following MM on patients with chronic asthma, although peak flows did not significantly change (13). Clinical experience has noted improvements in peak expiratory flow rates following treatments.

The manipulative treatment of these patients should include range of motion or articulatory techniques to the clavicles, as well as treatment of somatic dysfunction in the thoracic spine, thoracic inlet (T1), and ribs. Treatment applied to the sternum, paraspinal tissues, and diaphragm is important. This requires minimal time with no energy expenditure from the patient. It does not interfere with the delivery of oxygen, beta-agonists, intravenous medications, or diagnostic studies. Rib release techniques and respiratory motion augmentation in the patient with severe asthma can be applied in the sitting position and are well tolerated. Avoid direct stimulation of the upper cervical unit (occipitoatlantal joint, C1-C2) in severe asthmatics, as vigorous stimulation of the vagus nerve can theoretically generate a somatovisceral-mediated exacerbation of bronchospasm. However, inhibitory techniques to the lung viscerosomatic regions T1-T4 may help ablate bronchospasm.

MUSCLE TENSION HEADACHE AND SINUSITIS

Headache is a very common presenting complaint in emergency medicine. The active individual often develops a muscle tension headache as a consequence of overuse syndromes or strains. Additionally, 30% of patients presenting with acute sinusitis complain of a suboccipital headache as part of their chief complaint (14). In athletes with acute sinusitis, the fifth cranial nerve innervating the sinus walls can cause neural facilitation of the upper cervical nerves C1 and C2. This facilitation of the trigeminal nucleus is likely responsible for the somatic dysfunction that tends to develop at the cervico-occipital junction.

Be aware that the athlete who comes into the ER with headache from competition should be evaluated for neutologic insult to rule out traumatic brain injury. Any history of trauma to the head should be considered a concussion until proper evaluation can be made.

Manual Medicine Treatment

Purulent symptomatic sinusitis treatment involves use of antimicrobials, air humidification, elimination of exposure to noxious environmental agents, pain control, and consideration for use of topical and/or oral decongestants.

Manipulative treatment of these patients should affect the healing process by (a) improving the range of motion of the key anatomic areas affecting lymphatic drainage and venous return, (b) decreasing adnexal impingement on the frail lymphatic channels, (c) improved medication, (d) metabolite and oxygen transfer through a normalization of the structure–function relationship, (e) improved biochemical efficacy of antibiotics in the relatively hypoxemic sinusoids, (f) decreasing pain by alleviating somatic dysfunction, and theoretically by (g) improvement of the autonomically mediated circulatory control mechanisms of the surrounding areas.

Treating motion restrictions of the upper cervical unit (occipitoatlantal joint, C1-C2), clavicles, and cervicothoracic junction (T1) is an important component of the therapy. This can be done using either direct or indirect treatment to any dysfunctional cervical spinal tissues. Restoring normal motion at the sphenobasilar synchondrosis in conjunction with venous sinus drainage and ethmoid-vomer augmentation using craniosacral techniques is conceptually helpful. Lymphatic drainage treatment of the face, neck, and thoracic inlet is an essential adjunctive therapy.

DISCUSSION

The goal of this discussion is to create a conceptual marriage between manual medicine and

the traditional treatment approaches in emergency medicine. The ER is a logical place for this relationship to merge. There are many reasons used by clinicians to omit manual medicine from the practice of emergency medicine, the most common being time constraints and presumed liability.

However, osteopathic clinicians have been shown to provide cost-effective management of worker's compensation injuries, which are common to the ER and frequently involve musculoskeletal symptoms (15). Time constraint is a real factor in the emergency room, but no more than 2 to 6 minutes are needed to perform the necessary manual therapies. High liability for using manual medicine is a myth. It is within the lowest risk class of all treatments used in the medical profession today as designated by medical malpractice carriers. National clinical practice guidelines have clearly outlined that manipulation is recommended in the management of acute back pain (16); therefore, manipulative procedures should be included in the emergency medicine standard of care guidelines. Other factors play a tole in the disuse of manual medicine, such as disinterest, inadequate skill level or training, standard of care breach, inadequate or absent reimbursement for the procedures, unfamiliarity with the contraindications, and patient unfamiliarity with manual medicine.

Treating the musculoskeletal complaint benefits the athlete by removing the cause of pain that prompted the ER visit, ameliorating the neuromusculoskeletal components contributing to the physiologic and autonomic dysfunctions of the disease, providing efficacious therapy, and improving the physician-athlete relationship. Manual medicine also provides aggressive management of acute pain to prevent chronic pain pathways from developing. Finally, it can help decrease the social, financial, physical, and psychological morbidity caused by protracted dysfunction while assisting the body in its innate healing process. Emergency clinicians are motivated by immediate gratification and enjoy helping their patients. Manual medicine can provide this satisfaction for the emergency athlete and the health care provider.

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44

THE PEDIATRIC ATHLETE

PIERCE M. SHERRILL

This chapter is devoted to children, defined as those who have not finished their physical growth. Children are not miniature adults. They are growing—sometimes rapidly—while negotiating a complex landscape of physical, mental, social, emotional, and spiritual hurdles. Many are in puberty, which is second only to infancy in the amount of growth and development a child completes (1).

There is only one universal rule in treating sports injuries in children: "Tailor the process to the child's specific needs." Muscle, tendon, and ligament may become tight or lax as growth proceeds; this mandates changes in the standard exercise prescription. A child's skeletal muscle responds differently to strength training; routine adult conditioning may do more harm than good to a child. Some injuries, such as apophysitis, only develop in the skeletally immature; others, such as osteochondrosis, develop diffetently in children.

DEMOGRAPHICS

The 2000 census reported 61,297,467 children aged 5 to 19 in the United States. They comprised 21.78% of the total population. Of these, 69.22% were white; 15.10%, African American; 3.45%, Asian; 1.17%, American Indian/Alaska Native; 0.18%, Hawaiian/Pacific Islander; 7.36%, other; and 3.52%, "two or more races." There were slightly more males than females (2).

EPIDEMIOLOGY

The Centers for Disease Control and Prevention reports that almost 6 million high school students play team sports. Another 20 million participate in recreational or competitive sports outside the school setting. There are over 775,000 emergency department visits per year for sports injuries to children under the age of 15. Given the 41,077,577 children aged 5 to 14 in the 2000 census, this yields an annual incidence of 1.88% (3).

Gender and Racial Differences

There are distinct gender differences, and more subtle racial/ethnic differences in the kinds and mechanisms of injury in the pediatric population. Females participate in fewer sports involving contact, collision, or combat. Despite this, their injury rates approximate and sometimes exceed those of their male counterparts. Offer et al. (1) observe that many girls succumb to social pressures against vigorous exercise. This lack of conditioning contributes to injury risk. Females are also more likely to participate in appearance-sensitive sports such as gymnastics, figure skating, and dance. These sports emphasize flexibility and slenderness over strength. This increases the risk for injuries such as spondylolisthesis and joint instability.

Girls enter puberty about 2 years earlier than boys. They begin their final growth spurt earlier, and their epiphyses close about 2 years earlier as well. As a result, girls have 2 years less growth than boys. Women are, in the aggregate, shorter in stature and have less muscle mass than men.

Girls have a wider pelvis, greater femoral anteversion, and more genu valgum. There is less vastus medialis development, and a greater incidence of patella maltracking. Girls also have more lumbar hyperlordosis, and a greater risk

for nutritional disorders. All of these factors increase a girl's risk for musculoskeletal injury (4).

Despite the earlier puberty, girls at age 16 average 75% of the strength of their male counterparts, and they have proportionately less muscle mass. However, Offer (1) observes that when girls engage in strenuous and vigorous exercise through childhood, they develop a magnitude of strength similar to that of boys. It is not yet clear how much of the observed difference is sociocultural, and how much is hormonal or developmental.

Racial and ethnic differences include sport of preference; method of training; emotional response to injury; social and family support; folklore about health, injury, and healing; and expectations for recovery.

UNIQUE ISSUES

Physical

Children have open epiphyses, or bone growth plates. The epiphyses consist of uncalcified cartilage, and are the most fragile segment in the bone-joint-ligament-tendon-muscle complex (Fig. 44.1). Epiphyses are vulnerable to compressive and shear forces. In children, a joint sprain may actually be an epiphyseal fracture. It is critical to establish the appropriate diagnosis, since epiphyseal fractures carry the risk of premature closure and asymmetrical bone growth.

Similarly, children have apophyses. These tendon insertions into bone share many features of the epiphysis, but are most vulnerable to traction. During puberty, skeletal muscle increases in bulk and strength before its apophyseal insertion calcifies. If the strength of the muscular contraction exceeds that of its anchor, a traction injury (apophysitis) can result (Fig. 44.2).

Articular cartilage is softer in children; their subchondral bone resembles the epiphysis and is just as vulnerable to shear and compression injury. The injuries that produce chondromalacia in adults can cause subchondral fractures and osteochondritis dissecans in children (4). These injuries may need more aggressive management, including surgical débridement (5).

A child's muscular strength, VO_{2max}, and cardiac output are proportionately lower than an adult's; however, surface area-to-body mass is greater in children (6).

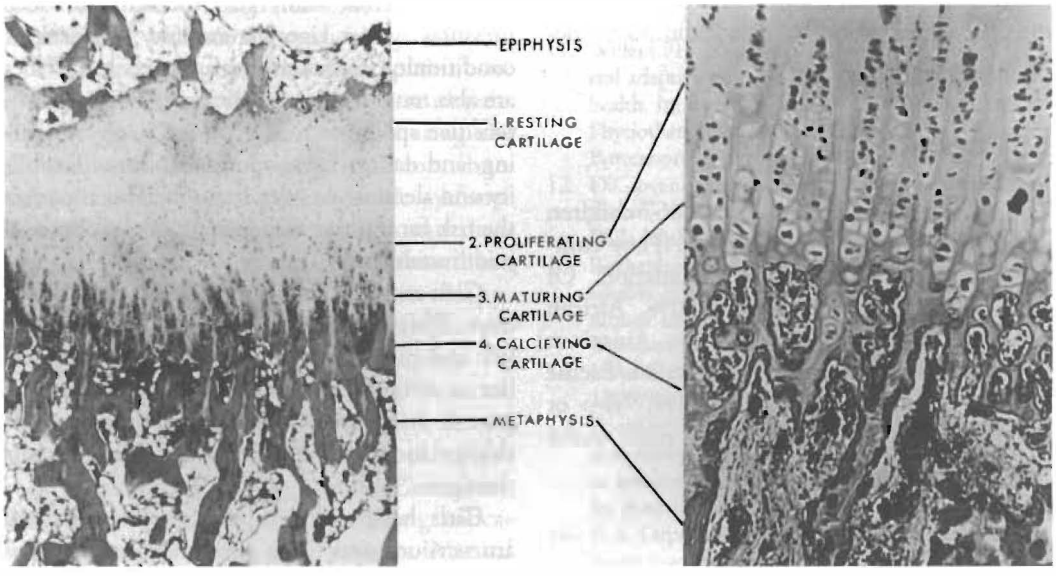

FIGURE 44.1. Histology of an epiphyseal plate from the upper end of the tibia of a child. **A,** Low power. **B,** High power. (From Salter RB. *Textbook of disorders and injuries of the musculoskeletal system,* 2nd ed. Baltimore: Williams & Wilkins, 1983.)

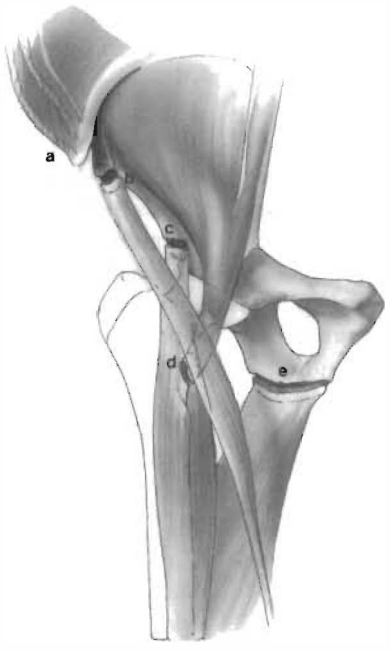

FIGURE 44.2. Avulsion fractures of the growing pelvis result from traction injuries where major muscle groups insert into or originate from apophyses about the pelvis. Abdominal and trunk muscles insert into the iliac apophysis **(a)**. The sartorius originates from the anterior superior iliac apophysis **(b)**. The direct head of the rectus femoris originates from the anterior inferior iliac apophysis **(c)**. The iliopsoas inserts into the lesser trochanter apophysis **(d)**. The hamstrings originate from the ischial apophysis **(e)**. (From Morrissy RT, Weinstein SL, eds. *Lovell and Winter's pediatric orthopedics,* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2001.)

Skeletal muscle in preadolescents does not hypertrophy in response to strength training. The strength gains achieved in children are the result of enhanced neuromuscular recruitment and motor-unit synchronization (5). Preadolescent weight training has also been shown to facilitate Wolff's law of soft tissues, that is, the body's tendency to remodel connective tissues along lines of greatest force (7). Prepubescent weight training (in a carefully supervised setting that emphasizes technique over power) can enhance late-adolescent strength gains. Once androgenic adult hormones appear, muscle hypertrophy can occur.

Social

More people are involved in the treatment of the pediatric athlete than the adult athlete. Permission to treat comes from parent or guardian, as does transportation to and from treatments. Parents and coaches may need to be included in return-to-play decisions. Also, they may have unreasonable expectations and may need more education than the athlete. Family, teammates, and coaches may need to help ensure compliance with treatment. On the other hand, these same people may provide conflicting advice or enable self-defeating behaviors.

The injured child will have evolving relationships with family, coaches, and peers. The injury may affect the child's self-image, or social standing as a successful athlete. The injury may have impact on peer relationships within or outside the team.

Athletic competition places children on opposite teams. Aggressive behavior may be inherent to the sport. The pediatric athlete may not

understand the difference between healthy competition and unhealthy aggression.

Mental

The injured child will not have an adult's comprehension of the injury. Erickson and Piaget each developed an outline for the stages of a child's psychosocial development (3). Both note that the adult's ability to deal with a complex issue such as an athletic injury develops relatively late in adolescence.

A child may see a painful procedure as punishment, or misconstrue the physical contact between caregiver and patient. Education and instructions may be misinterpreted or forgotten, especially if they conflict with what is heard from parents or coaches. Younger children may not believe the injury will heal.

Emotional and Spiritual

When injured and unable to compete, adolescents can respond with behaviors that are out of proportion to the actual injury (9). Most injured athletes go through a grief process like that described by Elizabeth Kübler-Ross (10) in her work with the dying patient. The denial, anger, bargaining, depression, and acceptance seldom follow a smooth, orderly progression: the patient may exhibit several stages at once, or become locked in one stage. Such behaviors may also need treatment.

Another normal coping technique in the pediatric athlete is regression, in which the child temporarily adopts behaviors from a younger age (11). What appears to be immaturity is actually healthy coping; it allows the child some emotional breathing room as he or she tries to deal with unfamiliar stresses.

Magical thinking can persist well into adolescence. Some children see their injury as punishment for a sin that is completely unrelated to the problem at hand. They may try to buy a cure with ritual behaviors. Children sometimes see the caregiver as an adult authority figure and rebel directly or passively-aggressively. Feelings of invulnerability increase the risk for injury in adolescents.

APPROACH TO THE PEDIATRIC ATHLETE

The basic precept in caring for the pediatric athlete is to tailor the process to the child's specific needs, as previously mentioned. This process includes noting the patient's stage of musculoskeletal development, and whether he or she has begun or ended a growth spurt.

Strength and flexibility imbalances need to be addressed in the treatment plan. The following points deserve mention:

1. Aggressive strength programs begun during a growth spurt may contribute to the injury, because they may aggravate tight muscle-tendon units (12).
2. Hypermobility in an adolescent may be present as the normal part of a growth phase, and does not in and of itself indicate somatic dysfunction.
3. If the hypermobility is truly pathologic, direct-action manipulation, or any technique that increases mobility in a hypermobile segment, is inappropriate (3).
4. Adverse changes in strength and flexibility during treatment may be due to differential growth, and not due to noncompliance or treatment failure.
5. Problem areas that seem resistant to treatment may spontaneously improve as the patient's growth continues.

The young athlete may never have been in a continuing therapeutic relationship. The terms of that relationship must be spelled out clearly and carefully. Many problems with noncompliance can be prevented by making it clear that the caregiver is an advocate, not a boss. This relationship may be the first one in which the child interacts with an adult as an equal.

Privacy may be a pivotal issue, especially in a male-female therapeutic relationship. A respect for privacy can bridge the trust question and strengthen the relationship. This is particularly true with injuries near the groin and chest.

When possible, the athlete should continue to participate with the team in some way. A limited training routine still allows contact with

teammates and coaches. These social connections give incentive to complete the rehabilitation and return to play.

If treatments last longer than a week, negotiate a written return-to-play contract that spells out the steps in rehabilitation and the criteria for return to play. Setting goals on a visit-to-visit schedule allows the athlete to see progress, and can discourage nonproductive behavior (18).

The athlete should understand the nature of the injury and the purpose of the treatments. By asking what the patient knows about his or her injury, providers can address misconceptions and teach at an age-appropriate level. A useful prompt is In this clinic, there's no such thing as a silly question. I'll give you a straight answer to anything you want to know about. If the child is unable to make use of the information, it should go to a parent, coach, or capable family member.

The athlete experiences a wide variety of emotions as the stages of grieving progress. There may be a disconnection with what he or she is feeling. Support can be as simple as identifying his or her emotions. Support may be more complex if acting-out, hostility, or other negative behaviors appear. If the injury does not permit return to the sport, this needs to be made clear to the child and alternative activities explored.

This is a good time to talk to the athlete about maladaptive issues in sports; a well-informed athlete is less prone to reinjury. Topics to explore include aggressive behavior in sports; expectations of players, coaches, and parents; participation despite medical limitations; sports nutritional supplements and performance enhancers such as anabolic steroids; eating disorders; and inappropriate relationships on and off the team.

The History: Special Considerations

A history of previous injury, especially if the athlete is incompletely rehabilitated, is the single most reliable predictor of reinjury (15). A history of noncompliance or treatment failure is also cause for concern.

If the athlete is a minor, consent and confidentiality become an issue. The parents or guardians are entitled to review routine medical records. If the child wants to keep something off the record, this needs careful consideration of the child's age and situation. Any suspicion of physical or sexual abuse or neglect is subject to the usual reporting requirements.

The athlete may forget or deny critical details. It is helpful to corroborate history with teammates, coaches, and family. This is also an opportunity to learn what the athlete is hearing from these sources, and what pressures or expectations are being brought to bear. Because many young athletes are poor historians, use things like events (holidays, birthdays, and games) to estimate onset of symptoms and other temporal issues.

Physical Examination: Special Considerations

Always assume that the patient has never been through an examination before. Explain all procedures that may cause pain or embarrassment, especially when dealing with sensitive areas such as the groin, buttocks, or chest. Ask permission to touch. Respect the child's privacy and need for comfort. Encourage the parent to be present.

Look thoroughly for signs of spinal abnormalities during the examination, such as dimples, hair tufts, café au lait spots, and spinal curves. Lower back strength is roughly equal between boys and girls until about age 9 or 10, then it changes. Boys have the greatest amount of change around 15 to 16 years old, while girls increase markedly around age 11. At age 18, female back strength is roughly 70% of males (16).

MANUAL MEDICINE TREATMENT PARADIGM

The general approach to children's athletic injuries is similar to the adult model. The following three-phase program provides early restoration of comfort, then targets rehabilitation and return to full activity (15).

Phase 1: Acute

In the acute phase, goals are to control pain, restore range of motion, and preserve strength. Pain can usually be controlled with ice, analgesics, and modalities such as interferential current or transcutaneous electrical nerve stimulation. Acetaminophen, alone or in combination with narcotic analgesics, may be combined with nonsteroidal anti-inflammatory drugs in age-appropriate doses. Because of its association with Reye's syndrome, aspirin is best avoided. Ultrasound is unproven in acute pain, and in children, it should be avoided over the epiphyses because it may cause growth disturbances (6).

Immobilization, when necessary, should be brief. Strength losses as much as 17% have been documented after 72 hours of immobilization (15). If a muscle injury is to be immobilized, splinting with the muscle in a stretched position helps to reduce edema and bleeding (17).

Manual medicine techniques in the acute phase focus on restoration of gross motion with a minimum of discomfort. Indirect techniques (counterstrain) and gentle isometric (muscle energy) are preferred over mobilization with impulse, which can cause spasm and pain. Lymphatic techniques are invaluable to address swelling without causing significant pain.

Early mobilization—first passive, then active range of motion—reduces scar formation and speeds strength recovery. Once active range of motion has begun, gradual increases in muscle loading can be added. When pain is controlled and range of motion is near-normal, the patient can begin phase 2.

Phase 2: Recovery

The goal of the recovery phase is restoration of preinjury function. In this phase, pain should be controlled or absent. Tissues are progressively loaded to direct remodeling and restore strength. As already noted, strength gains in the preadolescent and early adolescent child will be due to enhanced synchronization and recruitment, not muscle fiber hypertrophy. Strength training should initially be directed toward high-repetition, low-weight routines. Late-adolescent athletes (Tanner stages 4 and 5) may then transition into a high-weight, low-repetition power routine if appropriate to the athlete's sport.

Manual medicine techniques in this phase may appropriately place more demands on the injured muscle, tendon, and bone. Soft tissue techniques such as massage and isolytic stretching are useful once acute inflammation has resolved. After mobilization, strengthen the injured area with graduated isokinetic and plyometric routines. These techniques increase circulation to the area and direct remodeling of connective tissue.

The recovery phase is complete when the child is pain-free and has essentially full, painless range of motion with good flexibility. He or she may then progress to the rehabilitation phase.

Phase 3: Functional Rehabilitation

In the rehabilitation phase, the athlete begins sport-specific training. The goal is performance-level function. This is the time to correct biomechanical problems that either led to the injury or resulted from it. It is also the time to address manual medicine techniques to areas distant from the injury. Mobilization-with-impulse techniques are safe to use at this stage of healing, if appropriate.

The child may be released to full participation and discharged from care, once the following criteria are met: the athlete is pain-free; there is no edema or swelling; normal range of motion has been achieved; the child has normal strength on the injured side when compared with the uninjured side; and appropriate functional testing shows no residual deficit in comfort or performance (18).

COMMON PEDIATRIC INJURIES

Apophysitis

Apophysitis is usually the result of traction on the apophysis. Less often, a direct blow causes the pain and inflammation. The most common

sites are the anterior tibial tubercle (Osgood-Schlatter disease), inferior pole of the patella (Sindig-Larsen-Johanssen disease), os calcis (Sever's disease), iliac crest, fifth metatarsal, and olecranon. Any area where tendon inserts into immature bone is vulnerable.

Apophysitis usually occurs in children aged 10 to 15, at Tanner stage 3 for females, and Tanner stage 4 for males. At these ages, the child is growing rapidly: skeletal muscle is beginning to hypertrophy under the influence of androgenic hormones. Rapid strength gains often coincide with tight muscle-tendon-joint relationships. These asymmetries can be exaggerated by differential growth, as bone lengthens faster than its muscle (15). The apophysis is the weakest structure in this system. It becomes symptomatic if the tendon tries to pull away from its anchor.

There may be other factors involved. For example, a football player who feels pressured to bench-press his body weight may overtrain and develop an olecranon apophysitis. A female distance runner with genu valgus, femoral anteversion, and forefoot pronation is at risk for Osgood-Schlatter disease. A volleyball player with worn-out knee pads may develop Osgood-Schlatter disease from repetitive impact to the tibial tubercle, as he or she drops to the floor for a ball. A history of apophysitis in one area increases the risk for a subsequent episode elsewhere (5).

Therapeutic goals (beyond those listed previously) include unloading the affected tendon; avoiding impact on, or explosive contraction of, the affected muscle group; stretching the agonist/antagonist muscle groups; padding as necessary; and correction of biomechanical problems with bracing or orthotics.

Manual medicine techniques should initially address tight muscle-tendon groups with submaximal isometric stretches. Other soft tissue techniques may improve circulation in the area. When pain is well controlled, a graduated program of isokinetic strengthening, progressing to plyometric training, allows the apophysis to harden with a minimum of discomfort.

It may not be reasonable to expect complete pain relief. If the athlete is willing to accept mild discomfort as the price of the sport, he or she may be cleared to compete if all other discharge criteria are met. Once the athlete's epiphyses close, the problem resolves.

Two complications deserve mention. The first, chronic apophysitis, is a painful apophysis that persists after skeletal maturity. This is usually self-limited, although some patients require steroid injection or surgery for pain relief.

The second, apophyseal avulsion, is rare. When it occurs, it may need surgical attention. In avulsion, the body's protective mechanisms have been overwhelmed. If there is no obvious explanation (such as isolated trauma), consider anabolic steroid abuse, which blunts pain perception and aggravates strength imbalances. This can allow the athlete to ignore the body's warning signs and progress to rupture.

Epiphysiolysis

As already noted, the epiphysis is often the "weakest link" in the muscle-tendon-ligament-joint-bone complex. Repetitive multidirectional forces can cause the cartilage of the epiphysis to weaken and lyse. Little league shoulder occurs in the proximal humeral epiphysis, and is the result of rotation-with-distraction, usually occurring in pitchers (Fig. 44.3). Gymnasts can develop lysis of the distal radial epiphysis (gymnast's wrist) from repetitive compression-with-rotation during handsprings and cartwheels (20). Other vulnerable areas include the vertebral body (Scheuermann's disease) and the proximal femur (slipped capital femoral epiphysis).

The most important therapy is to stop the offending activity. Lysis of the epiphysis carries a significant risk of premature physeal closure, with subsequent growth arrest. This can cause length discrepancy in the long bones as well as vertebral wedging.

The injury is extremely variable, ranging from mild physeal inflammation to complete disruption. Treatment is equally variable, depending on the location and severity of the injury. In slipped capital femoral epiphysis, surgery is usually necessary; in Scheuermann's disease, there may be no need for treatment.

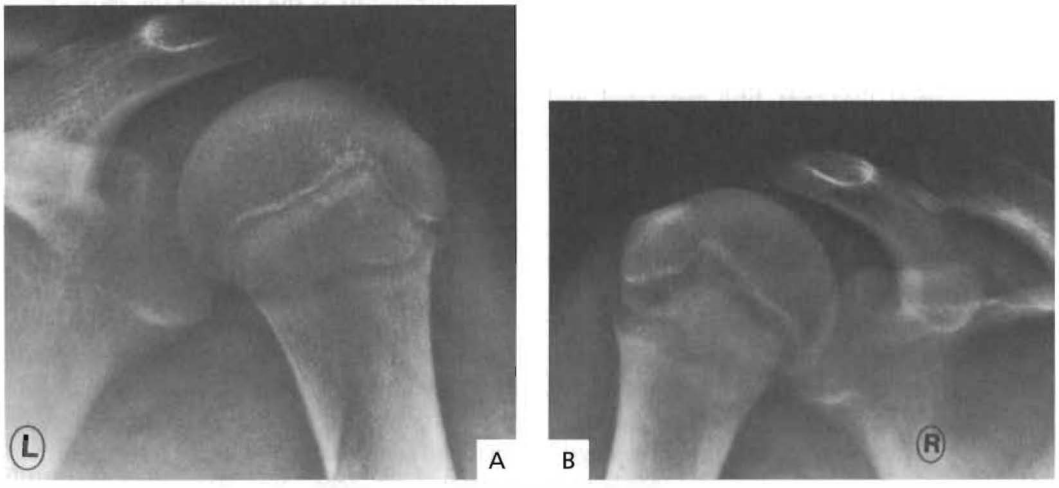

FIGURE 44.3. Radiographs of the proximal humerus in a 14-year-old right-handed baseball pitcher with shoulder pain that progressed over the final few weeks of the season. **A,** Widening and irregularity of the physeal plate are present in the right shoulder. **B,** Radiograph of the left shoulder is provided for comparison. (From Morrissy RT, Weinstein SL, eds. *Lovell and Winter's pediatric orthopedics*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2001.)

Each injury's rehabilitation requires careful coordination with the orthopedist. Once the athlete is cleared for rehabilitation, the treatment paradigm outlined earlier can be tailored to the athlete's specific needs.

Joint Sprains

In large joints, the collateral ligaments often insert into the epiphyses (4). These ligaments have near-adult tensile strength, which makes the epiphysis the weakest part of the system. Any injury capable of spraining a collateral ligament is equally capable of fracturing the epiphysis, which is why pediatric sprains are uncommon.

Stress Fractures

When a bone is subjected to repetitive loads that exceed its remodeling ability, the cumulative overuse may result in a stress fracture. Over 40% of all stress fractures occur in preadolescents and adolescents aged 19 and younger; 6% occur in children below the age of 16 (5). Tibial stress fractures are by far the most common, with metatarsal march fractures coming in a distant second. Most stress fractures occur in endurance running sports such as cross-country and soccer.

Stress fractures are a signal to search for biomechanical problems, such as hyperpronation, or training errors, such as improper footwear, inappropriate running surfaces, or poor advice from coaches or teammates. Search for strength and flexibility imbalances in the lower extremities: poor quadriceps-to-hamstring strength is a frequent contributor. Adequate warm-up/stretch is an essential part of the rehabilitation education.

Tenosynovitis

In tenosynovitis, the body of the tendon, not its insertion, is inflamed. This is the result of friction between the tendon and its synovial wrapping. There is no great difference between children and adults in either the cause or the treatment.

The most common cause is inadequate warm-up and stretch prior to exercise. Stretching before exercise stimulates the synovial membrane around the tendon. This increases lubrication to the area. If warm-up is not adequate,

the tendon runs dry of lubricant and a friction injury can result.

Direct pressure over the tendon can also cause tenosynovitis. If a runner's shoe fits too tightly at the heel, the pressure can cause Achilles tenosynovitis. Shoes laced too tightly can cause a friction injury in the extensor tendons of the toes. In these situations, padding or replacing the footwear can resolve the problem. Biomechanical problems can contribute to the injury. For example, a foot that hyperpronates may aggravate tibialis tendinitis. Orthotics may be needed to avoid hyperpronation.

Muscle Contusion

A direct blow to the belly of a muscle can tear the fibers with subsequent bleeding, edema, pain, and limited strength/motion. The primary difference between a child's muscle contusion and an adult's is the response to strength training, as previously mentioned. Treatment is otherwise identical: ice/analgesics, brief immobilization in a stretched position; passive-then-active range of motion, and compressive bandage if needed.

Muscle avulsion injuries deserve mention. An avulsion is a tear in the belly of the muscle from violent stretch or explosive contraction of the muscle fibers. As in apophyseal avulsion, if there is no obvious explanation, anabolic steroid abuse should be excluded.

Spondylolysis and Spondylolisthesis

A child's physes calcify and close in a fairly predictable order. One of the last areas to calcify in the vertebra is the pars interarticularis, which lies between the superior and inferior articular facets. As with other physes, this area is vulnerable to compression, traction, and shear forces. Traumatic or congenital spondylolysis can be described as a complete epiphysiolysis of the pars; spondylolisthesis occurs when the fragments move apart. Each causes spinal instability and pain.

The most common area affected is the lumbar spine. The most common sports are those that involve repetitive flexion, extension, rotation, and compressive loading. Gymnasts, cheerleaders, dancers, weightlifters, and football linemen are the most frequently affected.

The usual history is insidious back pain of 3 weeks or longer without a specific injury. Pain may be worse with weight bearing and exercise. The lumbar spine may show increased lordosis, pain with palpation of the affected spinous processes, and anterior translation of the affected vertebrae (step-off sign).

Most athletes respond to conservative treatment, but the offending activity must be stopped. Relative rest can include conditioning activities such as pool exercise and limb-specific weight machines that avoid stressing the spine. Once pain-free, the athlete can be conditioned and then gradually returned to sport participation.

In pars defects, the connective tissues are required to provide support to spinal elements ordinarily carried by bone. In this situation, manual therapies that increase mobility—mobilization-with-impulse, isolytic, and muscle energy—may be counterproductive. Strengthening should precede use of stretching. Spinal motion asymmetries may best be treated by strengthening the relatively mobile side, rather than mobilizing the tight side. As the athlete is transitioned back to full participation, observe carefully for signs of increased pain. Any sign of recurrence should be evaluated promptly. Acute pars fractures often require a period of time in an extension brace while rehabilitating the athlete's core. Restriction from sport for as long as 6 months may be needed, especially if the problem threatens to become chronic. These rarely require surgical fixation.

Spinal Strains and Sprains

Back pain coming from a single, identifiable injury, with a normal neuromotor examination, can be treated initially as a muscle strain. As with muscle contusion, the most important difference between a child's muscle injury and an adult's is the immature muscle's response to strength training. Remember that the immature spine is also subject to the more worrisome injuries detailed in the previous section. If pain

persists beyond 2 or 3 weeks, a more detailed evaluation should exclude skeletal injury.

Be sure to search for the strength and flexibility imbalances. Many back sprains are caused by an activity that the child could perform safely weeks to months before, but the athlete may have entered the growth spurt and lost significant flexibility. Many dance teachers, for example, limit their students' training during rapid-growth periods to reduce the risk of such injuries. If differential growth has caused flexibility problems that do not respond to modalities, stretching, and manual therapy, it may be wise to limit the athlete's training schedule until the growth spurt is complete.

Scoliosis

Adolescents with scoliosis may need treatment for athletic injuries unrelated to their spine problems. In this regard, a few points deserve mention:

- If scoliosis is detected on initial evaluation, always inquire whether evaluation has been performed by an orthopedist or neuromuscular specialist. If not, it deserves a full evaluation.
- If a spinal curve is noted to be progressing (worsening) during treatment, it deserves prompt evaluation by an orthopedist or neuromuscular specialist.
- The milder the curve, the more likely it will respond to conservative intervention such as night bracing.
- Similarly, the more pronounced the curve, the more likely it will require surgical fixation.
- There is limited (but inconclusive) evidence that physical therapy techniques can correct scoliosis (21).
- The few studies published on chiropractic-type manipulation for scoliosis show no significant benefit when compared to untreated controls (22).
- Techniques that increase the mobility of a spinal segment that is already too mobile are inappropriate and may worsen the problem.
- Treatments applied in an area distant from the curvature should be watched carefully to make sure they do not cause a postural imbalance that allows the curve to progress.

Torticollis

Acute torticollis is a sudden unnatural positioning of the head due to cervical muscle spasm. In adults, there are many causes such as arthrosis, dental pathology, trauma, and infection. However, the cause in the pediatric athlete without trauma is almost always infection. Enlarged and inflamed lymph nodes or evidence of pharyngeal infection can be seen on examination. Manipulation is *contraindicated* in these cases.

CONCLUSION

Children represent an important, unique population for the sports medicine physician. Vigilant, individualized attention given to special developmental needs, both physical and psychological, assists as the athlete in the transition from growing pediatric athlete to healthy active adult. Manual medicine techniques are helpful when applied thoughtfully and appropriately.

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THE PREGNANT ATHLETE

LORI BOYAJIAN-O'NEILL

Pregnancy alters the female body in many significant ways. For the female athlete, it is an inevitable issue to face, and for years, pregnancy meant limited physical activity. Despite the anatomic and physiologic stresses associated with pregnancy, the pregnant athlete has become more active over the years to the point where she can run long distance and work out daily up until delivery. The body's ability to adapt to and compensate for these stresses is a model of its inherent predisposition to maintain homeostasis. Seasoned athletes or those who are training to prepare for the physical stresses of labor and delivery may enjoy the benefits derived from exercise but will face physical challenges. These demands affect neuromusculoskeletal, circulatory, respiratory, gastrointestinal, and other physiologic functions, all of which have an impact on athletic participation. The pregnant athlete presents challenges to the physician as well, who must have knowledge of the anatomic and physiologic demands of pregnancy and sports medicine.

Improved understanding of the anatomic and physiologic changes that affect exercise during pregnancy and of maternal and fetal adaptation to exercise has led to recommendations for exercise in pregnancy (1,2). This greater understanding has led to guidelines for safe participation that have evolved from restrictive and extremely conservative (3) to those that encourage physical activity and exercise (1,4).

The application of osteopathic principles, including manipulation, provides comprehensive care for the pregnant athlete, and assists in restoring and maintaining homeostasis as well as optimizing anatomic and physiologic function, the goals of which are to enable continued participation in sport and exercise (5). I support the implementation of exercise prescription and manipulation into obstetric care.

Recent scientific evidence supports the use of manipulation for the treatment of acute low back pain (6). Improved understanding of the benefits of manual therapies, including manual medicine, has led to the development of guidelines related to the use of manipulation in obstetrics (7).

EPIDEMIOLOGY

In 1967, when emphasis on physical activity was much less than it is today, Sady and Carpenter reported that 15% of women of reproductive age exercised regularly and expressed a desire to continue exercise into pregnancy (8). Today, there are pregnant athletes who engage in exercise and sports activities such as marathon running, and the effects of these activities in pregnancy should be considered.

One aspect of pregnancy that may affect activity is musculoskeletal pain and dysfunction. More than 50% of all pregnant women report musculoskeletal pain during pregnancy (9). There are numerous studies investigating the incidence of musculoskeletal pain in pregnancy, especially back pain. Low back and pelvic pain are the most common musculoskeletal complaints during pregnancy. Overall, studies show that between 48% and 90% of pregnant athletes experience back pain at some time during pregnancy, most commonly after the sixth month (7,10).

FACTORS

There are many risk factors associated with low back and pelvic pain in pregnancy, including

prior history of low back pain, strenuous work, smoking, and multiparity. Women with a history of low back pain prior to pregnancy experience more severe low back pain during pregnancy (11).

Physically strenuous work coupled with a history of low back pain are factors associated with increased risk of developing low back and sacroiliac (SI) joint dysfunction and pain during pregnancy (12). Smoking is also associated with low back pain (12), among other side effects. The amount of maternal weight gain does not appear to be correlated with low back pain (9,13); neither does age, height, race, fetal weight, and socioeconomic status (13).

Multiparity has been identified as a risk factor. Some studies suggest that multiparity alone is a factor in predicting back pain, while other studies link multiparity and prior low back pain as predictors of low back pain during pregnancy (14). Fast et al. reported more disability caused by backache in multiparas versus primiparas (9). They suggest that change of posture or weaker trunk muscles (core body strength) may explain this observation.

Although exercise may predispose any athlete to increased musculoskeletal injury or pain, studies show that active pregnant athletes report fewer overall musculoskeletal complaints than their sedentary counterparts (15). There has been no reported increase in injury rates when exercising during pregnancy. The National Collegiate Athletic Association does not keep statistics on numbers of athletes who become pregnant, and there are no prospective randomized controlled studies tracking injuries and injury rates of pregnant women during athletic activity (16). There are, however, presumed risks of certain athletic activities to maternal and fetal well-being. The increased understanding of these risks, both quantified and hypothetical, guides the recommendations for exercise and manipulation during pregnancy.

MUSCULOSKELETAL ADAPTATIONS TO PREGNANCY

The avetage woman gains 10 to 12 kg (27.5 lb) during a singleton pregnancy. There is a 20% increase in body mass by the ninth month (17), which alters normal biomechanics of the spine and extremities. Increased weight and mass of the uterus cause increased stress on soft tissues and bony structures unaccustomed to carrying this extra load. Workload during pregnancy is increased, while load-bearing capacity of the musculoskeletal system is decreased (18). The center of gravity (COG) shifts, which is clinically obvious in the usual postural changes observed in pregnancy.

As pregnancy progresses, the pelvis tilts forward, which increases lumbar lordosis and moves the thoracic spine posteriorly (19). These slight alterations in biomechanics may cause primary low back pain and dysfunction and lead to compensatory or secondary changes in cervicothoracic spine and upper and lower extremity biomechanics, which can affect athletes engaged in sports and exercise.

There are no reports of increased incidence of falling during pregnancy. This may be due, in part, to athletes being more cognizant of the increased risk and adopting more safe activities. However, the potential for falls and subsequent maternal or fetal injury warrants athlete counseling regarding high-risk activities. Poorly compensated shifts in the center of gravity increase the risk of falling, especially in sports requiring balance such as martial arts, cycling, or downhill skiing. The degree to which an athlete can adapt to these inevitable musculoskeletal stresses will determine, in part, whether the athlete can engage safely in athletic activity during pregnancy.

The hormones relaxin, progesterone, and estrogen are believed to contribute to the increased ligamentous laxity
This laxity, which contributes to the widening of rhe pelvis in preparation for delivery, may lead to joint instability, pain, and dysfunction. The production of relaxin increases tenfold during pregnancy (19,21). After 3 months post partum, thete is no detectable level of relaxin (22). There appears to be a correlation between relaxin levels and isolated symphysis pain; however, a correlation has not been shown between relaxin levels and pain intensity, joint dysfunction, or disability in athletes with low back pain (22). There is

theoretical concern for hormonally induced joint and ligamentous laxity leading to increased ligament and joint injuries such as sprains and tears (e.g., of the anterior cruciate ligament). However, scientific proof is lacking.

Biomechanical Changes of the Pelvis

Pelvic pain has been reported to be approximately 20.1% at 33 weeks of pregnancy (23) and refers to pain of the symphysis pubis and the SI joint. Joint laxity of the pelvis is most profound in the symphysis pubis and the SI joints, which suggests hormonal effects as a cause of pelvic instability (21). The symphysis pubis widens throughout pregnancy, from a normal width of 0.5 mm to a maximum width of approximately 12 mm (13). Symphysitis is associated with SI pain and dysfunction (12). There is increased risk of vertical displacement of the pubis, which may lead to increased rotatory stress at the SI joints, causing sacral torsions and unilateral sacral dysfunctions.

These primary changes may lead to compensatory changes causing altered biomechanics of the lower extremities. Widening of the symphysis pubis contributes to the increase in Q-angle observed in pregnancy, which can increase valgus stress and load on the medial knee. This may also lead to increased valgus stress at the medial ankle, possibly causing overpronation, potentially leading to conditions such as Achilles tendinitis, posterior tibial tendinitis, medial arch pain of the foot, and plantar fasciitis. Athletes engaged in running sports are particularly affected. These changes may prohibit continued participation in even low-intensity or low-impact activities such as walking, Tai Chi, or yoga, and the pregnant athlete may have to try non–weight-bearing activities such as water training or stationary cycling.

Biomechanical Changes at the Sacroiliac Joint

As the SI ligaments relax due to hormonal influence, the SI joints become more mobile during pregnancy, which increases instability of the joints. SI joint instability allows for increased movement, which can stretch pain-sensitive structures, causing SI inflammation and pain (13). Radiographic studies confirm the presence of anatomic changes and inflammation at the SI joints (24).

Pregnancy-related pelvic pain (PRPP) is caused by dysfunction at the SI joints and frequently radiates into the buttocks and thighs. This is the most common site of pain in pregnancy. Sacral torsion and flexion and extension dysfunctions are common causes of SI joint pain (25). Berg found that two thirds of pregnant athletes with severe low back pain have dysfunction of the SI joint (12). Asymmetrical SI joint laxity in athletes with moderate to severe pelvic pain during pregnancy is a predictor of moderate to severe PRPP, conferring a threefold higher risk that pelvic pain will persist into the postpartum period (26). Pelvic girdle syndrome, which refers to dysfunction and pain in all three pelvic joints, that is, the symphysis pubis and both SI joints, occurs in 6% of pregnant athletes and has the worst prognosis and most severe symptoms (10,23,27).

Biomechanical Changes of the Lumbar Spine

In the lumbar spine, joint laxity is evident in the anterior and posterior longitudinal ligaments. The static supports become weakened and are less able to withstand shearing forces as the center of gravity shifts. Disc or facet pain may develop as a consequence of the altered spinal biomechanics. Lumbar spinal mechanics are influenced by the natural lordotic curve and the enlarging maternal-fetal mass. This load alters forces and stresses the lumbar vertebrae and supporting structures. Disc herniations are rare; LeBan et al. reviewed the records of 48,760 deliveries and reported only one incidence of herniated lumbar disc (28).

Stabilization of the lumbar spine is important in maintaining neutral posture and counteracting the shifts in the center of gravity. Ligaments and muscles provide stabilization for the spine, which works to maintain center of gravity and normal posture. The abdominal and iliopsoas

muscles contribute greatly to countering the shift in the center of gravity and providing stabilization of the low back. When the ability of the ligaments and muscles to compensate is overwhelmed, pain and dysfunction can develop.

Core Strength

Poor core strength may be a predictor of poor posture and low back dysfunction and pain during pregnancy. Core strength is a factor contributing to the stabilization of the spine and may, in part, account for those pregnant athletes who do not complain of pain (9,29). As pregnancy progresses, there is a reduction in the capacity of the abdominal muscles to counterbalance the increased weight and mass. Fast et al. reported that approximately 17% of pregnant women could not perform a single sit-up compared to 0% of matched nonpregnant women (30). CSEP and the Society of Obstetricians and Gynaecologists of Canada (SOGC) both recommend that women without contraindications be encouraged to participate in strength-conditioning exercises as part of a healthy lifestyle during their pregnancy (1).

The main stabilizing center is at L5-S1 with secondary centers at C7-T1 and T12-L1. These are balance points for the anterior-posterior curves of the spine and reference points for the longitudinal center of gravity (31). Chapter 13 discusses core stabilization in more detail.

FETAL RESPONSES TO MATERNAL EXERCISE

Most of the potential fetal risks in uncomplicated pregnancy are hypothetical (2). There are no reported increases in abdominal injuries among pregnant athletes. The possibility, however, for injury that may cause catastrophe such as abruptio placentae does exist and should be discussed during the exercise screening examination.

Basal metabolic rate and heat production increase during pregnancy. Intensity of exercise has the greatest impact on body temperature. In nonpregnant women, the core temperature increases by an average of 1.5°C during the first 30 minutes of moderate-intensity aerobic exercise in thermoneutral conditions (32). If exercise is continued for an additional 30 minutes, core temperature then reaches a plateau. The few studies on the fetal effects of maternal exercise and core temperature during human pregnancy are limited (33). In animal studies, an increase in maternal core temperature of more than 1.5°C during embryogenesis has been observed to cause major congenital malformations including increased neural tube defects (33). The first 45 to 60 days of gestation are particularly critical to neural tube development, and temperatures above 39°C have been shown to be teratogenic in animals and may also be teratogenic in humans (34). However, there have been no reports that hyperthermia associated with exercise is teratogenic in humans (35,36). There are no prospective studies to date that have found any association between increased maternal temperature induced by self-paced exercise and teratogenicity. It appears that sustained exercise does not increase maternal core body temperature to detrimental levels (37).

Physical exercise diverts blood flow to large muscles and has the potential to decrease uteroplacental perfusion and thus the transport of oxygen, carbon dioxide, and nutrients to the fetus. This raises concerns about the lasting effects, but the indirect evidence shows no lasting fetal effects. This concern mainly applies to out- of-condition women who begin a vigorous exercise program while pregnant. Such women should limit themselves to exercise that is no more strenuous than walking. Well-conditioned pregnant women generally are able to continue their exercise routines if their pregnancy is uncomplicated.

The fetus is well protected during exercise. Clapp and co-workers reported that fetal heart rate always increased in women who exercised regularly throughout pregnancy as long as the duration of the exercise was 10 minutes or longer and the intensity of the exercise exceeded 50% of maximum aerobic capacity (38). Minor decreases in P_{O_2} cause a fetal sympathetic response, which increases fetal

heart rate. There is also an increase in fetal baseline parasympathetic tone with advancing gestation, which causes a lower baseline fetal heart rate. Therefore, fetal sympathetic stimulation that occurs during maternal exercise causes an increase in fetal heart rate that is of greater magnitude due to the increased parasympathetic tone (39).

EXERCISE AND PREGNANCY

Louisa Burns, DO, supported exercise during pregnancy as far back as 1944, when she wrote, "Normal pregnancy and especially normal labor require strong muscles, both striated and non-striated. It is extremely important that all these muscles be strengthened during pregnancy. The striated muscles, which are completely voluntary, can be developed by well-planned exercises" (quoted in reference 7).

Active athletic women who are pregnant should be encouraged to continue their activities, and those who are not physically active should be encouraged (provided there are no contraindications) to begin an exercise program, particularly if they are planning a pregnancy. A moderate level of exercise on a regular basis has minimal risks for the fetus and beneficial metabolic, psychological, musculoskeletal, and cardiorespiratory effects for the mother during a low-risk pregnancy (15).

Women who exercise throughout pregnancy may require less medical intervention such as forceps or cesarean section (15,40), improved aerobic and muscular fitness, facilitation of labor (5), prevention of gestational glucose intolerance (5,41), and pregnancy-induced hypertension (5). Weekly physical exercise before pregnancy reduces risk for back pain in pregnancy. In fact, the American College of Obstetricians and Gynecologists (ACOG) (18,41) and CSEP (1) promote regular exercise during pregnancy for its overall health benefits (18). The Centers for Disease Control and Prevention and the American College of Sports Medicine recommend at least 30 minutes of moderate physical activity daily three times per week (42). Moderate intensity is defined as activity with an energy requirement of 3 to 5 metabolic equivalents (METS), equivalent to walking at a rate of 3 to 4 miles per hour.

Exercise prescriptions are useful for setting parameters and guidance for safe participation and providing encouragement for exercise as well as security. Maternal adaptations to pregnancy occur throughout the trimesters. These adaptations necessitate ongoing adjustments in physical activity and exercise prescription. Pregnant athletes should be counseled that modifications in type, frequency, intensity and duration of physical activity are expected in order to address the changing conditions of pregnancy.

Pregnant athletes need to adapt their training to maintain sport-specific fitness and conditioning. Cross-training can be beneficial in maintaining the athlete's aerobic and anaerobic capacity to avoid environments unsafe for a pregnant athlete. For example, cross-country runners should be discouraged from running on uneven terrain, which may be unpredictable and predispose to falls. Water training may be particularly beneficial. It provides for continued aerobic activity in a relatively weightless environment where there is less stress and strain on joints and muscles. Non–weight-bearing or low-impact endurance exercises using large muscle groups (e.g., walking, stationary cycling, swimming, aquatic exercises, low-impact aerobics) are generally safe and should be recommended and included as part of the exercise prescription (43).

Exercise Screening

Athletes should be screened for any contraindications to exercise including medical, obstetric, and neuromusculoskeletal that may become problematic as pregnancy progresses. Ideally, this occurs at a preconception visit where the athlete can be assessed for medical problems and for current levels of fitness and athletic participation. Preexisting conditions, including neuromusculoskeletal, may worsen during pregnancy and may lead to pain or dysfunction that may prohibit the pregnant athlete from engaging in athletic activity or even light activity.

Screening should also include inquiry into the use of supplements. Ten percent of all Americans use dietary or herbal supplements or neutraceuticals along with prescription medications (44), yet almost 50% of athletes studied did not report their use of herbal medicines, even when specifically asked on written forms (45). This has the potential for serious health consequences (45) including herb-drug interactions and teratogenicity.

TABLE 45.1. ABSOLUTE CONTRAINDICATIONS TO EXERCISE IN PREGNANCY

Hemodynamically significant heart disease
Restrictive lung disease
Incompetent cervix/cerclage
Multiple gestation at risk for premature labor
Persistent second- or third-trimester bleeding
Placenta previa after 26 weeks' gestation
Premature labor during current pregnancy
Ruptured membranes/amniotic fluid leakage
Pregnancy-induced hypertension
Evidence of intrauterine growth retardation

Contraindications to Exercise

There are some sports that are too threatening to maternal and/or fetal well-being, even in low-risk pregnancies. These include sports where contact or collision is a concern and those that require balance or otherwise place the athlete at increased risk of falls and abdominal injury. Abdominal trauma can cause direct placental and fetal injury, including abruptio placentae. Contact-collision and limited contact sports that are contraindicated include basketball, ice hockey, soccer, downhill skiing, and horseback riding (46). Environment must be controlled when formulating exercise prescription and advising athletes on safe activity. Hyperbaric, hyperthermic, humid, or hypoxic environmental conditions should be avoided (43). The athlete should limit activities to those occuring in a climate-controlled environment (indoors) and run on flat, even surfaces. High-altitude climbing (6,000 ft or higher) is contraindicated because of the risk of acute mountain sickness and the limitations pregnant women have in performing high-intensity physical activities at that elevation (47). The development of high-altitude pulmonary edema (HAPE) or high-altitude cerebral edema (HACE) could be disastrous. No adverse fetal response has been noted in activity below 2,500 meters. Scuba diving is contraindicated because of the risk of fetal decompression sickness (4).

Certain medical and obstetric conditions prohibit or limit physical activities. Ongoing assessment and evaluation for these conditions are important in adapting exercise prescriptions. Both ACOG and CSEP have issued absolute and relative contraindications to aerobic exercise during pregnancy based on medical and obstetric conditions. Pregnant athletes should be screened for these conditions and be aware of them should they develop during exercise (Tables 45.1 and 45.2). The CSEP recommendations in Tables 45.1 and 45.2 are also listed on the PARmed-X for Pregnancy (48) documents for convenience, which CSEP developed and which is endorsed by SOGC as the basis of safe and practical exercise prescription in pregnancy.

ACOG has issued warning signs for athletes to discontinue exercise during pregnancy and to seek immediate medical attention (Table 45.3).

Exercise Prescription

Athletes who have not been exercising prior to pregnancy are advised against implementing a program during the first trimester (43). It is not advisable to begin a *new* exercise program or

TABLE 45.2. RELATIVE CONTRAINDICATIONS TO EXERCISE IN PREGNANCY

Severe anemia
Unevaluated maternal cardiac arrhythmia
Chronic bronchitis
Poorly controlled type 1 diabetes
Extreme morbid obesity
Extreme underweight (body mass index <12)
History of extreme sedentary lifestyle
Intrauterine growth restriction in current pregnancy
Poorly controlled hypertension/preeclampsia
Orthopedic limitations
Poorly controlled seizure or thyroid disorder
Heavy smoker
Anemia or iron deficiency (Hb under 10 g/dL)

TABLE 45.3. WARNING SIGNS TO DISCONTINUE EXERCISE IN PREGNANCY

Vaginal bleeding
Dyspnea before exertion
Dizziness
Headache
Chest pain
Muscle weakness
Calf pain or swelling
Preterm labor
Decreased fetal movement
Amniotic fluid leakage

Adapted from Canadian Society for Exercise Physiology.

increase the amount of current exercise prior to the 14th week of pregnancy or after the 28th week. Those women who have been previously exercising are advised to continue but not increase their intensity or frequency during the first trimester (43). The best time to increase activity is during the second trimester because risks and discomforts of exercise are lowest at that time. The preconception visit is the best time to establish exercise prescriptions.

A screening assessment tool such as the Physical Activity Readiness Medical Examination for Pregnancy (PARmed-X for Pregnancy) is helpful in determining whether there are any underlying general medical problems or previous or current obstetric complications that may be contraindications for exercise. Such an assessment can help develop the exercise prescription by tailoring it to address medical conditions and somatic dysfunctions. Exercise prescriptions should take into account the status of maternal conditioning, maternal-fetal factors, and desired physical activity. They should include advice for type, frequency, duration, intensity, and rate of progression of activity as well as recommendations for aerobic activity, muscular conditioning, and stretching.

Pregnant athletes must avoid overexertion from high-intensity activity because of risk of increased maternal temperature. Studies show that there is poor correlation between heart rate and exertion during pregnancy. The ACOG previously had recommended maintaining a heart rate of less than 140 beats per minute even though there were insufficient data to support this hard and fast rule. In its latest *Committee Opinion on Exercise During Pregnancy and the Postpartum Period* (2) released in January 2002, there is no specified maximum heart rate at which exercise should be stopped.

Perceived exertion during exercise may be more useful than heart rate in preventing overexertion. Rate of perceived exertion (RPE) is increased during weight-bearing activities such as walking, jogging, or running (49,50) and can be used to gauge exercise intensity. CSEP suggests using both heart rate and the Borg rating of perceived exertion as an approach to prescribing and monitoring exercise. The Borg scale increases linearly with physiologic measures such as heart rate and Vo_{2max} as exercise intensity increases. Borg RPE scores should be maintained between 12 and 14 on the 6- to 20-point scale (5,51) to maintain safe levels of exercise intensity. The "talk test" is even simpler, as the inability of an athlete to talk during exercise indicates overexertion (5,52).

Aerobic exercise should be gradually and progressively increased during the second trimester from a minimum of 15 minutes per session to a maximum of approximately 30 minutes per session (1,60). CSEP recommends that aerobic activity be preceded by a brief (10- to 15-minute) warm-up and followed by a short (10- to 15-minute) cool-down. Low-intensity calisthenics, stretching, and relaxation exercises should be included in the warm-up/cool-down (5).

Although there are no studies directly implicating relaxin, progesterone, or estrogen in increased joint injuries, there is concern that the physical changes associated with these and other hormones may predispose joints to injury. Increased joint laxity is theoretically thought to increase the risk of sprains and joint injury during exercise and sporting activities. Data support hormonally linked ligament laxity changes in pregnancy (21); however, minimal data link these changes to increased injury rates. Activity involving bouncing and quick changes of direction, such as high-impact aerobic exercise, is

discouraged because it may cause joint or soft tissue injury (5). A sample exercise prescription is given in Appendix A.

MANUAL MEDICINE TECHNIQUES IN PREGNANCY

The goal of manipulation is to restore the whole body to a state of homeostasis (25). In pregnancy, baseline is consrantly changing and the body is constantly trying to adapt. Manual medicine is used in the pregnant athlete to optimize function with the intention of having the athlete safely continue athletic activity to the degree that maternal and fetal conditions allow. Manipulative prenatal care can correct faulty body mechanics, keep the pelvis balanced and unrestricted, and treat the athlete's discomfort (53).

Massage has been shown to decrease discomfort such as low back pain and leg cramps (54). Manual manipulative techniques have demonstrated improvement in acute low back pain (55), chronic low back pain (56), and pelvic pain (57). Mobilization techniques show promise in decreasing low back pain in pregnancy (58).

Manual medicine techniques are directed toward the normalization of two different types of dysfunction: those present in any individual (nonpregnant) and those caused by the changes in body balance due to pregnancy (39). These changes in balance are sufficient in themselves to decrease the mobility of the vertebral articulations, and when such postural deviations are superimposed upon chronic dysfunctions, regions of acute dysfunction may result (5). Osteopathic manual medicine is also useful in reducing stress and improving neuromusculoskeletal pain, joint motion, cardiovascular function (arterial, venous, and lymphatic), and respiration. Multimodal techniques can be directed to treat dysfunctions causing neuromusculoskeletal, cardiorespiratory, vascular, and gastrointestinal problems (59).

The goals of manual medicine include general relaxation of the muscles of the back, correction of any somatic dysfunctions, and improvement of passive motion in areas of postural strain. Zink noted that osteopathic manual medicine techniques for the obstetric athlete had been limited to soft tissue relaxation and occasional corrections of the isolated somatic dysfunctions, but today they are used comprehensively to optimize respiration, facilitate drainage of the venous and lymphatic systems, and address "gravitational strains" (60).

Manual techniques should always be accompanied by an exercise stabilization program, especially for the lumbosacral region. Manipulation can restore function and alignment, but keeping the body that way depends upon muscle strength, conditioning, and firing patterns. Strengthening and stretching exercises designed to increase abdominal and low back strength during pregnancy with the purpose of decreasing frequency and intensity of low back pain and improving ability, function, and mobility of the athlete with chronic low back pain (56).

The evaluation of respiration should be done first because the pulmonary system is greatly affected by biomechanical changes during pregnancy and treatments directed to optimizing respiratory effort would be beneficial for the athlete (6,60).

Manipulation in athletes with acute low back pain has been shown to improve pain (61,62). Women who reported back pain and received multimodal osteopathic manipulative treatment during pregnancy reported a decrease in pain (63) and a significant increase in their abilities to perform activities of daily living during the third trimester (63). Women athletes who received lumbar soft tissue manipulation reported a decrease in both pain intensity and use of pain medication during labor (64). Myofascial techniques such as massage have been shown to decrease pain during pregnancy and labor (65,91). Sacroiliac pain has been shown to be improved after manipulation (6,59).

Indications for and contraindications to manual medicine treatment can be evaluated at the preconception visit. Reassessment at each prenatal visit is needed to follow changes in pregnancy status that could affect exercise and fetal health. Indications for osteopathic manipulative treatment are listed in Table 45.4.

TABLE 45.4. INDICATIONS FOR AND CONTRAINDICATIONS TO OSTEOPATHIC MANIPULATIVE TREATMENT DURING PREGNANCY

Indications

Somatic dysfunction during pregnancy
Scoliosis or other structural condition associated with pregnancy
Edema, congestion, or other pregnancy-associated condition amenable to osteopathic manipulative treatment

Contraindications

Undiagnosed vaginal bleeding
Threatened or incomplete abortion
Ectopic pregnancy
Placenta previa
Placental abruption
Premature rupture of membranes (preterm)
Preterm labor (relative contraindication)
Prolapsed umbilical cord
Eclampsia and severe preeclampsia
Surgical or medical emergencies (other than those listed above)

Adapted from Tettambel M. Obstetrics. In: Ward RC, ed. *Foundations for osteopathic medicine*. Philadelphia: Lippincott Williams & Wilkins; 2002:450–461.

Techniques

In general, most manual techniques, as well as positions, are well tolerated by obstetric athletes (63). There are, however, issues of athlete comfort and maternal-fetal safety that must be considered. As maternal weight increases and as shifts in posture occur, positions and manual techniques that were tolerable early in pregnancy may not be possible in later pregnancy. Early in pregnancy, the athlete may tolerate most techniques and positions very well, including lying in prone or supine positions. Later, however, as the uterus moves into the abdomen, the athlete does not tolerate the prone position. Prone techniques should not be used once the uterus expands out of the pelvis, about the 12th week of pregnancy, because of potential for uteroplacental injury.

Supine techniques may also become more problematic as pregnancy progresses (66), and the athlete should not lie supine for an extended time. During the second and third trimesters, the risk for orthostatic hypotension increases due to increased uterine size and pressure on the inferior vena cava, which decrease venous return and causes a decrease in preload and cardiac output. This may lead to symptoms associated with presyncope and syncope including lightheadedness, dizziness, nausea, and diaphoresis. The clinician must monitor the athlete throughout the procedure to assess for any symptoms. The athlete who has been lying down is moved to seated and standing position slowly so as not to induce a syncopal episode due to orthostatic hypotension.

The clinician should avoid techniques that put pressure on the abdomen in the second and third trimesters, to avoid the possibility of uterine injury, including abruptio placentae (66,67).

SOMATIC DYSFUNCTION IN PREGNANCY

Exercise prescription may change as pregnancy progresses as a consequence of biomechanical changes and somatic dysfunction. Somatic dysfunction should be anticipated and evaluated at each prenatal visit. Somatic dysfunctions in pregnancy occur primarily in the pelvis, sacrum, and lumbar areas (9,33,84), with secondary compensatory dysfunctions in the thoracic and cervical spines, ribs, and extremities (68).

Cardiovascular, respiratory, gastrointestinal, and neuromusculoskeletal functions are all affected by pregnancy. Visceral changes may manifest as neuromusculoskeletal changes. Facilitated segments may exhibit somatic dysfunctions through viscerosomatic reflexes. Knowledge of viscerosomatic reflex patterns can assist in the recognition of somatic dysfunction patterns. Sympathetic innervation to pelvic structures is supplied through T10-T12 and L1-L2. S2-S4 supply innervation through the pelvic splanchnic nerve. Nociceptive fibers follow these tracts and share interneurons with musculoskeletal nociceptive fibers at the same levels. Somatic dysfunction may be manifest as changes in tissue texture at these corresponding sites. Referred back pain from pelvic structures

is well recognized clinically and discussed extensively in the literature (69,70).

There are some common conditions in pregnancy that may benefit from evaluation and treatment of associated somatic dysfunctions. By evaluating viscerosomatic reflexes, effects in the somatic structures following a visceral disorder can be resolved, and the viscus can be influenced through stimulation of somarovisceral effects (71).

First Trimester

Nausea/Vomiting. Nausea and vomiting occur in about 50% of women, most likely due to rising levels of human chorionic gonadotropin and a hyperactive autonomic nervous system (72,73). This can affect physical activity and limit participation in exercise. Athletes may present with parathoracic muscle spasm, Jones's tender points, Travell's trigger points, and other evidence of viscerosomaric reflex. Relieving pressure on the vagus nerve may possibly alleviate nausea.

Manual medicine treatments should try to decrease the sympathetic effect and normalize the parasympathetic influence (74). Techniques directed to decreasing sympathetic outflow may offer some relief (75). Acute gastritis may exhibit facilitated segments at T6-T9 (75). Rib raising and inhibition of both posterior muscle spasm and of midline collateral ganglia are the two main techniques used to target sympathetic supply (74). Dietary adjustments and supplements such as vitamin B may also be beneficial.

Cephalgia. Tension-type headache may develop as a consequence of postural changes or compensatory changes of the cervical spine and supporting musculature that cause increased stress and strain on upper thoracic and cervical suboccipital soft tissue. Spasm and multiple tender points of the paracervical and trapezius muscles are common. The upper cervical areas (C1-C3) are primarily involved (77). Sphenoid, temporal, occipitomasroid, and sacral dysfunctions may be present (77). Suboccipital soft tissue traction (33), counterstrain, cervical muscle energy, and high-velocity, low-amplitude (HVLA) techniques are generally well rolerated throughout pregnancy and may be considered. Stretching and massage techniques before and after exercise may be of benefit. Craniosacral techniques may also offer amelioration of discomfort (78). Abdominal pillows, such as the Bzzlo pillow, have been shown to alleviate low back pain and decrease the development of compensatory changes. Neck pillows that supporr the neck and head during sleep may help promote more biomechanically normal posture.

The American Academy of Neurology reports that cervical manipulation for headaches may be an option; however, evidenced-based treatment recommendations for cervical manipulation as preventive or acute therapy for migraine have not yet been formulated (59).

Second Trimester

The uterus expands into the abdomen in the second trimester. This begins the strain on bony and soft tissue supporting structures of the pelvis, sacrum, and lumbar areas. The physical structure and orientation of the pelvis begin to change and continue to do so throughout pregnancy. The pelvis rotates forward and compensatory increases in lumbar lordosis follow. Prepregnancy obesity and weight gain during pregnancy contribute to these adaptive structural changes. As pregnancy continues, muscular, fascial, and ligamentous counterbalances to these bony changes frequently are overwhelmed by increased abdominal size. Sacral, sacroiliac, and sacrococcygeal dysfunctions frequently occur. Common findings include sacral flexion, extension, and torsion. Evaluation of thoracic, rib, and cervical areas may reveal compensatory dysfunctions (53). The primary changes observed in the pelvis may also lead to somatic dysfunction of the extremities, discussed in more detail subsequently.

In the athlete who has had prior pelvic surgery, pain from stretching of adhesions about the uterus and adnexa may occur. Direct and indirect myofascial release, especially early

in pregnancy, before pain develops, may delay the development of such symptoms or offer some relief of discomfort that arises during pregnancy.

In the late first trimester or early second trimester, the iliohypogastric nerve may become entrapped, causing pain. The athlete may complain of a burning sensation deep in the pelvis. This likely occurs as a consequence of the expanded uterus into the abdomen and is generally relieved after delivery. Anterior counterstrain points at L3-L4 (79) may be present. Counterstrain techniques may be well tolerated in the early second trimester, but may not be possible later, as the abdomen becomes more protuberant and positioning is less tolerated.

Third Trimester

The third trimester is marked by profound postural changes and weight gain. Breast size increases dramatically, which may lead to exaggerated thoracic kyphosis. This change also contributes to redistribution of weight, changes in posture, and a shift of the center of gravity commonly causing thoracic and rib dysfunctions.

The burden of increased weight and changes in metabolic and respiratory physiology may cause the athlete to complain of decreased endurance with workouts and early muscle fatigue. Breathlessness is common. In addition to the direct pressure of the uterus and other displaced abdominal organs on the diaphragm, there are physiologic changes in the diaphragm and thoracic cage that contribute to decreased respiratory capacity and lead to earlier fatigability.

Uterine enlargement causes decreased venous and lymphatic return from the lower extremities, causing edema, increasing foot size, leg cramps, and hemorrhoids. Muscles become fatigued more easily. Shoes should be adapted to remain properly fitted. Simply loosening the laces is not the answer to poor-fitting shoes. Athletes should be advised to monitor foot size and comfort and wear only properly fitted shoes to ensure adequate arch and heel cup support. Runners, in particular, are susceptible to injury as a consequence of ill-fitting shoes including blisters, plantar fasciitis, and heel problems.

Reflux esophagitis is common in the third trimester. This may be due to direct pressure on the lower esophageal sphincter or it may be a consequence of overactive sympathetic outflow. Facilitated segments are present at T6-T9. Treatment is directed toward decreasing sympathetic outflow and normalizing parasympathetic activity (72). Attention should be given to somatic dysfunction potentially affecting the vagus nerve at the occipitoatlantal junction.

INJURIES IN PREGNANT ATHLETES

A comprehensive holistic approach to the injured pregnant athlete is important because many musculoskeletal injuries or athletic-based illnesses have a somatic component (85). Diagnosis is ideally obtained during the prepregnancy evaluation, so that risk assessment related to sports activities, medical history, and physical condition can be made and an exercise prescription formulated. Some injuries and conditions that are common in nonpregnant athletes may have special significance in the pregnant athlete. These merit more detailed, specific discussion.

During the initial prenatal examination, the pregnant athlete should be asked about running surface and environment, shoes, training schedule, and preexisting conditions. Runners are particularly susceptible to sacral, pelvic, and lower extremity pain and injury as pregnancy progresses. Preexisting hip, knee, ankle, or foot problems may be exacerbated during pregnancy. Runners who have overpronation, for example, are more susceptible to foot and ankle problems. History of Achilles' tendinitis, medial foot pain, or plantar fasciitis should warrant examination for overpronation. Running shoes with quality heel cups, heel counters, and adequate arch support are important for the maintenance of normal static and dynamic ankle and foot biomechanics and the prevention of ankle and foot problems. Shoes should be examined for uneven wear patterns, condition, and fit. As pregnancy progresses, foot size may increase necessitating new shoes to ensure proper fit.

Other conditions and injuries frequently present themselves in runners and may be observed in the pregnant athlete.

Patellofemoral Syndrome

Patellofemoral syndrome is the most common condition associated with running. It is characterized by anterior knee pain and frequently caused by abnormal patellar tracking. Athletes may complain of increased pain with climbing stairs. Patellofemoral syndrome, whether prepregnancy or pregnancy related, may be secondary to the increased Q-angle of pregnancy which causes increased load at the medial knee. Increased weight of pregnancy also contributes, as discussed previously.

Iliotibial Band Syndrome

Iliotibial band syndrome is the second most common knee problem of runners. The iliotibial band (ITB) can be a source of recurrent pre- and postpregnancy dysfunction. Genu varum and leg-length discrepancy contribute to the development of ITB syndrome. As pregnancy progresses and the pelvis widens, causing a physiologic increase in the Q-angle, there is more stress on the ITB.

Sprains

Sprains are among the most common injuries in sports. The ankle is particularly at risk in runners. Inversion injuries at the ankle are common and can be caused by running on uneven terrain. Ideally, pregnant athletes should limit their running to flat surfaces in controlled environments. A thorough examination is imperative to elicit information that will determine the extent of injury.

Wrist sprains from sports such as tennis, golf, and weightlifting may also occur. Treatment is directed at improving mobility and function of the joint. Limitation of or reduction in offending activity may be necessary. Manual techniques that may be considered include lymphatic techniques to relieve edema, as well as soft tissue massage and myofascial release to improve range of motion. Taping and bracing may improve symptoms and allow for return to activity. Strength training may also be beneficial in providing more musculoskeletal support for the joint. Rehabilitation exercises directed at improving proprioception may shorten the recovery period. This may have an added benefit for pregnant athletes for whom balance and proprioception are particularly important.

Overpronation

Overpronation of the ankle, whether anatomic or physiologic, may develop or be exacerbated during pregnancy. The widening pelvis, increased Q-angle, and increased weight cause tremendous stress at the ankle and foot. Physiologic laxity of the ligaments coupled with increased load can cause anatomic and functional instability. These altered mechanics may lead to Achilles tendinitis, posterior tibial tendinitis, plantar fasciitis, and other foot problems. Ankle pain early in pregnancy most likely will increase in the absence of intervention. Orthotics should be considered in any athlete with overpronation in order to normalize the biomechanics of the foot and ankle and decrease stress at the Achilles' tendon, subtalar joint, and plantar fascia. Proper footwear is critical. Counterstrain and HVLA techniques may be helpful. Stretching of the hamstring muscles may decrease stress at the Achilles' tendon. Deep tissue massage directed at stretching the plantar fascia is helpful.

Spondylolysis

Spondylolysis is caused by a stress reaction or stress fracture of the pars interarticularis, most common at L5. It is characterized by unilateral, localized paraspinal pain that is worse with extension or twisting activities. Athletes who play sports with violent, explosive extension at the lumbar spine, such as gymnastics and volleyball, are at risk to develop spondylolysis. Athletes may have spondylolysis before pregnancy, which may worsen during pregnancy. Bracing is not used in pregnancy. This finding warrants close clinical and radiographic monitoring before the athlete returns to full activity post partum.

Osteitis Pubis

Osteitis pubis is poorly understood inflammation of the symphysis pubis. The characteristic finding is pain at the pubic symphysis. The pain may occur during pregnancy or after pregnancy as a consequence of labor and vaginal delivery. It is seen in cyclists and runners and can be exacerbated during pregnancy. Generally, the inflammation is self-limiting and improves over time after pregnancy. Athletes with this condition who wish to rapidly return to activity post partum may be impeded in their return to exercise, which can be quite painful. Osteitis pubis may be particularly problematic for cyclists, whose positioning on the bike and side-to-side motion of the pelvis during peddling can exacerbate this condition. Treatment of osteitis pubis can be reviewed in Chapter 22.3.

Nerve Compression Syndromes

Thoracic Outlet Syndrome

Thoracic outlet syndrome (TOS) occurs when the divisions of the brachial plexus or subclavian artery are compressed by the clavicle, first rib (cervicobrachial junction), scalene, or pectoralis minor. Athletes engaged in overhead activities such as serving in tennis, weight training, or throwing may be susceptible to TOS. Posture also has been recognized as contributing to the development of TOS, especially anterior and internally rotated orientation of the shoulder. Pain is frequently referred to the lateral aspect of the hand and forearm; motor symptoms are rare. Treatment directed to the first rib and other associated thoracic or clavicular dysfunctions should be considered. Cervical, thoracic, costal scapular, and brachial mechanical relationships should be considered when developing a treatment approach and selection of modalities. Treatment can include myofascial release and stretching techniques (84).

Ulnar Nerve Compression

Ulnar nerve compression within Guyon's canal at the wrist is most common in bicyclists due to compression caused by pressure exerted against the handlebars. It is usually found in road bikers who assume positions that stress the wrist for long periods of time, but it can also be observed in athletes using stationary training cycles. Symptoms are localized to the ulnar one-and-a-half digits and depend on the nerve branch involved. The deep branch supplies motor innervation, and the superficial branch supplies sensory innervation. An adjustment in activity or positioning to take pressure off of the wrist is necessary. Soft tissue techniques to promote lymphatic movement and decrease edema, rest from the offending activity, and bracing to protecr the nerve from further insult help.

Carpal Tunnel Syndrome

Carpal tunnel syndrome (CTS) is the second most common musculoskeletal diagnosis in pregnancy (79,81). It is most commonly diagnosed in the second trimester, and may be secondary to a multiplicity of conditions common in pregnancy, including thoracic/rib dysfunction or fluid retention (48,80). Pregnant women are at higher risk of developing CTS than their nonpregnant peers. Overuse activities are a well-known risk factor for CTS; however, there are no studies correlating the incidence with pregnancy and athletic activity. Racquet sports may or may not confer an increased overall risk of developing CTS, but participation in racquet sports certainly is affected. Increasing the space within the carpal tunnel by using direct release techniques may ameliorate symptoms. Direct and indirect techniques including myofascial release, directed at the thoracic outlet, may be considered (79, 82–84). Correcting upper thoracic and upper rib dysfunctions by addressing facilitated segments and removing cervical somatic dysfunction to improve brachial plexus function may also alleviate symptoms (80).

Tarsal Tunnel Syndrome

Tarsal tunnel syndrome is caused by the entrapment neuropathy of the posterior tibial nerve as

it passes through the fibro-osseous tunnel created by the flexor retinaculum. Overpronation is often a factor. Symptoms usually occur in the forefoot and may include shooting, burning, and radiating pain from the medial plantar aspect of the foot to the toes. A positive Tinel's sign may be present. The increased weight of pregnancy and peripheral edema may exacerbate symptoms, and relief may not be achieved until after delivery. Stretching the plantar fascia can help release the tunnel restriction, while lymphatic techniques can decrease compressive edema. If unresolved after delivery and conservative treatment, surgery may be considered for attempt at definitive treatment.

Peroneal Nerve Compression

Peroneal nerve compression occurs as a consequence of compression at the fibular head, where the nerve is superficial and wraps around this prominence. Improper positioning or protection of the athlete's legs in stirrups may cause this condition. Symptoms include paresthesias of the lateral foot and footdrop. Treatment priority is alleviating the source of compression, which could take weeks or months post partum to resolve. Orthotics including ankle-foot orthoses, physical therapy, and bracing are recommended. Athletes are advised to refrain from crossing their legs while sitting to prevent further injury. Proprioceptive techniques are important in maintaining or reestablishing normal neurologic function. A posterior or anterior fibular head may contribute to symptoms and is frequently treated using HVLA techniques (86). Muscle energy, myofascial, and HVLA techniques are effective in treating talar and subtalar dysfunctions that can affect fibular and tibial motion.

Meralgia Paresthetica

Meralgia paresthetica is caused by the entrapment of the lateral femoral cutaneous nerve as it exits the pelvis near the anterior superior iliac spine. Symptoms include pain and dysesthesia in the anterior thigh. It can be caused by pressure from the protuberant abdomen or the overenthusiastic application of retractors during a cesarean section. Removing the source of nerve compression is the primary basis of treatment. If it is caused by abdominal pressure, the condition may not resolve until after delivery. The use of a maternity belt to alleviate pressure on the nerve may be considered. Traction neurapraxia after surgery almost always leads to resolution, but may take several months. The lateral femoral cutaneous nerve is sensory and does not affect motor activity.

OTHER PREGNANCY ISSUES

Labor and Delivery

There are studies showing that exercise during pregnancy leads to premature labor in athletes not already at increased risk for preterm labor (40,87). Pain associated with labor has been studied extensively. Contraction of the uterus and dilation of the cervix activate pain receptors (88). Anesthetic techniques and agents alleviate pain while causing no adverse effects on maternal or fetal health and without protracting labor.

Fifteen percent of pregnant athletes complain of extremely severe low back pain during labor (89). Melzack and Schaffelberg studied the correlation between labor pain and low back pain during menses and the correlation between labor pain and episodic low back pain before and during pregnancy (89). They found a significant correlation between pain during menses and labor pain, suggesting they have a similar physiologic etiology. They found no correlation between episodic low back pain prior to or during pregnancy and labor pain, which is consistent with similar studies (11,52) There is, however, an association between low back pain during pregnancy and labor (69).

Manipulation has been reported to lead to shorter, easier labor (90,91). Manual pressure over L1-L5 has been shown to alleviate pain in laboring athletes and is associated with the use of less analgesia (64,92). Manipulation has been recommended as an adjunct to those approaches used to alleviate lumbar pain during labor, but there have been no reported prospective randomized studies (93).

Postpartum Period

Osteopathic physicians have long recommended the use of manipulation post partum to alleviate pain and correct dysfunctions caused or exacerbated by labor and delivery. This promotes recovery and encourages the athlete to return to prepregnancy levels of activity quickly.

Almost 10% of athletes experience postpartum back pain (10). The dorsolithotomy position and vaginal delivery can lead to somatic dysfunctions including sacral extension, iliac flares, sacroiliitis, and pubic symphysitis. During delivery, the hip, pelvic, and abdominal musculature undergoes maximum physical strain that stresses the back and the pelvic and sacroiliac ligaments. This presents challenges for postpartum rehabilitation, as the athlete works to regain prepregnancy activity levels. Evaluation and management of somatic dysfunctions may begin while the athlete is still hospitalized. Gentle, passive techniques such as soft tissue technique and Still's technique can be applied to the athlete lying in the hospital bed. Other techniques, such as muscle energy, are generally well tolerated, but must be used with care immediately post partum due to increased ligamentous laxity.

The athlete's exercise prescription should be modified during the postpartum period. Exercise prescription should be made on an individual basis with consideration given to athlete motivation, prepregnancy fitness levels, and medical and obstetric conditions. There are no specific guidelines regarding the resumption of exercise in the postpartum period. ACOG recommends a gradual return to activity but acknowledges that there are no studies to indicate that rapid resumption of activities will result in adverse effects (2,3,94).

In addition, the psychosocial benefits of return to activity are significant for the athlete. Emotional wellness is promoted through postpartum physical activity. Koltyn and Schultes reported a decrease in postpartum depression in athletes who engaged in a stress-relieving aerobic exercise program (95).

Maternal dieting and aerobic exercise have not been found to adversely affect lactation performance or neonatal weight gain (96). Athletes may experience upper back discomfort with increased breast size and may have associated thoracic and rib dysfunction. Additionally, incorrect maternal positioning during breastfeeding may lead to upper extremity discomfort including forearm, wrist, and hand pain. Lactation consultants may offer detailed instruction on proper positioning techniques for breastfeeding to alleviate stress on the mother's back, neck, and upper extremities.

Lactating athletes should be advised to wear support bras, especially during exercise. Nipple irritation due to friction with the bra cup may cause skin tears and pain. This may be ameliorated by the use of a well-fitted support bra and the use of lubricant on the nipple to decrease friction.

Nutrition

The importance of proper nutrition in health promotion and disease prevention cannot be overstated. Pregnant athletes should be informed of the basics of nutrition during pregnancy. At the preconception visit, the athlete should be given a prescription for prenatal vitamins including 4 mg of folic acid daily, which should be continued through the first trimester. Inquiry should be made as to the use of supplements and ergogenic aids. The athlete should be advised that she requires an additional 300 kcal per day during pregnancy and an additional 500 kcal per day during breastfeeding.

Pharmacology

Athletes experiencing pain during pregnancy have limited pharmacologic options. Analgesia is most often addressed with acetaminophen. In more severe pain, codeine or morphine may be considered. They are both classified as category C drugs in relation to their teratogenic potential. The use of nonsteroidal anti-inflammatory drugs and aspirin in pregnancy is generally not indicated. Consultation and collaboration with the obstetrician is important in maintaining a safe pregnancy without drug side effects.

Modalities

Modalities frequently used by physicians and physical therapists are safe if they are applied to the extremities and axial skeleton with avoidance to the abdominal region. Therapeutic ultrasound is not recommended over or near the fetus because cavitation in the amniotic fluid could harm the fetus (97). A transcutaneous electrical stimulation unit applied over the abdomen is not recommended by the Food and Drug Administration due to lack of data on safety (97). Ultrasound, phonophoresis, electrical stimulation, and iontophoresis are considered to be safe if not applied over the pregnant uterus. They are not indicated for back or abdominal conditions because of concerns such as induction of uterine contractions and potential for teratogenicity. An improvement back discomfort may be facilitated by the use of elastic or Velcro lumbar support early in pregnancy (43).

SUMMARY

Holistic care of the pregnant athlete involves integrating the principles of wellness into all aspects of obstetric care, from prenatal screening to the post partum. Athletes benefit greatly from maintaining fitness in pregnancy and incorporating exercise into their prenatal care plans. Clinicians must consider the effects of neuromusculoskeletal influences on pregnancy and sports participation. The challenge is incorporating their knowledge of sports and exercise and their knowledge of manual medicine, including manipulation, into a comprehensive obstetric care plan that promotes optimal maternal and fetal health. This is a uniquely holistic approach that provides the pregnant athlet with the most comprehensive care.

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Appendix A
SAMPLE EXERCISE PRESCRIPTION

Prescription for Aerobic Activity

Frequency: Monday, Wednesday, Saturday

Intensity: Stay within a score of 12 to 14 on the Borg rate of perceived exertion scale; also gauge exercise intensity using the talk test.

Time: Try to exercise for 15 minutes. Rest or decrease intensity, if you need to complete 15 minutes.

Type: Swimming with 10- to 15-minute warm-up of stretching before swimming and 10- to 15-minute cool-down after swimming.

Prescription for Anaerobic Activity

Frequency:	Daily
Upper back:	Shoulder shrugs, 3 sets of 10
	Shoulder blade pinch, 3 sets of 10
Lower back:	Modified standing on opposite leg and arm lifts, 3 sets of 10
Abdomen:	Leg lifts (4 to 6) off of floor, 3 sets of 10
Pelvic floor:	Wave elevator, 3 sets of 10
Upper body:	Shoulder rotations, 3 sets of 10
	Push-ups against wall, 3 sets of 10
Buttocks:	Buttocks squeeze, 3 sets of 10
	Heel raises, 3 sets of 10

Prescription for Stretching

Frequency: Daily

- Iliopsoas stretch
- Thoracolumbar stretch
- Sacral stretch

Adapted from the PARmed-X for Pregnancy.

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