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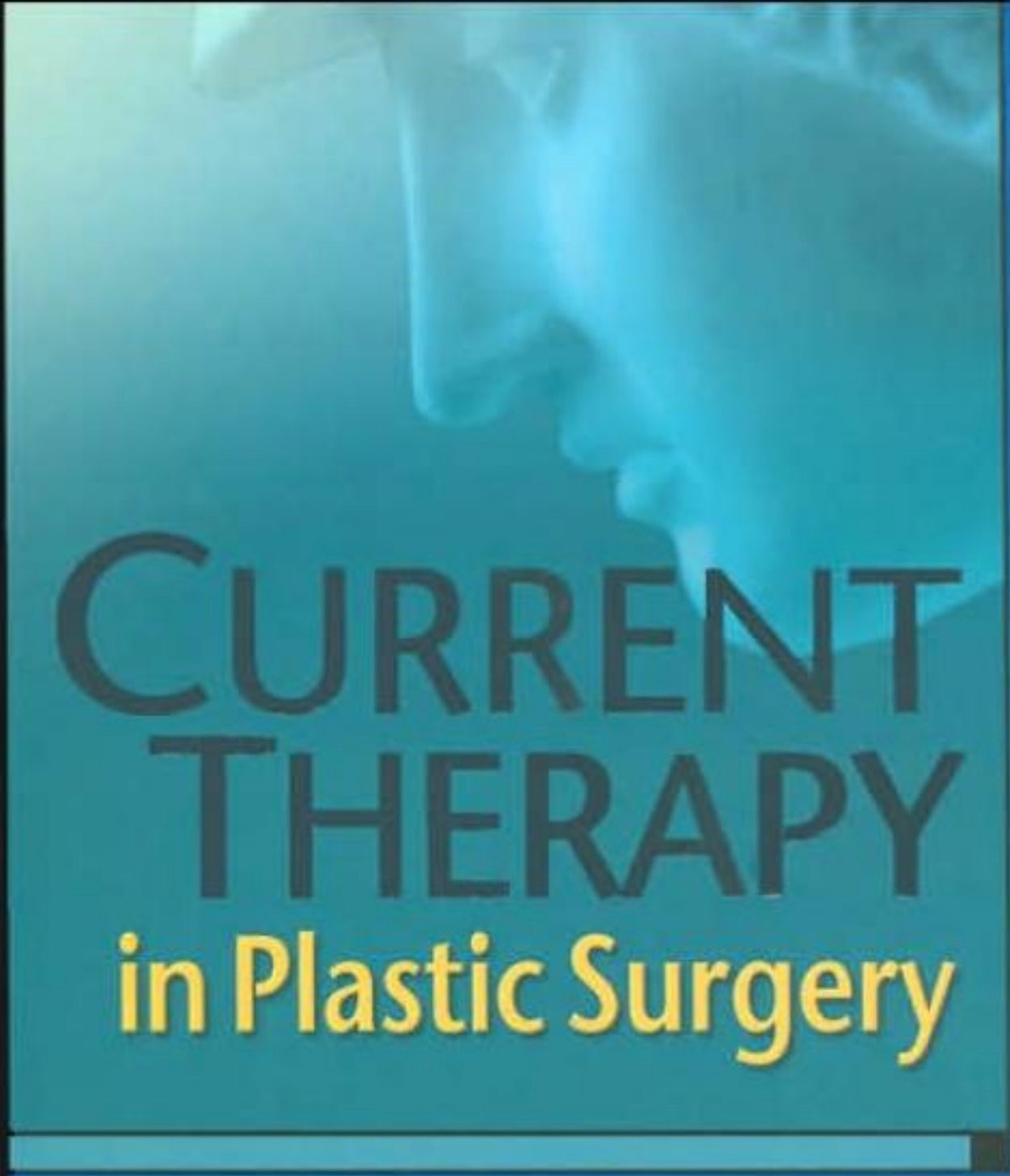

Current Therapy in Plastic Surgery

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This book is dedicated to three generations of McCarthys:
Karlan – my soulmate and partner for over 40 years
Cara and Brit, Christina and Stephen
Remi, Lilia, Aidan and Tim

JGM

This book is dedicated to:
My wife Susan, whose love and kindness have graced each and every one of our days together. You are the best thing that has ever happened to me.
My daughter Inès, who is my pride and source of unending joy.
My parents Jaime and Hilda, for their support, sacrifices, and example.

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This book is dedicated to:
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Preface

The genesis of a book can be a fascinating story. Where does the idea of a book begin? This text had its origins as the idea of two plastic surgery residents who approached their Chief one day with a proposal. They pointed out to a somewhat skeptical Chief the need for a plastic surgery component to the *Current Therapy* series. They cited that, as general surgical residents, they had found its counterpart invaluable in their daily activities both on the wards and in the operating room. The residents brought to the project the enthusiasm and creativity of youth. Moreover, they provided the invaluable perspective of the coming generation of plastic surgeons. They recognized the up-and-coming leaders and innovators in the various subspecialties of plastic surgery. In the organization of the book, they emphasized the need to introduce new topics and to place less emphasis on other areas or techniques that are less practiced today. Without them this text would not exist – the Chief acknowledges his gratitude and admiration for them.

The other heroes of this book are the contributing authors. The editors well recognize the time and effort that were put into completing their chapters. We, as readers, do not always appreciate the nocturnal and weekend writing and the time spent in obtaining the requisite illustrative material. They have the gratitude of the editors and the readers alike.

The editors strove to condense the subject of plastic surgery into a single volume text. Hemingway said that a book should be comfortable if resting on one's chest while reading in bed. The editors strove to reach the Hemingway benchmark even though it entailed eliminating text and illustrations from submitted manuscripts. The goal of the editors was to develop a concise overview of plastic surgery that was relevant not only to the student but also to the practitioner of plastic surgery. In keeping with the theme of the *Current Therapy* series, much of the history, pathophysiology, and surgical approaches that have been consigned to historical irrelevance are not included. We strove to make each chapter targeted, concise, and hard-hitting, but without sacrificing content for succinctness. We believe that this text will be particularly useful for the busy practitioner, as well as for fellows and residents with significant time commitments. We looked at this book as the primary reference book; it should be the first book that is opened to research a topic and it should prepare the reader to benefit from other more in-depth sources when needed.

Acknowledgments

This project would not have succeeded without our editorial counterparts at Elsevier. We would like to extend our thanks to Sue Hodgson, Elyse O'Grady, Peter McElhenney, Catherine Carroll, Donna Ciccotelli, Marie Miller, Jodi Kaye, and Tina Rebane for their tremendous assistance. We would also like to thank Elizabeth Danes for her help here in New York. A special note of gratitude is due to Marilyn Ostrow, our long-suffering but ever-capable administrative assistant here at the NYU Medical Center, for graciously accepting yet another task in addition to her many others. We are grateful to all of you.

Plastic Surgery Strategies

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The term plastic surgery is derived from the Greek *plastikos*, meaning to shape or mold. Even though the term remains accurate in the sense that so much of plastic surgery is concerned with form, the true essence of all plastic surgical endeavors is the blending of form and function. Much of the history of plastic surgery parallels humanity's effort to "close holes" or repair deformity. Although the first surgical interventions were ritualistic and included amputation, circumcision, and trephination, more refined "plastic" interventions appeared as early as 650 BC when Sushruta described the reconstruction of nasal defects with a forehead flap. In Roman times, advancement flaps were described by Celsus, but the next milestone, more than a millennium later, was the 1597 publication of Tagliacozzi's classic text on reconstruction of the nose with an upper extremity tube flap. However, it was not until 1797 that the Indian technique of nasal reconstruction was first published in the Western literature in the *Gentlemen's Magazine* in London.

The 19th century represented a true renaissance for all of surgery. The scientific method, first introduced in German universities, was fortunately applied to the nascent discipline of surgery. There were classic publications of nasal reconstruction by von Graefe (1818) and Dieffenbach (1845). Probably the first comprehensive text of plastic surgery was the *Handbook of Plastic Surgery* by Zeiss in 1838. The use of anesthesia and the introduction of the concept of antisepsis in the second half of the 19th century encouraged the advancement of all surgical techniques. In the following decades, skin grafting and cleft lip/palate repair became more common, with reasonable success rates achieved around the world.

Trench warfare in World War I resulted in an inordinate number of maxillofacial injuries. The concept of a specialized facial surgeon was developed. Many of the military surgeons, such as Gilles, Kazanjian, Blair, and Ivy drawn from the disciplines of general surgery, dentistry, and otolaryngology, went on to become leaders of 20th century plastic surgery. The interwar years, ironically, did not see the growth of plastic surgery in the civilian population because the concept of elective or nonemergency surgery would not be developed until well after World War II.

Plastic surgery units were again organized during World War II, the most famous being the U.S. Army Hospital (1800 beds) at Valley Forge, Pennsylvania. Many of the young surgeons recruited to practice at this facility became the leaders of American plastic surgery in the second half of the 20th century: Brown, Cannon, Murray, Littler, and Edgerton. American plastic surgery in the 1970s and 1980s reaped the fruits of U.S. government investments in medical research (National Institutes of Health) and education in the postwar era. Anatomic and physiologic studies of the circulation of the skin resulted in a revolution in flap technology. Flaps with greater surface area, including myocutaneous and microvascular free flaps, were developed and provided methods of reconstructing almost any defect in the human body. With refinement in the aesthetic aspects of flap design, breast reconstruction after ablation became routine. Advances in microvascular technique resulted in widespread replantation of amputated parts. The development of craniofacial surgery, again resulting from anatomic studies, provided the plastic surgeon with techniques for reconstructing previously inaccessible anatomic sites: the orbits and cranial vault.

Unlike the period between World War I and World War II, the 1970s and 1980s witnessed a widespread demand for elective surgery. Aesthetic surgery of the aging face was especially the beneficiary of this change in societal attitude.

Modern plastic surgery is concerned with deformities that range from the "top of the head to the soles of the feet." In addition to the head and neck

region, plastic surgeons are involved in breast surgery and upper extremity repairs. In fact, upper extremity surgery has evolved as a surgical specialty in itself. Recontouring of the trunk and limbs is yet another branch of aesthetic plastic surgery. Plastic surgeons have also revolutionized the salvage of lower extremities after trauma.

Uniqueness of Plastic Surgery

Plastic surgery is unique in that it has no anatomic territory to which it can lay a particular claim. Unlike the cardiac surgeon, who alone operates on the heart but is also restricted to that anatomic site, plastic surgeons can truly practice their surgical skills over all areas of the human body and address problems in form, function, and even the physiology of the cranial vault, the chest cavity, and the upper and lower extremity.

Although plastic surgery is usually regarded as dealing with problems in human form, it should also be recognized that functional problems are often treated. A newborn child with sleep apnea may undergo mandibular distraction to avoid a tracheostomy; the victim of an automobile accident may require revascularization of a limb to prevent ischemia and amputation; and a patient with a brachial plexus injury may require nerve reconstruction to restore motor and sensory function.

Plastic surgery is also unique in that preoperative planning is not algorithmic. There is usually no set or prescribed way to correct a defect. A nasal deformity is a good example; treatment choices range from skin grafts to local flaps to distant flaps with or without cartilage grafts. The same problem, if presented to 20 different plastic surgeons, can elicit 20 different operative plans and result in 20 postoperative results of varying quality.

Hence, the preferred definition of plastic surgery is that of a problem-solving discipline. Its *raison d'être* is based neither on anatomic territory nor on a particular concept. Over the decades it has attracted innovative surgeons who have devised unique techniques to solve surgical problems. From transplantation to craniofacial surgery and from microsurgery to aesthetic surgery, plastic surgeons have demonstrated this uniqueness and have not only advanced the field but also created new ones. This progress continues today as new techniques are being developed in the areas of inductive surgery and tissue engineering.

Principles of Plastic Surgery

1 The doctor-patient relationship, based on integrity, remains fundamental to all plastic surgery activities. It is critical that the surgeon understand what concerns the patient and what the patient wants. Such understanding is realized only with a consultation that is not rushed and that provides an open forum for discussion by the patient and surgeon alike. The goal of the patient must be similar to that of the surgeon, and both should see "eye to eye." A second or third visit with review of photographs usually results in a mutually arrived at consensus. No procedure should be scheduled unless such an understanding has been achieved.

2 Elective surgery means that the patient enthusiastically and rationally "elects" to have a scheduled operation. If the enthusiasm resides only with the surgeon, there will be postoperative disappointment by not only the patient but also the surgeon. According to Sir Harold Gillies, the patient should be "on his knees begging for the operation." In other words, the patient should have a positive and optimistic attitude in the preoperative period, albeit tempered with some degree of anxiety or apprehension.

3 The surgeon must carefully weigh the risks associated with the procedure and the benefits. In many surgical procedures there is a "tradeoff." For example, a woman with large pendulous breasts is usually gratified after reduction mammaplasty because of increased comfort, improved breast form, and a wider choice of clothing, but she must be forewarned and be prepared to accept the postoperative scars—the "costs" of breast reduction. Usually, the "tradeoff" is readily accepted. This stands in contrast to an adolescent patient who undergoes cosmetic rhinoplasty in which the desired change in nasal form is achieved without any observable scars (or "costs"). A woman who wishes to have a benign skin lesion over the sternum removed and ends up with a hypertrophic scar that is visible with certain types of clothing will not feel that the tradeoff was in her favor.

The surgeon must ensure that the treatment is not worse than the problem or deformity and must also consider the importance of returning the patient to work and society as quickly as possible.

4 In the initial consultation the surgeon must define the deformity. This obviously sounds simple, but it can be an agonizing part of preoperative planning. For example, in craniofacial asymmetry, the surgeon must decide whether one side is too large or the contralateral side is too small. It is helpful to obtain a photograph and ask which side is preferred by the patient. Another useful aid is to use the unaffected side as a template to serve as the goal of reconstruction. The surgeon must also recognize the difference between the "true" versus the "apparent" defect. A postthermal scar in the antecubital fossa, once surgically excised and released,

results in a skin ("true") defect much larger than the original ("apparent") scar.

5 The concept of a group of clinicians from a variety of disciplines working on a single problem (i.e., a multidisciplinary team) yields the optimal care for complex problems. Examples are craniofacial, cleft lip/palate, vascular malformation, and nerve centers, which provide the optimum care of the patient and education of team members.

6 In preoperative planning the surgeon should consider a "reconstructive ladder" but on occasion must resort to a "reconstructive elevator." The principle of a reconstructive ladder is that simple procedures should be chosen before more complex ones. For example, if a full-thickness skin graft will satisfactorily resurface a small facial defect, it is preferable to a more complex flap transfer. However, on occasion, a reconstructive ladder, that is, proceeding rung by rung, is not always the preferred reconstructive route. Nonvascularized bone grafts applied to some reconstructive sites are associated with resorption or bone loss. In these situations it would be preferable to bypass the bone graft and use a vascularized bone transfer. An example would be reconstruction of a large full-thickness mandibular defect.

7 The surgeon must avoid overaggressive surgery. The phrase "less is more" particularly applies to plastic surgery planning, especially aesthetic surgery. A postoperative facelift patient with elevated brows and swept-back cheek skin (the "wind tunnel" look) has a deformity that is far worse than the preoperative signs of aging. A patient with a postrhinoplasty defect characterized by a saddle deformity and asymmetric nostrils is a victim of excessive resection of bone and cartilage and attracts attention from onlookers that is potentially disabling.

8 Replace like with like. For example, a full-thickness skin graft harvested from behind the ear and transferred to a defect in the tip of a sun-damaged nose will have an entirely different color and texture match. A local tissue flap, despite being a somewhat more complicated procedure, provides a more aesthetic result. A full-thickness skin graft applied to a defect in the male cheek would be unsatisfactory because it would not replace the hair follicles of the beard; a cervicofacial flap of bearded neck skin would be more desirable because like is replaced with like.

9 In facial reconstruction one must determine whether the deficit is soft tissue, skeletal, or both, and the reconstructive procedure should be designed to replace the missing part or parts. In craniofacial microsomia, in which there is a deficit of skeletal mandible and overlying cheek soft tissue, the reconstructive needs include skeletal replacement and the addition of soft tissue, such as a microvascular free flap.

10 Consider the aesthetic units in facial reconstruction. In general, reconstruct the entire aesthetic unit or subunit. The resulting scar should be designed to fall along the periphery of the aesthetic unit. The nose has aesthetic subunits, and any flap reconstruction should fall along these lines. Consideration should be given to convert a partial to a complete subunit defect by excision of residual skin.

11 Autogenous reconstruction is, in general, preferable to alloplastic reconstruction. It is only a matter of time before an alloplastic reconstruction becomes problematic; the passage of time results in thinning of the overlying soft tissue and erosion of the underlying skeleton. Autogenous reconstruction, although technically more demanding and more at risk for resorption, is less prone to complications.

12 When resurfacing complex defects, the reconstructive flap must fill the defect three-dimensionally. A good example is a pressure sore. The flap should not tent over the defect and leave a "dead space" in which fluid can accumulate. Persistent wound drainage eventually necessitates excision of a serosal-like lining and reapplication of a flap to seal the space.

13 The timing of surgical intervention can be problematic and should always be carefully considered. Variables include the age of the patient and the patient's psychological status.

In a younger patient one must take into consideration the higher incidence of hypertrophic scarring that follows an elective incision. Moreover, facial growth continues until late adolescence. For example, before a midface advancement is performed on a 6-year-old, the parents must be forewarned that mandibular growth will outstrip the midface and a second midface advancement may be required because of a resulting malocclusion. Conversely, surgery on a growing structure such as the nose can impair subsequent growth of that structure.

It is better to defer a surgical reconstruction if the patient is not pyschologically prepared for that procedure. A delay often allows the patient to fully appreciate the degree of difficulty and the limitations of the planned procedure and to give time for psychological counseling. For example, a patient with a nasal defect after a traumatic injury who lives with the deformity will have more appreciation for the results of the reconstruction. Parents who have lived for several months with an infant with a cleft lip are more likely to appreciate the operative result and accept the limitations of surgical repair.

Body dysmorphic disorder (BDD) is an obsessive preoccupation with a perceived imperfection

in some aspect of one's physical appearance. It has obsessive-compulsive features that are expressed as overconcern about a variety of physical features: a head that is too large, a nasal tip that is too pointed, a face that is asymmetric, and so forth. The individual often documents the perceived flaw with photographs on which drawings and sketches are made. There is usually a history of multiple plastic surgery consultations and, regrettably, surgical procedures that were ill conceived and "unsuccessful." As described, these patients are not candidates for elective plastic surgery.

The Best Friends of the Plastic Surgeon

Skin Lines of Minimal Tension

Simply stated, incisions planned in the lines of minimal tension result in the most satisfactory scars (Fig. 1). The plastic surgeon should always plan to make an incision in such lines. For example, a horizontal incision in the skin lines of the lower anterior aspect of the neck results in an almost imperceptible scar. Conversely, an incision placed at right angles to the lines of minimal tension (e.g., along the vertical midline of the anterior aspect of the neck or across the flexor surface of the joint) results in a foreshortened (often hypertrophic) scar or contracture.

A strategic variant would be to camouflage an incision (and the resulting scar) behind an anatomic structure. Examples include incisions in the retroauricular sulcus, the lower eyelid in a subciliary position, the inframammary fold, and the groin crease.

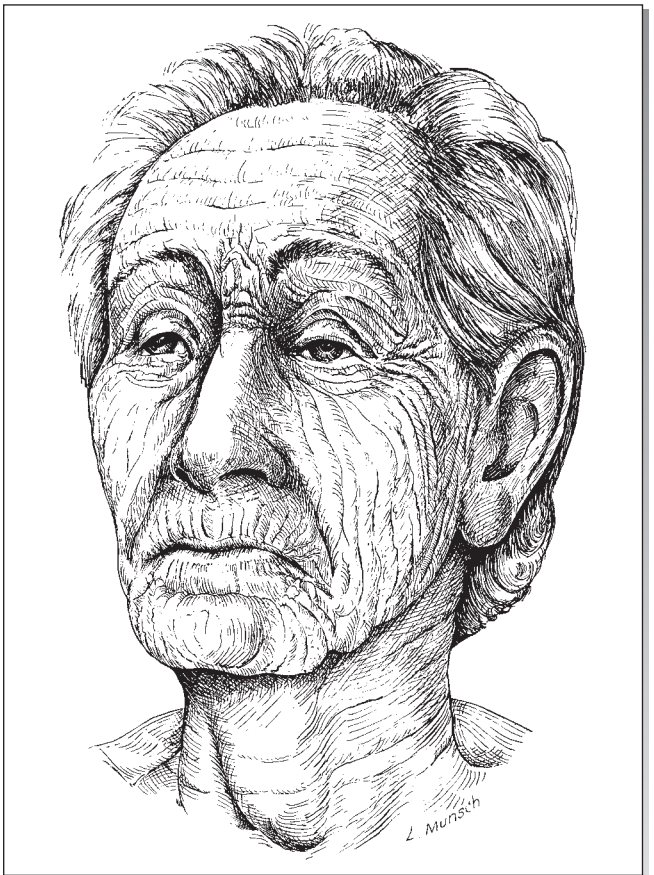


Figure 1 • Lines of minimal tension on the face and neck. (From McCarthy JG: Plastic Surgery. Philadelphia, WB Saunders, 1990.)

Suture Technique

Meticulous suture technique ensures the best possible scar. Proper technique must incorporate three principles: distribution of tension to the deeper layers, gentle or atraumatic handling of tissues, and eversion of skin edges.

Layered closure is a key element of both tension distribution and eversion of skin edges. Either absorbable or nonabsorbable sutures are used in the deep facial layers, approximating these layers and simultaneously coapting the skin. They should avoid encircling excess fat and instead approximate the strong fibrous tissues. Sutures in the subcutaneous fat serve only to devascularize the fat and add no strength to the closure. Additionally, sutures can be placed in the deep dermis to approximate the skin and allow for a tension-free skin closure.

Tissue handling is a critical, yet overlooked, principle of plastic surgery. Improper handling of tissue causes significant tissue injury, with resultant scarring. It is best to think of forceps as crushing tools that kill cells each time they are applied. Their use should be limited to direct manipulation of tissues, with hooks or retractors serving the function of tissue retraction. They should be toothed to limit the areas of injury with application.

Skin sutures should ensure both proper skin levels and eversion of skin edges. This can be done with passage of the suture in the proper orientation and by avoiding a skimming passage that will invert the edges (Fig. 2).

Z-plasty

A Z-plasty (Fig. 3) is one of the most commonly used plastic surgical techniques. It is used to lengthen a scar, as in correction of a contracture, or to break up a straight line scar and incorporate it partially in a line of minimal tension.

The geometric principle is simple: the scar becomes the central limb of the planned Z-plasty, and after transposition of the triangular flaps, the central limb is increased in length.

In the classic Z-plasty, the central limb lies along the scar or the line of contracture. Two limbs are designed to complete the Z, usually at an angle of 60 degrees, although the angles can vary. Wider angles theoretically allow more lengthening but

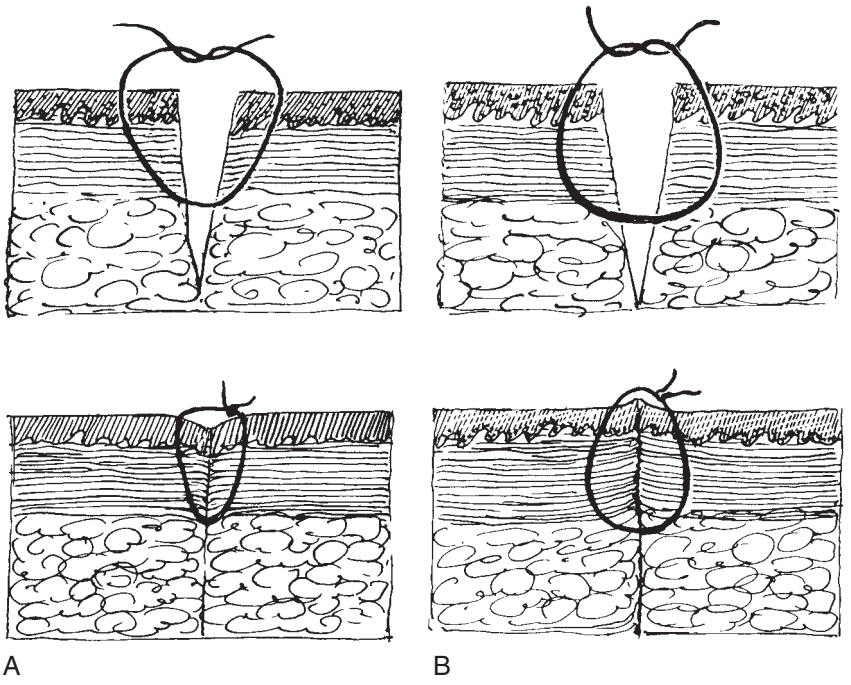


Figure 2 • A, Inverting suture: placing the suture to produce inversion. Inversion of wound edges is produced when the suture loop is wider superficially than it is deep in the tissues. **B,** The everting interrupted suture: correct manner of placing a suture to produce eversion. The needle should be placed to form a wider loop deep in the tissue to obtain eversion. The outline of the suture path is similar to that of an Erlenmeyer flask. (From McCarthy JG: Plastic Surgery. Philadelphia, WB Saunders, 1990.)

make flap transposition increasingly more difficult. In the vast majority of Z-plasties, 60-degree angles are preferred. The limbs should ideally lie along the lines of minimal tension. In the face one must exercise caution so that the length of the central limb (and the adjoining limbs) is never excessively large because there will be an unsightly large scar; accordingly, it is preferable to perform multiple small Z-plasties in the revision of facial scars.

Variations of the Z-plasty technique can be used, such as a four-flap Z-plasty, which has been recommended for correction of interdigital contractures in

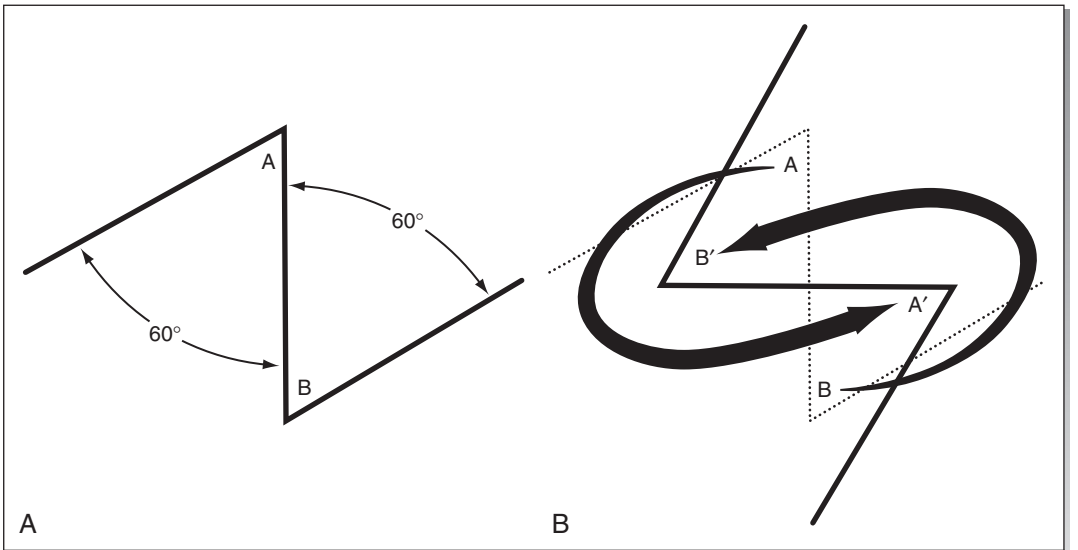


Figure 3 • Z-plasty technique. **A,** Central limb designed along the scar. Both limbs are equal in length, and angles A and B are 60 degrees. **B,** The flaps are transposed and sutured. Note that the original central limb has been lengthened and the scar is reoriented.

the hand. A two-flap Z-plasty is converted into a four-flap Z-plasty with angles varying from 45 to 60 degrees. Its advocates claim that it allows a greater increase in length of the central limb. Another variation is the double opposing Z-plasty, which is useful when elevation of large flaps is not possible, such as at the medial canthus. It allows two larger flaps to be dispersed into four smaller ones.

It should be noted that a Z-plasty is effective in reducing the hypertrophic nature of a scar. A dilemma, however, is posed in Z-plasty design in patients with extensive scarring in the area of the planned Z-plasty as scarring decreases the vascularity and mobility of the flaps. Judgment and experience must be used to design widely based flaps to allow adequate blood flow while simultaneously keeping flaps sufficiently narrow so that they are easily transposed.

W-plasty

A W-plasty (Fig. 4) is similar to a Z-plasty in that it reorients the direction of a scar. However, it has the disadvantage that it does not lengthen a constricted scar to the extent that a Z-plasty does. Another disadvantage of a W-plasty is that it requires considerable excision of scar and healthy skin and can thus lead to a relatively tight closure and the potential for a widened scar. It is especially indicated in a face when a Z-plasty, especially a large one, can result in a noticeable scar. It probably finds its greatest use in the correction of a trapdoor scar with chronic swelling. The resulting interdigitated scar appears to decrease the latter. A template is generally used to outline a W-plasty so that the apices on one side of the excised scar are opposite the base of the triangle on the opposite side. In general, W-plasties are not used nearly as frequently as Z-plasties, the latter being much more utilitarian.

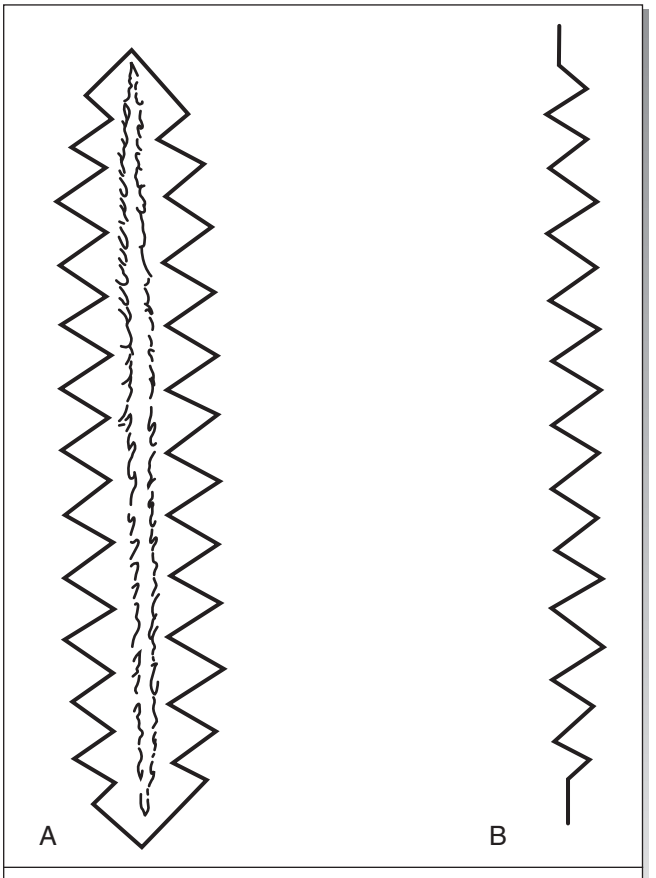


Figure 4 • W-plasty. A and B, W-plasty for repair of a straight scar. Triangles become smaller at the end of the scar. A W-plasty, however, results in excision of nonscarred skin.

V-Y Advancement

V-Y advancement is essentially a V incision that in closure is converted to a scar in the shape of a Y (Fig. 5). The resulting advancement can be used to provide extra soft tissue in the area of a tight scar. V-Y advancement finds its application in the correction of a whistle deformity of the upper lip or in lengthening of palmar skin in the correction of Dupuytren's contracture.

Split-Thickness Skin Graft

A split-thickness skin graft is used extensively to resurface large defects, especially on the trunk and extremities. It suffers the disadvantage in facial resurfacing of poor color, thickness, and texture match. Nonetheless, it is a technique that is especially suited for resurfacing a large burn deformity or for providing temporary coverage of a large, open wound before definitive coverage (see the chapter "Cutaneous Defects: Flaps, Grafts, and Expansion").

Full-Thickness Skin Graft

A full-thickness skin graft, the skin graft initially used in the 19th century, is associated with superior color and texture match. Its vascularization, or "take," is not as predictable as that of a split-thickness skin graft, and a full-thickness skin graft is limited in size because the donor site must be closed primarily.

Flaps

A simple classification of flaps (see the chapter "Cutaneous Defects: Flaps, Grafts, and Expansion") is as follows:

LOCAL FLAPS

Advancement Flaps. A V-Y flap is a classic example of an advancement flap. There is no single point of limitation of movement, and the entire tissue slides to fill a defect. These flaps lack adequate mobility and often require excision of Burow's triangles.

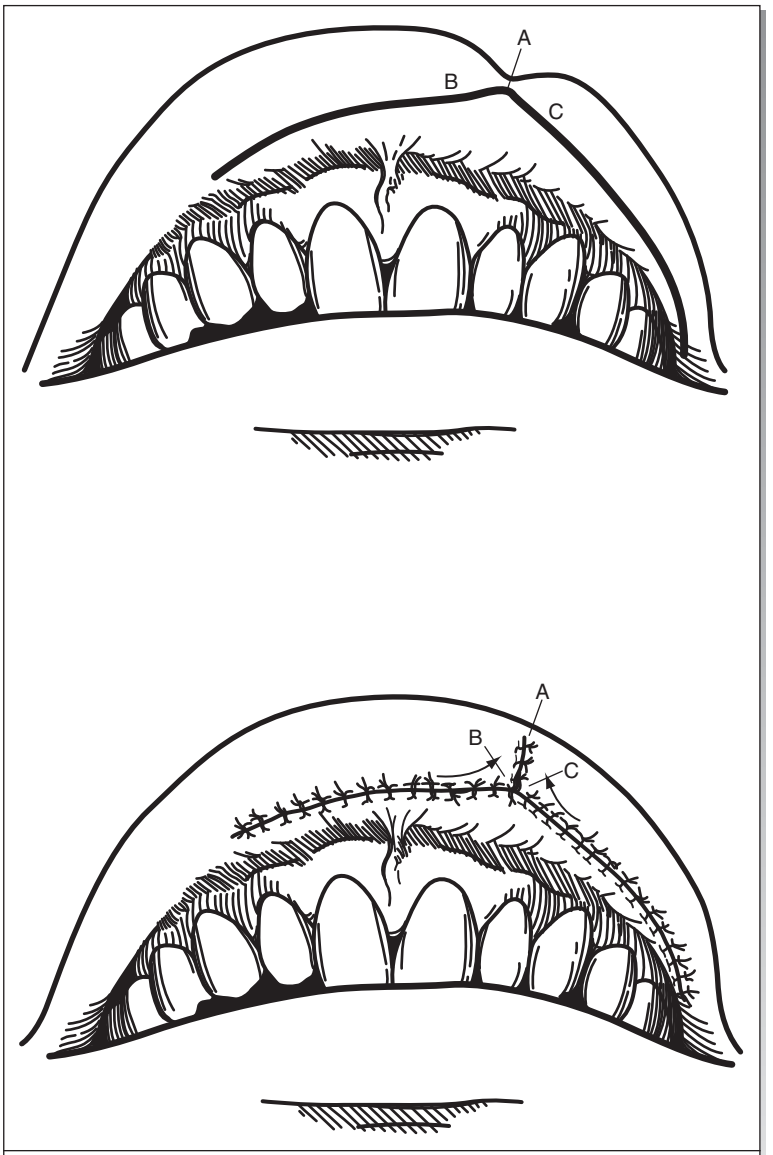


Figure 5 • V-Y advancement for correction of notching of the upper lip. Design of the V-shaped incision on the inner aspect of the upper lip. With advancement directed toward correcting the defect, the incision is closed as a "Y".

Rotation or Transposition Flaps. These flaps are transferred to an adjacent defect, the rhomboid flap being a classic example. Movement of the flap is limited by the arc of rotation. A bilobed flap is an example of a double rotation flap.

Interpolation Flaps. Interpolation flaps have interposed soft tissue between the donor area and the defect. These flaps typically have an axial blood supply and therefore a greater degree of movement.

DISTANT FLAPS

Distant Pedicled Flaps. These flaps are harvested from a distant anatomic region. They require a delay before they can be divided, which can vary from 10 days to 3 weeks, depending on the problem and the blood supply at the defect site.

Microvascular Free Flaps. Microsurgical techniques of free tissue transfer have allowed previously impossible reconstruction to become routine.

Avoidance of an Unfavorable Scar

There are several types of unfavorable scars:

1 Scars that are chronically red or erythematous, widened, and either hyperpigmented or hypopigmented can improve in appearance

with the passage of time; the efficacy of laser therapy or steroid injection is variable.

2. Hypertrophic scars (see the chapter "Hypertrophic Scars and Keloids") are defined as thickened scars that maintain the boundaries of the original incision or laceration. They occur most commonly in younger patients, in patients with darker skin, and in specific anatomic sites such as the sternum and deltoid region.
3. Keloids (see the chapter "Hypertrophic Scars and Keloids") are thickened scars that exceed the confines of the original wound (laceration or incision). They contain excessive amounts of collagen and, unlike hypertrophic scars, rarely improve in appearance with the passage of time. They are much more common in blacks than in whites and also have a strong familial predisposition. Keloids occur most commonly on the anterior aspect of the chest, earlobes, and face.

Variables

ANATOMIC SITE. The deltoid area, sternum, posterior aspect of the trunk, and limbs (except for the palms and soles) are at risk for the development of either a hypertrophic scar or keloid. It is remarkable, however, that eyelids are rarely the site of pathologic scars.

TYPE OF SKIN. Patients with oily or sebaceous skin are at risk for scars that are widened, depressed, and irregular in outline, especially in the nasal tip area. Scars in patients with pale, taut skin tend to remain red for prolonged periods. Sun-damaged skin in an elderly, often unhealthy, patient ironically lends itself to fine line scars.

AGE. The most satisfactory scars occur in elderly patients with limited elasticity of the skin. For example, excision of skin tumors from an older patient with heavily sun-damaged skin usually results in fine line scars. In contrast, scars in infants and children are generally erythematous and hypertrophic for prolonged periods. Caution should be exercised when making elective incisions on the face of patients younger than 15 years. The fetus is especially unique in that incisions in the earlier part of gestation can result in scarless healing.

Scars also mature with the passage of time. It is important to forewarn the patient, usually when sutures are removed, that the scar will look its worst somewhere between 4 and 8 weeks postoperatively. The patient must expect that scar maturation will require up to 2 years before the surgeon is willing to say that the final appearance of the scar has been achieved. In general, scar revision should not be undertaken within 1 year of the surgical procedure or traumatic event.

RACE. Dark-skinned individuals are more at risk for the formation of hypertrophic scars and keloids, especially in the earlobes, face, and sternal regions.

PATHOLOGIC DISORDERS. Among the various syndromes that should be approached with caution by surgeons planning an elective procedure are the following:

1. Ehlers-Danlos syndrome
2. Cutis laxa
3. Pseudoxanthoma elasticum

SUGGESTED READINGS

Gillies HD: Plastic Surgery of the Face. London, Oxford University Press, 1920.

Kazanjian VH, Converse JM: The Surgical Treatment of Facial Injuries. Baltimore, Williams & Williams, 1949.

McCarthy JG: Introduction to Plastic Surgery. Philadelphia, WB Sanders, 1990.

Millard RD: Principlization of Plastic Surgery. Boston, Little, Brown, 1986.

Cutaneous Defects: Flaps, Grafts, and Expansion

JASON A. SPECTOR
JAMIE P. LEVINE

The plastic surgeon is often consulted to assist in the closure of a complex cutaneous wound that is caused by either trauma or ablative surgery, or is congenital. Although the goal of reestablishing continuity of the integument and underlying soft tissue is straightforward, the manner in which this is accomplished may be complex if reliable closure is to be attained without compromising the aesthetic outcome. Variables to be considered include the size and location of the defect, the availability of adjacent tissue, and the presence of exposed vital structures. Closure of the wound takes priority over aesthetic considerations. As plastic surgeons, many tools are at our disposal to balance these often conflicting goals.

As advances in plastic surgery continue to be made, the reconstructive options likewise increase. The traditional view of reconstructive surgery as a ladder with rungs of increasingly complex and challenging options has matured, and the ladder concept is now considered less as dogma and more as a road map with several routes to the desired destination. In select instances, use of the reconstructive "elevator" is mandated; for example, microvascular free flap reconstruction is often the procedure of choice in patients with loss of integument over vital structures.

Assessment of the Defect

Each defect is individualized according to the missing components (e.g., lining, support, and cutaneous coverage), as well as the underlying cause (cancer, trauma, infection), anatomic location, aesthetic visibility, associated functional disabilities, and availability of local and distant donor sites. Compatibility of the donor tissues with the area being reconstructed should be considered with regard to skin color, texture, thickness, and hair density. The overall health of the patient is also evaluated. Only after careful consideration of these factors can one choose the appropriate reconstruction for a given defect.

Skin Grafts

Choice of Graft and Donor Sites

Skin grafts require neovascularization to remain viable in their new location. Skin grafts are classified as either split-thickness skin grafts (STSGs), which contain only a portion of the dermis and range from very "thin" (approximately $^{6}/_{1000}$ inch) to thick ($>^{20}/_{1000}$ inch), and full-thickness skin grafts (FTSGs), which contain all skin components, including the epidermal appendages. Because the appendages resume growth in the recipient area, the surgeon must consider the cosmetic implications of transferring hair follicles to the recipient area when using a FTSG. A FTSG contains the entire dermis, so *primary* (immediate) *contraction* (what is observed in the operating room) is greater than the contraction seen with a STSG. *Secondary contraction*, however, which occurs as the graft heals, is minimal. Thus, a FTSG is ideal for locations where significant graft contraction would cause major functional impairment, as in the digital web spaces or eyelids.

All STSG donor sites, although they contain epithelial elements for reepithelialization, heal with deposition of scar and have noticeable pigmentary changes. In general, the thighs and buttocks are ideal donor sites for a STSG because they are camouflaged by clothing and provide an ample surface area that allows for multiple harvests. An

alternative donor area such as the scalp can also be harvested with minimal cosmetic sequelae.

Meshing of the graft increases the graft surface area and improves graft "take" (vascularization) by allowing egress of fluid beneath the graft. However, meshed grafts are aesthetically inferior to nonmeshed or "sheet" grafts and require longer healing times because the interstices must be reepithelialized.

Technique of Harvest

STSGs are best harvested with an air-powered or electric dermatome. Infiltration of the donor site before harvest with a dilute solution of a long-acting anesthetic such as bupivacaine (Marcaine) with epinephrine (0.25% Marcaine, 1 mL/kg, in 50 to 100 mL Plasma-Lyte) facilitates graft harvest by increasing skin turgor (tumescence) and provides increased postoperative pain relief and decreased blood loss. The donor site is covered with an occlusive dressing to promote more rapid reepithelialization and decrease patient discomfort. Leaving the donor site dry to form an eschar is an obsolete approach that should no longer be practiced because it results in greater patient discomfort and protracted healing.

FTSGs have the advantage of leaving only a linear scar at the donor site, which is typically placed in an inconspicuous location such as the groin crease, supraclavicular area, or the skin anterior or posterior to the ear. The latter two sites are ideal for FTSG transfer to the face because they share similar texture and color with facial skin. When using groin skin as a donor site for a FTSG, the surgeon should be aware that these grafts tend to darken and may contain unwanted hair follicles.

As mentioned previously, FTSGs are ideal for areas where graft contracture would be detrimental to function and appearance; once healed, they are more durable than STSGs. Thus, a FTSG may be used to cover poorly padded areas where a STSG would be prone to breakdown and where a flap may be infeasible or undesirable. Aside from their somewhat limited donor areas, another important consideration in using a FTSG is the less consistent "take," or survival, of FTSGs. Although graft survival can be maximized by meticulous surgical technique, an otherwise completely viable FTSG may display epidermolysis, which can be treated expectantly with dressing changes.

Factors Influencing Graft Survival

After an initial avascular period of *plasmatic imbibition*, during which the graft survives solely on nutrients derived by diffusion from the local transudate, capillary buds from both the graft and the wound bed form new vascular connections, a process known as *inosculation*. Ultimately, successful graft take depends on several factors, including secure fixation, which permits inosculation of the delicate neovasculature. Unmeshed STSGs and FTSGs may be minimally "pie crusted" with a No. 11 blade to allow egress of any fluid collection beneath the graft that might retard graft take.

Meticulous insetting, with care taken to tailor the graft to fit the defect exactly, optimizes the aesthetic result. The graft is typically covered with a bolster dressing to prevent desiccation and protect the graft from shearing forces that might disrupt neovascularization. All shearing forces must be minimized for the first 5 days. In extremities, a wrapped splint that immobilizes the grafted portion of the extremity obviates the needs for a tie-over bolster. Alternatively, a subatmospheric pressure dressing such as the vacuum-assisted closure (VAC) dressing may be used to bolster STSGs. This modality may be especially useful for large and topographically irregular wounds that would otherwise be difficult to bolster.

Another factor required for successful skin graft vascularization is a healthy recipient bed. All devitalized tissue is thoroughly débrided before skin graft application. Even a wound that is apparently free of necrotic tissue may harbor sufficient bacteria to preclude successful skin grafting. A graft placed into a contaminated wound bed invariably fails. Although not usually a consideration in acute wounds, chronic wounds are colonized with bacteria even after diligent dressing changes. The bacterial load in chronic, otherwise clean wounds may be decreased by 24 hours of dressing changes with application of half-strength Dakin's solution. Débridement of excess granulation tissue should also be performed just prior to graft placement to optimize the wound bed.

The wound bed must also be able to support vascular ingrowth into the graft. Bone devoid of periosteum, tendon stripped of its paratenon, and cartilage will not support a skin graft. Chronic venous ulcers of the lower extremity will not support a graft unless the underlying edema is improved. Irradiated wound beds are notoriously poor skin graft recipient sites because of impaired neovascularization. Such wound beds may sometimes be optimized by treatment with a subatmospheric pressure dressing, which often promotes the formation of granulation tissue. Because the dressings are changed much less frequently than traditional moist to dry dressings, they should be placed only onto clean wounds or changed more frequently when used on more contaminated wounds. The only clear contraindication to the use of a subatmospheric dressing is placement on large blood vessels, which should be covered with vascularized tissue. In addition, wounds must be débrided of nonviable tissue.

Xenografts and Other Skin Substitutes

In situations such as a burn involving a large body surface area, sufficient donor skin may not be avail-

able to provide the necessary coverage. This deficiency in donor skin can be addressed by using xenograft skin or cadaveric allografts. These grafts are useful because they provide temporary wound coverage. However, because of their inherent immunogenicity, xenografts and allografts are universally rejected. The interval between placement of the graft and rejection may be as long as several weeks in immunocompromised patients, in whom the grafts are typically used.

The past decade has witnessed significant progress toward reaching the goal of engineering biologic skin substitutes. Nearly a dozen skin substitute products are currently available for clinical use, with many more under development. The products range from those composed entirely of cultured keratinocytes (Epicel, Genzyme Tissue Repair Corporation, Cambridge, MA), to acellular deepithelialized cadaver dermis (AlloDerm, LifeCell, Woodlands, TX), to combinations of dermal matrices with and without cultured cells covered by a silicone neoepidermis (Integra, Integra Life Science Corporation, Plainsboro, NJ; Transcyte, Advanced Tissue Sciences, La Jolla, CA; Biobrane, Dow Hickam/Bertek Pharmaceuticals, Sugar Land, TX). Perhaps the most elegant of the engineered biologic skin substitutes is Apligraf (Organogenesis, Canton, MA, and Novartis Pharmaceuticals Corporation, East Hanover, NJ), which is composed of an epidermal layer of neonatal keratinocytes overlying a dermal layer of collagen seeded with neonatal fibroblasts. Biologic skin substitutes hold immense promise. Their lack of immunogenicity allows tissue incorporation. Many contain a dermal layer, which once revascularized by the host, may also confer the advantages of a FTSG (increased durability, better aesthetic appearance). Finally, the products are available "off the shelf" without the need for donor harvest and further detriment to an already debilitated patient. However, the expense associated with their manufacture limits their current utility.

Flaps

A *flap* is a unit of tissue that may be transferred en bloc from a donor to another recipient site while maintaining an intrinsic blood supply. For the purposes of this chapter, emphasis is placed on local flaps developed from adjacent tissue, although the anatomic basis of microvascular free flaps is briefly described. Numerous types of flaps and classification schemes exist. Flaps may be classified by their component parts (e.g., cutaneous, myocutaneous, osseocutaneous), by their spatial relationship to the defect (local, regional, or distant), by the nature of the blood supply (random versus axial), and finally by the movement placed on the flap (e.g., advancement or pivot) to fill the defect. The first two classification schemes are self-explanatory and will not be addressed further. This chapter focuses on the latter two flap classification systems.

Blood Supply: Random Flaps

All flaps require an intact blood supply at the time of transfer to ensure viability and successful healing. Random cutaneous flaps, as the name suggests, are based on random, nondominant contributions from the dermal plexus and subdermal plexus (Fig. 1). The combined contribution of experimental studies and clinical experience has resulted in the observation that the ratio of flap length to width is critical for complete flap survival. Although random flaps in the face can be designed with a length-to-width ratio as high as 5:1, most random flaps are not as robustly vascularized and are therefore limited to lesser length-to-width ratios, thus restricting their ability to cover large defects. When used appropriately and when an appreciation of their limitations is acknowledged, random flaps are reliable first choices for coverage of smaller defects throughout the body.

To extend the somewhat restricted size of random flaps, surgeons rely on the *delay phenomenon*, most commonly achieved by interrupting a portion of the normal blood supply to the flap without transferring the flap from its native position. The stress of elevation and sublethal ischemia with the release of angiogenic and vasodilatory factors results in the opening of "choke" vessels which, although normally closed, dilate to allow blood flow into the ischemic flap from a neighboring territory. Furthermore, the ischemic areas of the flap probably experience a sprouting of new vessels through angiogenesis and perhaps via vasculogenesis.

Vessels within the flap also respond to the stress of delay by increasing in caliber. The resulting

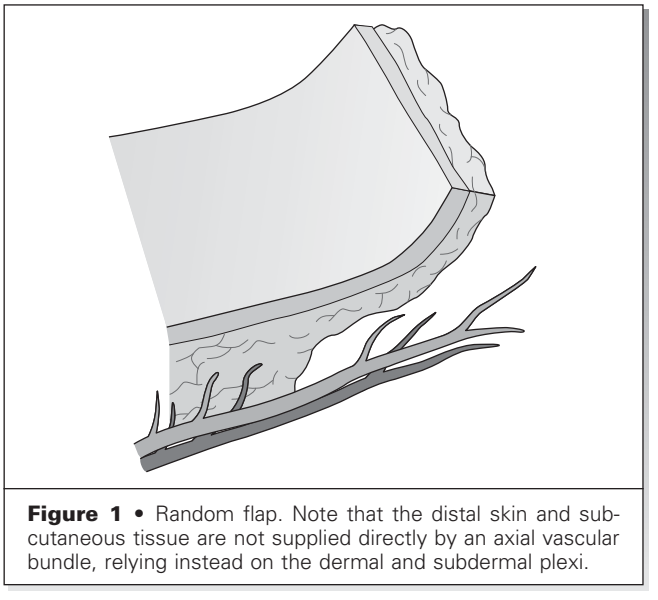


Figure 1 • Random flap. Note that the distal skin and subcutaneous tissue are not supplied directly by an axial vascular bundle, relying instead on the dermal and subdermal plexi.

increase in blood flow is seen as early as 2 days after the initial delay, with a maximal increase observed by 4 days. However, most surgeons find it prudent to delay a flap for at least 10 days to 3 weeks before final transfer, thereby permitting maturation of the process of neovascularization.

Incorporating a planned delay can significantly improve the chance of survival of a large random cutaneous flap. This is especially important in patients with impaired microcirculation, such as smokers and diabetics. Furthermore, a delay should always be considered a safety net if a flap demonstrates signs of ischemia or venous congestion after elevation. Reconstruction in such cases is best performed in a staged manner.

Blood Supply: Axial Flaps

In contrast to random-pattern flaps, axial-pattern flaps are based on a reliable, anatomically discrete arterial contribution that extends beyond the base of the flap (Fig. 2). Since description of the first axial flap (the deltopectoral flap) 4 decades ago, knowledge of the body's various cutaneous territories (angiosomes) and subsequent exploitation of axially based flaps have grown exponentially. Advances in anatomic science have increased the reliability of axial flaps and eventually fostered the development of microsurgical free flap transfer. Because of their significantly greater reliability, axial flaps are preferred for coverage of moderate to large defects.

The nature and distribution of the vessels that supply axial flaps vary with the anatomy of the flap. Arterial cutaneous flaps (e.g., a groin flap) are based on vessels that course parallel to the skin far beyond the base of the flap; they give off numerous cutaneous branches that supply the overlying skin. Fasciocutaneous flaps rely on inclusion of the deep fascia and its rich (suprafascial and subfascial) vascular plexus for maintenance of vascularity. Fasciocutaneous flaps can be characterized according to the pattern of blood flow to the deep fascial plexus. The radial forearm flap, for example, is a fasciocutaneous flap that receives its blood supply from the radial artery through numerous (septocutaneous) perforating vessels that reach the overlying tissue via a fascial septum connecting the flap to the radial artery. In other fasciocutaneous flaps, such as the parascapular flap, the deep fascia may be supplied by a vessel coursing longitudinal to the long axis of the flap that subsequently arborizes to provide cutaneous perforators.

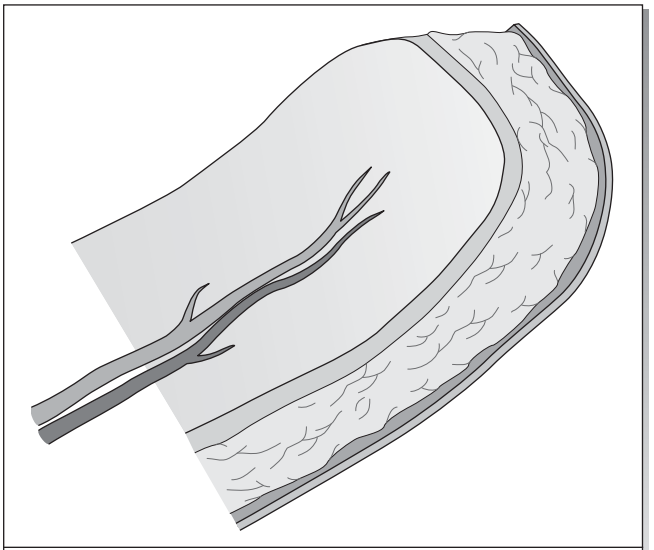


Figure 2 • Axial flap. Note that the distal aspect of the flap is supplied directly by an axial vascular bundle.

The vascular pattern of muscle flaps lends itself well to a formal classification system. The most universally accepted system was developed by Mathes and Nahai. In this system, muscle flaps are grouped according to whether they are supplied by a single dominant artery (type I), a dominant artery and minor pedicles (type II), two dominant pedicles from different vascular sources (type III), multiple segmental pedicles (type IV), or a single dominant pedicle and a secondary segmental pedicle (type V). In this system, a flap may be successfully raised on either its dominant pedicle, all of its segmental pedicles, or a few of its minor pedicles. Obviously, type IV flaps offer the least flexibility in design.

Myocutaneous flaps are axial flaps that consist of a composite of muscle and overlying subcutaneous tissue and skin. In most cases, the muscle at the base of the flaps is supplied by a single dominant vessel that gives off one or more perforating vessels to supply the overlying subcutaneous tissue and skin. Examples of myocutaneous flaps include the transverse rectus abdominis muscle (TRAM) flap and the latissimus dorsi flap. The perforating vessels of the flaps can be meticulously dissected from the surrounding muscle, with the result that the muscle-sparing flap supplied by a single perforator becomes a direct arterial cutaneous flap. The so-called *perforator flaps*, although technically more challenging to execute, may decrease some of the morbidity associated with the harvest of muscles in a formal myocutaneous flap.

Classification of Flaps Based on Movement

Flaps can also be characterized by describing the type of movement required to fill a defect. Such classification is somewhat of an artificial delineation because many flaps, such as the rhomboid flap, have qualities of both a pivot flap and an advancement flap.

ADVANCEMENT FLAPS. An advancement flap is a flap in which direct uniplanar movement of tissue fills the defect. It may be created by merely undermining skin on either side of the defect to obtain

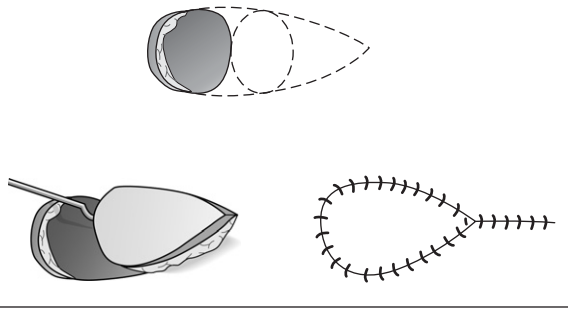


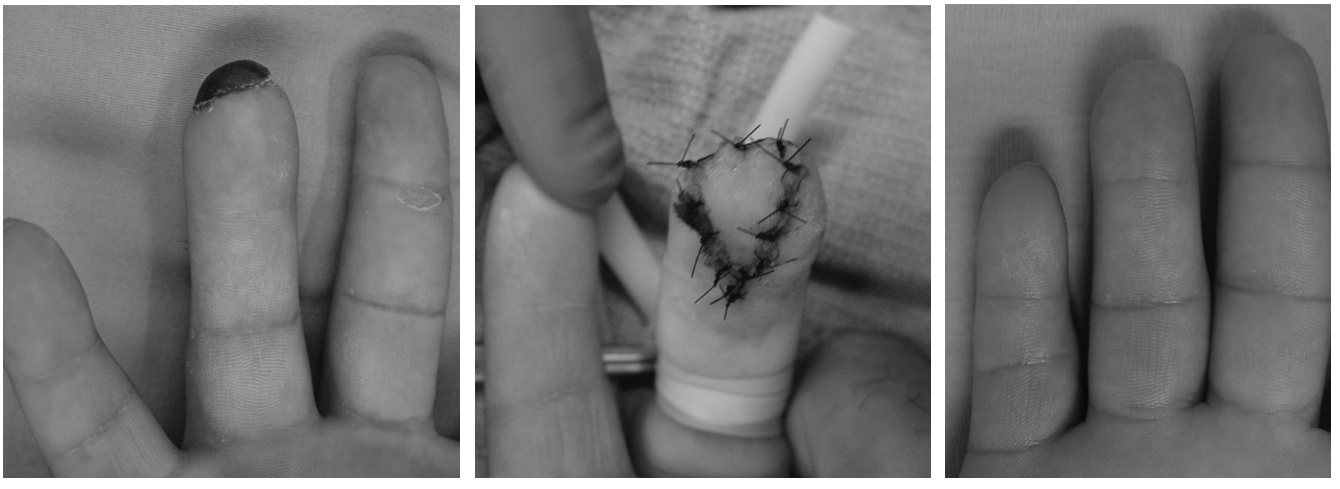
Figure 3 • V-Y advancement flap. The flaps are limited to minimal tissue advancement.

closure or by using a flap with a rectangular design. Advancement flaps may also be designed in a "V-to-Y" fashion or, conversely, in a "Y-to-V" fashion, depending on where the regions of relative tissue deficit and excess are located with respect to each other (Figs. 3 and 4). The flaps take advantage of regional tissue laxity, and the optimal design for any given flap should incorporate flap movement along the skin lines of maximal extensibility. Flap advancement can be supplemented and dog-ears contoured by excising Burow's triangles through the appropriate placement of a back cut or by performing Z-plasties at the base of the flap (Fig. 5).

PIVOT FLAPS. In contrast to an advancement flap, in which tissue is moved in one direction, a pivot flap moves tissue in two dimensions along an arc. A pivot flap may be a straightforward *rotation* flap (Fig. 6), in which case a nearly semicircular-shaped donor site rotates about a pivot point to fill an adjacent defect, or a *transposition* flap, in which case a more rectangular-shaped block of tissue rotates through an arc (Fig. 7). Several types of transposition flaps deserve special mention.

Z-Plasty. A Z-plasty is a transposition flap in which two adjacent triangular-shaped flaps are transposed with a resultant change in scar orientation, as well as an increase in length of the scar. The former property may be especially useful when a scar occurs perpendicular to relaxed skin tension lines. The increase in scar length is suited to address functional impairment or aesthetic deformity caused by scar contracture. It is crucial to release the scar tissue below the transposition flaps to allow for tension-free movement of the flaps. Properly designed, a Z-plasty may improve the appearance of a linear scar by breaking it up into more easily camouflaged segments of alternating orientation.

The "Z" of a Z-plasty consists of a central limb, which includes the scar/contracture, and two parallel peripheral limbs drawn from either end of the central limb. The angle between the central and peripheral limbs determines the amount of scar lengthening that may be achieved (Fig. 8). An angle of 60 degrees, used most commonly, provides a theoretical increase in scar length of 75%. Similarly, angles of 30, 45, 75, and 90 degrees provide theoretical increases of 25%, 50%, 100%, and 125%, respectively. It is also important to note that whereas a 60-degree Z-plasty changes the orientation of the original scar by 90 degrees, Z-plasty designs incorporating smaller angles between the central and peripheral limbs result in a central limb oriented somewhat less than 90 degrees from the original (approximately 60 degrees for a 45-degree Z-plasty and 45 degrees for a 30-degree Z-plasty). The surgeon should tailor the design of the Z-plasty to provide the appropriate amount of scar lengthening and rotation of the scar central limb.



A B C

Figure 4 • Example of V-Y advancement flap: Atasoy flap used to cover distal fingertip defect. **A,** Preoperative appearance. **B,** Intraoperative. **C,** Appearance 1 year postoperatively.

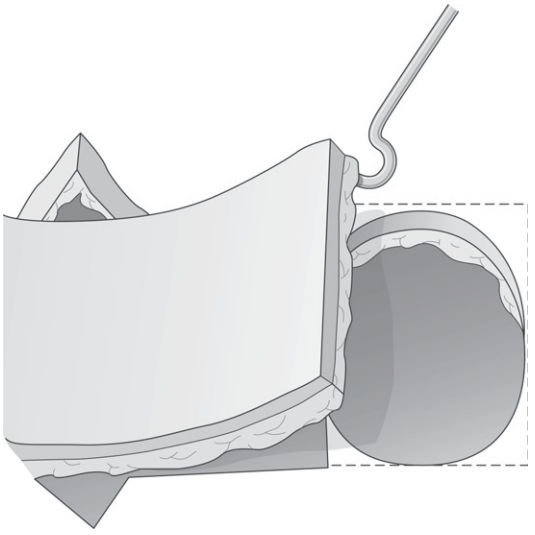


Figure 5 • Rectangular advancement flap complemented by Burrow's triangles.

Rhomboid Flap. A rhomboid flap is designed with internal angles of 60 degrees and 120 degrees and with equilateral sides (Figs. 9 and 10). In the design of a rhomboid flap, a line is drawn that extends from the short axis of the rhombus, equal to the length of the short axis. From this line, a second line is drawn parallel to the near side of the rhombus. Because lines can be extended from either side of the rhombus short axis, four possible parallel lines can be extended to create four possible flaps that may be moved to cover the defect. The design of a rhomboid flap should place one pair of the parallel sides of the rhombus along the lines of maximal extensibility, if possible, to allow for maximal movement of the flap and closure of the donor defect. The local anatomy and flap drainage should be taken into account when deciding which of the available rhomboid flaps is the best option.

Since the original report of the rhomboid flap, several other modifications have been described that vary largely in the angles of the rhomboid defect created. For closure of circular defects (for example, on the vertex of the scalp), several (three) rhomboid flaps can be combined. The use of rhomboid flaps has

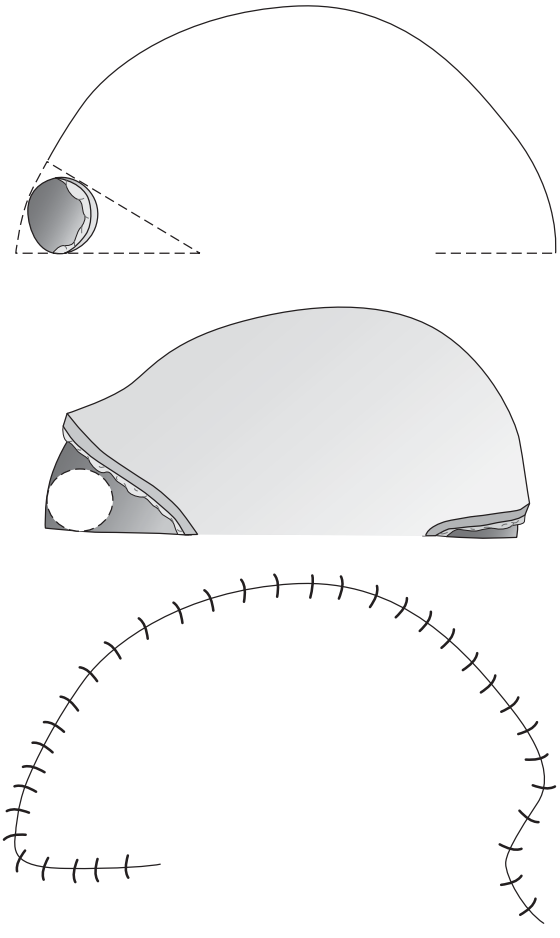


Figure 6 • Rotation flap. Note that, as the flap rotates into the defect, the radius of rotation decreases. Also note the back cut used to extend the arc of rotation.

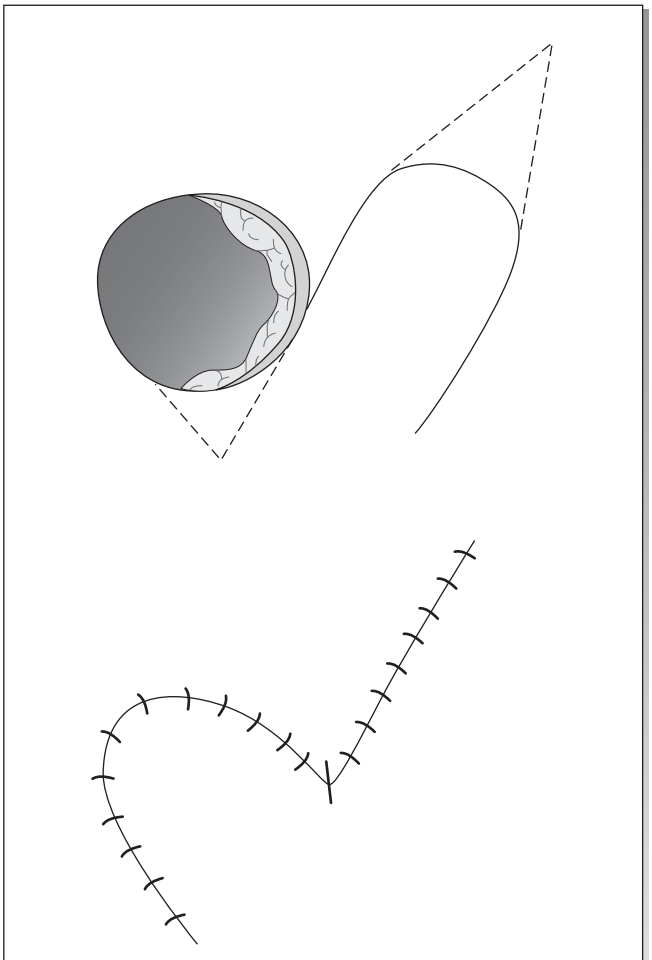


Figure 7 • Pivot flap with the characteristics of rotation and advancement.

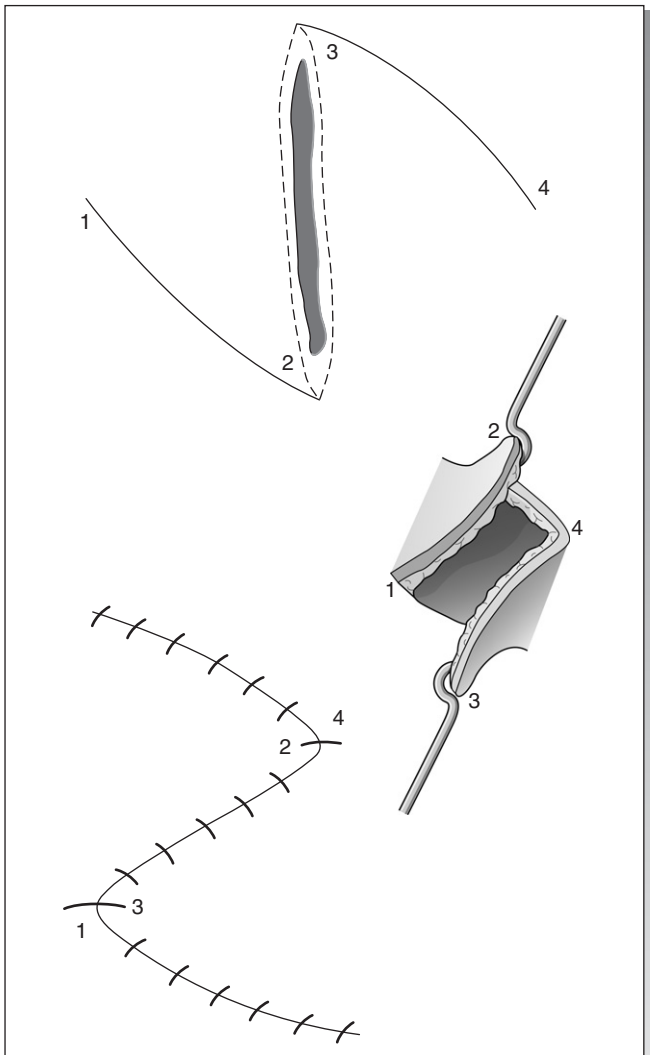


Figure 8 • Z-plasty. Note that the distances 1-2, 2-3, and 3-4 are equal. The central limb (scar) has been reoriented.

drawbacks, notably the potential for trapdoor scar formation and a resultant wound with multiple scars in various directions. These considerations mandate judicious use of this flap in situations where other alternatives do not suffice.

INTERPOLATION FLAPS. In contrast to the previous flaps, the donor site of an interpolation flap is not immediately adjacent to the defect. It may be transferred with either a random or an axial pedicle, and in most cases reconstruction is performed in two stages. A classic example of an interpolation flap is the (para)median forehead flap, which is used for reconstruction of large nasal defects (Figs. 11 and 12). Although the use of interpolation flaps generally requires at least two operative stages, in appropriate scenarios the pedicle may be thinned, deepithelialized, and tunneled subcutaneously, thereby allowing for a single-stage procedure.

In both rotation and interpolation flaps, the design of the donor site must incorporate the fact that the greater the distance the flap rotates, the shorter its effective long axis becomes. Accordingly, the donor flap should be designed "longer" than the defect that it will fill. This problem may be offset somewhat by using a back cut, which, however, decreases flap blood supply by narrowing its base. As is the case for advancement flaps, excision of Burow's triangle may also provide extra length.

In any type of flap, complete closure of both the defect and the donor site generally requires wide local undermining, when feasible, to recruit surrounding tissue laxity. The tension on the flap at closure should not be excessive because undue tension could compromise flap survival. In cases in which improper flap design has resulted in excessive tension, the flap can be reset (delayed), or the flap can be transferred and the donor site skin grafted.

Tissue Expansion

The primary advantage of tissue expansion is that it provides a source of adjacent donor tissue whose

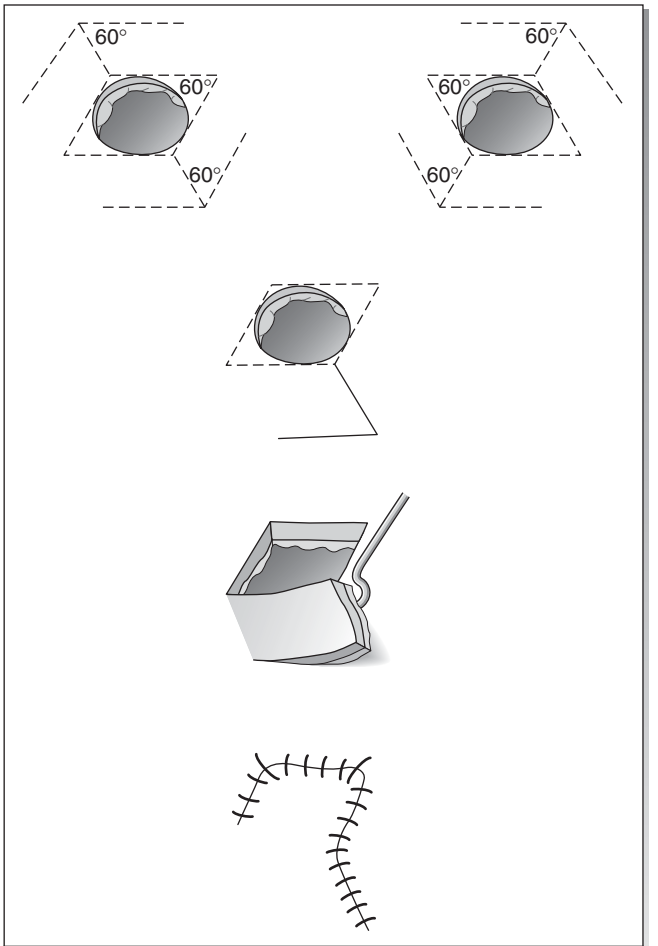
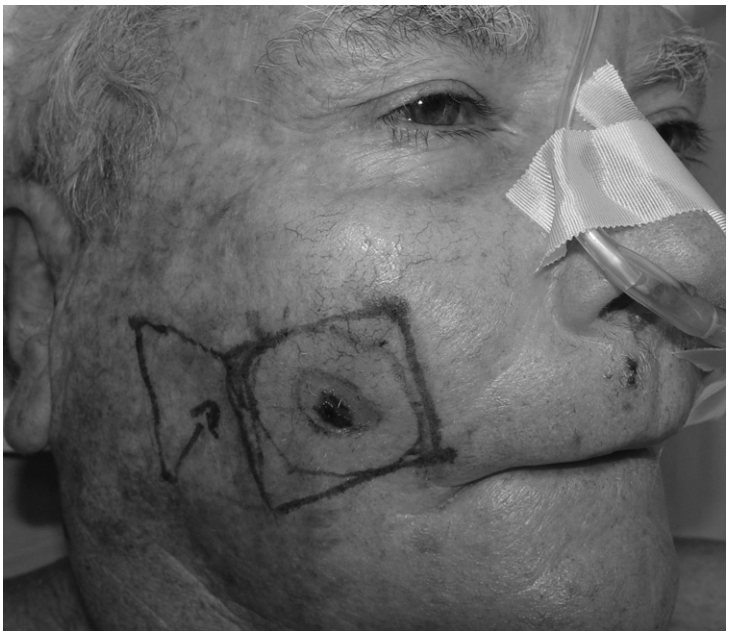


Figure 9 • Rhomboid flap. (See text for details.)



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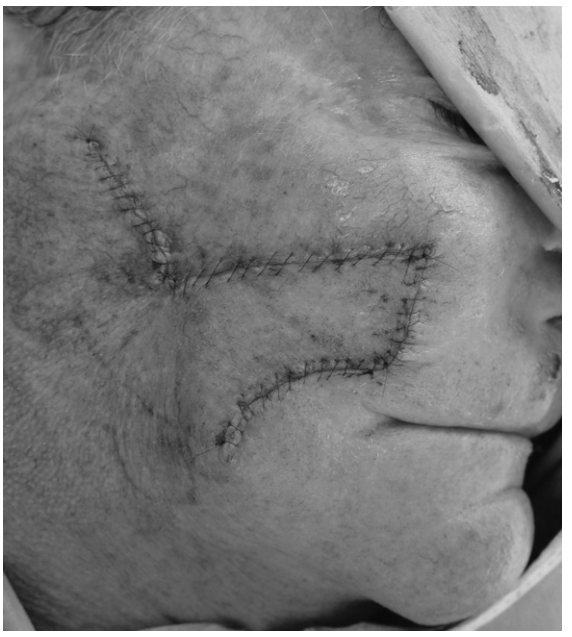

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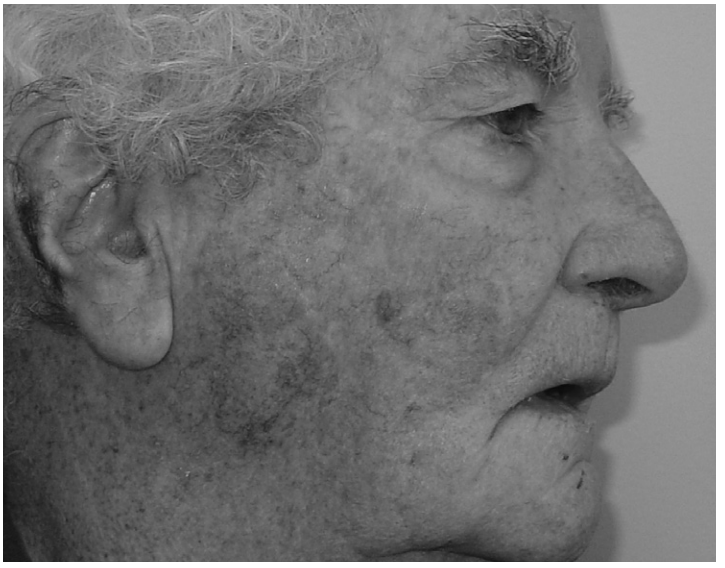


C

Figure 10 • Clinical example of rhomboid flap. Note that the flap chosen to fill the defect (of the four possibilities) allows for maximal movement as well as optimal lymphatic and venous drainage. **A,** Preoperative. Basal cell carcinoma. **B,** Intraoperative appearance of the cheek. **C,** One year postoperatively.

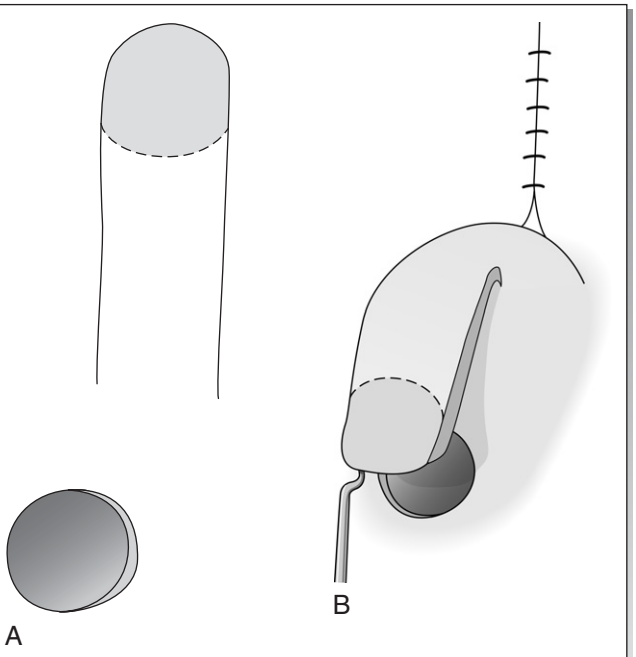


Figure 11 • A, Axial flap with defect and interposing tissue. **B,** Flap elevated and roated to fill defect (first stage).

color and texture are usually well matched to the recipient site. In addition, tissue expansion recruits both sensate and hair-bearing tissue into the recipient site when needed. It gives reliable results, can decrease donor defects, and, depending on the amount of tissue required, can be completed within a few months. Tissue expansion has found widespread application in reconstruction of the breast, head and neck region, and the trunk, usually with acceptable morbidity. Use of tissue expansion in the extremities, especially distally, tends to be more problematic.

Expanded tissue displays several characteristic changes. In general, tissue expansion results in an increase in epidermal thickness, thinning of the dermis, and possible atrophy of the overlying fat or underlying muscle. Atrophy of subcutaneous fat is usually permanent and may result in disfigurement; muscle atrophy may lead to fibrosis and irreversible loss of function. This is especially important in the face with the thin underlying facial mimetic muscles. Expansion in the scalp region should be performed with awareness that the expander can erode the underlying calvarial bone, particularly in children.

The expansion process promotes a significant increase in vascularity of the overlying skin and capsular tissue that surround the expander, similar to

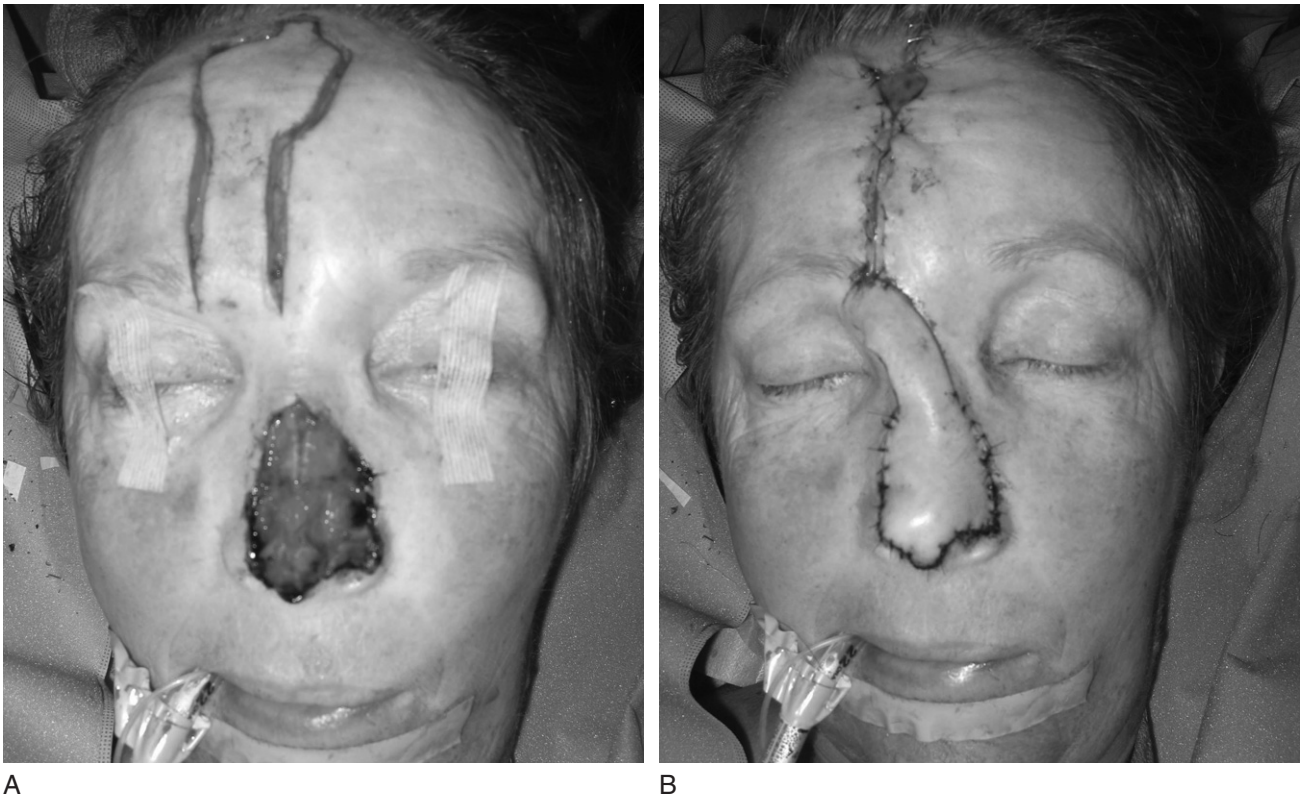


Figure 12 • Clinical example of interpolation flap; paramedian forehead flap used to resurface a large nasal defect. **A,** Nasal defect. **B,** Intraoperative transfer of flap.

what is seen when a flap is "delayed." Although the initial stages of expansion are thought to occur via creep and stress relaxation of the expanded tissue, the increased rate of cellular mitosis observed within the expanded tissue suggests the formation of new tissue over a period of days to weeks.

Choice of Expander and Technique of Placement

Standard tissue expanders are composed of a silicone polymer shell. Various sizes and shapes of expanders are available, depending on the exact needs of the reconstruction. In general, rectangular expanders generate the most usable tissue, although the shape of the expander should be tailored to the reconstructive needs. In the breast, for example, where expansion aims to develop a soft tissue envelope for subsequent placement of a permanent implant, an anatomic round expander is clearly superior to other shapes.

Several tenets of tissue expansion should be adhered to. When expanding tissue for resurfacing of adjacent tissue, the expander should be placed immediately adjacent to the defect. In the resurfacing of congenital melanocytic nevi, for example, the expander may be inserted through the lesion, if possible, to minimize scarring, but expansion of the pigmented lesion itself should be avoided. The areas in which the expander and its associated port are to be placed should be outlined as part of the preoperative marking (Fig. 13). Before commencement of the operation, a dilute epinephrine-containing anesthetic solution should be infiltrated into the marked areas. Such infiltration not only promotes hemostasis but, if injected into the proper tissue plane, also facilitates surgical dissection. A subcutaneous pocket of sufficient size should be dissected to allow the expander to be placed in its proper orientation, fully unfurled. Once in the correct plane, the use of a blunt dissector such as a urethral sound can assist appropriate dissection. Hemostasis must be rigorous because even small collections of blood around the expander increase the risk for infection, fibrosis, subsequent expander loss, or inability to expand.

Estimating Tissue Gain and Rate of Expansion

The tissue gain derived from expansion has been studied extensively but can be roughly approximated as half the width of the expander, regardless of its shape. Thus, a design that uses an expander roughly twice as wide as the anticipated defect should provide sufficient tissue for resurfacing. This may not always be feasible, especially for extremely large lesions. These will require serial expansion.

The duration of expansion can vary, depending on factors such as local tissue quality and patient tolerance. In general, expansion should commence intraoperatively to a point that allows tension-free

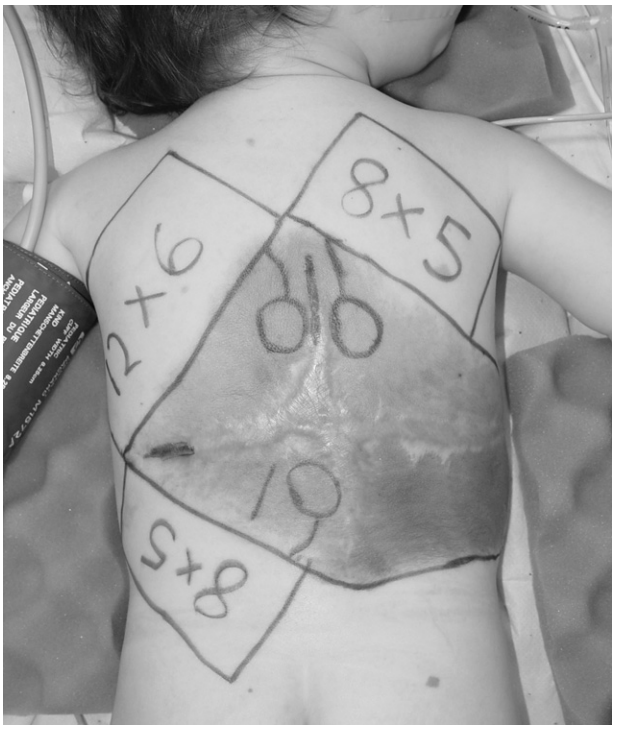


Figure 13 • Preoperative planning for placement of expanders for resurfacing of a large congenital melanocytic nevus of the back. Note dimensions and ports of the expanders.

closure, with serial expansion typically carried out every week thereafter. At each visit, the expander should be filled as much as possible without causing patient discomfort. Objective signs, such as skin blanching over the expander, indicate overfilling with the potential for skin ischemia. Rigorous sterile technique minimizes the possibility of bacterial contamination and subsequent expander infection.

Maximizing Tissue Gain from Expansion

In most cases, expanded tissue is moved into an adjacent defect as an advancement flap. To maximize the amount of expanded tissue that can be used for advancement, back cuts may be made. Although some authors have proposed making the back cut around the base of the expander, maximal advancement with minimal waste of tissue is obtained by using a back cut that is made from a point halfway along the side of the expander up to the height of the expander itself. A single back cut allows for effective rotation-advancement into a roughly triangular defect, whereas a double back cut, useful for more rectangular- to oval-shaped defects, allows only uniplanar advancement. In designing a double–back cut type of advancement flap, the surgeon may use areas of tissue adjacent to the (future) defect that can be inset into the roughly triangular defect in the

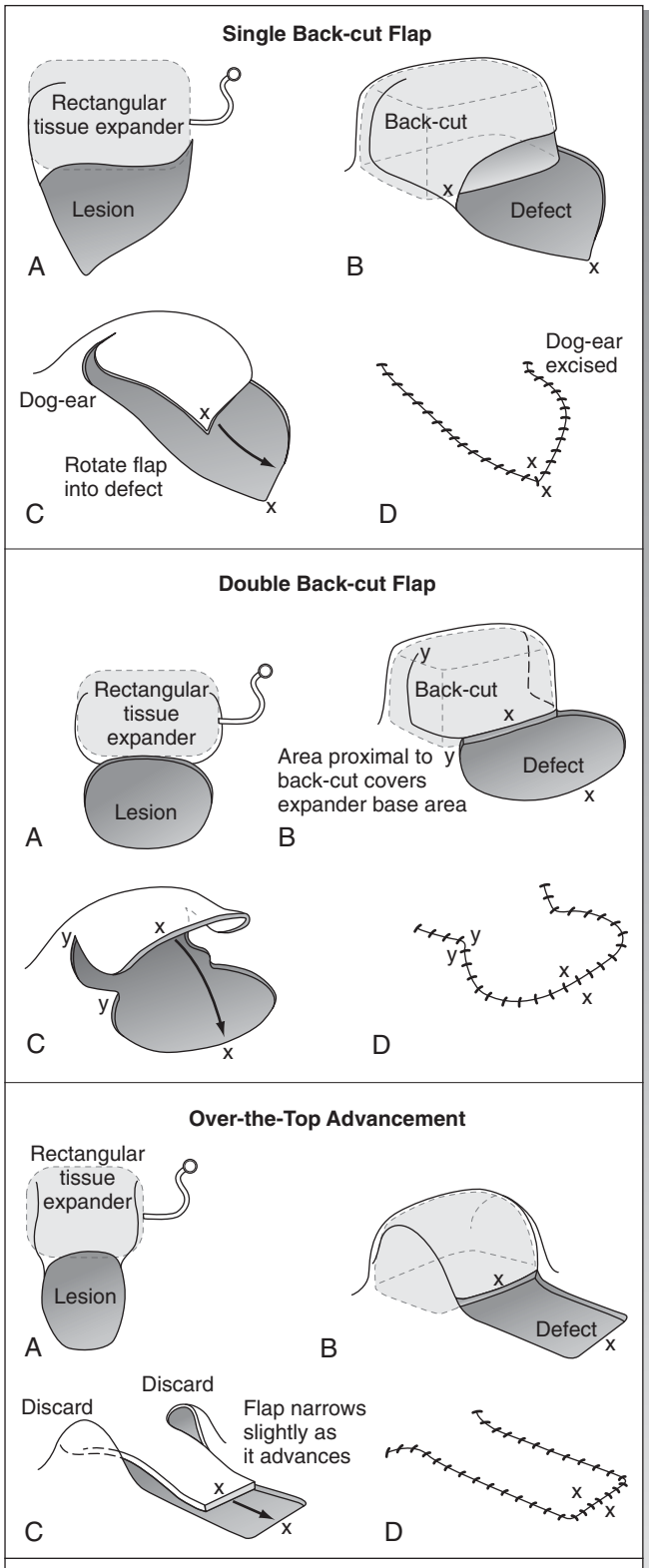


Figure 14 • Three methods of flap design after tissue expansion. Single back cut. Double back cut. "Over-the-top" advancement. (From Zide BM, Karp NS: Maximizing gain from rectangular tissue expanders. Plast Reconstr Surg 90:500-504, discussion 505-506, 1992.)

flap created by making the back cut (Fig. 14). Finally, the surgeon may use an "over the top" advancement flap, which has similar applicability as the double–back cut flap. Although it is easier to design than the latter flap, it is less efficient in its use of expanded tissue. In all cases, a significantly greater proportion of the expanded tissue is used to fill the defect than would be possible without the back cuts.

In addition, STSGs may be taken from the expanded skin, a technique particularly applicable to young children and infants, in whom the amount of donor tissue is limited even after expansion. A drawback to the technique is a reduction in the quality of the aesthetic outcome. Full-thickness grafts can also be harvested in larger sheets by using this technique with subsequent primary closure of the donor site.

Complications

The most common complication associated with tissue expanders is infection. A limited trial of conservative management with antibiotics is indicated for the treatment of mild early expander infections. If nonoperative management fails, the implant may be salvaged with deflation, irrigation of the expander pocket, and administration of parenteral antibiotics. If the infection is not eradicated by these measures, as is usually the case, the expander must be removed. Other complications associated with tissue expanders are hematoma, seroma, skin necrosis, expander exposure, and expander deflation, with an overall complication rate between 10% and 30%.

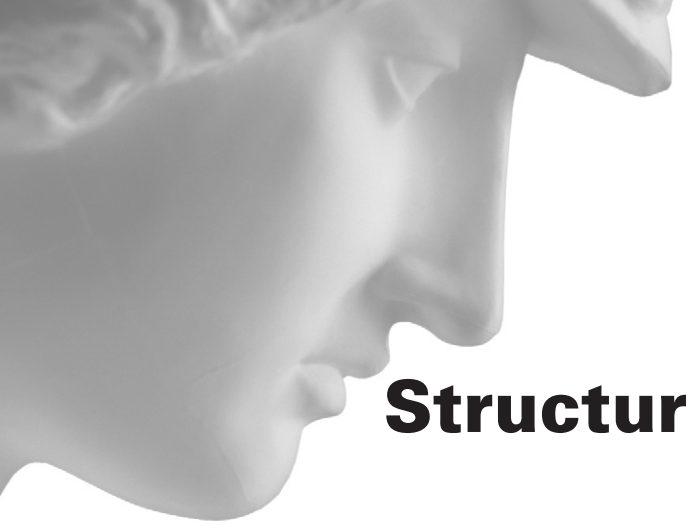
As mentioned previously, expansion may lead to local muscle atrophy, which may not only affect the aesthetic outcome but can also have serious functional consequences in the head and neck region. Other less common complications include changes in pigmentation and sensibility in the expanded skin. Complications are best minimized through meticulous planning, precise placement in a well-tailored pocket, subsequent atraumatic and sterile filling technique, and appropriate patient selection. Expansion in areas with questionable vascularity, previously irradiated tissue, and the distal end of the lower extremity is associated with high complication rates.

SUGGESTED READINGS

Baker SR, Swanson NA: Local Flaps in Facial Reconstruction. St Louis, CV Mosby, 1995.

McCarthy JG: Plastic Surgery, vol 1, General Principles. Philadelphia, WB Saunders, 1990.

Sasaki GH: Tissue Expansion in Reconstructive and Aesthetic Surgery. St Louis, CV Mosby, 1998.



Structural Support: Cartilage and Bone

SEAN G. BOUTROS

Establishment of structural integrity is essential to plastic surgical reconstruction. Often, reconstructive operations require structural support to prevent soft tissue collapse or to give contour, shape, and form. Other cases require rigid support that can withstand the forces of scar contracture and gravity to facilitate restoration of function. Bone and cartilage are unique tissues that provide support of an autogenous nature. They are often used as grafts or flaps. More recently, bone distraction has allowed the creation of new, structurally incorporated bone in many clinical situations.

Pathologic Anatomy

Structural defects, often congenital in origin, can also result from trauma, cancer resection, and infection. Most structural defects are not isolated to bone or cartilage but, in addition, are associated with deficiencies in the surrounding soft tissue. In many cases, scar contracture is present. When analyzing such defects, it is necessary to define the true defect in three dimensions in order to identify the soft tissue requirement, as well as any structural or skeletal requirements.

For as long as plastic surgeons have attempted to reconstruct defects, the question has remained whether to use autogenous material or commercially available alloplastic substitutes. The advantages of commercially available alloplastic materials are obvious. In addition to simplicity, there is no donor site morbidity and no limit to the amount of material that can be obtained. These substances are well tolerated in some clinical situations. However, all materials have shortcomings that need to be appreciated by both the practitioner and patient before implantation within the body. They require well-vascularized soft tissue envelopes of adequate bulk. Nevertheless, they are at higher risk for infection and, if they do become infected, are at higher risk for loss of the entire reconstruction. Alloplastic substances elicit unpredictable degrees of scar and tissue reaction. Over time, they are more likely to erode tissues, extrude, or become encapsulated by scar; the patient may often be left with a greater defect relative to the original one.

Autogenous material likewise has advantages and disadvantages. The main disadvantages are donor site morbidity, limited graft availability, and long-term resorption (bone) or warping (cartilage). Autogenous materials also require increased intraoperative time and add technical complexity to the reconstruction. They have a lower risk of infection than alloplast materials, however, and if they do become infected, a portion of the reconstruction can often be salvaged if appropriately managed. They also have a lower rate of tissue reaction and scar formation and do not erode overlying soft tissues over the long term. They are also more likely to be successful than alloplast materials when inserted into previously operated or scarred beds. Autogenous material adds no cost to the reconstruction. Each type of graft has its own properties, and therefore many of the risks and benefits can be optimized by careful graft selection.

In most situations, autogenous material remains the preferred option for surgical reconstruction.

Grafts

Cartilage

One unique property of cartilaginous grafts is that they do not resorb. In the hospitable environment under a vascularized soft tissue cover and in the absence of infection, cartilage grafts retain all

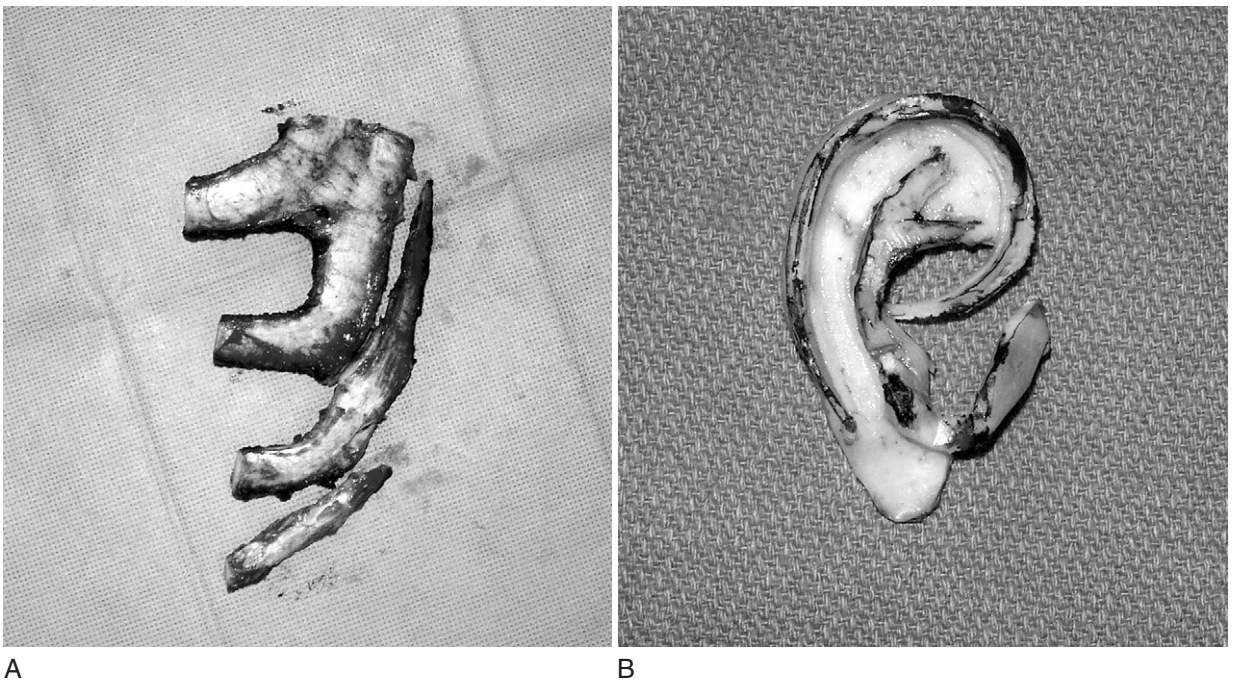
their initial volume over time. This is due to the physiology of cartilage. In its natural state, cartilage has no vasculature. The low metabolic rate of the chondrocytes is maintained by diffusion of nutrients from the surrounding tissues. The extracellular matrix of cartilage is designed to allow this diffusion. In essence, if a cartilage graft is placed in a well-vascularized bed, the supply of nutrients to the cartilage is similar to the supply in its anatomic donor state.

Rib is the most plentiful donor site for cartilage grafts but is the stiffest when compared with other sources. It is, however, prone to warping, and to prevent warping it is necessary to stabilize the graft by either inserting a rigid material (e.g., placing a K-wire in the center of a rib graft as on the dorsum of the nose) or making a multilayered construct in which the separate layers serve to stabilize and prevent warping (e.g., as in ear framework design). Septal cartilage is strong and straight, yet flexible. It is best reserved for either nasal or tarsal reconstruction. Ear cartilage has a characteristic shape that makes it useful for nasal and ear reconstruction. It is strong and has intermediate flexibility.

Rib cartilage should be harvested via either a submammary approach or a costal margin approach (Fig. 1). The submammary approach is used for harvest of a straight piece of the cartilaginous portions of ribs 5 and 6, as used in nasal reconstructions. The costal margin approach gives better access to the synchondrosis of ribs 7 and 8 when a large single piece of cartilage is needed, as in ear framework design. When the costal margin approach is used, it is necessary to divide the rectus abdominis muscle to gain access to the synchondrosis. The cartilage can be harvested without removal of the perichondrium. Sparing the perichondrium results in less pain and, in the case of the costal margin approach, less change in the contour of the thorax.

Ear cartilage can be harvested via the anterior or posterior approach. Advocates of the anterior approach argue that hypertrophic scars and keloids do not usually occur on the anterior surface of the ear. Proponents of the posterior approach point out that posterior incisions are hidden and the approach provides adequate visualization. With either method, the principles for harvest remain the same: it is critical to harvest the floor of the concha and spare the posterior conchal wall to prevent distortion of the ear. The cartilage harvested should extend to the ear canal anteriorly and to the margin of the conchal bowl superiorly, posteriorly, and inferiorly. With both methods, subperichondrial hydrodissection facilitates elevation of the cartilage with sparing of the perichondrium, which leads to less hematoma and pain. A compression bolster can be placed in the concha to prevent hematoma formation.

Harvest of septal cartilage is discussed in the primary and secondary rhinoplasty chapters.



A B

Figure 1 • A and **B,** A 6-cm costal margin incision was used for harvest of costal cartilage for ear reconstruction. A costal margin incision offers the best access for harvest of the cartilage. Alternatively, the submammary approach can be used for harvesting a smaller piece of straight cartilage.

Bone

Bone is unique in its ability to heal without scar formation. This characteristic allows healing of fractures with normal bone strength and incorporation of bone grafts that become truly integrated at the recipient site. It also allows distraction techniques to provide for deposition or generation of new bone. The biologic processes by which fractures heal, vascularized bone flaps are incorporated into existing bone, and distraction lays down new bone are similar.

Several processes are crucial for bone healing and physiology and must be understood and taken into account for successful results. *Osteogenesis* is the process by which new bone grows from existing bone. It is seen in the distraction zone and at the edge of a fracture site as new bone sprouts from existing bone. *Osteoconduction*, or creeping substitution, is the process by which cells populate and replace an existing bone matrix or artificial scaffold. It is seen when osteoblasts invade and replace cortical or cancellous bone grafts. *Osteoinduction* is the process by which factors stimulate and recruit mesenchymal cells to become osteoblasts that lay down bone. This process occurs in conjunction with bone grafting because bone grafts bring with them cytokines that initiate the process. It also can occur when commercially derived factors are added to a matrix and placed in the appropriate environment.

Healing of fracture sites begins with the formation of blood clot at the fracture ends. The clot forms a network of fibrin that acts as a temporary scaffold holding mesenchymal cells. The cells differentiate into cartilage cells and fibroblasts. Through the process of *endochondral ossification*, the fibrocartilage tissue differentiates into bone. Simultaneously, the bony edges proliferate and new bone grows toward the defect (*osteogenesis*).

In the case of vascularized bone transfers, healing occurs in the same manner, specifically, a combination of endochondral ossification and osteogenesis. With nonvascularized bone grafts, the graft first undergoes the process of osteoconduction, or creeping substitution. The scaffold of bone must be populated with viable bone cells. These cells may originate directly from surrounding bone or may be differentiated mesenchymal cells (*osteoinduction*).

Bone is more widely available as an autograft than cartilage is. It is stronger and can be used for soft tissue support or, more commonly, for grafting of bony defects. Bone grafts are classified as cortical, cancellous, or corticocancellous. Cancellous bone grafts can be harvested from several anatomic sites to fill small bony defects and to assist with fracture healing in bony nonunion. Cancellous bone grafts act in two ways to allow for healing or new bone formation. They release growth factors and cytokines such as bone morphogenetic protein, which recruits and stimulates mesenchymal cells to differentiate into bone (osteoinduction). In addition, they provide a framework into which the surrounding bone can grow (osteoconduction) via creeping substitution. As the bone grows from the periphery, the existing graft framework is degraded, and bone is laid down de novo. The principles for success are adequate vascularized soft tissue coverage and immobilized adjacent bony segments.

Cortical or corticocancellous bone grafts are also used to bridge bony gaps. However, they serve a structural function as well. Larger bony gaps can be bridged and serve as a scaffold to which new bone will grow from the margins (osteoconduction). The principles for survival of any cortical or corticocancellous graft are healthy, well-vascularized soft tissue coverage and rigid, multipoint stabilization with contact of the graft to incorporated bone. For bone grafts to maintain their volume over time, they must form a solid union with the existing incorporated bone and be exposed to stress, especially compressive force. These forces can be minimal (as in cantilever bone grafts applied to the nasal dorsum, where the graft forms a union at the base and is exposed to the force of the nasal envelope) or strong (as in bone grafts for defects in weight-bearing bones such as the mandible or tibia).

Harvest of Bone Grafts

The iliac crest is the most common bone graft donor site (Fig. 2). It provides a large volume of bone with ample amounts of cortical, cancellous, and corticocancellous bone. The access incision begins 1 cm posterior to the anterior superior iliac spine. The incision is made below the pelvic brim with the skin stretched over the iliac crest. This maneuver makes the final scar less conspicuous because it lies below the bony prominence. With the skin stretched over the crest, the incision is made directly from the skin through the subcutaneous tissue and muscle to the periosteum of the underlying bone. The thickest portion of the ilium is approximately 2 cm posterior to the anterior superior iliac spine.

If *cancellous* bone is required, the periosteum is not elevated. The bone is split with an osteotome parallel to the crest. Two other cuts are made perpendicular to the bone on the inner aspect only. The inner table is fractured inward so that the bone is attached solely by the inferior periosteum. The crest is, in essence, opened like a book. The cancellous bone is obtained either in blocks or with a curet. The divided bone is coapted by placing sutures from the overlying muscle cuff of the infractured inner cortex to the corresponding cuff on the outer cortex.

If *cortical* bone is needed, it is best to harvest only the inner table of the crest, thereby leaving the natural contour of the pelvic brim. However, if larger pieces are required, the entire top of the iliac crest can be harvested. The periosteum is split and elevated from the bone in continuity with the muscle insertions. The bone is harvested and the periosteum repaired. This technique also coapts the

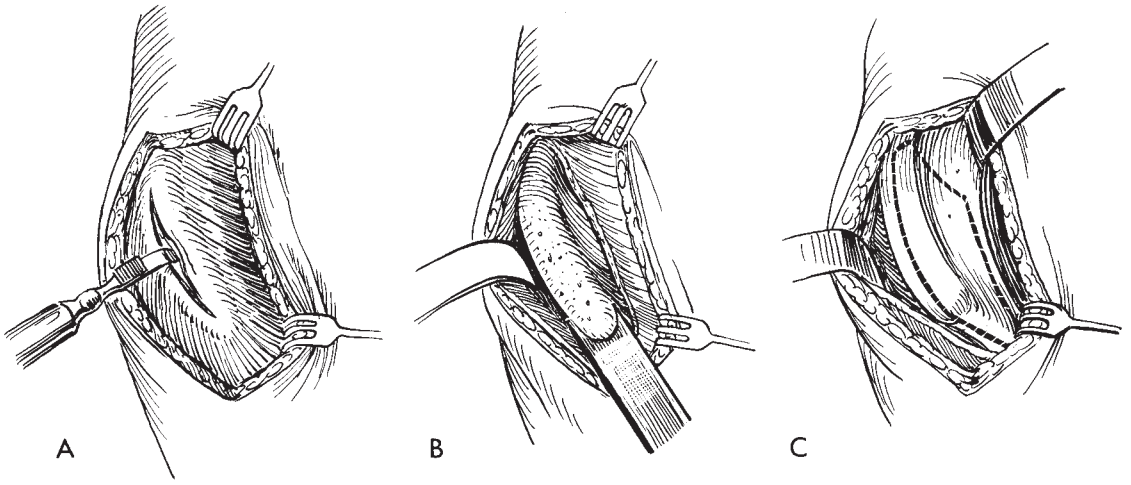


Figure 2 • Removal of bone from the ilium. **A,** The periosteum has been incised and is being raised with an elevator. **B,** Resection of bone from the crest. **C,** Resection of the bone from the inner table (From McCarthy JG [ed]): Plastic Surgery. Philadelphia, WB Saunders, 1990, p 607.)

muscles and thus restores muscular anatomy. By leaving the anterior superior iliac spine intact, donor site morbidity is greatly reduced.

Rib also provides useful bone grafts. These grafts are characteristically thin and narrow and have minimal strength. Multiple ribs can be harvested and split to increase their surface area. They are useful in areas with minimal force, such as in calvarial or nasal reconstruction. If multiple ribs are harvested, alternate ribs should be used. It is best to make an incision over the seventh rib laterally and harvest the fifth, seventh, and ninth ribs through this incision. The anterior periosteum is incised to the length of the rib to be harvested and elevated such that the periosteum is left attached only posteriorly. In the most accessible area, the periosteum is circumferentially elevated. A rib dissector is passed in the anterior and posterior directions and the posterior periosteum is dissected. The rib is divided at the bone-cartilage interface anteriorly and as far posteriorly as possible (Fig. 3).

Calvarial bone grafts are another useful graft both for adjacent calvarial defects and for orbital floor and nasal reconstructions (Fig. 4). The calvaria is the least painful of all bone donor sites, and no visible scars are left in patients with hair. It suffers the disadvantage of being thin and brittle. The bone is best harvested from the parietal bone that is thickest, approximately 2 cm posterior to the coronal suture. It can either be removed in full-thickness fashion as in a craniotomy and split on a back table with a sagittal saw or, more commonly, be split in situ with the inner table left intact. For the latter, a trough is created with a bur around the bone to be harvested. The trough should be sufficiently deep with a gradual curve and should extend completely through the diploic layer to the inner cortex. Thin curved osteotomes are used to cut through the diploic layer. Care must be exercised because an osteotome can act as a lever and easily fracture the graft. It is best to move the osteotome frequently to avoid fracture.

Distraction

Bone distraction is a technique in which normal fracture healing is manipulated so that as the fracture ends are pulled apart, callus is deposited and new bone is formed between the fracture ends. The principles are as follows: vascularized healthy bone of adequate stock provides the best results, sufficient time should be allowed for initial callus deposition (latency period), distraction should be performed at a slow rate (approximately 1 mm/day), and there should be a consolidation period in which the bone is stabilized in its distracted position. Bone distraction has the advantage of expansion of the surrounding soft tissues and creation of new, well-vascularized bone. It has no donor site morbidity, requires patient compliance, and is time consuming. Overall, it has revolutionized the treatment of many craniofacial conditions and opened a new era of bone surgery.

Pearls and Pitfalls

- Autologous material is preferable to synthetic substitutes.
- Cartilage grafts placed in a hospitable environment do not lose volume over time.

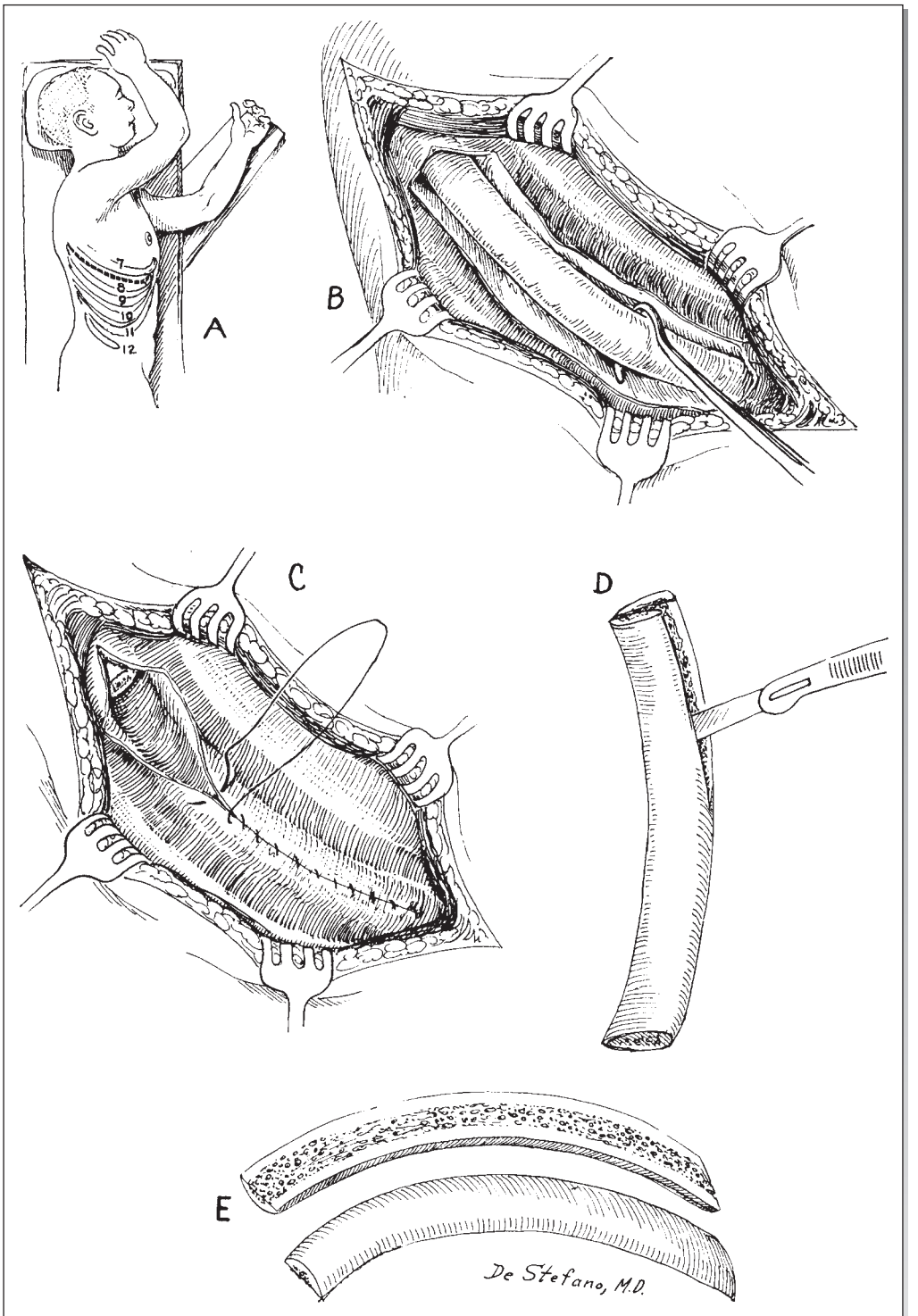


Figure 3 • A, B, Removal of a full length of rib by the subperiosteal technique. The Doyen elevator is used to free the periosteum of the rib bed. **C,** Closing the periosteal bed with a running chromic catgut suture. **D, E,** Technique of splitting the rib with the #10 knife blade. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, WB Saunders, 1990, p 616.)

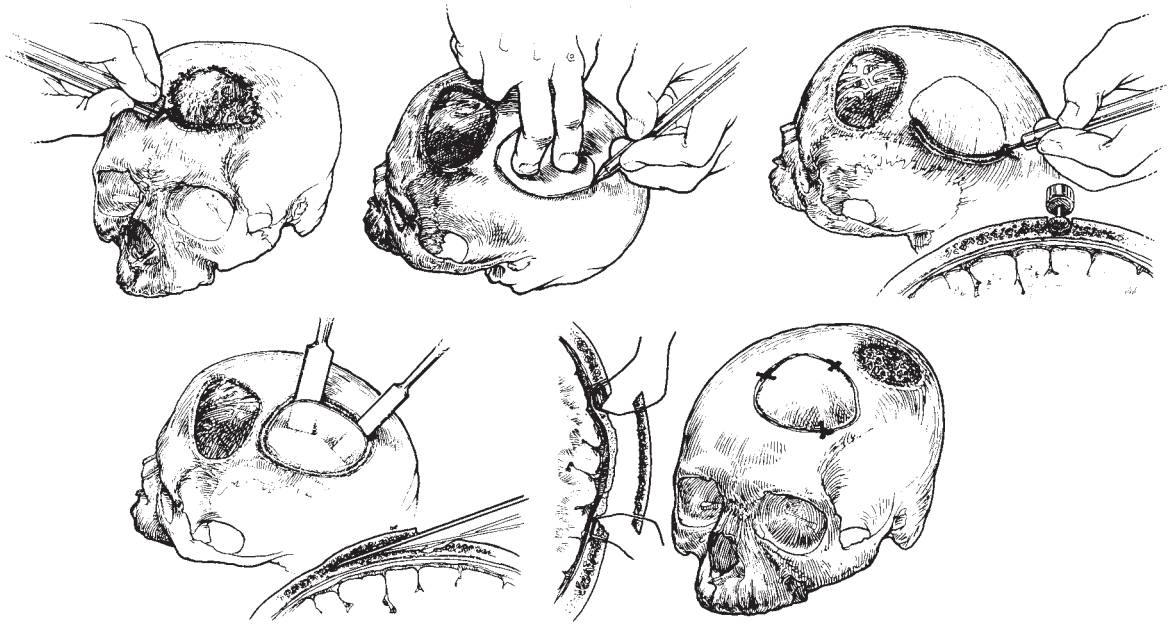


Figure 4 • In situ harvesting of a bone graft from the outer table of the calvaria. A trough is drilled around the periphery of the proposed graft into the diploë. The outer table is removed by passing an osteotome through the diploic space. (From McCarthy JG, Zide BM: The spectrum of calvarial bone grafting: Introduction of the vascularized calvarial bone flap. Plast Reconstr Surg 74:10, 1984.)

- Rib cartilage grafts do not warp if supported with rigid material or layered as a multilevel construct.
- Ear cartilage harvest should spare the posterior wall of the concha and include the floor of the external canal to provide a large piece of cartilage and prevent ear deformities.
- Bone grafts are rigidly fixated and exposed to stress force to avoid long-term resorption. Onlay bone grafts, with the exception of those placed in the nasal dorsum, tend to resorb over time.

SUGGESTED READINGS

Brent B: Repair and grafting of cartilage and perichondrium. In McCarthy JG (ed): Plastic Surgery. Philadelphia, WB Saunders, 1990, p 559.

Cutting CB, McCarthy JG, Knize D: Repair and grafting of bone. In McCarthy JG (ed): Plastic Surgery. Philadelphia, WB Saunders, 1990, p 583.

Anesthesia

JOHN B. HIJJAWI
THOMAS A. MUSTOE

A thorough knowledge of anesthesia techniques can facilitate many aspects of plastic surgery. Anesthesia for many plastic surgery procedures can be achieved through knowledge of the cutaneous patterns of innervation. Such knowledge, combined with conscious sedation, can provide an ideal experience for both the patient and surgeon.

Recently, a trend toward outpatient surgery has taken place. Although substantial improvements in general anesthesia allow more rapid recovery, there remain disadvantages, including the risks inherent in induction, intubation, blood stasis in the extremities, extubation, and postanesthesia nausea.

This chapter highlights techniques of local anesthesia which, when combined with conscious sedation, are applicable to aesthetic and reconstructive procedures. Many of these techniques do not rely solely on the use of sedative or analgesic medications; rather, moderate amounts of opioids and benzodiazepines are used to allow painless infiltration of high-volume, dilute lidocaine solution to create a reliable, anesthetic surgical field.

Spectrum of Anesthesia Techniques

Conscious Sedation

During conscious sedation, patients preserve the ability to protect and maintain a patent airway and remain capable of responding purposefully to verbal and tactile commands (such as repositioning in cases of liposuction). At no point is the patient's spontaneous ventilation or airway patency impaired. Sedation may or may not be combined with analgesic medications and local anesthesia, depending on the nature of the procedure to be performed. Typically, the patient has limited recall of the procedure and less postanesthesia nausea than with other techniques.

A critical component of successful conscious sedation for surgical procedures is the ability to achieve adequate local anesthesia through a combination of wetting solutions (dilute preparations of lidocaine and epinephrine) and nerve blocks, thus limiting the amount of opioid analgesia required.

Deep Sedation

In deep sedation, the patient experiences partial loss of protective airway reflexes and becomes unable to respond purposefully to verbal stimulation. Some assistance may be needed to maintain airway patency and spontaneous ventilation. The patient's responses may be limited to withdrawal from painful stimuli. Cardiopulmonary function may become depressed as the patient proceeds along the spectrum from deep sedation to general anesthesia.

"Overshooting" when attempting to achieve conscious sedation results in the patient progressing to deep sedation. The patient typically requires verbal or tactile stimulation to continue breathing spontaneously. Additionally, supplemental oxygen may be required. Reversal agents must be on hand, although their use is often avoided.

In contrast, "overshooting" a deep sedation protocol results in complete cessation of respiratory drive. This problem becomes especially challenging because deep sedation protocols are typically carried out with propofol, which despite being metabolized quickly, has no reversal agent. As a result, a significantly higher level of training (i.e., a certified registered nurse anesthetist [CRNA] or anesthesiologist) is required for medication administration and patient monitoring when using a deep sedation protocol.

General Anesthesia

General anesthesia is characterized by complete loss of protective reflexes, accompanied by an inability to

TABLE 1 Procedures Amenable to Conscious Sedation Techniques

RECONSTRUCTIVE
Expander implant exchange
Implant revision
Transverse rectus abdominis myocutaneous (TRAM) delay procedures
Nipple reconstruction
Mohs' reconstruction
Excision of large lipomas
Excision of large pigmented lesions
AESTHETIC PROCEDURES
Rhytidectomy
Brow lift
Blepharoplasty
Liposuction
Breast augmentation (submuscular and subglandular)
Mastopexy
Limited breast reduction

maintain a patent airway or spontaneous ventilation. Deeper states of general anesthesia are associated with depressed cardiovascular function. Deep sedation and general anesthesia are strictly limited to practice by anesthesiologists or by CRNAs under the direction of a surgeon or, more commonly, an anesthesiologist.

Procedure Selection

A common misconception is that conscious sedation is used in aesthetic surgery solely to decrease costs. Many would therefore assume that conscious sedation techniques are not used for reconstructive procedures with third-party payers. Such is not the case because the benefits extend well beyond cost, thus making conscious sedation useful in a variety of procedures (Table 1).

The common factor that makes safe execution of all these procedures possible under conscious sedation is the ability to achieve effective local anesthesia. In healthy patients, conscious sedation can be safely used in any procedure or combination of procedures in which effective local anesthesia can be achieved with an upper-limit lidocaine dose of 35 mg/kg and an operative duration of less than 5 hours.

Patient Selection

Medical Status

Properly selecting the method of anesthesia and clearly communicating what the patient can expect are important factors in a successful surgical experience. Patients who are candidates for conscious sedation must be American Society of Anesthesiology class I or II. More recently, physical status (PS) classifications have been used to stratify patients into risk-based categories. Within the PS classification, conscious sedation techniques are limited to patients qualifying as PS-1 or PS-2. Such restriction limits conscious sedation to either healthy patients or those with mild systemic diseases resulting in no functional limitations (i.e., well-controlled hypertension or diabetes, mild obesity). Particular attention is paid to preoperative electrocardiograms and cardiac status as indicated by a patient's functional status.

Relative contraindications to conscious sedation include either extremely young or old age, a history of heavy smoking, or significant cardiopulmonary, hepatic, renal, or central nervous system (CNS) disease. The technique also is not appropriate for those with a significant history of alcohol or illicit drug use, patients who are morbidly obese, have sleep apnea, have atypical airway anatomy, or those with a previous history of complications related to sedation or general anesthesia.

Preoperative Evaluation

Preoperative evaluation of candidates for conscious sedation includes a history and physical examination, age-appropriate laboratory testing, electrocardiography, radiographic evaluation, and consultation with appropriate medical specialists. Healthy patients younger than 40 years require no specific laboratory evaluation short of pregnancy testing in females. Between the ages of 40 and 65, a preoperative hemoglobin assay and electrocardiogram are obtained. Patients older than 65 years should also have a preoperative chest radiograph reviewed.

Safety Considerations and Preparation

As in all surgical care, the safety of the patient is of paramount importance when performing plastic surgery procedures under conscious sedation. Although protocols have been designed to maintain patients in the state of conscious sedation, it is mandatory that the physician providing conscious sedation be prepared to manage patients who may fall into the next level of sedation, deep sedation. Physicians providing conscious sedation must therefore be trained to manage a compromised airway, establish a patent airway, and provide adequate positive pressure ventilation and oxygenation.

At least one person trained in basic life support (BLS) must be with the patient in the operating

room and the recovery room. Advanced cardiac life support (ACLS) services must be available within a reasonable amount of time. In most office settings, emergency medical service (EMS) responders provide these services when needed. It is important that any facility performing procedures under conscious sedation communicate with the directors of local emergency departments so that they are aware that conscious sedation care is being provided in their community. Contact information for local EMS responders should be periodically verified. Procedures can be performed in a hospital setting with anesthesia backup immediately available. In an office situation, use of a nurse anesthetist adds an extra margin of safety, but each physician must make a decision regarding appropriate safety measures for that particular practice setting.

Any person ordering or administering sedative and narcotic medications should be thoroughly familiar with their pharmacology and the potential interactions of any agents given on the day of surgery. To maximize expertise with the medications used during procedures, they should be limited to one sedative and one analgesic agent (often a single benzodiazepine, midazolam, and a single opioid, fentanyl). These agents are discussed in detail in the next section. Both incremental and total doses, as well as the exact time that the medications are given, are charted on a large board that is visible to the entire operative team. A "medication-monitoring" nurse announces the total doses every 5 minutes.

The surgical team for conscious sedation procedures includes the surgeon and three nurses. The surgeon is responsible for determining the dosing and frequency of medication delivery. The "medication-monitoring" nurse has very strictly defined responsibilities during the intraoperative period, including monitoring the patient's level of comfort and consciousness; monitoring and recording vital signs; delivering, charting, and announcing the total doses of medications; and most important, communicating with the patient and surgeon. This nurse does not function as a circulating nurse at any point in the procedure, nor does this nurse need to be a CRNA.

Monitoring and Supportive Therapies

Basic monitoring includes the level of comfort and consciousness, pulse, blood pressure, and respirations measured at least every 5 minutes and continuous electrocardiogram and oxygen saturation monitoring. *Oxygen saturation is maintained at or greater than 92%.* Oxygen saturation below this level is the most sensitive indication of a transition from conscious sedation to deep sedation.

The corollary to monitoring the oxygen saturation level as an indicator of the patient's level of consciousness is avoidance of routine supplemental oxygen. Although deep sedation protocols routinely include supplemental oxygen to provide an added margin of safety in the event of apnea or airway obstruction, deep sedation techniques, by definition, hinder the patient's ability to independently maintain a patent airway and breathe spontaneously. In conscious sedation protocols, adding supplemental oxygen elevates a patient's oxygen saturation at a given level of sedation and, as a result, masks one of the most sensitive indicators of descent into deep sedation.

By eliminating oxygen supplementation, the ability to detect the critical transition into oversedation or narcotization at a relatively early point is maintained. Thus, respiratory depression is detected at a point at which it is more readily reversed with verbal stimulation and temporary withholding of further sedative or analgesic medications.

Essential equipment in the operating room includes a source of continuous oxygen, equipment to provide positive pressure ventilation (Ambu bag and facemasks), emergency airway equipment (oral airways, nasopharyngeal "trumpets," intubation equipment), suction, defibrillation equipment, reversal agents, and monitoring equipment with adequate emergency backup power. As noted, the equipment is only as good as the training of the surgeon and staff responsible for using it.

The training and equipping of staff in the recovery area is equally as important in the operating room. A registered nurse trained in BLS should be directly responsible for patients in the recovery area. Appropriate equipment for monitoring vital signs, including oxygen saturation, and a continuous supply of oxygen should be immediately available. Emergency medications and airway equipment should also be present.

Preoperative Procedures and Medications

Patients are required to abstain from solids for 6 to 8 hours and from liquids for 3 to 4 hours before procedures performed under conscious sedation. Factors that traditionally increase the period of restriction, such as pregnancy, obesity, and significant diabetes mellitus, are not an issue because these patients are not candidates for conscious sedation techniques.

Ondansetron (Zofran)

Nausea can be effectively dealt with in the preoperative period. Postoperative nausea and vomiting not only prolong recovery but can also increase the incidence of hematoma after plastic surgery procedures (e.g., rhytidectomy) and unintended admission after

planned outpatient procedures. Preoperative ondansetron reduces the incidence of postoperative nausea and vomiting in patients undergoing surgical procedures performed under conscious sedation.

Of note, in procedures that take less than 90 minutes, the risk of postoperative nausea and vomiting is virtually zero. Therefore, the expense of ondansetron is probably not justified for procedures lasting less than 90 minutes.

Clonidine

It is extremely important to maintain stable blood pressure and heart rate in patients undergoing facial rhytidectomy. Even brief episodes of hypertension can have catastrophic results in these patients. Hematoma and seroma formation can potentially lead to skin flap necrosis or delayed wound healing. Even when hematomas are detected and drained expeditiously, the overall experience of the cosmetic surgery patient is made significantly less pleasant. Therefore, the authors routinely premedicate all patients undergoing facial rhytidectomy with 100 to 200 μg of oral clonidine (Catapres) approximately 1 hour before surgery.

Clonidine is an α_2-adrenergic receptor agonist that acts centrally to inhibit sympathetic outflow. Inhibition of sympathetic outflow is responsible for many of the effects that make clonidine so useful perioperatively. Clonidine improves hemodynamic stability with a reduction in both baseline heart rate and mean arterial pressure. Unlike many β-blockers, there is no significant "rebound hypertension" associated with clonidine; in fact, its effect lasts well into the recovery period. In addition, clonidine has a sedative effect that serves as a useful adjunct in the conscious sedation technique.

Clonidine has also been shown to reduce the use of sedative, analgesic, and anesthetic agents in a wide range of surgical procedures performed under both general anesthesia and sedation techniques. Within plastic surgery, it has been shown to decrease propofol requirements in patients undergoing facial rhytidectomy. The shivering associated with recovery from anesthesia has also been reduced by the preoperative administration of clonidine. It is important to note that patients should always have their heart rate and blood pressure documented before the administration of clonidine, with doses adjusted accordingly.

Intraoperative Procedures and Medications

Opioids and Benzodiazepines

Multiple agents are available for use in conscious sedation protocols, including benzodiazepines, opioids, barbiturates, phenothiazines, and hypnotics. The agents selected for use in any conscious sedation protocol should achieve the goals of anxiolysis, anesthesia, analgesia, and amnesia. These goals are ideally achieved by using the most limited number of drugs possible. Agents can be strictly limited to diazepam (given only in the preoperative period), midazolam, fentanyl, and lidocaine. By limiting the protocol to three classes of agents, surgeons can maximize the familiarity of the surgical and nursing staff with the appropriate doses, onset of action, and duration of action of these agents. Furthermore, all agents are limited to intravenous delivery to eliminate the sometimes unpredictable onset of action of orally delivered drugs (i.e., diazepam).

Fentanyl and midazolam are ideal agents. Their rapid onset of action and short half-lives greatly facilitate the titration of drug delivery to a given effect. The use of small, incremental doses separated by at least 5 minutes is key because of the rather steep dose-response curve of both these drugs. Typical incremental doses of midazolam are in the range of 0.5 to 1 mg. Generally, once the total dose of midazolam reaches 15 mg, amnesia is complete.

Fentanyl administration is limited to doses of 25 μg. The total administration of fentanyl is restricted to 250 μg for a 2-hour case and 300 μg for procedures lasting up to 4 hours. Patients older than 55 years are given relatively smaller doses, whereas younger adults or those with a significant history of alcohol consumption are given relatively higher doses.

Reversal Agents

Flumazenil and naloxone specifically antagonize benzodiazepines and opioids, respectively. Their use is reserved for situations in which clear oversedation or narcotization has occurred. These situations are indicated by the following: a loss of patient-maintained ventilation that is refractory to verbal stimulation, hypoxemia refractory to oxygen supplementation, and the patient's inability to maintain a patent airway.

Reversal agents do have significant side effects. The sudden blockade of opioid receptors in a patient who has recently or is currently undergoing a surgical procedure can induce significant pain-mediated hypertension and tachycardia. Additionally, naloxone can cause nausea and vomiting, pulmonary edema, and ventricular dysrhythmias. Flumazenil has been associated with dizziness, agitation, and seizures. Therefore, rather than administering these agents at the first hint of a potentially oversedated patient, supplemental oxygen should be administered and the patient verbally encouraged to breathe deeply. Next, a painful stimulus such as injection of local anesthetic or a sternal rub is applied. Basic airway maneuvers such as a jaw thrust and application of oxygen by nasal cannula are simultaneously performed. Positive pressure ventilation can be administered if these maneuvers

fail. Only after the failure of these interventions should reversal agents be considered. However, there should be no hesitation in administering these agents after it is clear that positive pressure ventilation cannot be maintained.

These agents are given incrementally and titrated to effect. It is critical to realize that reversal agents have durations of action that are significantly shorter than those of the agents that they antagonize. Consequently, they may need to be readministered several times before the benzodiazepine or opioid has been completely cleared to avoid the phenomenon of resedation. Reversal agents need to be kept available and the patient closely monitored for up to 2 hours after the completion of surgery.

The endpoint of any conscious sedation protocol is a hemodynamically stable patient who remains cooperative and maintains the ability to verbally confirm comfort and relaxation. Indeed, lack of cooperation by the patient is one of the earliest signs of oversedation and typically represents the disinhibition sometimes induced by benzodiazepines. The tendency to further sedate an uncooperative patient must be avoided. Rather, the patient should be assessed for hypoxemia and signs of lidocaine toxicity or oversedation. All these factors may potentially contribute to agitation. Finally, the quality of local anesthesia in the surgical field is reassessed.

Local Anesthetics

Two major groups of local anesthetics are available. The *ester group* includes cocaine, procaine, tetracaine, and benzocaine. These agents are derived, in part, from *para*-aminobenzoic acid (PABA). Allergic reactions to these agents are known to occur and are largely thought to be reactions to the PABA component of ester anesthetics. Ester anesthetics are metabolized by plasma pseudocholinesterase, and therefore impaired metabolism is rarely an issue.

The isolation of lidocaine in 1948 introduced the *amide group* of local anesthetics. Amide anesthetics such as lidocaine, mepivacaine, and bupivacaine are virtually never associated with allergic reactions. They are metabolized via a hepatic route, however, and thus should be used cautiously in patients with significantly impaired liver function.

Ester and amide anesthetics both function by blocking sodium channels, inhibiting threshold potentials, and thereby reversibly blocking nerve conduction. Both anionic and cationic molecules exist in any preparation of local anesthetic. Anionic molecules penetrate nerve membranes, and thus it is the anionic portion of a given anesthetic preparation that provides the true effect of local anesthesia. Adding sodium bicarbonate to these agents achieves two goals. First, the acidity of commercial local anesthetics is decreased, thus diminishing the discomfort associated with infiltration in awake or sedated patients. Second, the proportion of local anesthetic existing in an anionic form is increased, thus maximizing the effect and onset of action of a given volume of agent. Ultimately, the single most important factor in potency and onset of action of any particular local anesthetic is the total dose delivered.

Tumescent Anesthesia

Tumescent anesthesia, familiar to any surgeon performing liposuction, is a fundamental component of the conscious sedation technique. No longer unique to liposuction procedures, tumescent anesthesia has been extensively used in aesthetic surgery, oncologic breast surgery, and even pressure sore treatment. The tumescent technique provides the advantages of excellent local anesthesia delivered over a broad anatomic region, hemostasis and decreased intraoperative blood loss, reduction in the need for intravenous fluid resuscitation, and elimination of the need for more toxic long-acting local anesthetic agents such as bupivacaine.

The tumescent technique is especially useful in facial plastic surgery and aesthetic breast procedures. Not only are anesthesia and hemostasis facilitated, but hydrodissection of surgical, avascular planes expedites operative dissection in many cases.

Typical solutions consist of 100 mL of 1% lidocaine (Xylocaine), 1 mL epinephrine (1:1000), and 250 mL saline for face lifts, breast augmentations, or mastopexies. For abdominoplasties, breast reductions, mastopexies with liposuction, or liposuction, a standard mix of 50 mL of 1% Xylocaine and 1 mL epinephrine in 1000 mL lactated Ringer's solution or saline is used as a supplement to a more concentrated solution for the skin incisions. As with traditionally delivered lidocaine, addition of sodium bicarbonate to more concentrated tumescent lidocaine solutions increases the proportion of anesthetic in the more functional anionic form. Additionally, sodium bicarbonate decreases the discomfort typically associated with the injection of acidic agents in awake or even sedated patients. Normal saline or lactated Ringer's solution should be stored at 40° C. Warming fluids not only minimizes the potential for hypothermia associated with the infiltration of relatively large volumes of room-temperature fluids but also reduces the discomfort associated with infiltration.

Traditionally formulated preparations of lidocaine are considered safe in doses of 5 mg/kg without epinephrine and 7 mg/kg with epinephrine. Lidocaine delivered in tumescent solution, however, is safe in doses of approximately 35 mg/kg up to 50 mg/kg (although the lower limit is the highest used by most practitioners). The safety of such high doses of lidocaine was initially thought to be due to

evacuation of the anesthetic agent along with the tumescent fluid and aspirated fat during liposuction procedures. However, peak plasma concentrations of lidocaine occur approximately 12 hours after infiltration, regardless of whether aspiration is performed. When higher doses are delivered more rapidly, peak plasma concentrations occur about 6 hours after infiltration.

The safety of high-dose lidocaine delivered in wetting solution is now thought to be due to massive dilution of the anesthetic agent, the relatively slow rate of administration, the vasoconstriction attendant with the addition of epinephrine, and the relatively avascular spaces in which the wetting solution is delivered.

Lidocaine Toxicity

Recognition

Despite the documented safety of the tumescent technique, it is important to recognize the signs of lidocaine toxicity whenever delivering such high doses. At plasma concentrations of 3 to 5 μg/mL, dizziness and lightheadedness are observed, whereas levels of 5 to 9 μg/mL are associated with muscle fasciculations and tinnitus. At levels of 9 to 12 μg/mL, seizure activity is manifested, and levels higher than 20 μg/mL are accompanied by respiratory arrest and cardiovascular collapse. Of note, benzodiazepines have been shown to increase the seizure threshold for lidocaine, and they may contribute in part to the safety record of high-dose lidocaine delivered via the tumescent technique.

Treatment

When lidocaine toxicity does occur, it is critical to act quickly to counteract the resulting CNS and cardiovascular sequelae. Seizure activity is treated initially by hyperventilation with 100% oxygen delivered by Ambu bag. This maneuver circumvents the deterioration in CNS status associated with hypercapnea. If seizure activity continues, diazepam or thiopental can be administered intravenously in doses of 0.1 mg/kg and 2 mg/kg, respectively. Hypotension is treated with aggressive intravenous fluid resuscitation and Trendelenburg positioning. The judicious use of peripheral vasoconstrictors may need to be considered. Although inducing hypertension is certainly not desirable in a patient undergoing surgery in a wide or richly vascularized field (i.e., abdominoplasty or rhytidectomy), maintenance of mean arterial pressure and thus cardiac output is always the first priority. Finally, if arrhythmias develop, resuscitation may be prolonged because the toxicity may not resolve until significant redistribution of the local anesthetic medication can occur.

As noted earlier, the peak plasma concentration of lidocaine delivered via tumescent technique occurs approximately 12 hours after infiltration. This sustained effect completely eliminates the need for longer-acting anesthetic agents such as bupivacaine to provide supplemental, immediate, or postoperative anesthesia. Bupivacaine and the longer-acting local anesthetics typically result in CNS depression and cardiovascular toxicity at significantly lower doses than those used with lidocaine. The associated toxicity makes their use impractical in broad surgical fields, such as those encountered in abdominoplasty or liposuction.

Pearls and Pitfalls

- Limit the variety and types of sedative and analgesic agents to maximize surgeon familiarity and safety.
- Use small, incremental doses of fentanyl and midazolam.
- Clearly chart both incremental and total doses of intraoperative medications in full view in the operating room.
- Be prepared to treat patients progressing into deep sedation.
- Local anesthesia delivered by high-volume, dilute lidocaine solution is the foundation of the technique, *not* opioids or benzodiazepines.
- Do not provide routine supplemental oxygen to patients undergoing conscious sedation.

SUGGESTED READINGS

Gottlieb LJ, Gurley JM, Parsons RW: Patient selection and risk factors. In Achauer BM, Ericksson E, Guyuron B, et al (eds): Plastic Surgery: Indications, Operations, and Outcomes. St Louis, Mosby, 2000.

Hasen KV, Samartzis D, Casas LA, Mustoe TA: An outcome study comparing intravenous sedation with midazolam/fentanyl (conscious sedation) vs. propofol infusion (deep sedation) for aesthetic surgery. Plast Reconstr Surg 112:1683-1689, 2003.

Klein JA: Tumescent technique for regional anesthesia permits lidocaine doses of 35 mg/kg for liposuction. J Dermatol Surg Oncol 16:3, 1990.

Marcus JM, Few JW, Chao JD, et al: The prevention of emesis in plastic surgery: A randomized, prospective study. Plast Reconstr Surg 109:2487-2494, 2002.

Benign Cutaneous Neoplasms

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DINA BEGAN

This chapter discusses common benign cutaneous neoplasms that clinicians are likely to encounter during examination of the skin.

It is important to ensure that the proper diagnosis is made before pursuing therapy or making a decision not to treat cutaneous neoplasms. Many malignant neoplasms can mimic benign growths and vice versa. In addition, many benign neoplasms have malignant potential. Thus, it is of paramount importance for physicians to have a high index of suspicion when examining skin lesions, particularly if the patient gives a history of the lesion growing or changing in any way. Biopsy should be performed in all cases in which malignancy cannot be ruled out by physical examination.

Because most benign cutaneous neoplasms pose no health risk to the patient, they are often left untreated or are removed solely for cosmetic purposes. Noncosmetic indications for treatment include pain, inflammation, infection, functional limitation, or the potential for malignant transformation.

Benign Epidermal Neoplasms

Seborrheic Keratoses

These lesions are benign proliferations of epidermal cells (keratinocytes) and have no malignant potential (Fig. 1). Seborrheic keratoses can be few or numerous and occur mostly on the trunk and head, but they can also occur on the extremities. Patients of Asian or African descent can have many small lesions over the face (*dermatosis papulosa nigra*).

Light application of liquid nitrogen is a viable treatment option for seborrheic keratoses. However, particularly in dark-skinned patients, care must be taken to avoid permanent hypopigmentation. Lesions can be removed under local anesthesia with gentle curettage. If curettage is too aggressive (i.e., into the reticular dermis), scarring can occur. Light electrodessication is the authors' preferred treatment. The lowest possible voltage that causes the lesion to change color to gray, white, or purple should be used. The keratosis can then be gently removed with gauze. When removed in this manner, scarring rarely occurs. If the lesions are small and thin, local anesthesia may not be necessary. If bleeding occurs, electrodessication is stopped and light pressure is applied because continued electrodessication results in scarring.

Actinic Keratoses

An actinic keratosis (AK) is defined histologically as atypia of the epidermis that does not involve the entire thickness of the epidermis. After full-thickness atypia occurs, the lesion is no longer AK but rather squamous cell carcinoma in situ (SCCIS). AKs are therefore precursors of squamous cell carcinoma (SCC). Between 1% and 10% of untreated AKs evolve to become SCCIS or invasive SCC. Therefore, it is advisable to treat these lesions to prevent the development of carcinoma.

In patients with few or discrete lesions, liquid nitrogen is commonly used. It can be applied with a cotton applicator or with a focused spray. It should be applied for 2 to 3 seconds in three successive treatments because cellular destruction is maximized by repeated freeze-thaw cycles. Superficial blistering after treatment is expected. Scarring can occur, usually as hypopigmented, round depressions in the area of treatment. If the lesion does not resolve with treatment or recurs after treatment, biopsy is indicated to rule out SCC.

Many patients with extensive photodamage have diffuse AKs, thus making spot treatment with liquid nitrogen impractical and ineffective. The most widely used treatment for large areas of involvement is topical chemotherapy with 5-fluorouracil (5-FU). Efudex (5-FU 5% cream) is applied twice daily for 2 to 6 weeks, or Carac (5-FU 0.5% cream) is

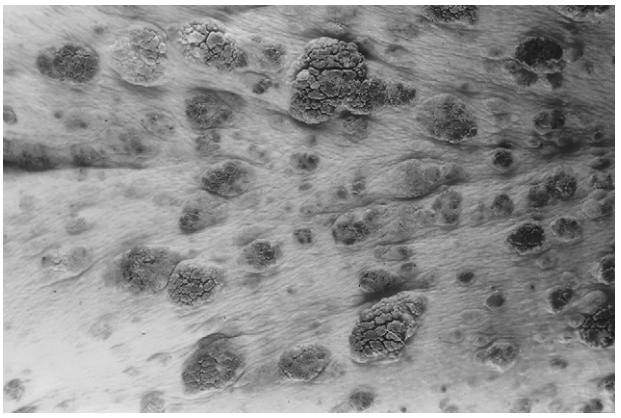

Figure 1 • Seborrheic keratoses. (Courtesy of the Department of Dermatology, New York University School of Medicine.)

applied once daily for 2 to 4 weeks. With any 5-FU preparation, treatment is continued until a plateau in the skin's response is seen.

5-FU preparations cause AKs to become erythematous, and if photodamage is severe, the skin may become diffusely red, crusted, and painful. The inflammation can take 2 to 4 weeks to heal, and the residual erythema may persist for 1 to 4 months. If discomfort is problematic for the patient, hydrocortisone 2.5% ointment can be applied either during or after the course of therapy. Patients must strictly avoid the sun throughout the course of therapy. Most patients tolerate 5-FU, and any photodamage is significantly improved after treatment. Scarring is exceedingly rare and generally occurs only when treatment is complicated by infection. If the patient has a history of herpes simplex, antiviral prophylaxis should be considered at the beginning of treatment.

Imiquimod (Aldara) is a topical immunomodulator that is now FDA-approved for treatment of AKs. It is usually applied three times weekly for 4 to 6 weeks. Diclofenac sodium, 3% gel, is a topical nonsteroidal anti-inflammatory compound also used for treating AKs. It is applied one or two times daily for 2 to 3 months.

Other less commonly used options for the treatment of AKs include curettage, particularly when a biopsy specimen is desired to confirm the diagnosis and rule out SCC; photodynamic therapy, such as the application of topical porphyrins followed by exposure to visible light; topical tretinoin or tazarotene; and chemical peels. In patients with multiple AKs attributed to xeroderma pigmentosum or immunosuppression, long-term oral isotretinoin or acitretin may be beneficial.

Keratoacanthomas

Keratoacanthomas are fast-growing papules with a keratotic or ulcerated center (Fig. 2). They are benign and spontaneously involute over a period of several months. However, they are often impossible to distinguish histologically from SCC on biopsy. Thus, solitary keratoacanthomas are best excised to the level of subcutaneous fat. In this way, invasive SCC can be definitively ruled out. Mohs' micrographic surgery is recommended for solitary keratoacanthomas on the face because some of them prove to be invasive SCC on excision.

In patients with multiple, eruptive, or giant keratoacanthomas, excision is not practical. After clinical and histologic diagnosis is established, the lesions may be treated with intralesional 5-FU, methotrexate, or bleomycin. Topical podophyllin, 5-FU, or tazarotene can be used if large areas are involved. Oral treatment of refractory cases includes retinoids and cyclophosphamide. A patient with keratoacanthomas and sebaceous neoplasms (including sebaceous adenoma/epithelioma/carcinoma or basal cell carcinomas with sebaceous differentiation) must undergo a workup for internal malignancies (Muir-Torre syndrome).

Warts and Condyloma

There are many subtypes of warts, each associated with distinct strains of human papillomavirus (HPV): common warts (verruca vulgaris), flat warts, plantar warts, and genital warts (condyloma). Warts often require prolonged treatment, and many treatment failures are due to discontinuation of treatment or undertreatment. Warts are particularly challenging to treat in immunocompromised patients. Continuous treatment is often required because new lesions constantly form. Treatment of warts is primarily destructive, although imiquimod and other immunomodulators are being used increasingly, often as adjuncts to destructive modalities.

Figure 2 • Keratoacanthomas of the hand. (Courtesy of the Department of Dermatology, New York University School of Medicine.)

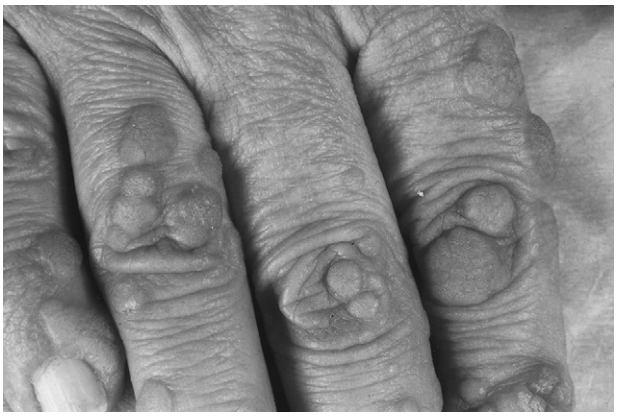

Figure 3 • Verruca vulgaris (common warts) of the fingers. (Courtesy of the Department of Dermatology, New York University School of Medicine.)

COMMON WARTS. First-line treatment of common warts (Fig. 3) is cryotherapy with liquid nitrogen. Before freezing, the wart should be pared as much as possible to permit deeper freezing. Adequate treatment is indicated by blister formation within 24 to 48 hours. Freezing time depends on the size of the wart and may vary from 5 to 30 seconds. It should take approximately 30 to 40 seconds for the wart to thaw after an adequate freeze, and two freeze cycles should be performed in a treatment session. An area of 2 to 3 mm around the wart should be included in the treatment. Treatments should be repeated every 2 weeks because prolonged periods between treatments allow regrowth of the wart. Hypopigmentation is common, but scarring is rare if the lesions are located on the hands or feet.

PERIUNGUAL WARTS. These lesions present an especially difficult problem because they involve the nail bed, which is protected by the nail plate. A flexor sheath nerve block is generally needed to administer adequate freeze. Whitening of the nail bed adjacent to the lesion must be seen during freezing. If the lesion fails 2 to 3 months of biweekly liquid nitrogen treatment, removal of the nail plate followed by aggressive curettage and desiccation may be required, but such treatment can lead to permanent nail dystrophy as a result of scarring of the nail matrix.

FLAT WARTS. Flat warts are more responsive to treatment than common warts and often spontaneously regress. Aggressive therapy (desiccation and curettage, intralesional chemotherapy) is generally avoided. Light cryotherapy (3- to 5-second application to each lesion) and 40% salicylic acid are satisfactory options for limited warts. For more extensive lesions, tretinoin (Retin-A, 0.05% or 0.1% cream) is effective. 5-FU (Efudex, 5% cream) applied daily or pulsed dye laser therapy can be used in refractory cases.

PLANTAR WARTS. Plantar warts are more refractory to therapy than common warts and aggressive desiccation and curettage can lead to painful scarring on the sole of the foot or palm. Therefore, a combination of liquid nitrogen cryotherapy, as described earlier for common warts, and home therapy between cryotherapy treatments is the best primary treatment. Home therapy consists of applying a 40% salicylic acid plaster daily after the cryotherapy blister has resolved. Each day when the pad is removed, the area should be soaked in water for 5 to 10 minutes. The keratotic, white material should be removed by the patient with a metal file or pumice stone. The patient should not use the stone or file elsewhere on the body because it may be contaminated with the wart virus. If combination therapy fails to work within 2 to 3 months, pulsed dye laser therapy may be considered.

GENITAL WARTS (CONDYLOMA). It is well known that genital warts or condyloma (Fig. 4) can lead to SCC of the cervix or rectum. Thus, Papanicolaou (Pap) smear screening is essential for screening. It is important to note that treatment of external condyloma has not been shown to decrease the risk of HPV transmission to sexual partners, nor does it decrease the risk of cancerous transformation. It is imperative that all female partners of men with condyloma and women with external condyloma be counseled to undergo Pap smears every 6 to 12 months to ensure early detection of cervical dysplasia. Condom use is advised but may not effectively protect partners from HPV transmission. Men with anal condyloma, particularly those who are human immunodeficiency virus positive, have a risk of rectal carcinoma similar to the risk of cervical dysplasia in women with HPV. Therefore, anal Pap smears and anoscopy are recommended every 6 to 12 months.

Trichloroacetic acid (TCA) in strengths of 25% to 50% followed immediately by the application of 25% to 50% podophyllin every 1 to 2 weeks is usually effective. Care must be taken to apply the medication only to the wart because application to normal skin results in ulceration. Precise application can be achieved by using thin calcium alginate swabs as applicators. The podophyllin must be washed off in 6 to 8 hours to avoid ulceration. TCA/podophyllin therapy, if used properly, rarely results in scarring. Because podophyllin cannot be used in pregnant women, it is advisable to avoid its use in sexually active women with reproductive potential.

Liquid nitrogen cryotherapy is a treatment option for light-skinned patients, in whom the resulting hypopigmentation is not noticeable. Clearance may be faster with cryotherapy than with TCA/podophyllin. Lesions are treated with approximately two applications of liquid nitrogen every 1 to 2 weeks. A 2-mm zone around the wart should be included.

Imiquimod can be a useful adjunct to the aforementioned therapies. It can also be used as

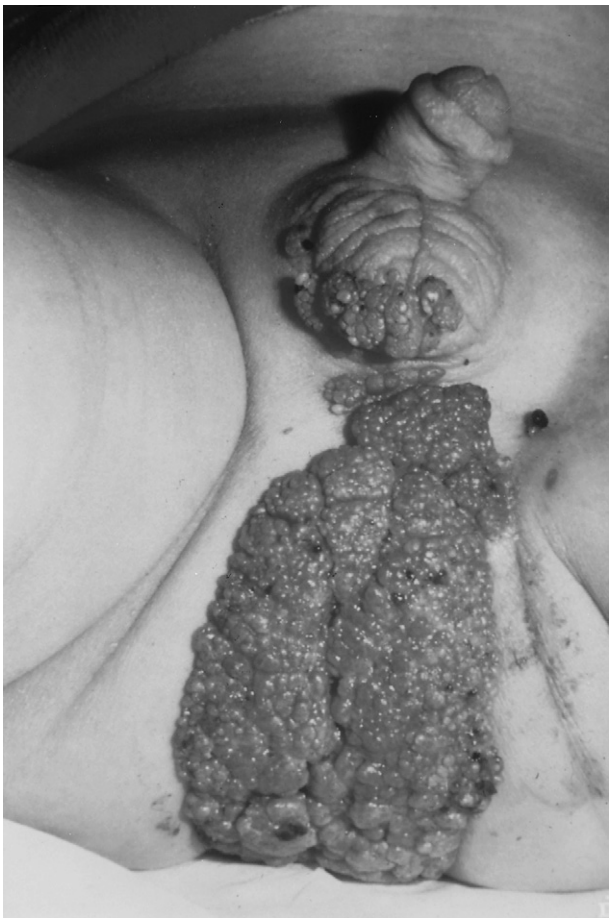

Figure 4 • Condyloma (genital warts) of the perineal-scrotal region. (Courtesy of the Department of Dermatology, New York University School of Medicine.)

monotherapy, but prolonged treatment of 3 or more months is often required. The main advantage of imiquimod is that it affords a private treatment option for the patient and minimizes office visits. However, its expense is prohibitive for many patients. For refractory condyloma or extensive lesions, desiccation and curettage or pulsed dye laser therapy is an option.

BOWENOID PAPULOSIS. This lesion arises in genital warts caused by HPV-16. It is histologically similar to SCCIS, although atypical keratinocytes are more scattered throughout the epidermis rather than confluent as in Bowen's disease/SCCIS. Bowenoid papulosis can resolve spontaneously or progress over time to SCCIS or invasive SCC. Conservative treatment with topical 5-FU is usually sufficient. If recurrent, excision or desiccation and curettage may be considered.

GIANT CONDYLOMA ACUMINATUM (BUSCHKE-LÖWENSTEIN TUMOR). This aggressive wart-like growth is a malignant verrucous carcinoma caused by HPV-6. It is treated by complete excision.

TREATMENT OF REFRACTORY WARTS. Pulsed dye laser therapy can be useful in refractory warts; however, multiple treatments (5 to 20) are often required. Intralesional bleomycin is another option for refractory warts. The treatment is administered every 3 to 4 weeks, and the warts usually clear in two to three treatments. Varying doses and administration techniques are reported in the literature with cure rates as high as 90%. A black eschar may occur after treatment. Care should be taken when treating distal digital warts with bleomycin because nail dystrophy, Raynaud's phenomenon, and necrosis of the digit have been reported.

Other therapies include cantharone or 40% salicylic acid, both of which can be applied daily by the patient. Imiquimod applied three times a week can be a useful adjuvant therapy that the patient can apply at home. Oral cimetidine (30 to 40 mg/kg/day), acitretin, isotretinoin, levamisole, topical cidofovir, and intralesional mumps or *Candida* antigen may also be useful treatments in selected cases.

Benign Dermal Neoplasms

Acrochordons (Skin Tags, Fibroepithelial Polyps, Soft Fibromas)

These benign lesions appear as soft flesh-colored to brown pedunculated papules, usually on the neck, trunk, or axillae. They occur particularly in areas of friction and are seen commonly in women of African or Mediterranean descent and in the obese. Acrochordons may be single or multiple and can vary from 1 mm to a few centimeters in diameter. They can have a smooth or verrucous surface. Acrochordons can become inflamed and painful as a result of trauma or thrombosis. On histologic examination they contain collagen fibers, fat, and blood vessels.

If the stalk of the lesion is thin, the tags can simply be clipped off or lightly electrodesiccated without anesthesia. Larger lesions require local anesthesia. If scissors are used, hemostasis is easily achieved with the application of aluminum chloride. Patients should be aware that new lesions are likely to develop over time.

Lipomas

Lipomas are benign fatty tumors. They can be easily diagnosed by their characteristic soft texture on examination. They may grow to large size (several centimeters), and direct excision is the treatment of choice. The entire lesion must removed or the lipoma can recur. They should be sent for pathologic examination to distinguish them from the rare liposarcoma.

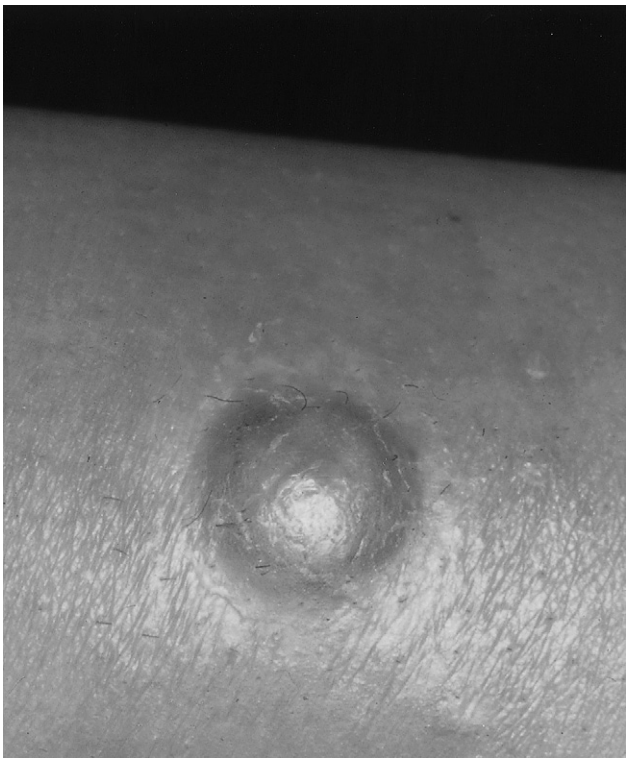

Figure 5 • Dermatofibroma (sclerosing hemangioma). (Courtesy of the Department of Dermatology, New York University School of Medicine.)

Dermatofibromas

Dermatofibromas are benign lesions that have no malignant potential and rarely pose any inconvenience or cosmetic concern to the patient (Fig. 5). Therefore, they are usually left untreated. Dermatofibromas appear as well-circumscribed round or oval papules or nodules (5 to 20 mm in diameter), red-brown in color. When the adjacent skin is pinched between the fingers, they have the tendency to recede into the skin (*the dimple sign*), which is helpful in making the diagnosis. If a dermatofibroma is growing gradually and becomes larger than 2 cm, excisional biopsy is recommended to rule out dermatofibrosarcoma protuberans or malignant fibrous histiocytoma.

Sebaceous Hyperplasia

Sebaceous hyperplasia is a benign tumor composed of enlarged sebaceous glands. It begins as a pale yellow, slightly elevated papule; with time it becomes yellow, dome shaped, and umbilicated. It may be mistaken for basal cell carcinoma. Biopsy is indicated if the diagnosis is in question. Treatment is by electrodessication, excision, or carbon dioxide or excimer laser. Oral isotretinoin may induce resolution of lesions, but they recur with cessation of therapy.

Angiofibromas

An angiofibroma most commonly manifests as a dome-shaped, flesh-colored papule (5 mm or less in diameter) on the nose or adjacent skin in a middle-aged person. The lesion may be shaved flat under local anesthesia. It is prudent to send the tissue for histologic examination to rule out early basal cell carcinoma.

Multiple angiofibromas on the face of a child are referred to as adenoma sebaceum, an unusual condition seen in tuberous sclerosis and multiple endocrine neoplasia.

Neurofibromas

Solitary neurofibromas (Fig. 6) are common and not associated with neurofibromatosis 1 (NF1). They appear as soft flesh-colored or pink nodules (2 to 20 mm in diameter). They can often be pressed into the skin through an underlying invagination—so-called buttonholing. The presence of two or more neurofibromas fulfills one of the two necessary criteria for the diagnosis of NF1.

The presence of any one of the following, in addition to two or more neurofibromas, establishes the diagnosis of NF1: six or more café au lait macules, optic gliomas, Lisch nodules, axillary or inguinal freckling, a characteristic bone lesion, or a first-degree relative with neurofibromatosis. An

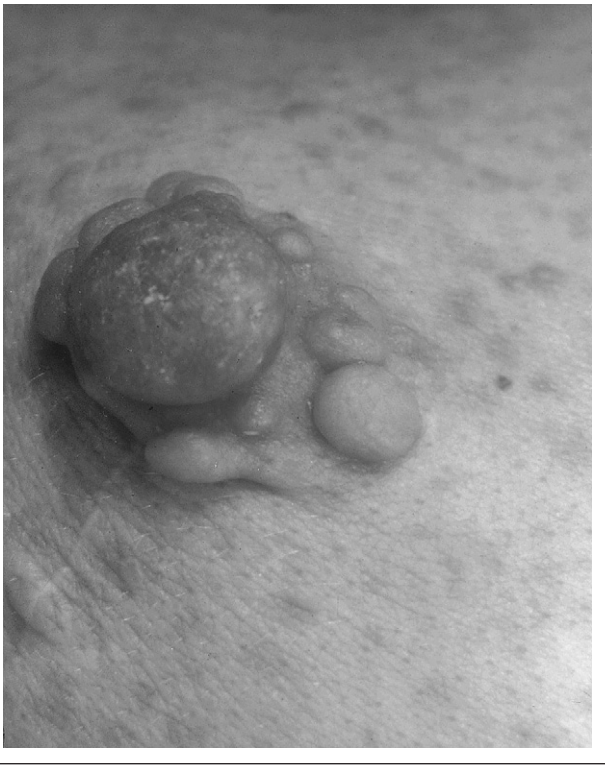

Figure 6 • Neurofibroma. (Courtesy of the Department of Dermatology, New York University School of Medicine.)

exception is the presence of a plexiform neurofibroma, which is a flesh-colored soft nodule, usually 1 cm or larger, that on palpation has a characteristic texture referred to as a "bag of worms." The presence of this lesion alone is sufficient to make the diagnosis of NF1.

Another exceptional entity is known as linear or segmental neurofibromatosis. This condition is a mosaic form of NF1 in which multiple neurofibromas are present in a localized area, often along Blaschko's developmental lines of the skin. Patients with segmental neurofibromatosis should be counseled that they may be carriers for NF1 if the mosaic mutation has affected the patient's germ cells.

Treatment of neurofibroma is not generally necessary for solitary lesions and impractical for patients with neurofibromatosis who have multiple lesions. Conservative surgical excision is the only treatment available.

Xanthelasma

Xanthelasma is a benign deposition of lipid in the superficial dermis, usually in the skin around the eyes. It can be associated with familial hypercholesterolemia, but most patients have normal cholesterol levels. It can be associated with an increased risk for atherosclerotic disease. Treatment is either direct excision or electrodessication. Carbon dioxide or excimer laser ablation may also be effective.

Benign Melanocytic Neoplasms

Melanocytic Nevi (Moles)

There are various clinical subtypes of melanocytic nevi that correspond to unique histopathologic patterns: junctional, compound, intradermal, congenital, blue, and halo nevi. These lesions are benign and have no malignant potential. They are removed when a malignant diagnosis is in question or when indicated for cosmesis. If complete removal is desired, surgical excision is usually performed. For small junctional and compound nevi, the cosmetic outcome is sometimes superior with a deep shave biopsy, depending on the location of the lesion. This technique is not advisable for intradermal, congenital, or blue nevi because they often penetrate deep into the dermis and recur after superficial removal. A punch biopsy to the level of fat with placement of superficial sutures is also an option for removal of small nevi. However, care must be taken to remove the entire lesion to avoid recurrence.

Dysplastic (Atypical) Nevi

Clinical differentiation between nondysplastic nevi, dysplastic nevi, and melanoma can be challenging. Dermoscopy (magnification of the lesion by the application of a dermatoscope to the skin surface) in the hands of an experienced physician can aid in distinguishing these lesions. Histologic examination of the lesion can generally establish the diagnosis.

Dysplastic nevi are those with cytologic or architectural atypia, or both, on histologic examination. Although they are benign, atypical or dysplastic lesions may carry a risk of transformation to malignant melanoma. The precise risk is not known but is thought to be proportional to the degree of atypia. Atypia is graded as mild, moderate, or severe by most dermatopathologists. However, the gradations extend along a continuum, and precise criteria have not been established for grading. It is important for the clinician to have familiarity with the grading system used by the pathology laboratory and to use this knowledge to influence subsequent treatment decisions. Many practitioners perform conservative excision when a biopsy report indicates the presence of moderate or severe atypia.

Patients with even a single dysplastic nevus have an increased risk for melanoma during their lifetime and should therefore be monitored closely with total body skin examination every 6 months. A melanoma may arise within the dysplastic nevus or may occur de novo. Patients with dysplastic nevi who have two or more relatives with melanoma have close to a 100% lifetime risk for melanoma. Thus, it is important to establish an accurate family history whenever possible. In the absence of a positive family history, the lifetime risk for melanoma in a patient with dysplastic nevi is increased 3- to 40-fold. The greater the number of dysplastic nevi present (by clinical or histologic diagnosis), the greater the risk. Patients with multiple nevi that are clinically dysplastic should be examined every 3 to 6 months or, if available, examined at a multidisciplinary pigmented lesion center where patients are monitored with the aid of dermoscopy and total body photographs.

Lentigines

Lentigines are benign proliferations of melanocytes along the basal layer of the epidermis. Unlike nevi, the melanocytes do not form nests. Unlike melanoma in situ and lentigo maligna, the melanocytes are not atypical, are symmetric in proliferation, are not confluent along the basal layer, and do not rise above the basal layer.

Lentigines are divided into lentigo simplex and solar lentigines. Most lentigo simplex lesions appear in childhood, but new lesions continue to develop in many patients throughout life. They can occur anywhere on the body. Lentigo simplex is clinically indistinguishable from a junctional nevus. Solar lentigines (also known as age spots or liver spots) accumulate over time in areas of sun exposure, particularly the face and dorsal surface of the hands. It can be difficult to distinguish a large irregular solar lentigo from lentigo maligna clinically. Therefore, it

is important to establish a firm diagnosis either clinically or histologically before treating a lentigo.

There is no indication for the treatment of lentigines other than cosmesis. Liquid nitrogen cryotherapy is usually sufficient treatment, although hypopigmentation is common. For darker lesions, more than one treatment may be required, and the risk of hypopigmentation is increased. Q-switched lasers, particularly ruby (694 nm) and Nd:YAG (532 nm), are effective in removing lentigines. The risk of scarring and hypopigmentation is minimal in light-skinned patients but common in patients with darker skin types. Intense pulsed light is also effective, but generally less so than ruby or Nd:YAG laser therapy.

Benign Vascular Neoplasms

Cherry Hemangiomas

Cherry hemangiomas are bright red macules or papules 1 to several mm in diameter. They occur over the trunk and upper extremities in adults and tend to increase in number with age. Cherry hemangiomas have no malignant potential and rarely become inflamed. They are collections of dilated capillaries in the upper dermis. The sole reason for removal is cosmetic. For small lesions, light electrodessication with the minimum voltage required can be used without anesthesia. For larger lesions, pulsed dye laser therapy is effective and has a low risk of scarring.

Pyogenic Granulomas

Pyogenic granulomas are rapidly growing bright red nodules that can achieve a size of 1 to 2 cm in diameter (Fig. 7). They occur most commonly on the fingers, lips, and gingiva. They often arise after cutaneous trauma or infection and are probably reactive capillary proliferations rather than true neoplasms. Pyogenic granulomas often ulcerate, are friable, and bleed easily. They can simulate angiosarcoma, Kaposi's sarcoma, and bacillary angiomatosis and should be biopsied. The optimal treatment is shave excision with electrodessication of the base, but local anesthesia is required. During excision, it may be evident that part of the tumor extends deeper into the dermis. This portion can be removed with blunt dissection before electrodesiccation; however, scarring is common with deep dissection and desiccation. Recurrence is common, but most lesions will resolve spontaneously with time.

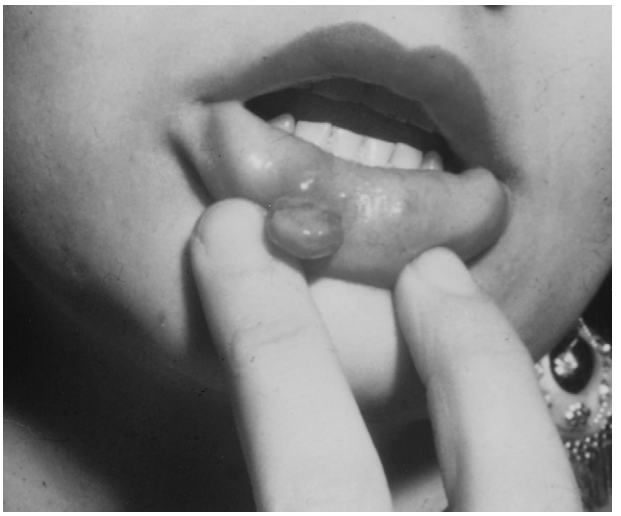

Figure 7 • Pyogenic granulomas. (Courtesy of the Department of Dermatology, New York University School of Medicine.)

Cysts

True cysts are spherical proliferations of epithelial cells without malignant potential. Pseudocysts are spherical cellular proliferations in which the cyst wall has no epithelial layer. The most common cysts in the skin are epidermal (infundibular) cysts, pilar (trichilemmal) cysts, milia, and steatocystomas. They are true cysts because their walls are composed of epithelial cells that simulate and may arise from different parts of the pilosebaceous unit.

Epidermal and pilar cysts are clinically indistinguishable and appear as subcutaneous nodules 1 to several cm in diameter. However, in contrast to epidermal cysts, pilar cysts are less common, tend to be multiple, and usually occur on the scalp. Steatocystomas appear as yellow nodules 2 to 6 mm in diameter and are primarily located on the upper part of the body and thighs. They can occur as single lesions. If multiple, steatocystomas are generally inherited in an autosomal dominant pattern (although sporadic cases exist), and the condition is referred to as steatocystoma multiplex. Milia are much smaller than other cysts. They appear as firm, white, 1- to 2-mm papules. Milia can occur spontaneously, after cutaneous trauma, or with deep blistering diseases such as porphyria cutanea tarda and bullous pemphigoid.

Inflammation of epidermal and pilar cysts is common. The lesions become very tender and may spontaneously open and drain. It is debatable what proportion of inflamed cysts are actually infected and whether antibiotics are necessary. If antibiotics are given, the drainage fluid should first be cultured. Most cultures yield *Staphylococcus epidermidis* or a combination of staphylococcal and streptococcal bacteria. Cephalexin or another first-generation cephalosporin, administered for 5 to 7 days, is the first-line therapy. If fluctuance is present, it is advisable to incise and drain the cyst because such treat-

ment greatly alleviates symptoms and accelerates healing regardless of whether an infection is present. If no area of fluctuance is observed, antibiotics are not generally required, and intralesional triamcinolone can be used to accelerate resolution of the inflammation. After the inflammation has subsided in 4 to 6 weeks, the cyst may be excised because repeated episodes of inflammation can occur.

Definitive treatment of cysts is excision. It is important to remove the entire cyst wall to prevent recurrence. In the case of steatocystoma multiplex, small incisions (5 mm or less) are often sufficient to remove the cysts. If the lesions are numerous, they may be incised and drained for amelioration; however, they do recur. Milia require only a very small incision in the epidermis followed by gentle lateral pressure to induce extrusion; sutures are not required.

Pearls and Pitfalls

- Suspicious lesions should be biopsied.
- Removed specimens should be submitted for pathologic examination.
- Ignoring aesthetic concerns in the removal of a benign skin lesion can result in a dissatisfied patient.

SUGGESTED READINGS

Callen J, Bickers D, Moy R: Actinic keratoses. J Am Acad Dermatol 36:650, 1997.

Gibbs S, Harvey I, Sterling J, Stark R: Local treatments for cutaneous warts: A systematic review. BMJ 325:461, 2002.

Hashimoto K, Mehregan A: Tumors of the Epidermis. Boston, Butterworth, 1990.

Morganroth G, Leffell D: Nonexcisional treatment of benign and premalignant cutaneous lesions. Clin Plast Surg 20:91, 1993.

Odom R, James W, Berger T: Andrews' Diseases of the Skin, 9th ed. Philadelphia, WB Saunders, 2000, pp 733-892.

Seykora J, Elder D: Dysplastic nevi and other risk markers for melanoma. Semin Oncol 23:682, 1996.

Malignant Cutaneous Neoplasms

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Malignant Melanoma

The incidence of cutaneous malignant melanoma has risen substantially over the last century. Despite accounting for only 5% of all skin cancers diagnosed annually, more than 75% of all skin cancer deaths are due to melanoma. Increased recognition of early lesions has resulted in diagnosis of the primary tumor while still confined to the skin in the majority (82%) of patients. Early diagnosis correlates with increased survival. The 5-year survival rate exceeds 90% in patients with localized disease but decreases sharply to 60% and 5% in patients with regional lymph node involvement and distant metastases, respectively.

Clinical Characteristics and Risk Factors

Melanoma arises most commonly on the skin of the back in men and on the lower extremities in women. In darker skinned ethnic groups (African American, Asian, and Hispanic), melanoma frequently arises in the volar and plantar skin (acral) or in the nail bed (subungual). Ninety-five percent of melanomas arise in the skin, but they may also occur in the eye and mucous membranes, including the vagina and anus. Three percent to 5% of patients have metastatic disease at initial evaluation in the absence of a primary lesion.

The typical patient with melanoma has a fair complexion that tans poorly and burns easily with brief sun exposure. Blistering sunburns sustained as a child are a significant risk factor, as is the development of atypical (dysplastic) nevi. Patients with more than 100 dysplastic nevi (dysplastic nevus syndrome) have a 10% to 15% incidence of melanoma in their lifetime, and the presence of even a single atypical mole increases the risk significantly. Children with large congenital nevi are also at increased risk for melanoma, although it appears to occur uncommonly (10% to 15%) and predominantly in the first decade of life in patients with truncal lesions. Any patient in whom melanoma has previously been diagnosed has a 3% to 6% lifetime risk for a second primary melanoma. In addition, there is a 3% to 10% incidence of melanoma in the first-degree relatives of affected patients. Therefore, lifelong cutaneous surveillance of all melanoma patients, as well as their immediate family members, is essential.

Histopathology

Cutaneous melanoma arises from the melanocyte, a pigment-producing cell of neural crest derivation most often located at the junction between the epidermis and dermis. Tumor thickness, or the distance from the epidermal-dermal junction to the lowest melanoma cell, is a measure of the vertical growth phase of melanoma and a powerful indicator of the potential for local recurrence, metastasis, and death. Accurate assessment of tumor thickness guides the scope of the appropriate metastatic survey, surgery, adjuvant treatment, and follow-up schedule. Thickness is assessed in millimeters (Breslow) or by levels of dermal penetration (Clark).

Melanoma is staged with the TNM system, revised in 2002, (Table 1), which stratifies groups of patients by delineation of the extent of lymph node metastases by recording microscopic nodal involvement of the sentinel node, as well as the number of nodes affected. Important prognostic features, such as the presence of ulceration of the primary lesion, the preoperative lactate dehydrogenase (LDH) level, and the anatomic site of distant metastatic disease, are also included in this staging system.

TABLE 1 Melanoma Staging

STAGE	CRITERIA	TNM
0	Melanoma in situ	Tis N0 M0
IA	Breslow ≤1.0 mm or Clark level II/III, no ulceration	T1a N0 M0
IB	Breslow ≤1.0 mm or Clark level IV/V with ulceration	T1b N0 M0
	Breslow 1.01–2.0 mm, no ulceration	T2a N0 M0
IIA	Breslow 1.01–2.0 mm with ulceration	T2b N0 M0
	Breslow 2.01–4.0mm, no ulceration	T3a N0 M0
IIB	Breslow 2.01–4.0 mm with ulceration	T3b N0M0
	Breslow ≥4.0 mm, no ulceration	T4a N0 M0
IIC	Breslow ≥4.0 mm with ulceration	T4b N0 M0
III	Clinically apparent, satellite, or in-transit nodal metastases*	Any T, N1–3, M0
IV	Distant metastases†	Any T, any N, M1

*N1, clinically occult/apparent metastases; N2, metastases in two to three regional nodes or intralymphatic regional metastases, without nodal metastases; N3, metastases in four or more regional nodes, matted metastatic nodes, in-transit metastasis, or a satellite or satellites with metastasis in a regional node or nodes.

†M1a, metastasis to skin, subcutaneous tissues, or distant lymph nodes; M1b, metastasis to lung; M1c, metastasis to all other visceral sites or distant metastasis at any site associated with elevated serum lactate dehydrogenase.

From American Joint Committee on Cancer: AJCC Staging Manual, 6th ed. Philadelphia, JB Lippincott, 2002.

Clinical Diagnosis

Physical examination begins with inspection of the entire cutaneous surface, including the volar and plantar skin, web spaces, and mucous membranes. The ABCD rule aids in the identification of lesions most likely to be melanoma. Lesions that are *A*symmetric with irregular *B*orders, *C*olor variation, and *D*iameters exceeding 6 mm are considered suspicious. In addition, any pigmented lesion that becomes darker or lighter in color, increases in size, or becomes raised, itches, or bleeds should immediately arouse suspicion.

Complete surgical excision is the biopsy method of choice for suspicious lesions. This technique, which can be performed rapidly in the office setting, enables an accurate assessment of tumor thickness should the lesion indeed prove to be melanoma. Punch or shave biopsies are often performed but may be inaccurate because tumor thickness may be underestimated. Incisional biopsy of unusually large or broad lesions is acceptable only if a diagnosis of melanoma is confirmed and tumor thickness is assessed accurately. For extremity lesions, surgical biopsy incisions should always be oriented longitudinally so that they do not interfere with or complicate subsequent definitive excision and reconstruction.

Metastatic Survey

The preoperative metastatic workup is determined by the thickness of the primary melanoma and the disease stage at initial evaluation (Table 2). No metastatic workup is required for patients with malignant melanoma in situ. Although the risk of distant metastases is minimal (<3%), patients with thin melanomas (<1 mm) should, nevertheless, undergo chest radiographs and routine blood analysis (including LDH). Patients with intermediate-thickness lesions (1 to 4 mm) and thick lesions (>4 mm) undergo chest radiographs; routine blood analysis; computed tomography (CT) of the chest, abdomen, and pelvis; and CT or magnetic resonance imaging (MRI) of the brain. Fludeoxyglucose positron emission tomography (FDG-PET) is useful in further characterizing indeterminate radiologic findings and may have a role in the staging of high-risk lesions.

Surgical Excision Margins

Malignant melanoma in situ is excised with 5-mm margins (Table 3). Thin (<1 mm) melanomas have an extremely low rate of local recurrence (<2%) and are excised with 1-cm margins. For intermediate-thickness (1 to 4 mm) lesions, the safety of 2-cm excision margins has been confirmed in prospectively randomized studies. For thick melanomas (>4 mm), the guidelines are less certain because no prospectively randomized study has specifically addressed the issue of excision margins for these lesions. Although 2-cm margins may be appropriate for these lesions based on currently available data, in selected circumstances (very thick lesions or the presence of multiple satellite metastases), many surgeons advocate wider excision margins of 3 cm. However, it is unlikely that margins exceeding 2 cm have a significant impact on the higher rate of local recurrence (12%) and poor survival (55% at 5 years) with which these lesions are associated.

Treatment of Regional Lymph Nodes

CLINICALLY NEGATIVE REGIONAL LYMPH NODES. The majority of patients with newly diagnosed melanoma exhibit no evidence of regional lymph node metastases. The potential for regional lymph node metastasis is most accurately assessed by tumor thickness. Malignant melanoma in situ has no significant potential for lymph node metastasis. For thin melanomas (<0.8 mm), the risk of regional lymph node metastasis is small (<5%); therefore, treatment of regional lymph nodes is not indicated.

Patients with intermediate-thickness lesions (0.8 to 3.9 mm) have a 20% to 25% incidence of micro-

TABLE 2 Surgical Treatment of Primary Cutaneous Malignant Melanoma (Clinically Negative Regional Lymph Nodes*)

MELANOMA THICKNESS	PREOPERATIVE WORKUP†	EXCISION MARGINS	RX—REGIONAL NODES
In situ	Not needed	5 mm	
<0.8 mm	CXR, LDH	1 cm	
0.8–3.9 mm	CXR, LDH CT (chest, abdomen, pelvis) CT/MRI (brain) FDG-PET‡	2 cm	Sentinel lymphadenectomy§
≥4 mm	CXR, LDH CT (chest, abdomen, pelvis) CT/MRI (brain) FDG-PET‡	≥2 cm¶	Sentinel lymphadenectomy§

*Patients with biopsy-proven (fine-needle aspiration) regional lymph node metastases (American Joint Commission on Cancer [AJCC] stage III) undergo formal (complete) lymph node dissection at the time of wide and deep excision of the primary lesion in the absence of distant metastases. Patients with biopsy-proven distant metastases (AJCC stage IV) undergo wide and deep excision of the primary lesion without sentinel lymphadenectomy. Patients with palpable regional lymph node metastases and distant metastases are candidates for palliative formal lymph node dissection only in the setting of *minimal* stage IV disease.

†Computed tomography (CT) is performed with intravenous contrast in patients with melanoma, magnetic resonance imaging (MRI) is more sensitive in the detection of central nervous system metastases, and MRI or fludeoxyglucose positron emission tomography (FDG-PET), or both, may be useful in cases in which CT findings are indeterminate.

‡FDG-PET may be indicated to assess patients with indeterminate radiographic findings or those at high risk for metastatic disease.

§Intraoperative mapping and sentinel lymphadenectomy are performed in the absence of confirmed palpable lymph node metastases or evidence of distant disease. Formal (complete) lymph node dissection is performed immediately if intraoperative microscopic analysis (touch preparation, frozen section, rapid immunostain) of the sentinel lymph node or nodes reveals evidence of metastatic melanoma. If sentinel node metastases are confirmed later on final pathology, formal (complete) lymphadenectomy is performed as a second procedure.

¶*Minimal* excision margins of 2 cm advised for melanomas 4 mm or larger in thickness. Wider (3 cm) margins are often used for these thick lesions, although no prospectively randomized data support this practice.

CXR, chest x-ray; LDH, lactate dehydrogenase; RX, treatment.

scopic regional disease and a 3% to 5% risk of distant metastasis. Sentinel lymphadenectomy serves to identify patients at high risk for systemic disease who may be candidates for adjuvant treatment.

SENTINEL LYMPH NODE BIOPSY. Cutaneous lymphoscintigraphy allows precise delineation of the primary lymphatic drainage of cutaneous melanomas and identification of a lymph node (or nodes) in the regional lymph node basin, termed the *sentinel node*, most likely to contain micrometastases. Intraoperative lymphatic mapping using a combination of intradermally injected vital blue dye and radiolabeled technetium sulfur colloid enables selective identification and excision of the sentinel lymph nodes. Rapid histopathologic staging is performed intraoperatively by microscopic examination of the sentinel node. If no microscopic evidence of metastatic melanoma is noted on histopathologic examination of the sentinel node, no other lymph nodes need to be excised because the incidence of so-called skip metastases to any other node in that particular regional drainage basin is less than 5%. Immediate therapeutic dissection of the regional nodes is performed if metastases are found in the sentinel lymph node because other nodes in the regional drainage basin may also harbor metastases.

In patients with melanomas 1 mm or thicker and no clinical evidence of regional lymph node metastases, lymphoscintigraphy consisting of dynamic images and spot views is performed preoperatively to define the lymphatic drainage and demonstrate the location of the sentinel lymph node or nodes. Sentinel lymphadenectomy is most accurately performed at the time of wide and deep excision of the primary melanoma. Consequently, patients who have already undergone definitive wide and deep excision of the primary lesion may not be ideal candidates for sentinel lymphadenectomy because lymphatic flow and drainage patterns may have been altered by the previous surgery.

TABLE 3 Recommended Melanoma Excision Margins

LESION THICKNESS	EXCISION MARGIN WIDTH
Lesion of uncertain diagnosis	5 mm
Melanoma in situ	5 mm
Melanoma <0.8 mm	1 cm
Melanoma ≥0.8 mm to <4.0 mm	2 cm
Melanoma ≥4.0 mm	3 cm*
Locally recurrent melanoma	1 cm†

*The available data do not support excision margins larger than 2 cm for thick melanomas.

†Authors' recommendation.

Several hours before surgery, the site of the primary lesion is injected with technetium Tc 99m filtered sulfur colloid in four divided doses. After induction of anesthesia, 1 mL of vital blue dye is injected intradermally at the site of the melanoma to stain the afferent lymphatics and sentinel node blue. The site is massaged manually for 20 minutes before incising the skin overlying the sentinel lymph node identified preoperatively by lymphoscintigraphy and confirmed intraoperatively with a handheld gamma detector. The sentinel lymph node is identified by the appearance of blue stain in the node or in the afferent lymphatics (or in both locations) and by detecting an enhanced gamma signal. After the sentinel lymph node has been removed from the field, no other evidence of blue dye should be observed, nor should there be any significant residual radioactivity (exceeding background) detected with the gamma probe. The sentinel node is submitted for rapid microscopic examination, which may include touch preparation, frozen section analysis, or rapid immunostaining (or any combination of the three). If no definitive evidence of metastatic melanoma is noted, wide and deep excision of the lesion is performed and the procedure is terminated. If micrometastases are confirmed in the sentinel lymph node, formal (therapeutic) lymphadenectomy is performed at that time.

The sentinel lymph node is examined in detail postoperatively. Patients with confirmed micrometastases in the final analysis routinely undergo complete regional lymph node dissection as a second procedure.

Patients with thick primary melanomas (>4 mm) in the absence of clinical regional lymph node metastases or radiographic evidence of distant disease are still at high (50% to 75%) risk of having microscopic nodal metastases and are also appropriate candidates for intraoperative lymphatic mapping and sentinel lymphadenectomy.

CLINICALLY POSITIVE REGIONAL LYMPH NODES. Any palpable lymph node in a patient with melanoma should be considered indicative of metastasis until proved otherwise. Fine-needle aspiration (FNA) biopsy is an accurate, reliable method of confirming metastatic melanoma. If FNA is not readily available or if the results are indeterminate, excisional biopsy is performed. Patients with regional lymph node metastases are at high risk for distant metastases and should therefore undergo imaging, including CT of the chest, abdomen, and pelvis and CT or MRI of the brain. FDG-PET is also recommended for further evaluation of patients with advanced melanoma. In patients with cytologically or histologically proven regional nodal metastases, complete lymph node dissection is performed. The development of palpable lymph node metastases is significantly correlated with substantially diminished survival (10% to 50%), which is strongly influenced by the number of and extent to which the lymph nodes are involved, as well the thickness of the primary melanoma.

Regional lymph node dissection should not be performed routinely in patients with documented distant metastases that are extensive or in patients with large fixed lymph nodes. Significant palliation of inoperable bulky or bleeding regional nodal metastases may be achieved with radiation therapy.

Postoperative Follow-up

For patients undergoing wide excision of malignant melanoma in situ, routine dermatologic surveillance is sufficient. For patients with thin melanomas (<0.8 mm), inspection of the total cutaneous surface as well as the surgical excision site and regional lymph nodes is performed at 3- to 6-month intervals for 2 years and at 6- to 12-month intervals thereafter. Chest x-ray and routine blood analysis are performed on a yearly basis. Patients with intermediate-thickness lesions (0.8 to <4.0 mm) should have the same follow-up protocol at 3-month intervals for 2 years and at 6-month intervals thereafter. A chest x-ray and LDH assay are performed each year, as are CT scans for 2 to 5 years after surgery. FDG-PET scanning is another modality that may have a role in screening high-risk patients for metastatic disease. Systemic disease ultimately develops in the majority of patients with thick primary lesions (>4 mm), as well as those with lymph node metastases. In these high-risk patients, follow-up physical examination is performed at 3- to 6-month intervals for life, along with CT, MRI, or PET scanning (or any combination of these imaging modalities) on at least a yearly basis.

The risk for development of a second primary melanoma necessitates that *all* patients, irrespective of lesion thickness, continue lifelong follow-up with total cutaneous examination.

Treatment of Locally Recurrent Malignant Melanoma

Local recurrence after adequate surgical excision in a patient with malignant melanoma is almost always associated with the development of systemic metastases. Survival is extremely poor, with rates averaging less than 5% at 10 years. Primary melanoma thickness remains the most significant prognostic indicator of local recurrence and death; other important predictive variables are the presence of ulceration and the anatomic location of the primary lesion.

Local recurrence most often appears clinically as a blue-tinged subcutaneous nodule arising in close proximity (within 2 to 5 cm) to the excision site of a primary melanoma (satellite metastasis) or en route to the regional lymph node basin (in-transit metastasis). Any subcutaneous nodule arising in the vicinity of a melanoma excision site should be considered to be disease recurrence or progression until proved

otherwise. The diagnosis is accurately accomplished with FNA. A complete metastatic survey, including CT or MRI and FDG-PET scans, should be performed because the majority of these patients will also have evidence of systemic metastases.

Complete surgical resection with primary wound closure is the most straightforward means of treating single recurrent lesions. Patients with multiple subcutaneous metastases clustered within a defined area can be treated by wide local excision with a skin graft or flap closure. Resection margin widths have not been as well defined in the resection of locally recurrent disease as they have in the treatment of primary cutaneous melanoma. However, recurrent lesions should be resected with a margin of normal tissue to avoid tumor spillage and wound contamination. Despite complete surgical resection of cutaneous recurrence, subsequent local and regional disease recurs in up to 67% of patients and is strongly associated with disease progression. The majority (70% to 82%) of such patients ultimately succumb to distant metastases. Although systemic chemotherapy in the adjuvant setting is rarely effective, systemic immunotherapy in the form of interferon alfa-2b (Intron A) or a variety of experimental melanoma vaccines may have a role in the treatment of patients subsequent to complete surgical resection of locally recurrent melanoma. The effectiveness of such modes of immunotherapy in improving survival in these patients remains unproven.

Selected Clinical Situations

SUBUNGUAL MELANOMA. Subungual melanoma accounts for only 3% of cases of melanoma in the white population but a higher proportion of melanoma (15% to 35%) in dark-skinned ethnic groups. More than 75% of subungual melanomas involve either the great toe or the thumb. Early signs of this lesion include darkening of the nail bed. Dark pigmentation of the posterior nail fold, or *Hutchinson's sign*, is a classic sign of subungual melanoma. Many patients with subungual melanoma report a recent history of trauma to the digit and attribute the lesion to a poorly healing wound. The differential diagnosis of subungual melanoma includes benign pigmented lesions of the nail bed mechanism (melanonychia striata), chronic bacterial and fungal infection, and subungual hematoma.

Although nodular and amelanotic lesions do occur, the majority of subungual hematomas appear as a sharply demarcated blue-black to brown discoloration of the nail that does not involve the adjacent cuticle. Formal biopsy of the nail bed is performed under digital or regional anesthesia blockade. The nail plate is elevated from the nail bed and removed so that the lesion is clearly visualized. An elliptical incision in the nail bed down to the underlying periosteum is performed to allow complete excisional biopsy of the lesion, as well as primary closure of the defect. Larger defects may be repaired with a nail bed flap or skin graft. Generous incisional biopsy through the central portion of pigmentation is performed for larger lesions not amenable to simple excision.

Melanoma in situ of the nail bed is treated by wide local excision. Negative surgical margins of at least 5 mm are optimal. The surgical defect may be repaired with a local skin flap or skin graft.

Invasive subungual melanomas of the lower extremity are most easily treated by amputation of the toe. The appropriate surgical resection margin width of 1 or 2 cm for lesions with a thickness less than 1.0 mm or 1.0 mm or greater, respectively, is achieved by complete amputation of the affected toe. Ray amputation may be necessary to treat thick lesions extending into the web space. In the majority of patients, the resulting surgical defect is easily closed, without any need for a specialized prosthesis or orthotic device. For upper extremity subungual invasive melanomas, surgical treatment is more individualized. Amputation is performed through the joint most proximal to the lesion. Wound closure is achieved with a flap of volar tissue while ideally maintaining a margin of at least 1 cm of normal tissue.

Sentinel lymph node mapping and excision are appropriately performed for patients with melanomas approaching 0.8 mm or greater in thickness in the absence of clinically palpable regional nodes. This procedure is optimally performed prior to amputation. Patients with palpable nodal metastases in the absence of significant distant metastases undergo formal regional lymphadenectomy.

PLANTAR MELANOMA. Melanoma arising on the sole of the foot, characteristically in the acral lentiginous growth pattern, is a rare clinical entity in white individuals and accounts for only 2% to 8% of melanoma cases. However, in dark-skinned ethnic groups, plantar lesions account for 35% to 90% of all cases of melanoma.

These lesions are often diagnosed at later stages and therefore generally have a less favorable prognosis. The frequent delay in diagnosis may be explained by their rarity, as well as by their infrequently examined location. In addition, the increased thickness of the epidermis of the plantar surface may distort the characteristic clinical appearance of melanoma. A major pitfall in the early diagnosis of this lesion is the failure to obtain a satisfactory biopsy specimen.

The preferred method of biopsy is complete excisional biopsy. Definitive wound closure may be deferred until rapid histologic diagnosis and margin inspection are complete. Once the diagnosis of melanoma is confirmed, the lesion is excised and staged according to the guidelines established for other cutaneous primary melanomas of comparable thickness. Sentinel lymph node mapping and

excision are recommended for patients with melanomas approaching 0.8 mm in thickness in the absence of clinically palpable regional nodes. Patients with palpable nodal metastases and no other evidence of distant metastases undergo superficial inguinal lymph node dissection. Dissection of the deep inguinal nodes is performed in patients with involvement of Cloquet's node or extensive disease in the upper aspect of the femoral triangle.

Lesions confirmed to be melanoma on shave, punch, or incisional biopsy that approach or exceed 0.8 mm in thickness may be treated definitively as outlined earlier and do not require a preliminary excisional biopsy procedure.

MELANOMA ON THE FACE. Patients with melanomas on the face approaching 0.8 mm or greater in thickness are at significant (at least 20%) risk for occult micrometastases in the regional lymph nodes. Although the anatomic location of the primary melanoma in the head and neck predicts the most likely site of regional nodal metastasis, cutaneous lymphoscintigraphy has better delineated the complex lymphatic drainage patterns unique to this region. Accordingly, elective lymph node dissection of the presumed sites of micrometastatic disease in the head and neck region has been replaced by cutaneous lymphoscintigraphy and sentinel lymphadenectomy with the use of a combination of intradermally injected vital blue dye and a radiolabeled tracer substance such as technetium sulfur colloid. Regional lymphadenectomy is performed selectively in patients with histologically documented micrometastases in the sentinel nodes. Superficial parotidectomy with identification and preservation of all facial nerve branches is recommended for patients with micrometastases in the periparotid nodes. Selective cervical lymph node dissection, based on precise localization of a positive cervical sentinel node, has replaced modified radical and formal radical neck dissection. Patients with palpable lymph node metastases in the absence of significant distant metastases should undergo formal regional lymphadenectomy.

In a small but not insignificant number of patients with melanoma of the face, preoperative cutaneous lymphoscintigraphy reveals a widespread pattern of lymphatic drainage from the primary lesion to multiple sentinel nodes dispersed throughout the head and neck region. Diagnostic accuracy may decline and the risk of facial or spinal accessory nerve injury (or both) rises as the complexity and number of individual sentinel nodes to be identified and excised increase. Therefore, it is reasonable to forgo sentinel lymphadenectomy in individualized circumstances when more than two sentinel node sites are revealed by preoperative lymphoscintigraphy. The reasons for this decision, as well as the risks and benefits of not identifying potential microscopic regional nodal metastases, should be discussed with the patient and documented.

Basal Cell Carcinoma

Basal cell carcinoma, the most common cutaneous malignancy, accounts for more than three quarters of skin cancers in the United States and affects nearly 800,000 people yearly. Although the incidence of basal cell carcinoma has risen over the last several decades, the average age at diagnosis has steadily decreased. Chronic exposure to sunlight is the principal factor in the development of basal cell carcinoma. Lesions principally occur on sun-exposed areas such as the face, scalp, ears, neck, shoulders, and back. Other known etiologic factors include exposure to ultraviolet light, chemical carcinogens (arsenic and hydrocarbons), ionizing radiation, cigarette smoking, chronic skin irritation or ulceration, and human papillomavirus. Immunocompromised individuals are at increased risk for this tumor.

The lesions classically have raised borders around a pearly central area with associated telangiectasia. The term *rodent ulcer* describes a particularly ulcerative, nodular lesion. Basal cell carcinoma may also appear more subtly as a scaly cutaneous lesion with areas of atrophy or scarring associated with chronic inflammation.

Basal cell carcinomas arise from the basal layer of the epidermis and are further classified within five histologic subtypes: nodular, micronodular, superficial, infiltrating, and morpheaform. Histologic subtyping aids in the clinical management of these tumors. The infiltrating and morpheaform subtypes often exhibit focal areas of invasion and may be locally aggressive; they have the highest rates of recurrence and positive margins. Nodular and superficial tumors are more indolent lesions.

Definitive histologic diagnosis of basal cell carcinoma may be achieved through a variety of simple techniques such as shave or punch biopsy. Small lesions may be excised completely at the time of initial biopsy. However, larger lesions should be confirmed histologically before complete excision is attempted.

Treatment of basal cell carcinoma involves complete removal of the lesion to obtain clear histologic margins by a variety of methods. Cryotherapy is often used to ablate primary lesions smaller than 2 cm in diameter. Although excellent results (greater than 95% cure rate) have been reported, hypopigmentation, scarring, and local nerve injury may occur. These techniques are appropriate for treatment of the less aggressive subtypes, but they are not recommended in patients with cryoglobulinemia or in anatomic areas in which contracture of the scar may create an unacceptable functional or cosmetic result. Topical chemotherapy (5-fluorouracil), used to treat less aggressive variants such as small nodular and superficial lesions, is also effective in the treatment of premalignant or borderline lesions.

Electrodessication, curettage, and Mohs' micrographic surgery may be used to treat lesions that

are not well circumscribed, nodular lesions smaller than 2 cm in diameter, and superficial lesions of any size. The Mohs technique is particularly useful in cosmetically sensitive areas such as the face. Defects are closed after negative resection margins are confirmed histologically. Although cure rates in excess of 90% may be expected, local recurrence has been reported in up to 30% of patients with lesions exceeding 3 cm in diameter.

Surgical excision of basal cell carcinoma results in a cure rate greater than 95%. As with other forms of treatment, the size, location, and histologic subtype contribute to the overall prognosis. There is no uniform recommendation regarding the optimal width of surgical margins. A reasonable approach is to obtain margins of at least 3 to 5 mm for small, well-circumscribed lesions and 1-cm margins for larger or more aggressive histologic variants of basal cell carcinoma.

For lesions of the face, especially those in critical areas such as the nose, lips, eyelids, and ears, Mohs' micrographic surgery is an excellent option. It has cure rates of approximately 95% and is extremely useful in morpheaform lesions. It allows maximal preservation of uninvolved tissue and does not compromise the clearance of tumor. Other indications include recurrent lesions or a history of irradiation.

Regional lymph node dissection is performed only in the setting of palpable nodal metastases positively confirmed preoperatively by FNA biopsy. Intraoperative lymphatic mapping with sentinel lymphadenectomy is possible technically but cannot be recommended in the standard treatment of basal cell carcinoma because of the extremely low incidence of regional lymph node metastasis associated with this tumor.

Radiotherapy may also be used to treat patients with basal cell carcinoma, although overall cure rates are usually suboptimal. However, radiotherapy may be offered to treat large or aggressive lesions in patients who are deemed to be poor surgical candidates.

Squamous Cell Carcinoma

Squamous cell carcinoma currently accounts for 20% of the total number of skin cancers diagnosed in the United States and affects approximately 200,000 Americans annually. Risk factors for squamous cell carcinoma are similar to those associated with the development of basal cell carcinoma, with exposure to sunlight being the principal cause.

Primary cutaneous squamous cell carcinomas arise from keratinocytes that migrate from the proliferating basal layer of the skin and grow continually in an uncontrolled manner. Lesions typically develop on exposed areas of the skin and appear as a firm nodular plaque on an erythematous base with raised borders. An area of central ulceration and necrosis may be present. On histologic examination, irregular nests of epidermal cells invade the dermis. Lesions are graded histologically to reflect the extent of cellular differentiation observed. *Low-grade*, well-differentiated lesions demonstrate less invasive growth patterns and are associated with an excellent prognosis when compared with more aggressive high-grade, poorly differentiated lesions. Cutaneous squamous cell carcinoma metastasizes to regional lymph nodes in approximately 2% to 5% of patients. Patients with confirmed lymph node metastases should be surveyed for more distant sites of disease, including the brain, lungs, and bones.

Marjolin's ulcer describes a cutaneous squamous cell carcinoma that arises in an area of chronic inflammation, drainage, or scarring. These unusual lesions are biologically aggressive and associated with a 50% risk of metastasis.

The differential diagnosis includes a variety of benign and premalignant lesions that clinically resemble the appearance of squamous cell carcinoma. *Keratoacanthoma* appears as a rapidly growing nodular cutaneous lesion with rolled edges and a debris-filled necrotic center. Although keratoacanthomas typically involute spontaneously after a period, they may be extremely difficult to distinguish clinically from a poorly differentiated cutaneous neoplasm. Any suspicion of carcinoma should promptly lead to an appropriate biopsy procedure. Complete but conservative surgical excision is often required to exclude malignancy from these lesions. *Actinic keratoses* are more subtle lesions that may also resemble squamous cell carcinoma in appearance. These lesions require excisional biopsy or close follow-up because the risk of squamous cell carcinomas arising from actinic keratoses approaches 20%. *Bowen's disease* defines lesions exhibiting the histologic characteristics of squamous cell carcinoma in situ. Because invasive squamous cell carcinoma develops in up to 10% of these lesions, they also require complete ablation and close surveillance.

Squamous cell carcinoma is generally treated with curettage or electrodessication, Mohs' technique, or simple surgical excision. Curettage is used most successfully in the treatment of lesions less than 2 cm in diameter with clear, well-defined borders. The Mohs technique is most useful in challenging anatomic locations such as the face.

Surgical excision yields excellent results for small, well-differentiated lesions. A resection margin of 5 mm is generally adequate for small (<2 cm in diameter), well-differentiated squamous cell cancers. Higher risk tumors, including poorly differentiated lesions, that are larger than 2 cm in diameter or more than 4 mm thick or that demonstrate histologic evidence of perineural invasion require a surgical margin of at least 1 cm. Although prophylactic or elective regional lymphadenectomy is not recommended in patients with high-risk lesions, lymphatic mapping with sentinel lymph

node biopsy is currently being evaluated in these patients, who are at risk for the development of regional nodal micrometastases. Lymph node dissection is appropriate, however, in patients with palpable, biopsy-proven regional lymph node metastases. Adjuvant radiation therapy may be offered to patients after resection of bulky lymph node metastases. In selected cases, radiotherapy may be used to palliate patients who are poor candidates for resection or those with extremely large, locally advanced, unresectable lesions.

SUGGESTED READINGS

Melanoma

Ackerman AB, Su WPD: Histopathology of cutaneous malignant melanoma. In Kopf AW, Bart RS, Rodriguez-Sains R, Ackerman AB (eds): Malignant Melanoma. New York, Masson, 1979, p 25.

American Joint Committee on Cancer: Manual for Staging Cancer, 6th ed. Philadelphia, JB Lippincott, 2002.

Balch CM, Soong SJ, Bartolucci AA, et al: Efficacy of elective regional lymph node dissection of 1 to 4 mm thick melanomas for patients 60 years of age and younger. Ann Surg 224:255-266, 1996.

Breslow A: Thickness, cross-sectional areas and depth of invasion in the prognosis of cutaneous melanoma. Ann Surg 17:902-908, 1970.

Briggs JC: Subungual malignant melanoma. A review article. Br J Plast Surg 38:174, 1985.

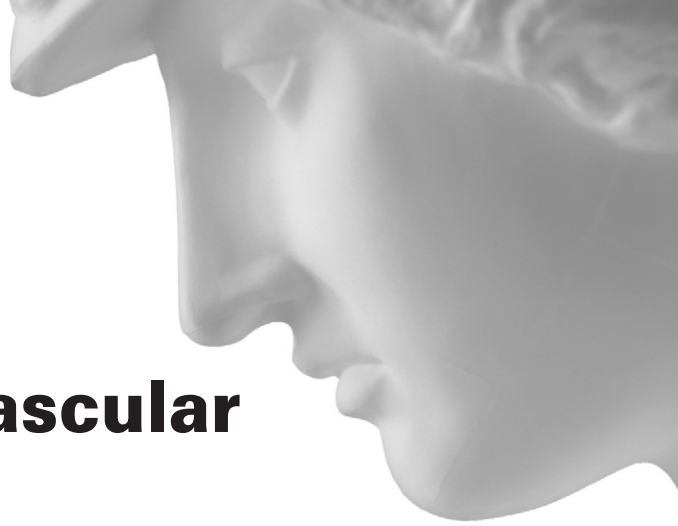
Morton DL, Wen D-R, Cochran AJ: Management of early stage melanoma by intraoperative lymphatic mapping and selective lymphadenectomy: An alternative to routine elective lymphadenectomy or "watch and wait." Surg Oncol Clin North Am 1:247-259, 1992.

Basal Cell and Squamous Cell Carcinoma

Fleming ID, Amonette R, Monaghan T, Fleming MD: Principles of management of basal and squamous cell carcinoma of the skin. Cancer 75(2 Suppl):699-704, 1995.

Luce EA: Advanced and recurrent nonmelanoma skin cancer. Clin Plast Surg 24:731-745, 1997.

Sexton M, Jones DB, Maloney ME: Histologic pattern analysis of basal cell carcinoma. Study of a series of 1039 consecutive neoplasms. J Am Acad Dermatol 23(6 Pt 1):1118-1126, 1990.



Hemangiomas and Vascular Malformations

JOHN B. MULLIKEN

The field of vascular anomalies is no longer obscured by its own outdated nomenclature, a bewildering admixture of descriptive and histologic words. Nonetheless, terminologic confusion continues to be responsible for imprecise diagnosis, illogical treatment, and misdirected research.

Hemangiomas

Etiopathogenesis

Hemangioma of infancy is characterized by rapid proliferation and slow involution, with phase-specific expression patterns of angiogenesis-related factors and inhibitors of blood vessel growth. There is evidence that hemangiomas arise from clonal expansion (a somatic mutation) of endothelial precursors that may originate in the fetus or possibly in the placenta. Regression may be inherent in the altered cell line or may be triggered by the tumor.

Histopathology

The *proliferating phase* (0 to 1 year) is typified by endothelial hyperplasia and upregulation of angiogenic peptides and enzymes involved in remodeling the extracellular matrix. During the *involuting phase* (1 to 7 years), there is downregulation of angiogenesis, endothelial apoptosis, and accumulation of mast cells (and other stromal cells). *Involuted-phase* specimens (after 7 years) demonstrate mature, capillary-sized vessels (surrounded by multilaminated basement membranes) and a few residual draining veins encased in fibrofatty tissue.

Diagnosis

Cutaneous hemangiomas occur in 4% to 10% of white infants, with a 3:1 or higher preponderance of females. The tumor is usually solitary, but approximately 20% grow in multiple cutaneous sites. If five or more lesions are present, there is an increased likelihood of involvement of other organ systems, especially the liver, gastrointestinal tract, and brain. A third or more of hemangiomas are nascent at birth as manifested by a pale spot, a telangiectatic or macular red stain, or a pseudo-ecchymotic patch. Most hemangiomas appear at a median age of 2 weeks; however, a deep tumor may not be seen until the infant is approximately 3 to 4 months old. A rare variant called *congenital hemangioma* (defined as fully grown at birth) is a raised, bluish, dome-shaped tumor, often with telangiectasia and a pale rim. These prenatal (fetal) hemangiomas behave differently; most regress rapidly, whereas some do not regress at all.

Hemangioma grows rapidly until 6 to 8 months of age (*proliferating phase*). A superficial tumor expands as the skin turns a vivid crimson color. A deep hemangioma spreads in the lower dermis, subcutis, and subcutaneous layer; the overlying skin is slightly raised, warm, and bluish. There is also a flat, telangiectatic form of hemangioma. Draining veins often radiate from the tumor. Spontaneous crusting and ulceration can occur, particularly in the lips, perineum, lateral cheek, and intertriginous areas.

Hemangiomas grow in proportion during late infancy; the first signs of the involuting phase appear at approximately 1 year. Over the ensuing 5 years (*involuting phase*), the bright red color fades, a patchy gray mantle forms, and the lesion is less tense on palpation. There are few indicators that accurately forecast the outcome of involution. The rate of regression is unrelated to the appearance, cutaneous depth, site, or size of the tumor. Regression usually ends and the last traces of color disappear by 5 to 7 years (*involuted phase*). Nearly normal skin is restored in an estimated 50% of hemangiomas. In the other 50%, the skin is damaged as evidenced by telangiectasia, crepe-like

laxity (destruction of elastic fibers), and yellowish discoloration or scarring (if ulceration occurred during the proliferating phase). There is also a variable underlying fibrofatty residual mass.

Radiographic Features

Imaging is sometimes needed to confirm the clinical diagnosis of a vascular anomaly, but it is essential that the radiologist have expertise in this field.

ULTRASONOGRAPHY. Ultrasonographic hallmarks of a proliferating-phase hemangioma are a discrete mass, solid parenchyma, and fast-flow vessels within and adjacent to the tumor. Ultrasonography is useful in differentiating a deep hemangioma from a vascular malformation. However, even an experienced ultrasonologist may have trouble distinguishing a young hemangioma, especially a congenital type, from an arteriovenous malformation (AVM) because both exhibit rapid flow.

MAGNETIC RESONANCE IMAGING. A proliferating-phase hemangioma is characterized as a parenchymous lesion of intermediate intensity on T1-weighted magnetic resonance imaging (MRI) and moderate hyperintensity on T2-weighted MRI. Flow voids, indicative of high-flow vessels, are confirmed by gradient-recalled echo sequences. There is dense, homogeneous enhancement with gadolinium. Involuting-phase hemangioma exhibits diminishing flow, decreasing size and number of feeding/draining vessels, and increasing lobularity with fat. An involuted hemangioma is almost avascular on MRI.

Associated Malformative Anomalies

"Hemangioma" is often erroneously listed as being associated with a particular syndrome, but these vascular lesions are more likely to be malformations. Furthermore, because of the frequency of hemangiomas, they sometimes coexist with, but unrelated to, a syndrome. However, there are rare clinical examples in which hemangiomas are associated with malformations. Cervicofacial hemangioma can coexist with abnormalities of the eye (e.g., microphthalmos, congenital cataract, and optic nerve hypoplasia), with persistent intracranial and extracranial arteries, with a variety of abnormalities of the great vessels and their branches, and with cystic malformations of the posterior cranial fossa. Midline thoracic anomalies, in a spectrum ranging from a minor supraumbilical raphe to sternal nonunion, can occur with hemangioma of the anterior portion of the chest. Lumbosacral hemangioma is one of several ectodermal lesions known to overlie spinal dysraphism (e.g., lipomeningocele, tethered cord, and diastematomyelia). An extensive telangiectatic hemangioma of the lower extremity and perineum can be accompanied by anogenitourinary anomalies.

Differential Diagnosis

One must remember that "not all hemangiomas look like strawberries" and "not all fruit-like lesions are hemangiomas." A deep hemangioma, particularly when located in the cervical region or trunk, can be confused with lymphatic or venous malformations. Congenital hemangioma can often be mistaken for a vascular malformation, particularly if major arteriovenous shunting is present. Telangiectatic hemangioma imitates a capillary malformation. In the lower limb, this macular form is often manifested as perineal ulceration and can cause sufficient direct arteriovenous flow to produce congestive heart failure.

Kasabach-Merritt phenomenon (thrombocytopenic coagulopathy) is not associated with infantile hemangioma. It is caused by other infantile vascular tumors: kaposiform hemangioendothelioma (KHE) and tufted angioma (TA). These tumors have overlapping clinical and histologic features, and it is likely that KHE and TA are part of a neoplastic spectrum. Unlike infantile hemangioma, KHE and TA affect both sexes equally. They are single, often extensive tumors that typically involve the trunk, shoulder, thigh, or retroperitoneum. Ecchymosis appears over and around the tumor along with generalized petechiae. Thrombocytopenia is profound (typically, $<10{,}000/mm^3$), fibrinogen levels are low, D-dimers are increased, and the prothrombin time and activated partial thromboplastin time are variably, but minimally elevated. MRI differentiates KHE and TA from common hemangioma; biopsy is rarely necessary.

Infantile fibrosarcoma can masquerade as fetal hemangioma when detected by prenatal ultrasonography or when seen at birth. Congenital hemangioma of the rapidly involuting type presents as a large, spherical firm mass with a predilection for the extremities, chest, and head/neck area. The overlying skin can be normal, but it is often tense with a dark violaceous hue and ectatic veins. Moderate thrombocytopenia and other hematologic findings suggest minor consumptive coagulopathy. Congenital hemangioma can also cause cardiac overload. Radiologic examination is usually diagnostic. Biopsy of any neonatal vascular lesion is mandatory if there is any suspicion of malignancy by history, physical examination, or imaging.

Treatment

Most hemangiomas are small, harmless lesions that complete their life cycle under the care of a pediatrician. The majority of these lesions leave normal or slightly blemished skin. Other hemangiomas should be treated because of either their size, rapidity of growth, anatomic location, or associated complications. Others cause cosmetic deformity, such as irrevocably damaged skin, deformed cartilage, or fibrofatty residuum. It is often difficult to predict the

behavior of hemangioma in the early proliferating phase.

Pain is often a problem with an ulcerated hemangioma. Topical viscous lidocaine is helpful but should not be applied more than twice a day. Other remedies include cleansing, topical antibiotic, or hydrocolloid dressing. Some ulcerated hemangiomas require more intensive care; débridement dressings can be effective in some locations. Flashlamp-pumped pulsed dye laser may accelerate healing but may also exacerbate ulceration. Antiangiogenic therapy is often included to control the tumor. None of these therapies is predictable; an ulcerated hemangioma often takes weeks to heal. Resection is an alternative for a small ulcerated tumor that responds slowly to topical treatment. Punctate bleeding from hemangioma is rare but, if encountered, can be controlled by compression; on occasion, a suture is needed.

Drug therapy is indicated if the hemangioma causes destruction, distortion, or obstruction. For example, ulceration can damage an eyebrow, eyelid, nose, lip, cheek, or ear. A large hemangioma can cause a mass effect on the nose or ear. Circumferential or localized subglottic tumor can obstruct the airway. Periorbital hemangioma can block the visual axis (causing obstructive amblyopia), infiltrate the extraocular muscles (causing strabismus), or extend into the retrobulbar space (causing ocular proptosis). A small hemangioma in the upper eyelid or supraorbital area can distort the cornea and produce astigmatic amblyopia. High-output cardiac failure is a life-threatening complication with hepatic hemangioma, but can also occur with a large or extensive cutaneous hemangioma.

Intralesional injection of corticosteroid should be considered for a small hemangioma (1 to 2 cm in diameter) located in the nasal tip, cheek, lip, or eyelid. The goal is to control the size of the tumor and minimize postinvolutional consequences. Triamcinolone (3 to 5 mg/kg) is injected slowly at low pressure with a 3-mL syringe and a 30-gauge needle. Whenever feasible, the periphery of the lesion should be compressed with the ring of a surgical instrument to reduce the slight chance for embolization of colloidal particles. Usually, three to five injections given at 6-week intervals are required. The response rate is similar to that for systemic corticosteroid. Subcutaneous atrophy occurs rarely, but is temporary. An ultrapotent topical corticosteroid can be used to treat a small, superficial facial hemangioma.

First-line therapy for problematic hemangiomas is *systemic corticosteroids*. Prednisolone is initiated at a dosage of 2 to 3 mg/kg/day. In an acute situation, such as respiratory or visual obstruction, an equivalent dose of intravenous corticosteroid can be given. Tumor stabilization and regression can occur within 1 to 2 weeks and is manifested by a diminished growth rate, fading color, and softening. At this point, the corticosteroid therapy is tapered slowly, usually every 2 to 4 weeks, and terminated when the infant is approximately 10 months old. If rebound growth occurs after discontinuation, another short course of the drug can be given.

The overall response rate is 85%. Cushingoid facies is expected in all infants, but it regresses as the dose is lowered. Approximately 35% of infants exhibit a diminished rate of gain in height and 25% in weight. However, all children return to their pretreatment growth curves for height and weight by 1 to 2 years of age. Approximately 15% of infants grow faster while undergoing therapy. Other serious complications, such as infection, hypertension, myopathy, and bone resorption, have not been observed with the aforementioned corticosteroid regimen.

Recombinant interferon (IFN) alfa, 2a or 2b, is considered second-line therapy for an endangering or life-threatening hemangioma or hemangioendothelioma. IFN is indicated for patients who fail to respond to corticosteroid therapy, those with a contraindication to prolonged systemic corticosteroids, patients with complications from corticosteroids, or children whose parents refuse to agree to corticosteroid therapy. The dose of IFN is 2 to 3 million U/m^2 injected subcutaneously daily and given up to 10 months of age. The dose usually does not have to be adjusted for the infant's growth. The response rate is 90%, even for tumors that failed corticosteroid treatment.

IFN causes low-grade fever during the initial period of administration. Other transient toxicities include elevation of hepatic transaminase levels, neutropenia, and anemia. The most serious complication is spastic diplegia (incidence of up to 20%), but it is usually reversible if detected early and IFN therapy is discontinued.

IFN therapy is somewhat less successful for KHE/TA with Kasabach-Merritt phenomenon. Chemotherapeutic agents such as vincristine and cyclophosphamide are effective alternatives. No single drug regimen has proved to be consistently effective. In the long term, these tumors persist beneath the skin, usually remain asymptomatic, and only rarely recrudesce.

Flashlamp-pumped pulsed dye laser can lighten the color of a proliferating hemangioma. It penetrates only 0.75 to 1.2 mm into the skin. Overzealous photocoagulation may cause ulceration, partial-thickness loss, scarring, and hypopigmentation. Laser is effective for telangiectases that persist in the involuting/involuted phase. Intralesional photocoagulation with a bare fiber laser (Nd:YAG) can produce rapid shrinkage of a proliferating hemangioma, but there is a risk of thermal ulceration, even in the hands of an experienced operator.

The surgeon has a role in all three phases of the life cycle. The conventional method is to excise the tumor in a lenticular format with linear closure in the axis of the relaxed cutaneous tension lines, either as a single or staged procedure. This technique is effective in some anatomic locations (such

as the lips and eyelids). Most hemangiomas, however, are spherical, and lenticular excision (to minimize "dog-ears") can cause central tension that will flatten a convex area, such as the cheek or forehead, or cause distortion of nearby structures. Hemangioma acts as a tissue expander. For this reason, circular excision and purse-string closure should be considered. This technique acts in the longitudinal and transverse dimensions and converts a circular lesion to a small ellipsoid scar. Thereafter, excision can be either circular or lenticular. The result is the smallest possible scar with minimal distortion of surrounding structures.

INFANCY. Excision of localized hemangioma in the proliferative phase is indicated for painful ulceration, repeated bleeding, anatomic deformation, or a pedunculated tumor. Another indication is a focal tumor in the upper eyelid that is unresponsive to pharmacologic therapy. The indications for resection of a nonproblematic hemangioma are debatable. The expected scar after early excision must be compared with that possible if excision were to be postponed.

EARLY CHILDHOOD. Excision of an involuting hemangioma is considered in the preschool period if (1) it is obvious that resection is inevitable (i.e., there is postulcerative scarring or irrevocably expanded, inelastic skin or fibrofatty residuum), (2) the scar would be the same were excision delayed until the involuted phase, or (3) the scar is easily hidden.

Resection of an involuting hemangioma involving the nasal tip, eyelids, lips, or ears requires multidimensional planning. If the excision is subtotal, additional regression of the remaining hemangioma must be taken into account.

LATE CHILDHOOD. The surgical strategy is usually obvious after regression of the hemangioma is nearly complete. The tumor often leaves behind irrevocably expanded skin or fatty tissue, or both. Resection may be possible in a single stage; however, staged excision is usually indicated to minimize distortion and scarring. Uncommonly, destruction of an anatomic structure, such as the nose, eyelid, or ear, necessitates more complex reconstructive techniques.

Vascular Malformations

Etiopathogenesis

Vascular malformations arise as a result of faulty embryonic development at some stage of *vasculogenesis* (alignment of mesodermally derived endothelial precursors to form primitive blood vessels) or *angiogenesis* (formation of new blood vessels from preexisting vasculature). Thus, vascular malformations are present at birth. Although most are seen in the newborn nursery, others manifest without warning in childhood and adulthood. Most vascular malformations are sporadic (not familial) and their cause is unknown. Some are inherited in a mendelian autosomal dominant pattern, and the causative genes are known. Other vascular anomalies probably arise by somatic mutation. Molecular studies suggest that vascular malformations are caused by abnormal signaling processes that regulate cellular proliferation and apoptosis, differentiation, maturation, and adhesion.

Histopathology

A *capillary malformation* (CM) consists of dilated capillary- to venular-sized vessels in the superficial dermis. The nerves in the vessel walls are deficient, which probably explains the gradual darkening in color and nodular ectasia that occur with age. A *lymphatic malformation* (LM) displays a wide spectrum of findings from microcystic to macrocystic channels containing eosinophilic, protein-rich fluid. The vessel walls are of variable thickness and composed of abnormally formed smooth and skeletal muscular elements and scattered collections of lymphocytes. A *venous malformation* (VM) is a sponge-like anomaly with thin-walled, dilated channels lined by flat, mature endothelium. The muscular layer is thin and the cells are arranged in clumps rather than in the normal circular architecture. There is often evidence of clot formation, as well as various stages of cellular aggregation, fibrovascular ingrowth, and pathognomonic phleboliths. A subtype of venous anomaly called *glomuvenous malformation* ("glomangioma") is distinguished by rounded (glomus) cells scattered in the distended walls. An *arteriovenous malformation* (AVM) is characterized by thick-walled dysmorphic arteries of variable caliber with a disrupted internal elastic lamina and disorganized smooth muscle layers. The abnormal veins exhibit progressive reactive hypertrophy of the smooth muscle layer with subsequent replacement by collagen, which results in thin, fibrotic, inelastic channels.

The endothelial lining in the various vascular malformations is usually quiescent. However, endothelial hyperplasia may be seen after injury, embolization, sclerotherapy, or attempted surgical resection.

Capillary Malformation

A CM is manifested at birth as a macular, pink to red cutaneous stain that blanches with pressure. It should not be confused with *nevus flammeus neonatorum* ("stork bite" or "angel kiss"), a common vascular birthmark on the upper part of the face and posterior aspect of the neck that usually fades by 1 year of age. Alhough most commonly located on the face, CMs can occur anywhere in the body and may

be associated with hypertrophy of soft tissue and underlying bone. In the face, there is often gradual enlargement of the affected cheek, lip, and gums, along with maxillary or mandibular overgrowth.

Sturge-Weber syndrome is characterized by unilateral/bilateral CMs located in the ophthalmic (V1) dermatome or extending into the maxillary (V2) division, often with gradual hypertrophy of soft tissue and underlying bone. Capillary staining (and hypertrophy) can also occur in other regions of the body. MRI (with gadolinium enhancement) is necessary to document vascular anomalies of the leptomeninges and choroid plexus. Biannual funduscopic examination and tonometry are essential because a child with a choroidal vascular malformation ("tomato catsup" fundus) is at risk for retinal detachment, glaucoma, and blindness.

CM of a limb can be associated with hypertrophy (both in length and girth) and is either an isolated finding or occurs in combination with deeper vascular anomalies. *Klippel-Trenaunay syndrome* is a capillary-lymphatic-venous malformation involving one or more limbs and occasionally the thorax. CMs are multiple, sometimes contiguous, and occur in a geographic pattern over the lateral side of the extremity, buttock, or thorax (or any combination of these locations). CM is macular in a newborn, but hemolymphatic vesicles usually erupt later in the stained area. The embryonal lateral vein (of Servelle) is a pathognomonic feature in the lower extremity and may be associated with deep venous anomalies. Lymphatic hypoplasia is found in more than half of patients and manifests as lymphedema or macrocystic/microcystic anomalies. Hypertrophy is obvious at birth, and axial overgrowth can be progressive. These patients are at risk for cellulitis, thrombophlebitis, and thromboembolism.

Parkes Weber syndrome is characterized by a geographic pink macular stain in a congenitally enlarged upper or lower extremity in association with multiple microscopic arteriovenous fistulas of the skin and muscle. Detection of a bruit or thrill confirms the diagnosis. On occasion, lymphatic anomalies are also present.

CM can signal an underlying abnormality. CM in the midline scalp region may cover an encephalocele or ectopic meninges. CM over the cervical or lumbosacral spine can be a clue to occult spinal dysraphism or a spinal AVM (*Cobb's syndrome*) in the thoracolumbar region. An isolated CM is not well visualized by MRI; however, imaging is indicated if there is suspicion of a deep abnormality or an obvious combined (eponymous) vascular anomaly.

TREATMENT. Tunable flashlamp-pumped pulsed dye laser is used for selective photothermolysis of CM. Some reports suggest better results if treatment begins in infancy; other authors state that age makes no difference after 1 year. In general, obvious lightening occurs in approximately 70% of patients. The results are better in the lateral aspect of the face than in the central region and less predictable in the trunk and extremities. Laser therapy usually requires serial sessions.

Facial soft tissue and skeletal hypertrophy can be improved. Small fibrovascular nodules are easily excised. If photocoagulation fails, hypertrophied facial areas can be resected in aesthetic units and resurfaced with skin grafts or flaps. Contour resection for labial hypertrophy and macrocheilia is effective. Maxillary hypertrophy can be diminished by osseous burring. Orthognathic surgical correction may be indicated for occlusal canting caused by maxillary overgrowth or for mandibular prognathism. Selected orthopedic procedures are used for overgrowth in the lower limbs, often in conjunction with ablation of hypertrophied soft tissue.

Lymphatic Malformation

Macrocystic LM can be diagnosed by prenatal ultrasonography in the late first to early second trimester. LM should not be confused with *posterior nuchal translucency*, a fetal finding during the first and early second trimester associated with aneuploidy and a guarded prognosis. Bilateral cervicofacial LM can compromise the airway. Cesarean delivery may be indicated, and the airway should be assessed before the umbilical cord is clamped. If laryngeal LM precludes neonatal intubation, tracheostomy can be performed under controlled circumstances (EXIT procedure).

Most LMs are apparent at birth, whereas others become evident by 2 years; however, LM can appear suddenly in childhood, adolescence, or adulthood. LM is most commonly located, in order of frequency, in the cervicofacial region, axilla/chest/mediastinum, retroperitoneum, buttock, and anogenital area. The overlying skin has a normal or a bluish hue. Macrocystic LM is soft whereas microcystic LM is firm-hard. There is often an associated capillary stain or melanocytic nevus of the skin. Tiny dermal or submucosal vesicles are telltale signs; they can become dark red and firm nodules as a result of intravesicular bleeding. LM of the trunk and extremities is often accompanied by fatty hypertrophy.

LMs occur in recognizable patterns as a result of variable combinations of microcystic and macrocystic elements. LM in the forehead and periorbital tissues causes swelling of the supraorbital area and upper eyelid, usually with intraorbital involvement and proptosis. Facial LM causes unilateral or bilateral hypertrophy, macrocheilia, and macrotia. LM in the floor of the mouth and tongue causes macroglossia with dorsal, dark-stained vesicles. Cervicofacial LM is associated with overgrowth of the mandibular body and results in an anterior open bite and anterior crossbite. Cervical LM often involves the supraglottic airway and mediastinum. LM of the chest wall usually extends into the axilla. LM in an

extremity causes diffuse or localized swelling or gigantism of both soft tissues and skeleton.

LM is best assessed by MRI with contrast; ultrasonography confirms macrocystic LM. Gadolinium often shows "rim enhancement" in the walls of large cysts and septa on T1-weighted sequences. Because of their high water content, LMs (like VMs) are hyperintense on T2-weighted sequences. Fluid levels caused by protein or old blood are often seen in macrocysts. Large or anomalous venous channels (or both) may be present.

TREATMENT. Sudden enlargement of an LM is caused by either intralesional bleeding or cellulitis.

Medication for pain, rest, and the passage of time are required after intralesional bleeding; in addition, preventive antibiotics should be considered. LM commonly swells with viral infection, and bacterial infection is sometimes difficult to treat. Prompt administration of an oral antibiotic can be successful; otherwise, the intravenous route is necessary. Antibiotic therapy is often necessary for 6 to 8 weeks.

Sclerotherapy and surgical resection are also used for the treatment of LM. Injection of sclerosant is successful only for macrocystic LM. Commonly used agents include absolute ethanol, sodium tetradecyl sulfate, doxycycline, and OK-432 (derived from a killed strain of group A *Streptococcus pyogenes*). Excision is usually staged and incomplete, but total resection is possible in rare cases. For each procedure, the surgeon should (1) focus on a defined anatomic region, (2) limit blood loss to less than the patient's blood volume, (3) perform as complete a resection as possible while preserving vital structures, and (4) be prepared to operate for as long as necessary. Postoperative complications include hematoma/seroma (and the need for prolonged suction) and cellulitis. Lymphatic regeneration and reexpansion are responsible for postoperative recurrence. Dermal LM (old term, "lymphangioma circumscriptum") must be widely resected; frequently, the defect requires primary closure with a skin graft or flap.

Venous Malformation

Although venous anomalies are present at birth, they do not usually manifest until later. VM grows in proportion to the child and frequently expands during puberty. VM is bluish and compressible; it can, however, be firm (and painful) as a result of intralesional clotting. Patients often complain of pain and stiffness in the affected area, especially on awakening in the morning. VM occurs in many forms, from localized to extensive lesions that permeate normal tissue planes. Most VMs are solitary, but multiple cutaneous and visceral lesions do occur and should raise the possibility of autosomal dominant inheritance, for example, glomuvenous malformation (or "multiple glomangioma"), cutaneous-mucosal VM, or cerebral-cavernous VM. The causative genes are known for all these disorders. Blue rubber bleb nevus syndrome may also be genetic. It is characterized by multiple dome-shaped cutaneous lesions (often on the palms and plantar surfaces) in association with chronic bleeding from intestinal VMs.

MRI is the most rewarding radiographic technique. VM is hyperintense on T2-weighted sequences and differs from LM by enhancement of the contents of the vascular spaces on T1-weighted images. Phleboliths or thrombi are seen as signal voids on all MRI sequences. Direct phlebography is needed for more detailed assessment if interventional therapy is planned.

Stagnant flow can cause localized intravascular coagulopathy. Therefore, a coagulation profile is needed for any patient with an extensive VM (or VM combined with LM), particularly if there is a history of easy bruising or bleeding during operations.

TREATMENT. Indications for intervention are abnormal swelling or color, pain, or functional problems. Sclerotherapy is the primary modality. Small cutaneous or mucosal VMs can be injected with a mild sclerosant, such as 1% sodium tetradecyl sulfate or ethanolamine oleate. Large VMs require formal sclerotherapy by an experienced interventional radiologist performed under general anesthesia with real-time fluoroscopic monitoring. Absolute ethanol is widely used. Local complications include blistering, full-thickness cutaneous necrosis, and damage to local nerves. Feared systemic complications are cardiac arrest and hemolysis causing renal damage. Several sessions are usually needed because VMs have a perverse propensity for recanalization (recurrence).

Surgical resection is generally best scheduled several weeks after completion of sclerotherapy. Indications for primary excision include VM near critical nerves (in which case alcohol should not be used), VM with thrombus, VM involving the palm and digits, or a small, focal, cutaneous or mucosal VM.

Custom-made elastic support garments are an indispensable aid for a patient with VM in a limb. Low-dose aspirin (81 mg every day or every other day) minimizes painful phlebothromboses. Preoperative control of intravascular coagulopathy by heparin may be indicated.

Arteriovenous Malformation

An AVM is often ignored as being only an innocent vascular birthmark. Evolution is unpredictable and is documented by the Schobinger clinical staging system: stage I (quiescence): pink stain, warmth; stage II (expansion): same as stage I, plus enlargement, pulsations, thrills, and tortuous/tense veins; stage III (destruction): same as stage II, plus either dystrophic cutaneous changes, ulceration, bleeding,

or persistent pain; stage IV: same as stage III, plus cardiac failure. Expansion may be triggered by puberty, pregnancy, trauma, or attempted intervention. Clinical diagnosis is confirmed by ultrasonography and duplex Doppler examination. MRI and magnetic resonance angiography document the extent of this fast-flow anomaly. Superselective angiography is not usually indicated until intervention is planned.

TREATMENT. In rare instances, prompt embolization is needed to control cardiac failure in a newborn with AVM. Stage I AVM should be monitored annually throughout childhood and adolescence. Resection in stage I is considered if it can be easily accomplished without causing major deformity. Intervention is withheld until symptoms or endangering signs are present, such as progression beyond stage I. There is no indication for proximal ligation or embolization of feeding vessels, which causes only rapid recruitment of flow from nearby arteries to supply the nidus and denies access for future embolization.

Superselective arterial embolization or sclerotherapy (direct puncture of the nidus on the venous side) is indicated to control pain, bleeding, or cardiac overload. Cure is possible for AVMs with macrofistulas. Complete resection can be curative but is not always possible. Preoperative embolization or sclerotherapy (or both) minimizes intraoperative bleeding but does not diminish the extent of resection. The nidus of the AVM and surrounding tissue must be widely excised, but unaffected overlying skin can often be preserved. Observation of the type of bleeding at the margins of resection is the most accurate way to assess the adequacy of removal. The wound is closed primarily with local tissue or a microvascular free flap. If there is any question about the margins of excision, one can consider temporary coverage with a split-thickness skin graft.

The outcome of combined embolization/surgical resection is better with localized stage I or stage II AVM. Patients must be examined yearly by ultrasonography or MRI as indicated. Recurrence can take place many years after embolization and resection. Unfortunately, some AVMs involve an entire anatomic region and permeate deep craniofacial tissues or the soft and skeletal tissues in an extremity. In these patients, embolization/sclerotherapy is usually only palliative.

Pearls and Pitfalls

- No single specialist has sufficient knowledge to treat the wide variety of vascular anomalies; patients should be evaluated only at interdisciplinary centers for vascular anomalies. In this setting, specialists can establish the diagnosis, exchange knowledge, agree on management, and assess outcomes.
- Incorrect diagnosis usually leads to inappropriate therapy.
- Words for vascular anomalies cover the trap into which an unsuspecting clinician can fall.

SUGGESTED READINGS

Breugem CC, van der Horst CM, Hennekam RC: Progress toward understanding vascular malformations. Plast Reconstr Surg 107:1509-1523, 2001.

Drolet BA, Esterly NB, Frieden IJ: Hemangiomas in children. N Engl J Med 341:173-187, 1999.

Enjolras O, Mulliken JB, Boon LM, et al: Non-involuting congenital hemangioma: A rare cutaneous vascular anomaly. Plast Reconstr Surg 107:1647-1654, 2001.

Mulliken JB, Burrows PE: Vascular malformations. In Goldwyn RM, Cohen MN (eds): The Unfavorable Result in Plastic Surgery: Avoidance and Treatment. Philadelphia, Lippincott-Raven, 2001, pp 271-287.

Mulliken JB, Fishman SJ, Burrows PE: Vascular anomalies. Curr Probl Surg 37:517-584, 2000.

Mulliken JB, Rogers GF, Marler JJ: Circular excision of hemangioma and purse-string closure: The smallest possible scar. Plast Reconstr Surg 109:1544-1554, 2002.

Hypertrophic Scars and Keloids

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Keloids and hypertrophic scars are abnormal tissue responses to wounding and result in significant cosmetic and functional deformity. Although early keloids and hypertrophic scars appear clinically similar, keloids eventually grow beyond the confines of the initial wound. Hypertrophic scars appear within 4 weeks after trauma, whereas keloids may not appear until 3 months and occasionally up to several years after injury. Hypertrophic scars usually undergo at least partial spontaneous resolution with time and are often associated with contractures of healing tissues. Most hypertrophic scars do not recur after appropriate surgical treatment, whereas keloids tend to recur. Table 1 summarizes the differences between hypertrophic and keloid scars.

Etiopathogenesis

Humans are the only species in which hypertrophic scars and keloids develop. There is no gender prevalence. The incidence is highest in patients with darker complexions, and lesions occur most commonly in the second decade. Although keloids have been reported to affect all ethnic groups (except albinos), African American, Hispanic, and Asian individuals are more susceptible. Both keloids and hypertrophic scars occur more frequently in wounds closed under tension. Anatomic areas that have high intrinsic skin tension, such as the deltoid region, sternum, and upper part of the back, are especially vulnerable to keloid and hypertrophic scar formation (Figs. 1 and 2). Other traumatized areas not associated with a significant degree of skin tension can also have a predisposition for keloid formation, such as the earlobes (Fig. 3).

Other correlative associations have been reported in patients with keloids, for example, associations with the immune system, hormonal influences, blood type, and HLA proteins, but the role these play remains uncertain. Both autosomal recessive and dominant transmission has been reported in families with a propensity to form keloids. Finally, hormones may influence keloid formation because keloids often appear in puberty, enlarge during pregnancy, and resolve after menopause.

Although the pathogenetic/pathophysiologic mechanism responsible for hypertrophic scars and keloids remains unknown, there has been considerable research on the biochemical differences between hypertrophic scars, keloids, and normal scars. Differences are observed at each stage of wound healing and are reflected in the cellular composition, synthesis and degradation of the extracellular matrix, and growth factor and proinflammatory cytokine profiles. For example, decreased levels of interleukin-1 (IL-1), as observed in patients with keloid scars, correlate with the extent of the deformity. IL-1 stimulates the release of matrix metalloproteinases and synergistically induces collagenase activity in conjunction with release of interferon-γ (IFN-γ) and tumor necrosis factor-α (TNF-α) from inflammatory cells, thereby resulting in degradation of the extracellular matrix. A decrease in IL-1 production may result in extracellular matrix accumulation and excessive scar formation at the site of injury. In addition, reduced interferon levels have been demonstrated in keloids. These mediators cause downregulation of collagen synthesis and upregulation of collagenase activity, such that a reduction in interferons could lead to elevated collagen accumulation. Collagenase inhibitors, such as α_1-antitrypsin and α_2-macroglobulin, are also present at higher levels in keloids.

TABLE 1 Differences between Hypertrophic and Keloid Scars

	HYPERTROPHIC SCARS	KELOID SCARS
Appearance after injury	Within 4 weeks after trauma	Months to years
Resolution over time	At least partial resolution	Rarely resolves
Associated with contractures	Yes	No
Boundary	Limited boundary	Overgrows boundary
Occurs in areas of motion	Yes	No
Areas of high predilection	Flexor surfaces, joints	Sternum, earlobes
Surgical result	Improves with surgery	Frequently worsened by surgery

Patients with keloids have a higher frequency of allergic symptoms than do individuals with hypertrophic scars. Mast cells, interspersed among dermal collagen bundles, are found in higher numbers in hypertrophic scars than in normal scar tissue. They have cytoplasmic granules that contain histamine, heparin, serotonin, acid hydrolase chymase, and several growth factors, most of which are involved in dermal matrix production. Histamine is capable of enhancing the formation of collagen by fibroblasts in vivo, and its levels are elevated in keloid tissue. Furthermore, the excess of mast cells and their mediators may account for the increased pruritus present in hypertrophic scars. Other cellular differences include increased quantities of macrophages, elevated density and activity of fibroblasts, and increased neovascularization. In addition, growth factor activity is probably different in keloids than in normal scar, and moreover, the cellular response to many growth factors appears to be exaggerated in keloids.

Diagnosis

The diagnosis of hypertrophic scars and keloids remains a clinical one, and no specific studies are needed. Hypertrophic scars and keloids can be differentiated by their time of onset and growth within or beyond the boundaries of the initial insult. Overall, evaluation over time is the best method of differentiation.

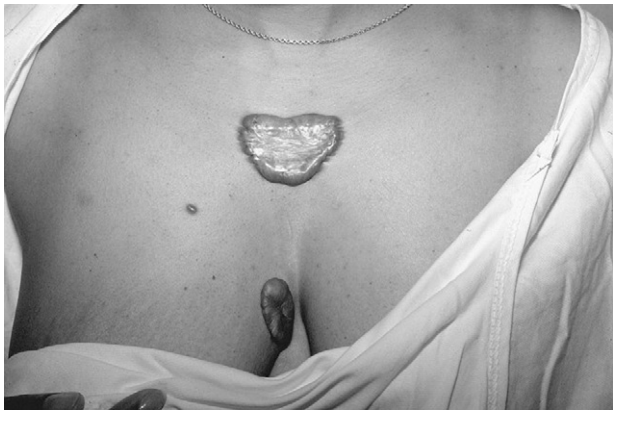

Figure 1 • Keloids of the sternum after local trauma.

Treatment

Molecular characterization of hypertrophic and keloid scars has progressed significantly over the

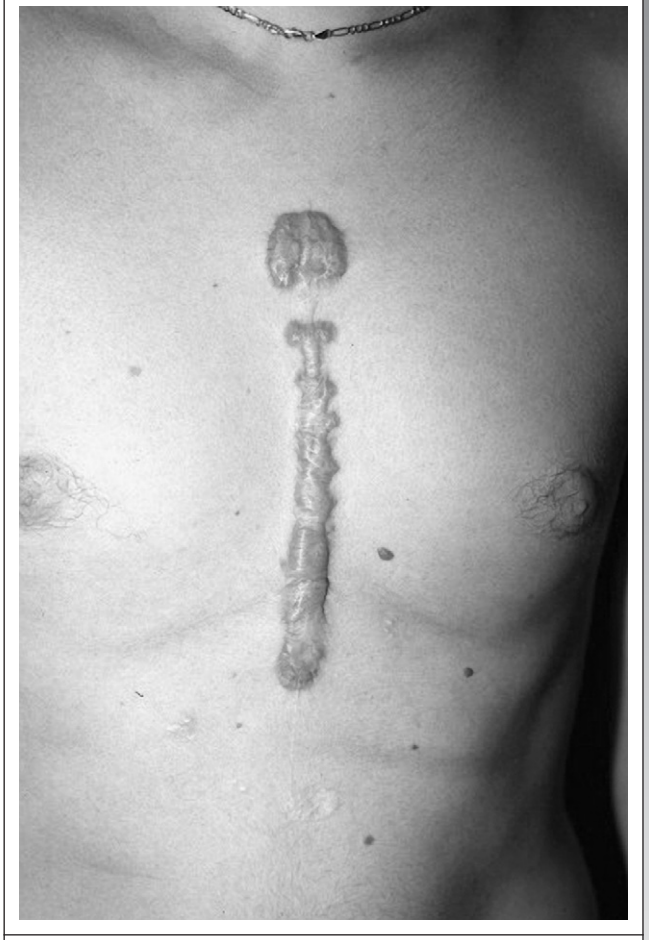

Figure 2 • Hypertrophic scar of the sternum after cardiac surgery.

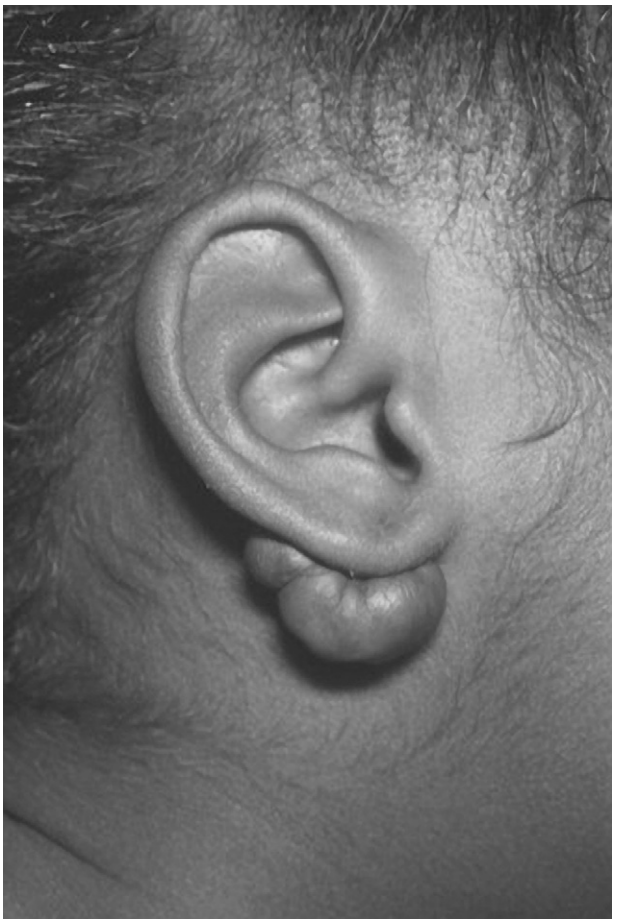

Figure 3 • Earlobe keloid after piercing.

past several years, particularly as biochemical and molecular biologic assays have become increasingly refined and readily available. This advance has broadened the approach to the development of different therapies. Current therapeutic options are based on one of the following concepts: (1) manipulation of the mechanical properties of wound repair, (2) correction of the abnormal balance of collagen synthesis and degradation, and (3) alteration of the immune/inflammatory response. Unfortunately, confusion in the classification of hypertrophic scars and keloids has resulted in contradictory and equivocal data. In addition, confounding variables such as the age of the scar and previous treatments are often not discussed. Moreover, follow-up periods are not uniform between studies, thus rendering evaluation and comparison of protocols extremely difficult. Most recurrences develop within the first year after surgery, although recurrence can take place 2 or more years postoperatively. With this in mind, the following is a summary of current therapeutic options.

Pressure Therapy

Although the precise mechanism of pressure therapy is imperfectly understood, success rates of 60% to 85% for at least partial reduction of hypertrophic and keloid scars have been reported. The pressure exerted should be at least 24 mm Hg to exceed inherent capillary pressure, but it must be lower than 30 mm Hg to avoid interruption of the peripheral circulation. Maintaining the pressure for a period of several months to 2 years has been reported to resolve hypertrophic scars permanently. It is thought that pressure causes ischemia, decreases tissue metabolism, and increases collagenase activity within the wound.

Pressure is most effective in hypertrophic scars with the use of specialized garments or devices. Pressure devices are most commonly used in combination with sponges, plastic plates, or silicone gels/sheets to increase the pressure level and provide a better fit with the scar. To achieve the maximum benefit, the pressure garments must be worn 18 to 24 hours a day for at least 4 to 6 months because early release of the garments tends to be followed by rebound hypertrophy. Pressure clip-on earrings, for example, are effective in smaller earlobe keloids, in conjunction with postexcision care.

Intralesional Corticosteroids

Hypertrophic and keloid scars can be treated with intralesional injections of corticosteroids alone or in combination with other therapies to control local discomfort and flatten scars. Steroids are thought to decrease collagen and glycosaminoglycan synthesis, reduce inflammation, and diminish fibroblast and growth factor involvement during wound healing.

The effectiveness of intralesional corticosteroid administration is highly variable, with response rates ranging from 50% to 100% and recurrence rates of 9% to 50%. Corticosteroids are less effective in older scars and keloids; they can only soften and partially flatten the scar and provide symptomatic relief.

Most injections are given intralesionally, although some authors prefer sublesional injections. Steroid preparations used include hydrocortisone acetate, methylprednisolone (Depo-Medrol), dexamethasone, and triamcinolone acetate, of which triamcinolone (Kenacort) is most commonly used. The steroids can be mixed with lidocaine to reduce pain with the injections. No clear advantage has been demonstrated for any particular type of corticosteroid. The dosage and administration should be adjusted to suit the individual patient. The initial concentration of triamcinolone acetonide ranges from 10 to 40 mg/mL, depending on the amount of scar formation, the area of the body, and the individual patient. Usually, two or three injections are sufficient, although injections for 6 months or longer are occasionally required, depending on the type of scar.

Adverse effects of corticosteroid injections include skin atrophy, hypopigmentation or depigmentation, telangiectasia, necrosis, ulceration, and cushingoid features. The risk of complications increases when corticosteroids are injected into the surrounding dermis or subcutaneous tissue.

Steroid injections are likely most useful in controlling local symptoms such as urticaria and burning/pain. When used in combination with intralesional excision, a 50% recurrence rate can be expected and should be quoted to patients before surgery.

Silicone Gel and Other Dressings

Studies on the application of silicone material to hypertrophic and keloid scars have generally reported a decrease in the scar's volume and an increase in elasticity in 60% to 100% of cases. Some authors have indicated that the use of silicone material after surgical resection can prevent the formation of hypertrophic scars and keloids in 75% to 85% of patients.

The silicone material is worn continuously for at least 3 months to prevent rebound hypertrophy. The mode of action of silicone materials is still unknown but does not appear to be solely due to pressure. Occlusion and hydration, a difference in oxygen tension or temperature, silicone leakage into the dermis, or electrostatic charges may be the principal modes of action of silicone gel sheet treatment.

Topical silicone treatment is most effective for hypertrophic scars. However, the need for at least 18 hours of daily scar coverage for up to 3 months makes this therapy cumbersome and difficult. Liquid silicone applications with tissue adhesives are becoming available and may make this treatment modality more useful, although data on their effectiveness have not been demonstrated.

Surgery

Recurrence rates after simple surgical excision of keloids vary from 50% to 80%. Several surgical approaches have been developed in an attempt to minimize recurrence, including reduction of wound tension through tissue reorientation and retention of a rim of keloid tissue to serve as a splint and thereby avoid new tissue trauma (intralesional excision). Excision and skin grafting have been advocated for large keloids. However, this approach seems counterintuitive because of the baseline fibrotic healing involved with skin grafts; the donor site is also a potential keloid. Another alternative is tissue expansion of adjacent normal tissue, followed by keloid excision and tension-free closure with the expanded flaps.

Radiation

Radiation alone does not produce marked regression of keloids. Most studies have shown that surgery followed by irradiation has a higher success rate than irradiation alone does. The most important factor appears to be the total dose of radiation rather than the radiation time or fraction. Radiation doses of at least 1500 cGy are sufficient to control 90% of keloid scars without requiring reexcision. The mechanism of action is destruction of sufficient cells so that a balance is created between collagen formation and breakdown in keloids. Radiation is purported to affect extracellular matrix gene expression and damage connective tissue stem cells. Side effects of irradiation included hyperpigmentation, localized pruritus, paresthesias, and pain.

Concern over the use of a potentially carcinogenic treatment such as radiation for a benign condition, as well as possible growth interference in children after irradiation, has relegated the use of radiation solely to keloids resistant to all other treatment modalities.

Pulsed Dye Laser

In a study of 16 patients with median sternotomy scars (either keloidal or hypertrophic), the 585-nm flashlamp-pumped pulsed dye laser was used, and pruritus, erythema, scar height, and scar surface texture were significantly improved. The flashlamp-pumped pulsed dye laser acts through selective photothermolysis by hemoglobin light absorption. This phenomenon leads to local heating of cutaneous blood vessels, which causes tissue ischemia and collagen breakdown. Presently, the CO_2 and Nd:YAG lasers are not used in the treatment of keloids or hypertrophic scars.

Cryotherapy

The rationale for the use of cryotherapy is a reduction in excessive scar bulk through ischemic damage and necrosis. Cryotherapy is used alone and in combination with other agents to treat keloids and is associated with amelioration of symptoms related to hypertrophic scars and keloids, as well as flattening of the scar. Scars of shorter duration have better responses. Cryotherapy is not a first-line therapy because of its side effects, which include hypopigmentation or hyperpigmentation (particularly in patients with a darker complexion), pain, edema, wound infection, hypoesthesia, milia formation, and skin atrophy.

Interferon and Other Medications

The lack of well-designed, evidence-based studies on the long-term effectiveness of INF-α or INF-γ, either as monotherapy or as combination therapy, does not permit definitive conclusions regarding the effectiveness of this method.

Topical retinoids (vitamin A derivatives), vitamin E, 5-fluorouracil, and bleomycin have been evalu-

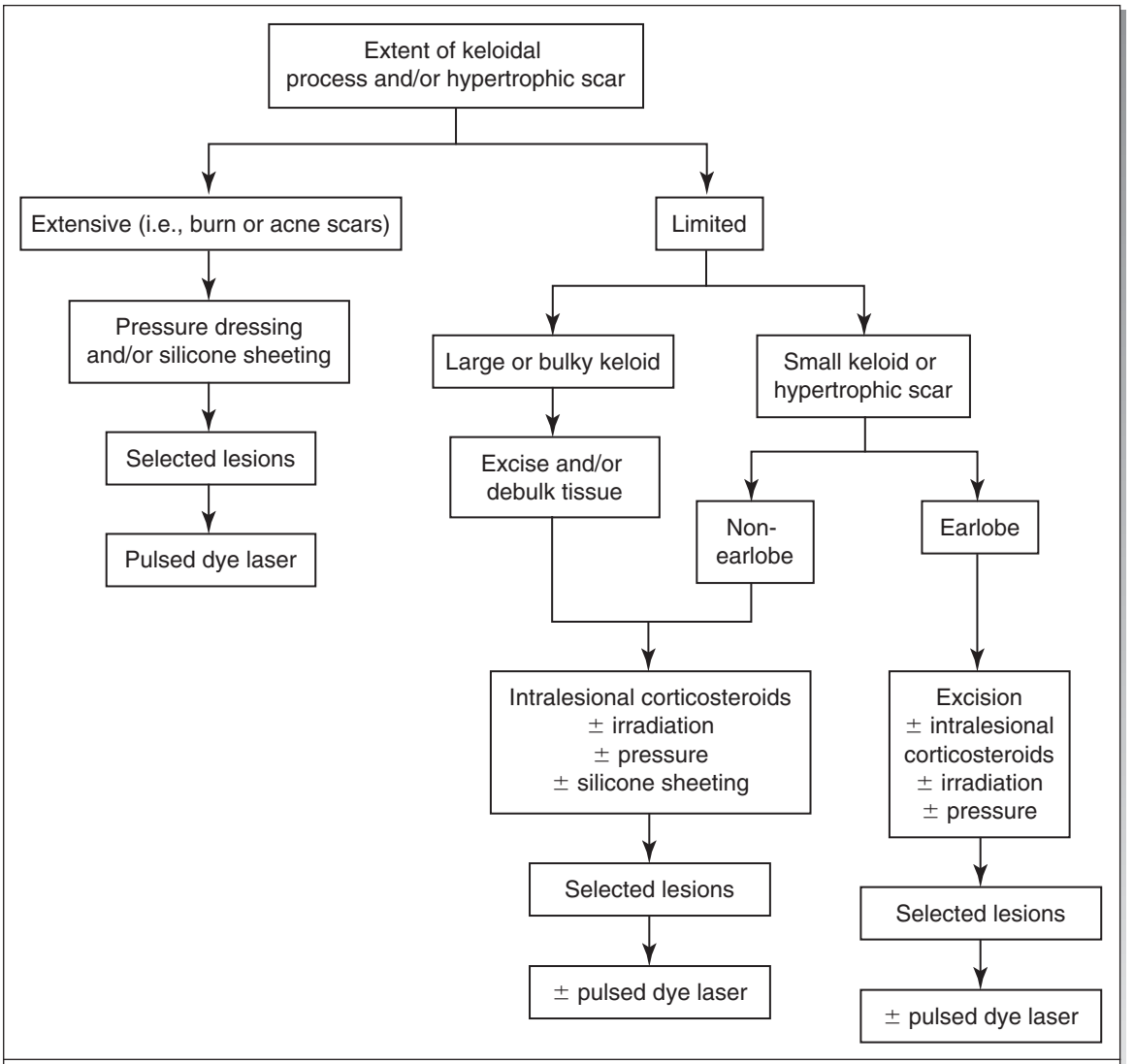

Figure 4 • Treatment algorithm for hypertrophic and keloid scars. (Modified from Urioste SS, Arndt KA, Dover JS: Keloids and hypertrophic scars: Review and treatment strategies. Semin Cutan Med Surg 18:159-171, 1999.)

ated in the treatment of keloids and hypertrophic scars. However, because of inconclusive and conflicting data, as well as poorly designed and incomplete studies, no meaningful demonstration of effectiveness has been provided.

Imiquimod

Imiquimod, the first member of the new class of immune response modifiers, stimulates innate and cell-mediated immune pathways and thereby results in potent antiviral, antitumor, and immunoregulatory properties. Imiquimod 5% cream, currently approved for the treatment of genital and perianal warts, has shown promise as an adjunctive treatment of keloids. It acts through the induction, synthesis, and release of cytokines such as IL-12, TNF-α, and IFN-α, particularly its induction of local levels of the latter.

After the surgical excision of keloids with primary closure, the application of imiquimod 5% cream directly to the suture line and the surrounding tissue each day for a total of 8 weeks led to no recurrences in preliminary studies. Although future studies with longer follow-up periods and comparison with other treatment modalities such as triamcinolone and radiation treatment after excision are warranted, it appears that topical application of imiquimod 5% cream is safe and effective in minimizing keloid recurrence after surgical excision.

Even though many therapeutic options for hypertrophic and keloid scars are available, the long-term efficacy of most remains to be determined. Controlled studies with adequate follow-up periods are needed to compare different treatment modalities as either single or combination therapy. A practical treatment approach to hypertrophic and keloid scars is to determine first whether the main treatment

goal is symptom relief or elimination of the scar and reduction of recurrence. Intralesional corticosteroids are effective in the treatment of pruritus and the associated burning sensation. Most hypertrophic and keloid scars treated by surgical excision also undergo combination therapy to reduce recurrences. A current practical approach to the management of hypertrophic and keloid scars is illustrated in Figure 4.

Pearls and Pitfalls

- Hypertrophic scars and keloids are best differentiated by history and serial examination.
- Hypertrophic scars most often improve with time or treatment.
- Silicone pressure therapy is a useful modality.
- Steroid injections most often have a beneficial effect in relieving pruritis and pain symptoms.
- Surgery alone should be avoided. It is most useful when combined with adjunctive treatments including pressure, steroids, or irradiation.

SUGGESTED READINGS

Berman B, Bieley HC: Keloids. J Am Acad Dermatol 33:117, 1995.

Ketchum LD: Hypertrophic scars and keloids. Clin Plast Surg 4:301, 1977.

Peacock EE Jr, Madden JW, Trier WC: Biologic basis for the treatment of keloids and hypertrophic scars. South Med J 63:755, 1970.

Rockwell WB, Cohen IK, Ehrlich HP: Keloids and hypertrophic scars: A comprehensive review. Plast Reconstr Surg 84:827-837, 1989.

Urioste SS, Arndt KA, Dover JS: Keloids and hypertrophic scars: Review and treatment strategies. Semin Cutan Med Surg 18:159-171, 1999.

Lymphedema

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The lymphatic system is responsible for returning fluid, proteins, and other large particulate matter from the interstitium to the intravascular compartment. It is also an integral component of the immune system in which foreign bodies are presented to lymphocytes to elicit an immune response. Lymphedema results from dysfunction of flow through the lymphatic channels in the face of normal capillary filtration. As such, not all cases of edema represent true lymphedema. Most cases of edema are due to physiologic factors that increase capillary filtration, such as increased capillary hydrostatic pressure, decreased capillary colloidal pressure, or increased capillary permeability. In these settings, capillary filtration overwhelms normal lymphatic channels with resultant edema formation. There is currently no cure for lymphedema; medical and surgical treatment focuses on reducing lymph formation, improving lymph drainage through existing lymphatic vessels, and preventing complications.

Etiopathogenesis

Lymphedema is categorized as primary or secondary. Primary lymphedema refers to cases of developmental lymphatic dysfunction or cases in which no underlying cause is identified. Primary lymphedema is subdivided according to the age of onset. Congenital lymphedema refers to edema that is present at birth or appears within the first 2 years of life. The majority of patients have bilateral involvement. *Milroy's disease* is an autosomal dominant form of lymphatic aplasia that manifests at birth as extremity edema. The most common form of primary lymphedema, *lymphedema praecox*, occurs around the time of puberty. It accounts for approximately 90% of all cases and often has unilateral involvement. *Lymphedema tarda* occurs after the age of 35 and accounts for less than 5% of cases. Primary lymphedema is more common in females.

Secondary lymphedema refers to cases in which an underlying cause can be identified. In the United States, the most common cause is edema after axillary lymph node dissection. Worldwide, however, filariasis is the most common cause of lymphedema. Tumors, trauma, and irradiation can also lead to lymphedema. Less common causes include tuberculosis, pregnancy, rheumatoid arthritis, and lymphogranuloma venereum.

Pathophysiology

Lymphedema is due to either a developmental or an acquired dysfunction of the lymphatic system. In the extremity, lymphatic vessels are located throughout the dermis and subcutaneous tissue. These superficial vessels drain into deep lymphatic vessels located in the epifascial plane that lies just above the deep fascia. There is also a subfascial system of lymphatics responsible for draining the muscles, fascia, bones, joints, ligaments, and tendons. These two systems normally function independently of one another. In lymphedema, the dysfunction in lymphatic flow is almost always restricted to the epifascial system.

Impairment of lymph flow leads to accumulation of interstitial fluid, proteins, and macromolecules in the dermis and subcutaneous tissue. The increased concentration of proteins and macromolecules further compounds the problem by increasing oncotic pressure and thereby leading to additional fluid accumulation. The stagnant lymph fluid also serves as a medium for bacteria. Because clearance of bacteria is impaired, a prolonged local inflammatory state persists. This results in recurrent damage to the lymphatic system and sets the stage for further episodes of cellulitis.

Chronic lymphedema leads to a local increase in fibroblasts, adipocytes, and macrophages within the dermis and subcutaneous tissue. These cells induce a chronic inflammatory state that leads to subcutaneous fibrosis, skin thickening, and hyperkeratosis. In the early stages of lymphedema, the skin is often soft and the lymph fluid is mobile, and pitting edema results. Over time, however, the skin and subcutaneous tissue progressively become indurated and fibrotic as the degree of pathologic collagen deposition increases.

Patient Evaluation/Diagnostic Studies

The diagnosis of lymphedema is usually ascertained by history and physical examination. In most cases, the edema initially occurs in the foot or ankle region and slowly progresses proximally. The edema often does not improve with leg elevation or with diuretics, and the patient may complain of increased pressure and fatigue in the extremity. Pain is not characteristic of lymphedema unless associated with cellulitis or lymphangitis. A family history of lymphedema should be ruled out, as well as a history of travel to countries where filarial disease is endemic.

Examination often reveals a firm and rubbery extremity with nonpittting edema. Hyperkeratosis, papillomatosis, or a peau d'orange texture of the skin may be apparent because of the subcutaneous edema and fibrosis. Lymphedema may also manifest as cellulitis or lymphangitis. It may be difficult to differentiate lymphedema in its early stages from other forms of edema because the chronic skin changes have not yet occurred.

In patients in whom the history and physical examination are not conclusive, diagnostic studies are indicated. Isotopic lymphoscintigraphy with radioactive iodine or technetium 99 is the most commonly used diagnostic test and is often considered the gold standard for the diagnosis of lymphedema. Magnetic resonance imaging and computed tomography may also aid in the diagnosis. The imaging techniques show a honeycomb distribution of edema limited to the epifascial planes, the characteristic absence of edema within the muscle compartment, and thickening of the overlying skin.

Lymphangiography was once the diagnostic tool of choice for lymphedema. It has since been shown to cause a severe inflammatory reaction in the lymphatic vessels that leads to further impairment in lymphatic drainage. Lymphangiography has been replaced by less invasive techniques, and its use is now limited to the preoperative evaluation of patients undergoing corrective surgery.

Lymphedema must be differentiated from other causes of edema. In chronic venous insufficiency, valvular incompetence leads to venous hypertension, which results in an increase in capillary filtration that overwhelms the lymphatic drainage system. Venous edema is characterized by pitting edema that progressively increases over the course of the day and improves on elevation. Hyperpigmentation and ulceration of the skin are also common. Systemic diseases such as congestive heart failure and renal disease should be differentiated by history and physical examination. Lipedema may also be mistaken for lymphedema. It is predominantly seen in woman and men with feminizing tumors. This type of edema is due to accumulation of fat in the subcutaneous regions of the hips, thighs, and legs and generally spares the feet.

Reconstructive Goals

There is no medical or surgical cure for lymphedema. Compliant patients who have exhausted conservative medical treatment without achieving the desired goals are potential candidates for surgery. Indications for surgery include impaired limb function, recurrent infection, intractable pain, lymphangiosarcoma, and cosmesis. The main goal of surgery is to improve function and quality of life. Surgery aims to decrease the incidence of cellulitis, reduce pain, and improve mobility, gait, and transfer. As a result of surgery, patient appearance, hygiene, and self-esteem should be enhanced.

Treatment

Medical

The goals of medical treatment include limiting the accumulation of edema fluid, improving existing lymphatic drainage, maintaining healthy skin, and avoiding complications such as cellulitis and lymphangitis.

In the early stages of lymphedema, leg elevation may be useful in reducing the buildup of edema fluid. As the disease becomes more chronic in nature, the benefits of elevation are lost. The main treatment used for reducing edema formation is compression garments. Stockings help to limit edema formation by opposing oncotic pressure and preventing backflow through existing lymphatic vessels. Recommended pressures range from 30 to 60 mm Hg. Patients should wear compression garments for 6 consecutive hours per day. Treatment with a diuretic has not been shown to be an effective long-term strategy for reducing lymph formation because the increased oncotic pressure in subcutaneous tissue results in rapid reaccumulation of edema fluid and progressive fibrosis.

Other treatment strategies are aimed at enhancing existing lymphatic drainage and recruiting

collaterals to return the edema fluid to the intravascular compartment. Complex decongestive physiotherapy (CDP) is the most comprehensive of these approaches. CDP consists of manual lymph massage, compressive dressings, exercise, and basic skin care to help to prevent infections. CDP works by stimulating existing lymphatic vessels in unaffected areas to drain the edematous tissue. Patients can expect an approximately 60% reduction in upper extremity and a 70% reduction in lower extremity lymphedema. The results are maintained in 90% of patients at 9 months. CDP is labor intensive and requires a licensed therapist, thereby limiting the practicality of its use in many patients.

Pneumatic compression devices have been used extensively in the treatment of lymphedema. Modern devices are multichambered and programmed to deliver sequential pressure that decreases by 10 mm Hg as one moves proximally along the extremity. Several studies support the role of pneumatic compression devices in the treatment of lymphedema, but others show no statistically significant difference. The degree of pathologic fibrosis present may account for this discrepancy. Patients with long-standing lymphedema and fibrosis may be refractory to this type of treatment, whereas those with lymphedema of recent onset may show improvement because of the lack of fibrosis. A recent randomized, prospective study demonstrated that the addition of pneumatic compression to CDP is more effective than CDP alone.

Patients with chronic lymphedema are prone to recurrent cellulitis and lymphangitis. The most common organism responsible for cellulitis in patients with lymphedema is β-hemolytic streptococci, followed by *Staphylococcus aureus*. Penicillin G or another β-lactam agent is the mainstay of treatment. The affected limb should be elevated and immobilized, and warm compresses should be applied for symptomatic relief. After a diagnosis of cellulitis is suspected, antibiotic therapy should be promptly initiated. Prophylactic use of antibiotics has not resulted in a reduction in the rate of infection.

The most common cause of secondary lymphedema worldwide is filariasis, which is due to transmission of *Wuchereria bancrofti* through a mosquito vector in endemic areas. Filariasis can be treated with four annual treatments of a single dose of diethylcarbamazine (6 mg/kg), and this treatment regimen also significantly reduces the clinical severity of lymphedema.

Pharmacotherapy as an adjunctive treatment strategy for nonfilarial lymphedema has, however, been disappointing. The ineffectiveness of diuretics has been discussed earlier. One class of drugs that has been used to treat lymphedema is the benzopyrones, specifically coumarin. This drug causes proteolysis by stimulating macrophage activity. The reduced protein concentration results in lower interstitial oncotic pressure and less edema formation. Previous studies have shown that benzopyrones clinically reduce limb edema; however, this effect remains unproven. The U.S. Food and Drug Administration does not currently approve the use of benzopyrones for the treatment of lymphedema, although additional studies are ongoing.

Surgical

Numerous techniques have been described for the surgical treatment of lymphedema; however, no procedure is curative. The patient must understand that these procedures are palliative and that aggressive medical management must be continued to ensure long-term reduction of lymphedema.

Excisional and debulking procedures are designed to remove redundant skin, subcutaneous tissue, and areas prone to repeated episodes of cellulitis. These procedures, however, do not improve lymphatic drainage. Physiologic procedures are designed to improve lymphatic drainage, and such procedures include the microsurgical creation of lympholymphatic and lymphovenous anastomoses to redirect lymphatic drainage. These procedures are obviously not indicated in patients with developmental defects in the lymphatic vessels, but they may play a role in secondary lymphedema.

EXCISIONAL OR DEBULKING PROCEDURES. Excisional procedures aim to remove redundant skin and subcutaneous tissue from the lymphedematous limb. Sir Richard Henry Havelock Charles described the first excisional procedure in 1912. It involved nearly total excision of the affected skin, subcutaneous tissue, and deep fascia to expose the underlying skeletal muscle, followed by coverage with split-thickness skin grafts. This procedure has many associated complications, and skin graft survival is unpredictable. The Charles procedure is reserved for patients who have poor skin quality and require extensive excision.

The most common excisional procedure currently used to treat lymphedema is staged skin and subcutaneous excision beneath skin flaps, as first described by Sistrunk in 1918 and later modified by Homans. This procedure effectively reduces the extent of lower extremity lymphedema with minimal associated complications and has become the mainstay of treatment. The results of staged excision in the upper extremity, however, have not been as promising. The skin and subcutaneous excisional procedure is usually performed in two stages. The first involves excision of the medial aspect of the extremity because a larger resection can be performed; this stage is followed by lateral resection 3 to 4 months later. The patient is admitted 1 to 3 days before surgery, and aggressive measures are taken to reduce the degree of lymphedema, including a combination of extremity immobilization, elevation, compression dressings, and pneumatic compression devices. These maneuvers result in increased skin

laxity and allow for more extensive excision. In an effort to reduce the length of hospital stay, the patient may perform some of these measures before admission.

The extremity is exsanguinated and a pneumatic tourniquet is placed on the proximal part of the thigh. An incision is made from the upper medial portion of the thigh and extended to a point 1 cm distal to the medial malleolus. Anterior and posterior flaps (1 to 2 cm in thickness) are elevated to the midsagittal plane. The subcutaneous tissue and deep fascia under the flaps are excised. Injury to the sural nerve can occur if it is not properly identified before resection of the subcutaneous tissue overlying the calf. Minimal resection of the deep fascia surrounding involved joints should be performed to reduce the risk of joint violation. The redundant skin is resected. Before closure, drains are placed in the dependent aspects of the extremity both proximally and distally. The skin is closed. Dermal sutures are not used because they can induce a prolonged inflammatory response in the incision. The leg is splinted and elevated. The procedure is repeated in 3 to 4 months on the lateral aspect of the leg. Care must be taken to preserve the sensory branches of the superficial peroneal nerve during this stage of the procedure.

In the United States, the most common form of lymphedema is secondary to axillary lymph node dissection. Some type of upper extremity lymphedema develops in approximately 6% to 40% of patients undergoing this procedure. With the poor success of staged excision in upper extremity lymphedema, liposuction has emerged as an effective treatment modality. The results appear to be long lasting, and liposuction does not seem to lead to further impairment in lymphatic flow. The importance of compression therapy after a surgical procedure is noteworthy because the reduction in lymphedema achieved by liposuction combined with compression therapy is significantly greater than treatment with liposuction alone.

The procedure is performed by making multiple 3-mm incisions spaced evenly throughout the affected extremity. Cannulas with a diameter of 3 to 4 mm are connected to a vacuum pump, and liposuction is performed in a circumferential manner beginning at the distal end of the extremity. The incisions are left open to allow for passive drainage. An elastic compression dressing is applied.

PHYSIOLOGIC PROCEDURES. Physiologic procedures aim to restore lymphatic drainage in the affected extremity. Early descriptions of physiologic procedures include the subcutaneous implantation of silk threads to facilitate lymph drainage (lymphangioplasty) and omental transposition flaps. The Thompson buried dermal flap is a combined excisional and physiologic procedure. Skin flaps are raised similar to the Homans procedure; however, instead of being completely excised, they are deepithelialized and buried in the underlying muscle in an attempt to connect the epifascial and subfascial lymphatic vessels. The effectiveness of this procedure, however, has not been established.

With the development of microsurgery, the possibility of lymphatic reconstruction was introduced. Multiple techniques have been described, including lymph node–venous, lymphovenous, and lympholymphatic shunts. Subjective improvement in appearance, objective reduction in volume, and a decreased incidence of recurrent infection have been reported. Some surgeons have reported long-term patency in their microlymphatic anastomoses by lymphoscintigraphy. However, until direct confirmation of long-term patency can be demonstrated in a large, multicenter study, the role of microlymphatic reconstruction remains limited.

In cases of secondary lymphedema, especially those caused by filiarial disease, benefit has been demonstrated with combined physiologic and reduction surgery. The patient first undergoes a physiologic procedure, specifically a lymph node–venous anastomosis, followed by a second-stage excisional (Homans') procedure.

For the first stage, an incision is made in the groin and a large lymph node is identified. The node is transected, and a curet is used to core out the node. It is anastomosed end to end to the saphenous vein stump. The extremity is elevated postoperatively, and compression garments, massage, and pneumatic devices are used for at least 3 days. During this time, significant diuresis, often several liters of urine output, is noted, along with a significant reduction in extremity size. After 3 to 4 days, the patient undergoes the second-stage excision procedure. This procedure has resulted in significant success in many patients, and long-term lymphovenous patency is not required.

Postoperative Care/Complications

After the staged skin and subcutaneous excisional procedure, the patient is placed on strict bed rest with the extremity immobilized in a splint and elevated. The drains are left in place for 3 to 5 days and the sutures are removed by day 10. Antibiotics are continued until the drains are removed. The patient is fit for an elastic stocking. Dependent ambulation can be started between days 10 and 12 and increased gradually. The elastic dressings must be worn continuously for the next 3 to 6 weeks to reduce seroma formation. Once the extremity has healed, the patient continues with compression therapy during the day and leg elevation at night.

Patients who have undergone liposuction for upper extremity lymphedema should continue arm elevation for 5 to 7 days. The patient should be remeasured for a compression dressing (32 to

40 mm Hg) before discharge and should continue its use indefinitely.

Postsurgical complications include seroma, hematoma, wound dehiscence, flap necrosis, wound infection, and exacerbation of the lymphedema.

The most dreaded sequela of chronic lymphedema is Stewart-Treves syndrome (STS), or the development of lymphangiosarcoma in patients with postmastectomy lymphedema. STS is estimated to have an incidence of 0.5% and develops, on average 9 years after the onset of lymphedema. It is hypothesized that chronic lymphedema provides an environment of impaired immune function that may lead to malignant transformation. The lesion often appears as a raised, red lesion that is surrounded by an area of discoloration. Suspicious lesions in a lymphedematous extremity require multiple deep biopsies to establish the diagnosis. Treatment traditionally involved radical amputation of the arm; however, no significant difference in survival has been demonstrated in patients who underwent radical amputation versus wide excision with 2- to 3-cm margins. The mean length of survival in treated patients is approximately 20 months.

Pearls and Pitfalls

- Lymphedema is an uncurable disease.
- Conservative medical management should be exhausted before surgical treatment is considered.
- Surgery serves only to enhance medical treatment. The ultimate goal of surgery is to improve quality of life.
- Liposuction with compression therapy is a useful modality for the treatment of upper extremity lymphedema.
- Microlymphatic reconstruction has limited application until its long-term efficacy is fully evaluated.

SUGGESTED READINGS

Brorson H: Liposuction gives complete reduction of chronic large arm lymphedema after breast cancer. Acta Oncol 39:407-420, 2000.

Campisi C, Boccardo F: Lymphedema and microsurgery. Microsurgery 22:74-80, 2002.

Greenfield LJ: Lymphatic system disorders. In Greenfield LR (ed): Surgery: Scientific Principles and Practice, 2nd ed. Philadelphia, Lippincott-Raven, 1997, pp 1966-1971.

Miller TA, Wyatt LE, Rudkin GH: Staged skin and subcutaneous excision for lymphedema: A favorable report of long-term results. Plast Reconstr Surg 102:1486-1498, 1998.

Rockson SG: Lymphedema. Am J Med 110:288-295, 2001.

Burns

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Of the more than 1.2 million people burned in the United States each year, approximately 60,000 are hospitalized and 5000 die. Mortality, however, has significantly improved since 1971, and deaths attributed to burns have decreased more than 40%. This is primarily due to preventive measures and advanced critical care. Improved survival results in more patients with problematic reconstructive and functional needs. The morbidity of burn injury ranges from devastating functional impairment to severe cosmetic deformity.

Etiopathogenesis

Burn wounds can be caused by the following sources: scald (hot liquids), flame (including flammable liquids and explosions), contact with a hot surface, electrical (including lightning), chemical, and sunburn. High-risk populations are those least able to avoid or escape burn threats: children, the elderly, the disabled, and military personnel. Recent emphasis on prevention of burn injury has contributed to a decline in morbidity and mortality. Flame-retardant clothing, cooler requirements of home water heaters, fire-safe cigarettes, smoke detectors, and fire sprinklers are proven prevention devices. Although emphasis on prevention should be a mainstay of burn care, the reality is that the majority of medical and surgical intervention is directed toward the postburn event.

Pathologic Anatomy

Burn wounds are classified by their depth of penetration. *First-degree* burns involve the epidermis only and result in erythema of the skin. Although desquamation may occur at a later time, initial blistering is absent. *Second-degree* or partial-thickness burns are subdivided according to the degree of severity based on the depth of the dermis involved. Superficial partial-thickness burns involve the papillary dermis, with most of the reticular dermis being spared, including the hair follicles and sebaceous and sweat glands. In contrast, deep partial-thickness burns destroy most of the reticular dermis and the majority of skin appendages. In *third-degree* or full-thickness burns, the entire dermis is destroyed, including the adnexal structures.

Clinical assessment remains the gold standard, but it may be difficult to differentiate the level of injury. Partial-thickness wounds are sensate. Superficial wounds blanch with touch, demonstrate capillary refill, and may also blister. Deep partial-thickness burns have diminished sensation, do not demonstrate capillary refill, and appear white in color. These wounds can produce blistering. Full-thickness burns do not blister and are charred with a gradation of color from white to brown or even black. There is little to no sensation. Burns that extend beyond the skin to involve deeper structures may exhibit a fixed red color as a result of coagulation of blood in the subdermal plexus. Careful initial examination of burn wound depth is critical for accurate calculation of resuscitation requirements and preoperative planning.

Burn wounds are divided histologically into three zones: a zone of coagulation (necrosis), a zone of stasis (edema), and a zone of hyperemia (inflammation). The zone of coagulation is characterized by nonviable necrotic tissue. The zone of stasis surrounds the zone of coagulation and is initially viable. The latter is clinically relevant because aggressive resuscitation can aid in perfusion of this tenuous tissue and prevent complete necrosis. The outermost zone of hyperemia is viable and has increased blood flow with a subsequently erythematous appearance.

Diagnostic Studies

Many burn patients experience other complicating trauma, and radiologic studies such as computed tomography and ultrasound can play an important role in evaluating the patient for intracranial, thoracic, or abdominal injury. Plain radiographic films should be obtained to assess for fractures or foreign bodies.

Initial Assessment

The initial evaluation of a burned patient should include a careful history and a complete physical examination. For patients with facial burns, fluorescent staining of the cornea is necessary. Extremities should be examined for signs of compartment syndrome. Wick compartment testing is often used, and a pressure higher than 30 mm Hg is an indication for escharotomy or fasciotomy. When inhalation injury is suspected, pulmonary assessment should include arterial blood gas evaluation, a chest x-ray, determination of carboxyhemoglobin, and bronchoscopy if indicated.

Careful calculation of the thermally injured body surface area is necessary to guide volume resuscitation. For adults, the rule of nines (Fig. 1) is a simple and accurate method of calculating the total surface area burned. However, in children, the Lund and Browder chart (Fig. 2) is more accurate because it accounts for the different body proportions in infants and children. For all burn patients, a burn diagram should be placed in the medical record and updated if the wound extends over time.

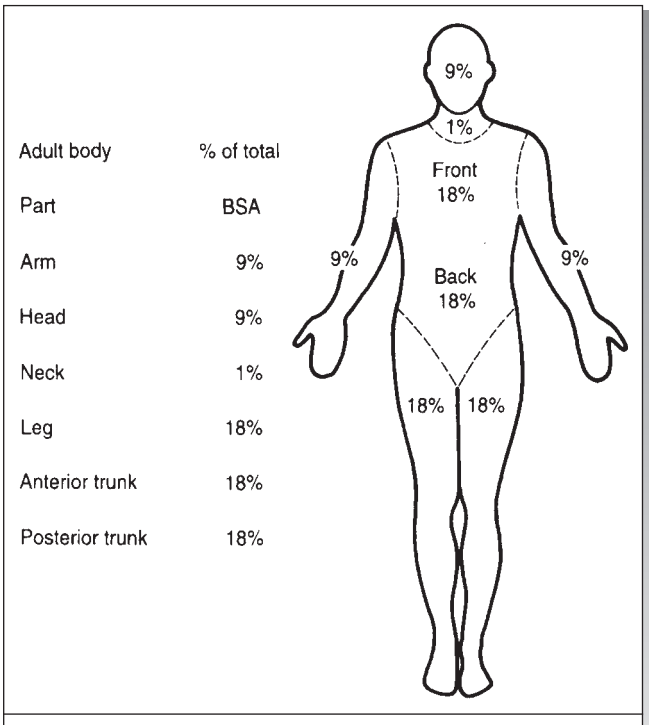

Figure 1 • Estimation of burn size with Wallace's "rule of nines." BSA, body surface area. (From Herndon DN [ed]: Total Burn Care, 2nd ed. Philadelphia, Saunders, 2002.)

Prevention of shock is of paramount importance. Routine assessment of electrolytes is necessary because large intravascular volume shifts occur. Intraoperative and postoperative care centers around preservation of intravascular volume, prevention of infection, and maintenance of the high metabolic demands of the burned patient. Sputum, blood, urine, and tissue cultures are critical to direct optimal antimicrobial therapy. Serum protein and albumin levels are useful in assessing nutritional needs.

Specific burn circumstances may dictate further diagnostic studies. For high-voltage electrical injuries, an electrocardiogram and continuous cardiac monitoring are indicated. Renal function should be monitored with urine myoglobin measurements. Chemical burns can mandate specific diagnostic testing. For example, serum calcium should be serially measured in patients with hydrofluoric acid burns because life-threatening hypocalcemia can occur.

Treatment

Treatment of a burned patient begins at the scene of the injury with safe removal of the patient from the heat source. Jewelry, watches, and belts that can retain heat and constrict the extremities should be removed. Inhalation injury is assumed, and 100% oxygen is administered by facemask. Heat can be dissipated in the burned area with clean water. Cold water or ice should not be used because hypothermia can quickly result. For chemical burns, the caustic agent, including all contaminated clothing, should be removed. The chemical agent should be identified so that the proper neutralizing solution can be used.

The airway should be secured because upper airway obstruction can develop quickly. Progressive hoarseness mandates endotracheal intubation. Intubation is also necessary for unconscious patients or those experiencing respiratory distress. For patients with possible spinal cord injury, cervical spine precautions are necessary.

After the patient arrives at the emergency department, the decision whether to transfer to a burn center should be made (Table 1), and fluid resuscitation with lactated Ringer's solution should be initiated. For adults, the Parkland formula is a widely accepted means of calculating fluid requirements in the first 24 hours after a burn injury (Table 2). For children, the Galveston formula provides a more accurate estimate based on body surface area (see Table 2). Adequate intravenous access should be established to accommodate these fluid

Age	0–1	1–4	5–9	10–14	15
A – $\frac{1}{2}$ of head	$9\frac{1}{2}$%	$8\frac{1}{2}$%	$6\frac{1}{2}$%	$5\frac{1}{2}$%	$4\frac{1}{2}$%
B – $\frac{1}{2}$ of one thigh	$2\frac{3}{4}$%	$3\frac{1}{4}$%	4%	$4\frac{1}{4}$%	$4\frac{1}{2}$%
C – $\frac{1}{2}$ of one leg	$2\frac{1}{2}$%	$2\frac{1}{2}$%	$2\frac{3}{4}$%	3%	$3\frac{1}{4}$%

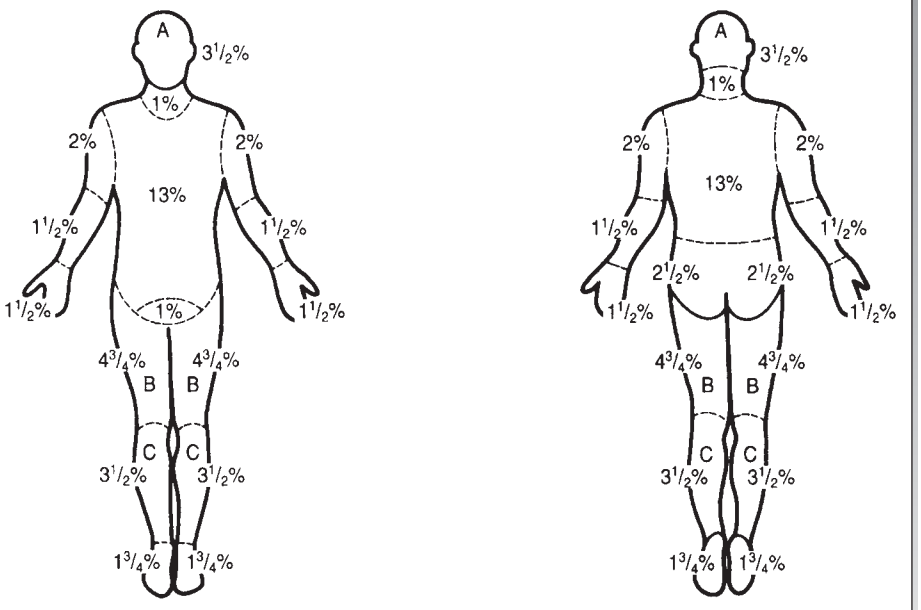

Figure 2 • Estimation of burn size with the Lund and Browder method. (From Herndon DN [ed]: Total Burn Care, 2nd ed. Philadelphia, Saunders, 2002.)

requirements. If central venous access is used, a chest x-ray should be obtained. The extremities should be evaluated for possible compartment syndrome and escharotomies or fasciotomies performed if necessary. Antimicrobial therapy and tetanus prophylaxis should be administered.

For first-degree and superficial second-degree burns, conservative management is indicated. These wounds should be cleaned and gently débrided with sterile saline gauze. The choice of wound dressing varies considerably and includes topical antimicrobials, biologic dressings, Vaseline gauze, or a skin substitute such as Biobrane (nylon mesh and Silastic bilaminar membrane). At our institution, Biobrane has proved to be a reliable and relatively painless method of treating superficial burns. Drawbacks include availability and the need to initiate treatment within the first 24 hours after the burn.

TABLE 1 Criteria for Transfer of a Burn Patient to a Burn Center

- Second-degree burns greater than 10% total body surface area
- Third-degree burns
- Burns that involve the face, hands, feet, genitalia, or perineum and major burns
- Chemical burns
- Electrical burns, including lightning injuries
- Any burn with concomitant trauma in which the burn injuries pose the greatest risk to the patient
- Inhalation injury
- Patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality
- Hospitals without qualified personnel or equipment for the care of burned children

TABLE 2 Fluid Calculations

PARKLAND FORMULA (ADULTS)

4 mL/kg/% BSA burned
Give half the total amount over the first 8 hours from the time of injury and the second half over the remaining 16 hours

GALVESTON FORMULA (PEDIATRIC PATIENTS)

5000 mL/m^2 BSA burn + 2000 mL/m^2 total BSA
Give half the total amount over the first 8 hours from the time of injury and the second half over the remaining 16 hours

BSA, body surface area.

Although this dressing is expensive, it can result in decreased length of stay with subsequent institutional savings.

Goals of Reconstruction

The most important step in burn reconstruction takes place at the initial excision. Although early excision has become a mainstay of modern burn care, it should be avoided in both the hands and face. An aggressive approach to either of these highly visible areas results in a less favorable functional and aesthetic outcome. In addition, the extent of excision should not exceed the ability to cover the wound with either autograft or an appropriate biologic dressing or skin substitute. Burn reconstruction should always be considered before the initial excision.

In reconstruction of the hands, function is paramount to all other concerns. Delayed excision can often preserve much needed length of the digits. Most important is maintenance of an opposable thumb or digit. Long-term functional impairment results from scar contracture, which can often be prevented by early splinting. Care must be taken to maintain a protected hand position (wrist extension, thumb abduction, metacarpophalangeal joint flexion, and interphalangeal joint extension) throughout the course of treatment. The role of therapists cannot be overstated in the long-term functional outcome of patients with hand burns.

For deep second- and third-degree burns, treatment is directed at excising and resurfacing the wound. When possible, early excision and grafting have resulted in decreased mortality and length of hospital stay. The decision regarding how much to excise at a single setting is determined by the patient's comorbid conditions, the resources of the hospital or blood bank, and the ability to cover the excised burn wound. Care must be taken to avoid excision of viable tissue and to limit blood loss. For partial-thickness wounds, the goal of *tangential excision* is achieved by conservative shaving of burned tissue until a viable wound bed is reached, usually after repeated passes with a dermatome set at intermediate depth or with a guarded skin knife until punctate dermal bleeding occurs. Full-thickness excision requires identifying a plane between the burn eschar and viable subcutaneous fat. Fascial excision is reserved for burns extending through subcutaneous fat into muscle. With the use of electrocautery, a fascial plane is identified and the overlying tissue removed. Although blood loss is less extensive with this method, it leaves a permanent contour deformity. For burns that destroy a limb or digit beyond salvage, amputation is indicated as a last resort.

The extent of excision should never exceed the ability to cover the excised wound. Autologous skin grafts are the gold standard for closing burn wounds because they provide permanent and durable coverage. Skin grafts adhere to the wound bed through the processes of plasmatic imbibition (first 48 hours), inosculation (postgraft days 2 to 3), and capillary ingrowth (postgraft day 4). Skin grafts can be harvested as split-thickness or full-thickness grafts. Split-thickness grafts are routinely used in the acute burn setting when large areas need to be covered. They can be widely meshed to allow coverage of large areas. With proper donor site care, split-thickness skin grafts can be reharvested every 5 to 7 days, a property ideal for burns in patients with limited donor sites. Full-thickness grafts require primary closure; therefore, their role in the acute setting is limited. Full-thickness grafts are better suited for reconstructive purposes because of their resistance to contracture.

When the size of the burn wound exceeds available or acceptable autologous donor sites, allograft can be used. In addition to providing temporary coverage, allograft aids in alleviating pain and encouraging granulation of the wound bed. Xenograft, skin, and human placenta can also be used as temporary biologic dressings. For massive burns (>90%) in patients with limited donor sites, cultured epithelial autografts can provide permanent wound closure. This technology is expensive and at this time has not been proved to be efficacious. Synthetic skin replacements such as Integra (acellular bilaminar membrane) and AlloDerm (decellularized sterile human dermis) provide dermal replacement for full-thickness burns but require a thin split-thickness skin graft for epithelial coverage.

Unique Burns

Chemical burns differ from thermal burns in that tissue damage can continue long after the source is removed. Copious irrigation with water is the first and most important step in management. Injuries involving large body surface areas may require immersion in a tank. Electrolytes must be monitored to prevent dangerous imbalances. Dry powder should be brushed away before irrigation. All affected clothing should be carefully removed so that unaffected skin is not contaminated

Alkali substances (lime, potassium hydroxide, bleach, and sodium hydroxide) are the most common agents implicated in chemical injuries. Copious irrigation is mandatory because the alkali penetrates deeply into the wound. Neutralization with weak acids is contraindicated because the heat from this reaction causes intensification of the burn. Cement (calcium oxide) burns are also common and treated in a manner similar to alkali burns.

Acid burns induce protein denaturation by hydrolysis and form a hard eschar that prevents deeper penetration; irrigation prevents additional

tissue damage. Hydrofluoric acid poses a unique problem because it chelates calcium and magnesium. Calcium gluconate (2.5%) gel or calcium gluconate (10%) intradermal injections should be used after initial water irrigation. All patients with hydrofluoric acid burns should be placed on a cardiac monitor and observed for prolongation of the QT interval. Serum calcium, magnesium, and potassium should be frequently checked. Phenol is a powerful oxidizing agent and causes extensive and deep tissue damage. In addition to irrigation, treatment with polyethylene glycol is helpful to prevent further injury to underlying tissue. Phosphorus is commonly found in military settings. After irrigation, phosphorus particles should be removed from the wound. A solution of 0.5% copper sulfate turns the particles black for easy identification and removal.

Electrical burns are unique because the visible injury represents only a small fraction of the injured tissue. The electrical current flows through the body along the path of least resistance: nerves, blood vessels, and muscles. Ventricular fibrillation is the most common cause of death in high-voltage injury, thus mandating cardiac monitoring. Because alternating current sources can cause violent muscle contractions, long bone fractures can occur. Compartment syndrome is common with injury to deep tissues. Fasciotomy and early débridement of nonviable tissue are necessary to preserve an affected extremity. Muscle damage results in release of myoglobin, which can cause obstructive nephropathy. Urine output should be maintained at 30 to 50 mL/hr with alkalization of urine by the intravenous administration of sodium bicarbonate.

Postoperative Care

Estimated blood loss for burn excision and grafting is approximately 0.5 mL/cm^2. Maintenance of intravascular volume and end-organ perfusion is critical in the perioperative period. Intraoperative blood loss can be limited by careful hemostasis and the use of tourniquets when excising an extremity. However, even with careful technique, blood product replacement is usually necessary. The patient's environment should be heated to maintain an adequate core body temperature because hypothermia is a common cause of coagulopathy. When necessary, invasive monitoring with a central venous pressure or pulmonary artery catheter can be performed. Urine output should be maintained at 1.0 to 2.0 mL/kg/hr.

Because of the skin's function as the primary barrier to microbial invasion, prevention of infection is still one of the most difficult challenges in burn care. Systemic and topical antibiotic therapy remains a mainstay of postoperative care. When possible, quantitative tissue culture should be performed to evaluate burn wound infection. Typically, a wound with greater than 10^5 organisms per gram of tissue does not heal; however, streptococcal species are harmful at any detectable amount. Selection of systemic antimicrobial therapy should be based on culture and reflective of resistance patterns specific to the institution.

Topical antimicrobials should be used for dressing a burn wound. Commonly used topical antimicrobials include silver sulfadiazine, mafenide acetate, 0.5% silver nitrate solution, and sodium hypochlorite. Silver sulfadiazine is soothing and has a broad spectrum of coverage, but it penetrates eschar poorly, a property that limits its effectiveness. Transient leukopenia is a common side effect. Mafenide acetate provides excellent coverage for most gram-positive organisms, readily penetrates eschar, but is painful when applied. Mafenide acetate is a potent carbonic anhydrase inhibitor and causes hyperchloremic metabolic acidosis and compensatory hyperventilation if applied over greater than 30% of the total body surface area. Silver nitrate solution (0.5%) is painless and effective for most *Staphylococcus* species and gram-negative aerobes, including *Pseudomonas*. Concentrations above 5% are histotoxic. Hyponatremia and hypokalemia can occur. Infrequently, methemoglobinemia can result and should be considered if the skin or blood appears cyanotic. At a concentration of 0.025%, buffered sodium hypochlorite is bactericidal without inhibiting native fibroblasts or keratinocytes. With liberal antibiotic use, selective pressure can lead to fungal infections, and nystatin cream or powder is commonly used for topical fungal infections.

Respiratory care begins at the initial burn assessment with a search for signs of inhalation injury. Facial burns, singed nasal hairs, carbonaceous sputum, and progressive hoarseness can indicate an inhalation injury. Endotracheal intubation should be performed early to prevent upper airway edema and loss of the airway if an inhalation injury is suspected. After intubation, surgery can proceed once adequate resuscitation is achieved. Extubation criteria after burn injury include the following: Pa_{O_2}/F_{IO_2} ratio greater than 250, maximum inspiratory pressure higher than 60 cm H_2O, spontaneous tidal volume greater than 5 to 7 mL/kg, and spontaneous vital capacity greater than 15 to 20 mL/kg.

The normal metabolic rate can be increased by as much as 200% after an extensive burn injury and may continue long after the burn wound has been closed. The increased energy requirement manifests clinically as increases in oxygen consumption, metabolic rate, urinary nitrogen excretion, lipolysis, and weight loss. The resulting caloric debt is met by mobilization of carbohydrate, fat, and protein stores, thereby leading to their depletion. The resultant malnutrition is reflected by delayed wound healing, organ failure, and immune system failure. Therefore, the nutritional demands of burn patients must be addressed early and accurately.

The Curreri formula is widely used for calculating the caloric needs of burn patients: 25 kcal/kg/day + 40 kcal/% total body surface area/day with 1 to 2 g/kg/day of protein to provide a calorie-to-nitrogen ratio of 100:1. Carbohydrates and fats make up the remainder of the nonprotein caloric intake. As a general rule, enteral feeding is vastly superior to total parenteral nutrition in a burn patient.

Complications

Burn care complications can be categorized as those involving the critical care process and those related to excision and grafting. The most common cause of death in severely burned patients is pneumonia resulting from either a burn injury to the tracheobronchial tree or hematogenous dissemination. The diagnosis can be problematic because sputum samples are often contaminated with oropharyngeal flora and the chest x-ray displays a diffuse pattern from increased pulmonary vascular permeability. The best diagnostic tool is bronchoalveolar lavage, which not only provides organism-specific information but also cleanses purulent material from the lung.

Sepsis may result from seeding of the blood stream from the burn wound, respiratory tract, gastrointestinal tract, urinary tract, or central venous catheters. The five cardinal signs of sepsis are hyperventilation, thrombocytopenia, hyperglycemia, obtundation, and hypothermia. Blood cultures should be obtained but may be negative in critically ill patients. Inotropic support is often necessary as a result of decreased systemic vascular resistance and hypotension.

Gastrointestinal complications are common because a burn injury causes mucosal atrophy and increased intestinal permeability. Mucosal ulceration and bleeding in the stomach and duodenum can be prevented with the early use of antacid therapy and early enteral feeding, which also alleviates the caloric imbalance. However, reduced gut motility and ileus can be problematic with attempted enteral feeding, and frequent checking of gastric residuals is necessary.

Acute renal failure (acute tubular necrosis) from underperfusion of the kidneys is another potentially lethal complication. Approximately half of all burned patients who require dialysis die, usually of multiorgan system failure. Acute renal failure can be prevented by early and aggressive volume resuscitation to maintain urine output and by avoidance of nephrotoxic agents such as intravenous contrast and aminoglycosides.

Skin graft loss is the most frequent operative complication and is caused by hematoma, infection, or graft shear. When possible, a graft should be examined on postoperative day 3 and any underlying blood clot carefully aspirated. After this critical period, capillary ingrowth has occurred and would be interrupted with graft movement. If examination reveals that skin grafts appear to be grossly infected, quantitative cultures can be performed and the topical antimicrobial changed. Finally, skin grafts should be carefully immobilized with bolsters or splinting to prevent graft shear.

Pearls and Pitfalls

- Aggressive resuscitation can prevent the conversion of a deep partial-thickness injury to a full-thickness injury.
- The most accurate gauge of adequate volume resuscitation is urine output; urine output must be maintained at 1.0 to 2.0 mL/kg/hr to ensure adequate tissue perfusion.
- Reconstructive concerns must be taken into consideration at the time of initial resuscitation.
- The head and upper extremity are highly visible areas; sheet grafting is essential in this area to avoid an unsightly meshed scar pattern.
- The scalp is an excellent donor site if care is taken to avoid harvesting at a depth below the level of the hair follicle.
- A burned hand should be splinted early to prevent stiffness and contracture.
- The role of therapists in regaining hand function cannot be overstated.

SUGGESTED READINGS

Boswick JA (ed): The Art and Science of Burn Care. Rockville, MD, Aspen, 1987.

Brou JA, Robson MC, McCauley RL, et al: Inventory of potential reconstructive needs in the patient with burns. J Burn Care Rehabil 10:555-560, 1989.

Feldman J: Facial burns. In McCarthy JG (ed): Plastic Surgery, vol 3. Philadelphia, Saunders, 1990, pp 2153-2236.

Herndon DN (ed): Total Burn Care, 2nd ed. Philadelphia, WB Saunders, 2002.

Herndon DN, Barrow RE, Rutan RL, et al: A comparison of conservative versus early excision therapies in severely burned patients. Ann Surg 209:547-552, 1989.

Ryan CM, Schoenfeld DA, Thorpe WP, et al: Objective estimates of the probability of death from burn injuries. N Engl J Med 338:362-366, 1998.

Tobin MJ: Advances in mechanical ventilation. N Engl J Med 344:1986-1996, 2001.

Principles of Head and Neck Oncology

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The care of a patient with a head and neck mass is a therapeutic endeavor that involves a broad spectrum of medical specialists, including the reconstructive surgeon. Close collaboration with the referring physician, radiation oncologist, medical oncologist, and head and neck surgeon is essential for optimal treatment. The advances in plastic surgery made during the past 20 years have permitted radical resections to be undertaken while allowing the realistic expectation of a return of form and function. In addition, the reliable transfer of well-vascularized tissue beds has permitted the use of aggressive irradiation protocols while minimizing postirradiation complications. Less morbidity has also been achieved with more refined use of selective neck dissection, analogous to the movement away from radical axillary dissection for breast malignancies. Future use of such modalities as sentinel lymphadenectomy and molecular detection of micrometastases may enhance staging and management of patients with occult metastatic cervical disease. This chapter outlines the tenets of care of patients with a known head and neck neoplasm, with emphasis placed on how oncologic concerns influence reconstructive considerations.

Etiopathogenesis

Head and neck cancers represent a significant health care burden and account for at least 4% of all cancers diagnosed yearly in the United States. The majority of tumors in the head and neck region are squamous cell carcinomas (SCCs). Epidemiologic studies have demonstrated that, in the Western world, the cause of these cancers is mainly attributable to the carcinogens present in tobacco. Although alcohol by itself has not been shown to directly be a carcinogen, in combination with tobacco it plays a synergistic role in head and neck carcinogenesis and increases the risk for cancer to a level 15 times greater than that of the general population. Other causes include chronic inflammation, poor hygiene, ultraviolet radiation (a particularly common cause of lower lip SCC), viruses (such as human papilloma virus, in particular HPV-16), and in rare instances, familial factors. The importance of cumulative environmental insults in the pathogenesis of SCC is supported by the association of SCC with advanced age, given that it is most frequently diagnosed in patients older than 50 years.

Other tumors found in the head and neck, which in aggregate represent less than 10% of all head and neck malignancies, include basal cell carcinomas, verrucous carcinomas, salivary gland tumors, melanomas, sarcomas (including rhabdomyosarcomas, osteosarcomas, angiosarcomas, and histiocytomas), lymphomas, and thyroid malignancies. With the exception of lymphomas, the tumors listed are inevitably encountered by the plastic surgeon performing head and neck reconstructions. A confounding factor to a discussion of these tumor types is that they are a heterogeneous group linked only by their anatomic location in the head and neck.

The cause of head and neck cancers is an area of intense investigation. Most do not have an infectious cause. Exceptions include nasopharyngeal cancer, which is almost certainly caused by Epstein-Barr virus and accounts for a large percentage of head and neck tumors outside the United States, and the infrequent SCC occurring in nonsmokers, which is more likely to be associated with HPV than with tobacco and alcohol use. Because the most common head and neck malignancy is SCC, this chapter emphasizes the biology and treatment of this tumor type.

TABLE 1 Common Genetic Changes in Squamous Cell Carcinoma of the Head and Neck

GENE PRODUCT	FREQUENCY	LOSS OR GAIN OF FUNCTION	TYPE OF GENE AND EFFECT
$P16^{INK4a}$	80%	Loss	Oncogene; increased cell cycle progression and decreased senescence
EGF receptor (erb-B1)	<10%	Gain	Oncogene; increased growth and enhanced cellular aggressiveness
p53	45%-70%	Loss	Tumor suppressor; decreased apoptosis and improper regulation of the cell cycle
ras	25%	Gain	Oncogene; dysregulated cell growth and increased aggressiveness
Cyclin D1	30%	Gain	Oncogene; increase in cell cycle transit
p63	30%	Loss	Tumor suppressor; increases proliferation and reduces differentiation
PTEN	10%	Loss	Tumor suppressor; increases migration and modulates cell-matrix interactions

Molecular Biology

SCCs arise from epithelial cells, specifically the keratinizing layer. A hallmark of the cells on histologic examination is the presence of intracellular keratin bundles ("pearls"). Table 1 lists common genetic changes identified in SCC of the head and neck. These changes are all implicated in different stages of SCC progression, and furthermore, all are potential therapeutic targets. The accepted model of SCC progression from hyperplasia to invasive carcinoma initially proposed by Califano is useful because it illustrates that the clinical behavior of the tumor cells is associated with discrete genetic and molecular alterations at each stage (Fig. 1). It is estimated that 6 to 10 independent genetic changes are involved in the development of head and neck SCC. Of particular note, aberrations in the *p53* gene are strongly represented in carcinomas of the head and neck. People who smoke and drink and develop oral SCC have a greater tendency to have alterations in this tumor suppressor gene present in tumor cells. Other important oncogenes include the epidermal growth factor receptor (*EGFR*), which is overexpressed in more than 80% of head and neck SCCs, as well as the tumor suppressor $p16^{INK4A}$, which normally functions as a regulator of the cell cycle. Several precancerous lesions lead to SCC, including leukoplakia and erythroplakia. Ninety percent of erythroplakia lesions harbor foci of in situ SCC or invasive SCC. In contrast, only 10% of leukoplakia foci develop in situ carcinoma or frankly invasive tumor cells, and in line with the model of SCC progression described earlier, these lesions do not contain the advanced perturbations in protooncogenes present in more invasive, dysplastic lesions.

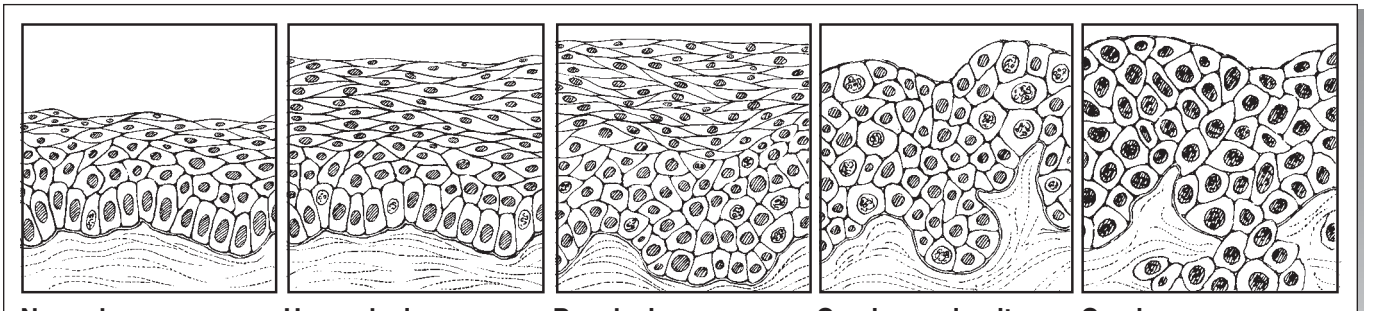


Figure 1 • Currently accepted model of histologic changes and associated molecular alterations involved in the pathogenesis of squamous cell carcinoma. Not depicted are chromosomal alterations, which are also found in each stage. Note that the progression depicted typically takes decades before carcinoma is reached. Furthermore, this model is applicable to the concept of field cancerization, where individual lesions may have any of the stages depicted.

Nowhere is the aphorism that you "can't chase cancer cells with a knife" more true than in the treatment of advanced head and neck tumors. The anatomic confines of the head and neck and the propensity for early metastases to the neck present formidable challenges to surgical resection and frustrate the ability to attain surgical margins free of residual disease. However, it is useless to perform an inadequate resection, and the goal of any surgical therapy should always be complete surgical resection to noninvolved margins. This aggressive approach is made possible by the availability of reconstructive options. Early lymphatic spread to local, regional, and distant lymph nodes mandates involvement of the radiation oncologist. Future improvements in patient survival will be attained not through improvements in surgical technique, but by the translation of the increased knowledge of the molecular pathogenesis of these tumors to therapeutics. In addition, the use of such technologies as polymerase chain reaction may facilitate detection of so-called micrometastases, which has implications for the future staging and treatment of early tumors. The era of molecular surgery is on the horizon, and it is therefore imperative that the surgeon understand the molecular aberrations responsible for head and neck cancer.

Pathologic Anatomy

The head and neck region is the most anatomically and functionally complex area of the human body. It includes the face, scalp, and neck externally, and within these investing layers a multitude of cavities and structures are present, including the orbital contents, nasal cavity, oral cavity, sinuses, and oropharyngeal area, as well as the brain. In general, SCC of the head and neck originates within either the facial skin, the oral cavity (including the lips and floor of the mouth, the tongue distal to the line of the circumvallate papillae, the trigone, and the hard palate), the pharynx (including the hypopharynx, oropharynx, and nasopharynx), or the larynx. The anatomic considerations of most of these areas will be expanded upon in subsequent chapters.

In terms of SCC of the oral cavity, the distribution of these tumors is depicted in Figure 2A. The overall distribution of all head and neck cancers and their anatomic frequency are shown in Figure 2B. More than 80% of tumors occurring in the oral/oropharyngeal region are SCC. Characteristics of the tumors associated with a poor prognosis include extension of tumor cells outside the tumor capsule and invasion of tumor cells along adjacent perineural sheaths or into blood vessels or lymph

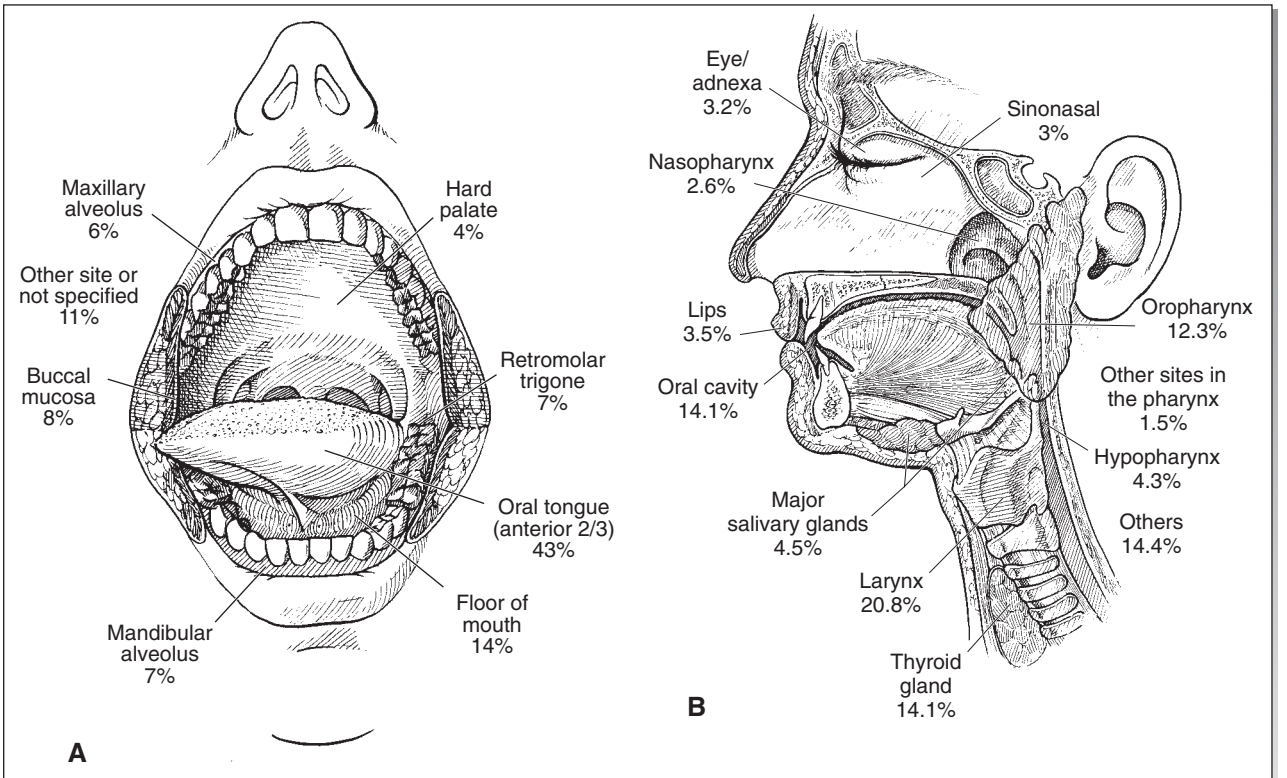


Figure 2 • A, Anatomic subsites of the oral cavity and relative frequency of cancer in each subsite. **B,** Incidence of cancer in various regions of the head and neck.

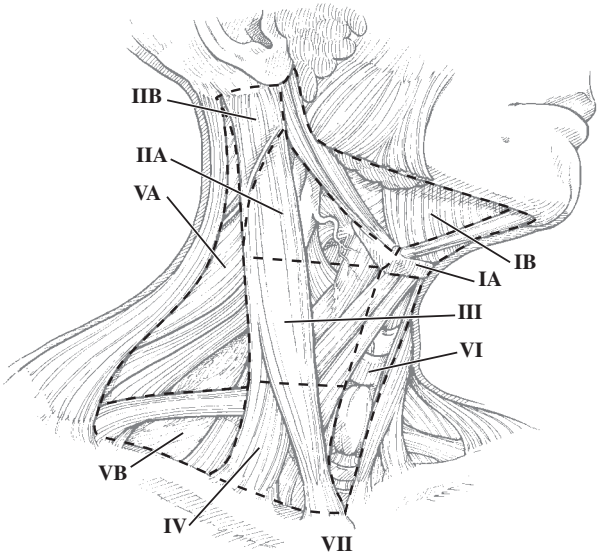


Figure 3 • The lymph node levels. Level I includes the submental and submandibular triangles, as well as the body of the mandible. Level II contains the upper jugular lymph nodes from the skull base superiorly to the hyoid bone inferiorly. Level III contains the middle jugular lymph nodes from the hyoid bone superiorly to the level of the lower border of the cricoid cartilage inferiorly. Level IV contains the lower jugular lymph nodes from the level of the lower border of the cricoid cartilage superiorly to the clavicle inferiorly. Level V contains the lymph nodes in the posterior triangle. It can be divided into upper, middle, and lower levels. Level VI contains the lymph nodes of the anterior central compartment from the hyoid bone superiorly to the suprasternal notch inferiorly, bounded laterally by the medial border of the carotid sheath. Level VII contains the lymph nodes inferior to the suprasternal notch in the superior mediastinum.

nodes. The most useful prognostic indicator in oral cavity SCC is tumor thickness, with thin lesions (1.5 to 3 mm, depending on the anatomic site) having a better outcome than thicker ones; otherwise, nodal status is the single most important prognostic factor.

There are six levels of lymph nodes. In addition, there are now six divisions as depicted in Figure 3. The levels and sublevels of lymph nodes include the following:

Level I: Nodes contained in the submental (level IA) and submandibular (level IB) triangles.

Level II: The superior jugular lymph nodes. Level IIA nodes are anterior (medial) to the plane formed by the spinal accessory nerve. Level IIB nodes are posterior (lateral) to this plane.

Level III: The middle jugular lymph nodes.

Level IV: The lower jugular lymph nodes.

Level V: The posterior triangle lymph node group. Level VA nodes (spinal accessory nodes) are separated from level VB nodes (the supraclavicular and transverse cervical groups) by a line in the horizontal plane marking the inferior border of the cricoid arch.

Level VI: The anterior compartment lymph nodes.

The precise anatomic borders used during surgical resection are described in Table 2. Other lymph node groups not accounted for in the schema presented include the suboccipital, retropharyngeal, parapharyngeal, buccinator, preauricular, periparotid, and intraparotid nodes.

It is accepted that tumors of the head and neck usually metastasize preferentially to the cervical region. Appreciation of the risk for neck metastasis is paramount for practitioners caring for patients with a head and neck malignancy. Studies by Shah and colleagues have shown that tumors located in certain areas will preferentially (although not exclusively) metastasize to particular lymph node groups in previously untreated patients (Fig. 4). Importantly, these drainage patterns are altered by surgery and irradiation. These anatomic observations are the basis for demarcation of the lymph node levels and sublevels, as well as for the evolution of selective neck dissections described later.

In addition to angiogenesis, lymphangiogenesis is now accepted to be a major mechanism by which head and neck tumors accelerate local and regional spread. The lack of a basement membrane on lymphatic vessels explains the high propensity of tumor cells to metastasize early to local and regional nodes via the lymphatic system. Accumulation and proliferation of these cells within the node will eventually lead to extracapsular breakout. It is notable that each of these characteristics (lymphatic spread and extracapsular extension) is associated with a 50% decrease in 5-year survival rates. Much effort is currently being placed on determining the pattern of spread of tumor cells to the cervical lymphatics. This is the basis of the evolution in neck dissection, as well as sentinel node techniques, both of which are discussed later. The discovery of lymphatic-specific growth factors and transcription factors (VEGF-C, VEGF-D, Prox-1, LYVE-1, and podoplanin) has revealed the molecular mechanisms underlying lymphatic growth and patterning and may in the future lead to novel therapeutic targets. The use of antiangiogenic agents in concert with antilymphangiogenic therapy may find a role in the pharmacologic treatment of head and neck tumors in the future.

Diagnostic Studies

Evaluation of the head and neck patient begins with a thorough history and physical examination. The history is directed toward the patient's possible risk factors for head and neck SCC, as delineated earlier, as well as toward obtaining information on underlying comorbid conditions. The triad of predisposi-

TABLE 2 Anatomic/Surgical Boundaries of the Neck Levels

	BOUNDARY			
LEVEL	**Superior**	**Inferior**	**Anterior (Medial)**	**Posterior (Lateral)**
IA	Symphysis of the mandible	Body of the hyoid	Anterior belly of the contralateral digastric muscle	Anterior belly of the ipsilateral digastric muscle
IB	Body of the mandible	Posterior belly of the digastric muscle	Anterior belly of the digastric muscle	Stylohyoid muscle
IIA	Skull base	Horizontal plane defined by the inferior body of the hyoid bone	Stylohyoid muscle	Vertical plane defined by the spinal accessory nerve
IIB	Skull base	Horizontal plane defined by the inferior body of the hyoid bone	Vertical plane defined by the spinal accessory nerve	Lateral border of the sternocleidomastoid
III	Horizontal plane defined by the inferior body of the hyoid	Horizontal plane defined by the inferior border of the cricoid cartilage	Lateral border of the sternohyoid muscle	Lateral border of the sternocleidomastoid or sensory branches of the cervical plexus
IV	Horizontal plane defined by the inferior border of the cricoid cartilage	Clavicle	Lateral border of the sternohyoid muscle	Lateral border of the sternocleidomastoid or sensory branches of the cervical plexus
VA	Apex of the convergence of the sternocleidomastoid and trapezius muscles	Horizontal plane defined by the lower border of the cricoid cartilage	Posterior border of the sternocleidomastoid muscle or sensory branches of the cervical plexus	Anterior border of the trapezius muscle
VB	Horizontal plane defined by the lower border of the cricoid cartilage	Clavicle	Posterior border of the sternocleidomastoid muscle or sensory branches of the cervical plexus	Anterior border of the trapezius muscle
VI	Hyoid bone	Suprasternal notch	Common carotid artery	Common carotid artery

From Robbins KT, Atkinson JL, Byers RM, et al: The use and misuse of neck dissection for head and neck cancer. J Am Coll Surg 193:91-102, 2001.

tion to SCC (advanced age, smoking, and alcohol use) also adversely affects other organ systems, most importantly the cardiopulmonary system and the vasculature. Clinical examination begins with evaluation of the skin for lesions. A direct examination with bimanual palpation is the only modality adequate for evaluation of mucosal lesions of the oral cavity, so its importance cannot be overstated. In addition to evaluation for lesions, the examiner should note the patient's oral hygiene with a careful manual examination, including the fit of any dentures that may be present. Because of the field effect of alcohol and tobacco, a diligent search is made for synchronous tumors. The oropharynx should be evaluated with a mirror exam, supplemented with endoscopy when the indirect mirror exam is inadequate.

The neck is evaluated for any lymphadenopathy. Note is made of the size and location of enlarged nodes. Occasionally, a patient has a neck mass in the absence of an obvious primary tumor. It is only in these instances that endoscopy and metabolic (PET) imaging play a vital role; after imaging, the primary tumor may often be found by directed biopsy of oral and pharyngeal landmarks. If a mass is palpated in the neck, it can be easily sampled by FNA (fine-needle aspiration) after diagnostic imaging studies have been obtained. It is important to obtain diagnostic imaging studies *before* any invasive surgical procedures (diagnostic or therapeutic) are carried out. If open biopsy is required, care is taken to place the incision within the design of a future neck dissection.

The neck may be scarred from prior neck dissections or irradiation, and the surgeon should be prepared to dissect out recipient vessels at the borders or outside the surgical field. Of note, modern neck dissections do not obligately sacrifice the vessels commonly used as recipient vessels by reconstructive surgeons performing microvascular free tissue transfers.

Emphasis on examination of the heart and lungs is necessary because of the frequent occurrence of comorbid disease in these organs in smokers. A base-

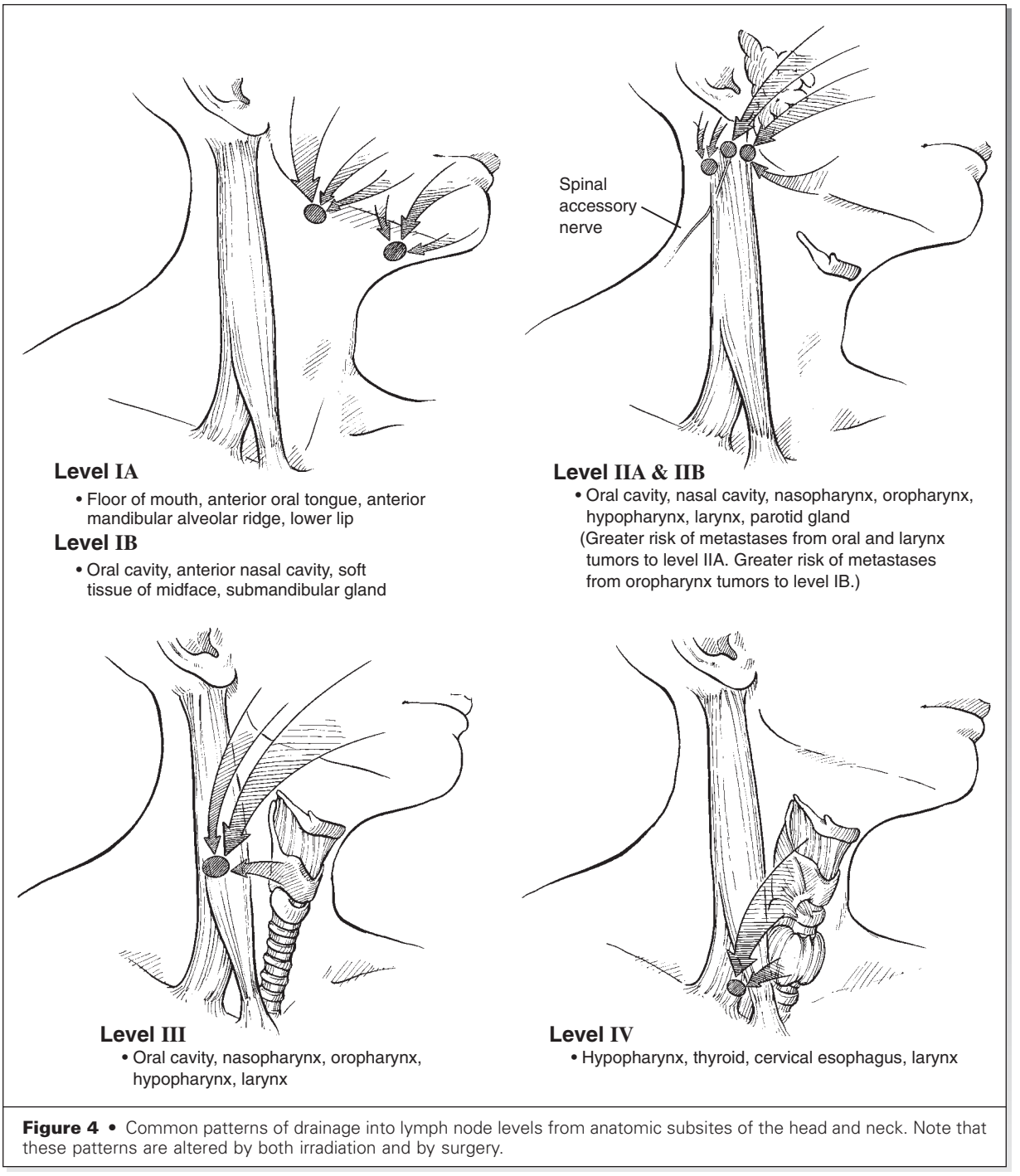
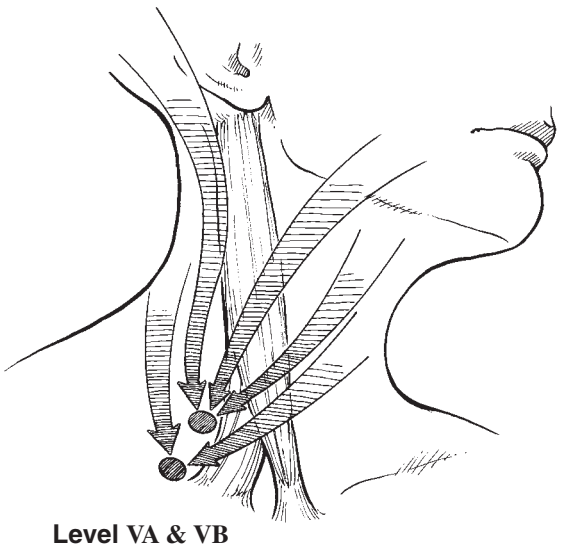


Figure 4 • Common patterns of drainage into lymph node levels from anatomic subsites of the head and neck. Note that these patterns are altered by both irradiation and by surgery.

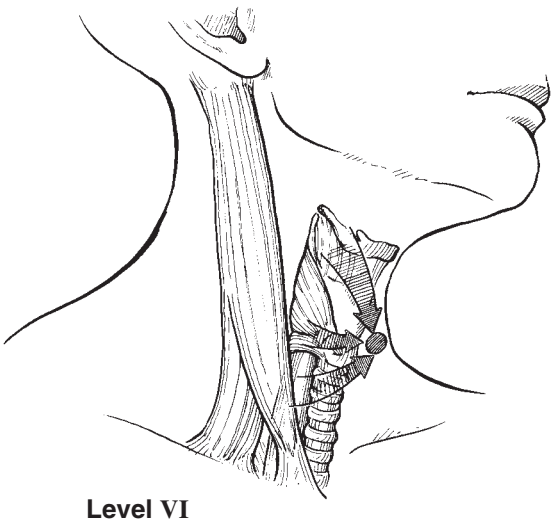
line chest radiograph or computed tomography (CT) is obtained at the first evaluation to search for the presence of lung metastases. These are the only screening diagnostic studies that routinely need to be performed.

Diagnostic Imaging

Diagnostic imaging studies, specifically, CT, magnetic resonance imaging (MRI), and positron emission tomography (PET), have an important role in the evaluation, staging, and eventual treatment plan for head and neck tumors. A CT scan with intravenous contrast is particularly useful to determine size, anatomic location, and nodal spread of a primary tumor. Contrast-enhanced CT studies are preferred for the delineation of bony anatomy and are therefore important in planning treatment strategies for tumors encroaching on the skull base, the mandible, and the maxilla. Axial views are



Level VA & VB
• Nasopharynx, oropharynx, posterior scalp/neck skin



Level VI
• Thyroid gland, glottic and subglottic larynx, apex of piriform sinus, cervical esophagus

Figure 4, cont'd • *For legend see opposite page.*

typically obtained from the skull base to the clavicles at 5-mm intervals. CT of the larynx at 3-mm intervals is also necessary. Coronal views are routinely obtained in studies of the paranasal sinuses for skull base, orbital, and maxillary tumors. Sinus series are non–contrast-enhanced, as opposed to most other head and neck studies.

MRI is advantageous in the evaluation of soft tissue invasion of tumors involving the tongue, salivary (parotid) glands, and the skull base for dural, perineural, and parenchymal involvement. MRI is also excellent for imaging nodal metastases, the cervicothoracic inlet, the brachial plexus, and spinal root areas. Multiplanar imaging (axial, sagittal, and coronal views) is readily obtained with MRI and is extremely useful to evaluate the anatomy surrounding a tumor. The ability to obtain differential information from variable weighting (T1, T2) and fat suppression techniques allows distinction between tumor mass and postobstructive mucus retention, which may appear identical on conventional CT examination. In this area, both studies are commonly obtained to evaluate a patient with tumors of the paranasal sinuses and skull base region. Most other sites, however, require only a single mode of well-planned imaging. At locations where bony resection or reconstruction is performed, posttreatment imaging at approximately 3 months is advised to serve as a posttreatment baseline for comparison in the event of recurrent disease.

Previous treatment, including radiation therapy (RT) or surgery (or both), results in changes in soft tissues that may be indistinguishable from persistent or recurrent tumor on anatomically based imaging (CT, MRI). Metabolic imaging techniques (PET) take advantage of the relative hypermetabolism characteristic of malignant tumors and the inability of such cells to metabolize 2-[^{18}F]-fluoro-2-deoxy-D-glucose (^{18}FDG) after it has gained intracellular access. Accumulation of this material in malignant tissues (primary or metastatic) has allowed sensitive detection of distant metastases, as well as locally recurrent or persistent disease when not clear on posttreatment anatomic imaging. The fusion of CT and PET images obtained contemporaneously has been a great addition to the management of previously treated patients. In addition, patients with metastatic disease from unknown primary sites will not uncommonly have the primary site disclosed on PET imaging, thereby allowing more focused treatment planning while often minimizing the morbidity of treating the nasopharynx (and bilateral parotids) by irradiation of the pharyngeal axis, which had been required for such diagnoses in the past. PET imaging obtained at least 8 weeks after treatment (chemotherapy/RT) may be used to evaluate response to treatment and guide further therapy.

Staging

The sixth edition of the TNM classification system of the American Joint Commission for Cancer (AJCC) contains the criteria used to stage head and neck cancers. Unlike other body areas, head and neck cancers are characterized by high rates of locoregional recurrence, which mandates a staging system sensitive to diagnostic studies unique to this

TABLE 3 TNM Classification of Oral Cancer

Tumor Size (T)	
TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
Tis	Carcinoma in situ
T1	Tumor 2 cm or less in greatest dimension
T2	Tumor more than 2 cm but less than 4 cm in greatest dimension
T3	Tumor more than 4 cm in greatest dimension
T4 (lip)	Tumor invades through cortical bone, the inferior alveolar nerve, the floor of the mouth, or the skin of the face
T4a (Oral cavity)	Tumor invades adjacent structures
T4b	Tumor invades the masticator space, pterygoid plates, or the skull base and/or encases the internal carotid artery
Regional Lymph Nodes (N)	
NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastases
N1	Metastasis in a single ipsilateral lymph node, 3 cm or less in greatest dimension
N2	Metastasis in a single ipsilateral lymph node, between 3 and 6 cm in greatest dimension; or in multiple ipsilateral lymph nodes, none more than 6 cm in greatest dimension; or in bilateral or contralateral lymph nodes, none more than 6 cm in greatest dimension
N2a	Metastasis in a single ipsilateral lymph node more than 3 cm but not more than 6 cm in greatest dimension
N2b	Metastasis in multiple ipsilateral lymph nodes, none more than 6 cm in greatest dimension
N2c	Metastasis in bilateral or contralateral lymph nodes, none more than 6 cm in greatest dimension
N3	Metastasis in a lymph node more than 6 cm in greatest dimension
Distant Metastasis (M)	
MX	Distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis

Reprinted with permission from AJCC Cancer Staging Manual, 6th ed, pp 24-25.

anatomic region and responsive to the benefits of tissue and organ preservation. A benefit of the staging system is that it can guide therapeutic interventions, permit accurate forecasts of prognosis for individual patients, and allow interinstitutional comparative analyses of different treatment protocols. The TNM staging system has the added benefit of highlighting tumors that are in high-risk and lower-risk subcategories. In general, stage I or II disease is associated with a better prognosis than more advanced stages and can often be treated by either surgery or RT with the expectation of equivalent rates of cure. Important exceptions are lesions of the oral tongue, alveolus, and maxilla, where the ease of surgery outweighs the risks of postirradiation changes, particularly to adjacent bone. More advanced stages require a multimodal approach. For head and neck SCC, 28% of cases are stage I, 17% are stage II, 18% are stage III, and 32% are stage IV at presentation. The staging system shown in Table 3 and Figure 5 is specific for oral cancer; staging systems specific for other sites in the head and neck are found in the AJCC manual.

Reconstructive Goals

Several goals are involved in reconstruction of the head and neck area, some of which are general principles of plastic surgery and some are specific to the treatment of tumors in this area. It is incumbent on the reconstructive surgeon that the operation chosen be reliable and not interfere with or compromise the oncologic aspects of patient care. Because many of the patients face dismal outcomes, little is gained by embarking on a heroic or ill-considered reconstructive plan that will prevent the patient from returning home in a timely manner or, even worse, compromise any adjuvant treatments that may be necessary. Some of the reconstructive goals are as follows:

1 Preservation of function. In the majority of cases, preservation of function has been made possible by the evolution of microvascular free tissue transfer. It allows preservation of function of structures such as the mandible, provides support to soft tissues, permits the reconstruction of an oral sphincter, provides the soft tissue bulk necessary to allow intelligible speech, and provides a properly shaped

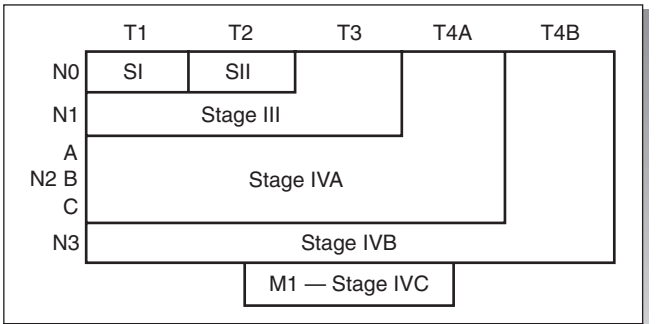


Figure 5 • Stage groupings of oral cavity, oropharyngeal, and hypopharyngeal tumors. (Used with permission from O'Brien JC [ed]: Selected Readings in Plastic Surgery, 2004, vol 10, no 4, p 11.)

and directed oral conduit for deglutition. The development of neurotized flaps has in some small series been shown to improve sensation.

2 Protection of vital structures. The head and neck area contains an abundance of important structures, including major vessels and nerves, as well as the intracranial contents and the orbit. It is imperative to provide well-vascularized tissues that can protect the major vascular structures of the neck from the damaging effects of salivary flow and radiation. The skull base and underlying dura are often broached by resections to attain clear margins. The reconstructive surgeon must transport well-vascularized tissue to protect these exposed areas, obliterate dead spaces, and ensure oro-nasal-cranial cavitary separation.

3 Permit uneventful healing. The oral and nasal cavities are populated by an abundant bacterial flora, and all procedures in the head and neck area are colonized if not outright contaminated. A vascularized free flap can bring healthy, well-vascularized, nonirradiated tissue to assist in wound healing. If properly planned and inset, tension-free closure should be achieved to prevent wound edge dehiscence and minimize the creation of fistulas. Irradiation is most effective if delivered *within 6 weeks* following resection. Because many patients need to be treated with primary RT after the results of surgical staging, the small window of time to permit healing of the resected and reconstructed tissues to allow the planned delivery of complication-free adjuvant treatment must not be extended.

4 Assistance with adjunctive therapies. The demonstration that functionally crippling radical neck dissections are not associated with increased cure rates or locoregional control means that more emphasis is placed on postoperative adjuvant RT to achieve a manageable rate of cure and local control. In many instances this has been made possible by the microvascular surgeon in the sense that a well-vascularized flap enables the routine use of high-dose RT.

5 Aesthetics. Amelioration of an often mutilating resection in conjunction with restoration of an acceptable appearance is not a trivial achievement. In many cases it has been possible to restore patients to their preoperative appearance, as most spectacularly exemplified by the ability to create a neomandible with a fibula microvascular free flap. Even reconstruction of an intraoral or oropharyngeal defect has implications for the appearance of the patient because the bulk provided by a flap prevents collapse of the surrounding soft tissues.

6 Support for prosthetics. A prosthesis can be functional (e.g., prosthetic teeth) or cosmetic (e.g., an eye). A well-designed reconstruction may achieve the functional goal of covering exposed structures and providing well-vascularized padding. In certain cases the final aesthetic result may be complemented by a well-designed prosthesis. When prostheses are necessary, the reconstruction should be planned so that the prostheses can be anchored into well-vascularized bone and soft tissue (i.e., osseointegration).

Treatment

In the near future, patients with precancerous lesions may be treated with chemopreventive regimens to minimize disease progression or the incidence of secondary lesions. Most early-stage SCC tumors without evidence of neck metastases can be treated by either RT or surgery with therapeutic efficacy. However, the presence of disease in the neck is associated with a 50% decrease in overall cure rates. Therefore, much of the focus in the treatment of head and neck tumors is currently placed on the detection and management of cervical metastases. The use of more selective neck dissections, in particular, sparing of the spinal accessory nerve and the sternocleidomastoid muscle (removed as part of a classical radical neck dissection), has decreased much of the morbidity of these operations without compromising oncologic outcomes. Expansion of the use of sentinel node biopsy may enable even more precise and targeted removal of affected tissues in the neck while sparing the patient the removal of nonaffected areas.

Radiation Therapy

The appropriate use of therapeutic radiation remains a cornerstone in the treatment of malignancies of the head and neck region. Recent technologic advances such as three-dimensional conformal treatment machines and intensity-modulated RT delivery promise to increase effectiveness while decreasing treatment-related morbidity through sparing of normal uninvolved tissues and critical anatomic structures. In many disease sites, concurrent chemotherapy/RT delivery protocols have replaced neoadjuvant (induction/anterior) chemotherapy regimens through clearly demonstrated improvements in disease control. Such protocols represent the state of the art in organ-sparing strategies (larynx) and in many hypopharynx and oropharyngeal sites. RT in a unique combination with chemotherapy remains the treatment of choice for nasopharyngeal cancer. Not surprisingly, combined modality regimens have been accompanied by increased severe toxicity and increased dependence

on feeding gastrostomy tubes and intensive supportive care. Primary RT alone is the treatment of choice for early-stage laryngeal cancer and for most supraglottic tumors less than 6 cm^2 in volume.

Postoperative adjuvant RT is indicated for most advanced-stage malignancies (T3, T4), for advanced metastatic disease (N2, N3), and for patients with nodes with extracapsular extension of disease. Therapeutic radiation is *not* a substitute for involved margins.

For sarcomas, RT provides an adjunct for high-grade tumors and aggressive histologic subtypes such as angiosarcomas. High-grade salivary gland tumors (mucoepidermoid, adenocarcinoma) are also appropriate indications for postoperative adjuvant RT or for fast-neutron RT. Many adenoid cystic carcinomas, which are notoriously neurotrophic, are appropriately treated with RT, which is more effectively able to treat large surface areas, thus covering regional structures potentially at risk for locoregional spread. Altered fractionation schemes (high dose per fraction) are used for advanced and metastatic melanomas of the head and neck region.

Intraoperative RT may be selectively used in the treatment of recurrent salvage cases (sparing additional doses to normal adjacent structures); yet, it is generally unavailable in most locations. Similarly, afterloaded brachytherapy catheters (high–dose rate iridium 192) may be selectively used in the same contexts, but flap coverage may be required for reliable wound healing and for protection of critical exposed structures. Acellular dermal allografts (AlloDerm) have been used for carotid protection and as a bolus for catheter placement and protection.

Complications attributable to RT include a variety of wound-healing problems such as fibrosis, chondronecrosis (larynx), and osteoradionecrosis (mandible), as well as the sequelae of destruction of specialized structures such as salivary glands and taste buds. Radioprotective agents have had minimal impact on these effects. Severe dysphagia as a result of fibrosis of delicate pharyngoesophageal musculature may make oral intake impossible. Indeed, for many patients, the functional cost of tumor control exceeds tolerable limits, in part because of treatment-related morbidity.

Chemotherapy

Over the past decade, the role of chemotherapeutic agents in the management of SCC of the head and neck has become more defined. Previously, medical oncology had been relegated to a third-tier role behind surgery and RT or to purely experimental protocols in inoperable patients. Organ preservation (larynx) studies in the early 1990s defined a role in predicting response to RT, with major partial responders entering radiation arms of treatment protocols and surgical management reserved for salvage. Those failing to achieve major responses entered surgical arms of such protocols and received postoperative adjuvant RT. Studies now coming to clinical maturity have further defined these agents in concurrent (simultaneous) treatment regimens with RT, with superior results obtained with induction (anterior) models.

Agents with particular activity in SCC of the head and neck include taxanes, platinum, 5-fluorouracil, and methotrexate. Although major responses are common, the durability of the response is lacking because once the agents are withdrawn, even more aggressive recurrent disease often follows in a short time. The promise of EGFR-targeting agents, cytokines, and other biologic response modifiers has thus far failed to improve response rates in ongoing clinical trials. Similarly, a significant impact on overall survival and on the development of distant metastatic disease has not been realized over decades of various combinations of agents and modalities.

Surgery

Surgical principles of tumor resection have been influenced by the development of reconstructive techniques. The overriding principle on the part of the extirpative surgeon is to ablate all apparent disease. This goal is facilitated by intraoperative pathologic guidance. The reconstructive surgeon should anticipate the defect that may result from attaining 1- to 2-cm uninvolved margins. These margins are attained in three dimensions and preclude primary closure in many instances. Incisions are generally placed in areas that minimize the deformity caused by the resulting scar. Therefore, an incision may be placed along the lateral subunits of the nose, or it may split the chin during a mandibulotomy. Attention to delicate handling of previously irradiated tissues is important. Drains are routinely used to minimize accumulation of hematoma. Neck incisions should be placed away from the great vessels whenever possible to prevent exposure in the event of wound dehiscence.

NECK DISSECTIONS. The decision to perform a neck dissection depends on whether there is clinical or radiographic evidence of nodal involvement, as well as the risk of occult nodal disease. A useful algorithm is presented in Figure 6. In the past, the rationale and definitions of neck dissection were fraught with interindividual and interinstitutional variability. In an effort to standardize this inconsistency and to improve on biologic correlation with known pathways of neck metastases, the classification of neck dissections has evolved. There are now four classes of neck dissection, with the "gold standard" still being classical radical neck dissection. Modifications developed and validated over recent decades have

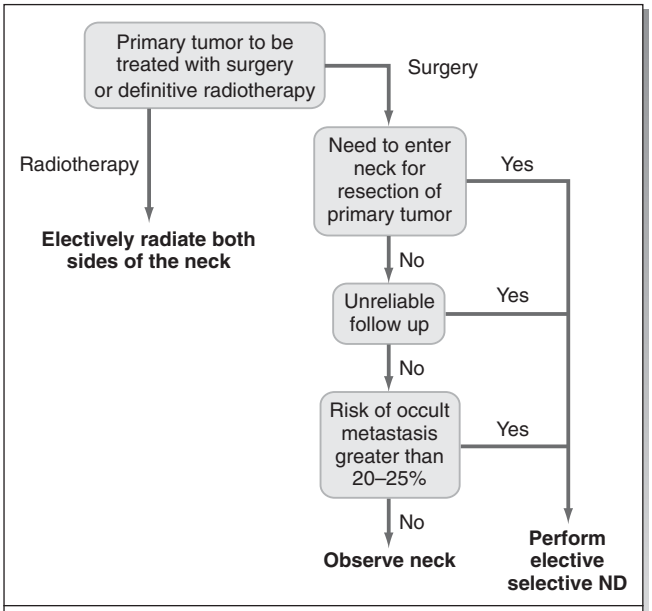


Figure 6 • Algorithm for treating an N0 SCC of the larynx/hypopharynx. (From Bobbins KT, Atkinson JL, Byers RM, et al: The use and misuse of neck dissection for head and neck cancer. J Am Coll Surg 193:91-102, 2001.)

attempted either to lessen the morbidity of radical neck dissection by retaining nonlymphatic structures, such as the sternocleidomastoid muscle and spinal accessory nerve, or take advantage of the propensity of previously untreated tumors in different areas of the head to metastasize in a stereotypic manner to specific lymph node basins. The four types of neck dissection are as follows:

1. *Radical neck dissection* is the standard operation for cervical lymphadenectomy. In this procedure, all of the ipsilateral cervical lymph nodes extending from the inferior border of the mandible to the clavicle and from the lateral border of the sternohyoid muscle, hyoid bone, and contralateral anterior belly of the digastric muscle medially to the anterior border of the trapezius muscle are dissected. Included are all lymph nodes from levels I through V. The spinal accessory nerve, internal jugular vein, and sternocleidomastoid are included. However, the suboccipital nodes, periparotid nodes, buccinator nodes, retropharyngeal nodes, and anterior compartment nodes are not included.
2. *Modified radical neck dissection* preserves one or more nonlymphatic structures routinely extirpated in radical neck dissection. These structures may include the spinal accessory nerve, the internal jugular vein, and the sternocleidomastoid. The structure spared should be so designated.
3. *Selective neck dissection* (SND) preserves one or more lymph node groups/levels routinely resected in radical neck dissection. This is one of the benefits of the new classification system because only lymph node groups and subgroups that are likely to drain the anatomic site resected may be rationally removed. Terms such as "supraomohyoid," "lateral," or "posterolateral" neck dissections are no longer favored. Instead, the levels or sublevels removed should be designated in parentheses after SND. For example, the classic supraomohyoid neck dissection is now formally designated SND(I-III). The three major nonlymphatic structures are typically spared.
4. *Extended neck dissection* removes additional lymph node groups or nonlymphatic structures beyond those typically resected in the radical neck dissection. These include lymph nodes outside the six levels, as well as the carotid artery, hypoglossal nerve, vagus nerve, and paraspinal muscles.

SENTINEL NODE BIOPSY. Sentinel node biopsies have received much attention, but their clinical utility remains mostly unproven in the head and neck. Until formal proof of accuracy and benefit, sentinel node biopsies for head and neck malignant disease should be performed only in the context of a clinical trial.

Efforts to rationalize and target selective minimally invasive therapies have a long history in head and neck oncology. The earliest efforts began in the 1960s and eventuated in modifications of the classic "radical" neck dissection that have resulted in functional preservation and improved cosmesis while not compromising oncologic principles and disease control. Head and neck primary tumor sites have a well-defined propensity to metastasize to particular nodal stations in previously untreated patients. This propensity has allowed staging procedures, such as supraomohyoid neck dissection (SND[I-III]), to direct prognostic and treatment planning. Because many of these tumors arise in elderly patients with significant and occasionally prohibitive comorbidity who may tolerate even nonsurgical therapies poorly, superselective lymph node biopsy techniques have been of intense interest. Similarly, tumors with a high incidence of occult metastatic spread (e.g., thick oral tongue tumors) lend themselves to such efforts. There are certain scenarios in the head and neck that appeal to the use of sentinel lymphadenectomy. For example, cutaneous primary lesions of the vertex of the scalp, which may drain to any combination of four quadrants, are a particularly good target for lymphoscintigraphy and the sentinel lymph node technique. Lesions of the skin of the periorbital and cheek regions, which may drain to either the facial

or the parotid/periparotid nodes, are probably candidates for application of these techniques. The greatest utility of sentinel lymph node techniques may eventually be for the management of the clinically N0 neck, where 33% to 50% of patients harbor occult metastatic disease in the context of a high-risk primary lesion.

Technical problems in clinical practice have been the proximity of primary sites to first-echelon nodes and the difficulty in resolution of this anatomy on lymphoscintigraphy cameras, as well as the rapid transit time characteristic of highly vascularized mucosal structures. The latter phenomenon has often limited efforts to use isosulfan blue chromatic localization to augment radioguided techniques. In short, the application of sentinel lymphadenectomy techniques to mucosal primary lesions requires greater attention to detail and technical mastery than the cutaneous analogues.

Postoperative Care and Complications

The postsurgical care is tailored to the operation undertaken. Tumors that were excised and closed primarily need minimal in-hospital care. Patients who underwent free tissue transfer are optimally monitored in a unit dedicated to the care of microvascular free flaps.

Certain postoperative complications are specific to the head and neck. One common complication is wound infection after neck dissection, typically resulting from contamination by oral flora. This complication is decreased most effectively with preoperative antibiotics and antimicrobial rinses in conjunction with the judicious use of drains to minimize hematomas and seromas. In addition, carotid blowout (more common in irradiated tissues and often fatal) and chyle leaks can also complicate the postoperative course. If bilateral neck dissections are performed, many patients display severe facial edema, which in addition to being rather disturbing can be severe enough to interfere with vascular flow.

Furthermore, both RT and chemotherapy can oftentimes be associated with disabling short- and long-term complications. Mucositis is a short-term complication that rarely persists and is treated with supportive care. Hypothyroidism, pituitary insufficiency, optic nerve damage, trismus, and soft tissue fibrosis can result from RT. Xerostomia is likely the most disabling complication. Moreover, osteoradionecrosis and persistent fistula formation can result. The use of cytotoxic drugs, particularly in conjunction with RT, can inhibit cellular functions necessary for optimal wound healing and immune defenses, further highlighting the need for diagnostic vigilance and optimization of parameters, such as nutrition, that are under control of the clinician.

Pearls and Pitfalls

- Although initially simpler, regional pedicled flaps are, in many cases, inferior to microvascular free flaps for reconstruction in the head and neck region because of their inferior vascularity and antigravitational orientation, particularly when radiation therapy will be used.
- Avoid the use of exotic flaps. The reconstructive surgeon should be familiar with harvesting and insetting of a few flaps (rectus, radial forearm, fibula) that reliably serve to reconstruct most defects in the head and neck area.
- Close cooperation with the anesthesia team is imperative to prevent the injudicious administration of fluids or blood products because many of the patients (particularly those with a history of irradiation or bilateral neck dissection) have a large amount of postoperative swelling, which can affect wound healing and flap survival.
- Open communication with the ablative surgeons facilitates the reconstruction. For example, emphasize the need to maintain adequate vessel stumps at the sites of major vessel take-offs, to use appropriately sized hemoclips vs. large sutures at the sacrifice of potential recipient vessels, and preserve vessels that do not need to be resected for oncologic reasons (e.g., the superior thyroid vessels and transverse cervical vessels).
- Failure of a microvascular free flap often delays postoperative adjuvant therapy and possibly compromises long-term survival. Therefore, it is important to simplify the reconstructive plan and focus on reliability. Large-caliber recipient vessels should be used at all times.
- All resections should be undertaken with complete extirpation of the tumor (all gross disease) and regional metastatic disease as the principal goal. Anything less should be considered a compromised therapeutic result and is unacceptable in terms of misallocation of resources and surgical insult to an oftentimes debilitated patient.
- The initial therapeutic intervention provides the best chance for control or cure of the disease. The patient is therefore best served by treatment undertaken by a multidisciplinary team and a practitioner who is committed to the care of the patient longitudinally through all phases of treatment.

SUGGESTED READINGS

AJCC Cancer Staging Manual, 6th ed. New York, Springer, 2002, pp 17-87.

Califano J, van der Riet P, Westra W, et al: Genetic progression model for head and neck cancer: Implications for field cancerization. Cancer Res 56:2488-2492, 1996.
Forastiere A, Koch W, Trotti A, Sidransky D: Head and neck cancer. N Engl J Med 345:1890-1900, 2001.
Gurtner GC, Evans GR: Advances in head and neck reconstruction. Plast Reconstr Surg 106:672-682, 2000.
Robbins KT, Atkinson JL, Byers RM, et al: The use and misuse of neck dissection for head and neck cancer. J Am Coll Surg 193:91-102, 2001.
Shah JP, Candela FC, Poddar K: The patterns of cervical lymph node metastases from squamous carcinoma of the oral cavity. Cancer 66:109-113, 1990.

The Forehead, Scalp, and Calvaria

RICHARD J. MACKOOL

Reconstruction of the scalp and forehead requires thorough knowledge of standard reconstructive procedures as well as surgical creativity. This chapter provides the foundation for the development of optimal reconstructive treatment plans.

Etiopathogenesis

Scalp and forehead defects can be divided into three broad groups: posttraumatic, congenital, and postablative. The most common group is traumatic defects, which include a wide range of injuries from simple lacerations to complete scalp avulsion injuries. They can result from blunt trauma, shearing injuries, and avulsion injuries, commonly from hair becoming entangled in mechanical equipment. Congenital defects include cutis aplasia and congenital nevi. Skin malignancy is relatively common on the scalp as a result of high sun exposure, especially in balding men. In addition, this area is often neglected because it is difficult to self-examine. As a result, lesions are often initially seen in more advanced stages.

Anatomy

The scalp consists of skin, subcutaneous tissue, galea, loose areolar tissue, and pericranium (periosteum). Scalp skin is thickest in the occipital region and thinnest in the temporal region. The subcutaneous layer contains the main arteries, veins, nerves, and lymphatics. The galea aponeurotica is a fibrous layer contiguous with the occipitalis and frontalis muscles, as well as the temporoparietal fascia, which in turn becomes the superficial musculo-aponeurotic system (SMAS) of the face. The loose areolar tissue plane consists of connective tissue and is the layer most often involved in avulsion injuries. This tissue plane continues deep to the temporoparietal fascia and is contiguous with the parotid-masseteric fascia. The pericranium invests the calvaria and fuses with the deep temporal fascia laterally.

The blood supply to the scalp is primarily from the occipital and superficial temporal arteries. The superficial temporal vessel bifurcates into a frontal and parietal branch at a point approximately 2 cm above the zygomatic arch; these branches supply the majority of the lateral aspect of the scalp. The occipital arteries supply the majority of the posterior portion of the scalp. Additional blood supply is provided by the posterior auricular vessels and by the supraorbital and supratrochlear vessels anteriorly.

The sensory nerve supply of the anterior scalp and forehead arises from the supraorbital and supratrochlear nerves. The supratrochlear nerves supply the central portion of the forehead, whereas the supraorbital nerves supply the anterior hairline and scalp. The lesser occipital nerves supply the posterior scalp, and the auriculotemporal nerve supplies sensation to the temporal region.

The frontal branch of the facial nerve runs on a line 0.5 cm below the tragus to 1.5 cm above the lateral aspect of the brow. It consists of two to five branches and innervates the frontalis muscle as it proceeds medially. The frontal branch runs under the SMAS at the zygomatic arch. Incisions and undermining for forehead reconstruction obviously require attention to the anatomic path of the frontal branch.

Diagnostic Studies

Management of scalp and forehead defects can most often be based solely on clinical examination. It is sometimes helpful to use a hand-held Doppler to document the presence of the superficial temporal and occipital arteries. For cases with loss of the underlying bony calvaria, it is necessary to evaluate the relationship of the defect to the underlying brain and venous sinuses. Radiographic studies can be helpful for these complex defects.

Reconstructive Goals

The goals of scalp reconstruction are:

1. Provision of stable soft tissue cover
2. Reconstruction of rigid support and protection of the brain
3. Correction of contour deformities
4. Restoration of the anterior hairline
5. Restoration of the remaining hair-bearing scalp

Scalp Injury and Repair

Lacerations

Scalp lacerations should be irrigated and débrided before layered closure. If substantial undermining of the scalp has occurred, drainage catheters are often necessary.

If soft tissue loss precludes primary closure, undermining in the loose areolar plane combined with scoring of the galea often provides sufficient laxity and allows primary closure. Scoring is performed perpendicular to the direction of desired advancement. Scoring the galea in a "checkerboard" pattern increases both the length and width of a scalp flap. Scoring should penetrate only the thick galeal layer to avoid damaging the vascular supply of the flap and it can be performed by placing tension on the flap and incising carefully with a No. 10 blade.

Partial-Thickness Scalp Loss

Partial-thickness scalp loss usually occurs below the galea in the areolar tissue plane, with the pericranium left intact. For small defects, local flaps may be used in the acute or subacute setting. For larger defects, flaps often result in widened scars with alopecia, and thus the final result may not be acceptable. In selected patients, the defect can be skin grafted. After the graft has healed, the patient can undergo tissue expansion of the hair-bearing tissue, followed by excision of the graft and coverage with the expanded tissue. In cases in which this technique is not an option, local flaps can be performed with secondary transplantation of hair to the widened scars.

Full-Thickness Scalp Loss

Full-thickness scalp loss leaves exposed calvaria. Although these defects can be skin grafted, this results in an unstable scar. This is often necessary in the acute setting and can be facilitated by creating perforations in the outer calvarial table followed by dressing changes, development of granulation, and placement of a skin graft. Alternatively, the outer calvarial table can be removed and a delayed skin graft placed for coverage. In all cases, the patient will be left with both alopecia and an unstable graft prone to recurrent ulceration.

Pericranial flap coverage, with subsequent skin grafting, of a full-thickness defect can provide stable coverage. Ideally, the pericranial flap is harvested without additional scalp incisions.

Scalp flaps are the preferred method of closure for coverage of denuded calvaria. The size of the defect dictates the design of the flap and the need for possible skin grafting of the donor defect (Fig. 1). The flaps are raised in the areolar plane between the galea and periosteum and generally require galeal scoring to fully cover a defect.

Larger defects may require subtotal scalp flaps as in Orticochea's three-flap technique (Fig. 2). The three-flap technique creates two flaps adjacent to the defect cut at an angle, the width of each equal to half the width of the defect. The third flap encompasses the majority of the remaining scalp. It is important to maintain a viable pedicle for each of the flaps. Again, many of these techniques result in wide scars with alopecia. These areas can be treated at a second stage by hair transplantation.

Total Scalp Avulsion

Scalp avulsion is usually the result of long hair becoming entangled in mechanical equipment. The cleavage plane is between the galea and periosteum. The eyebrows, upper lids, and portions of the ears can also be avulsed with the scalp. Microvascular replantation is the treatment of choice. Vein grafts are generally required to avoid anastomoses in the zone of injury. Multiple arterial and venous anastomoses increase the chance of success.

If the avulsed scalp is not available for replantation, skin grafts can be placed on intact periosteum. If the periosteum is not intact, microvascular free flap coverage is indicated. Free latissimus flaps provide adequate pedicle length and muscle size for coverage of large defects. Other options include omental, rectus, and scapular flaps.

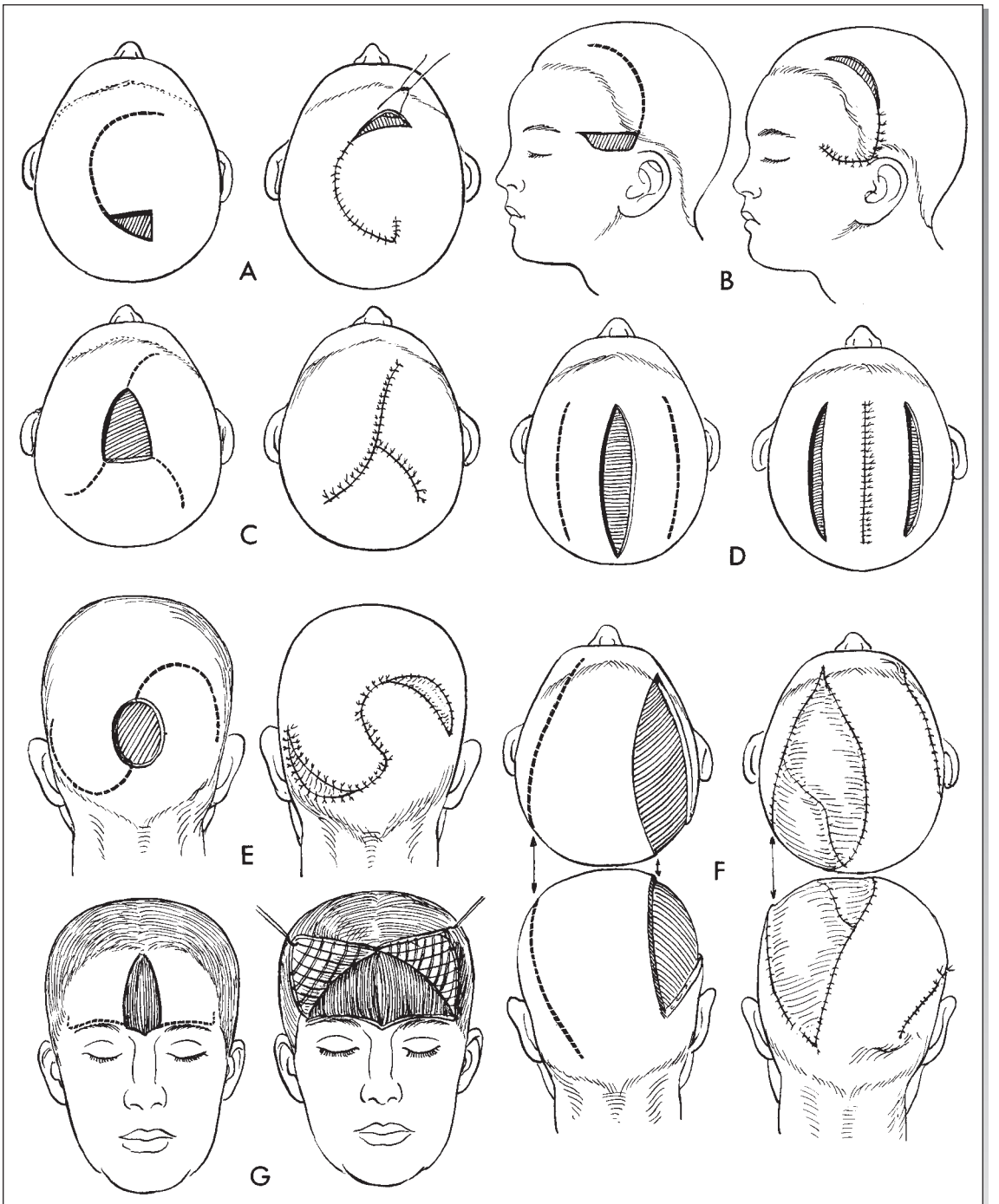


Figure 1 • A to **F,** Multiple methods of wound closure with local scalp flaps. All flaps are raised in a subgaleal plane. **G,** Forehead flaps raised and scored through the frontalis muscles to increase flap surface area. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, WB Saunders, 1990. Modified from Orticochea M: New three-flap scalp reconstruction technique. Br J Plast Surg 24:184, 1971.)

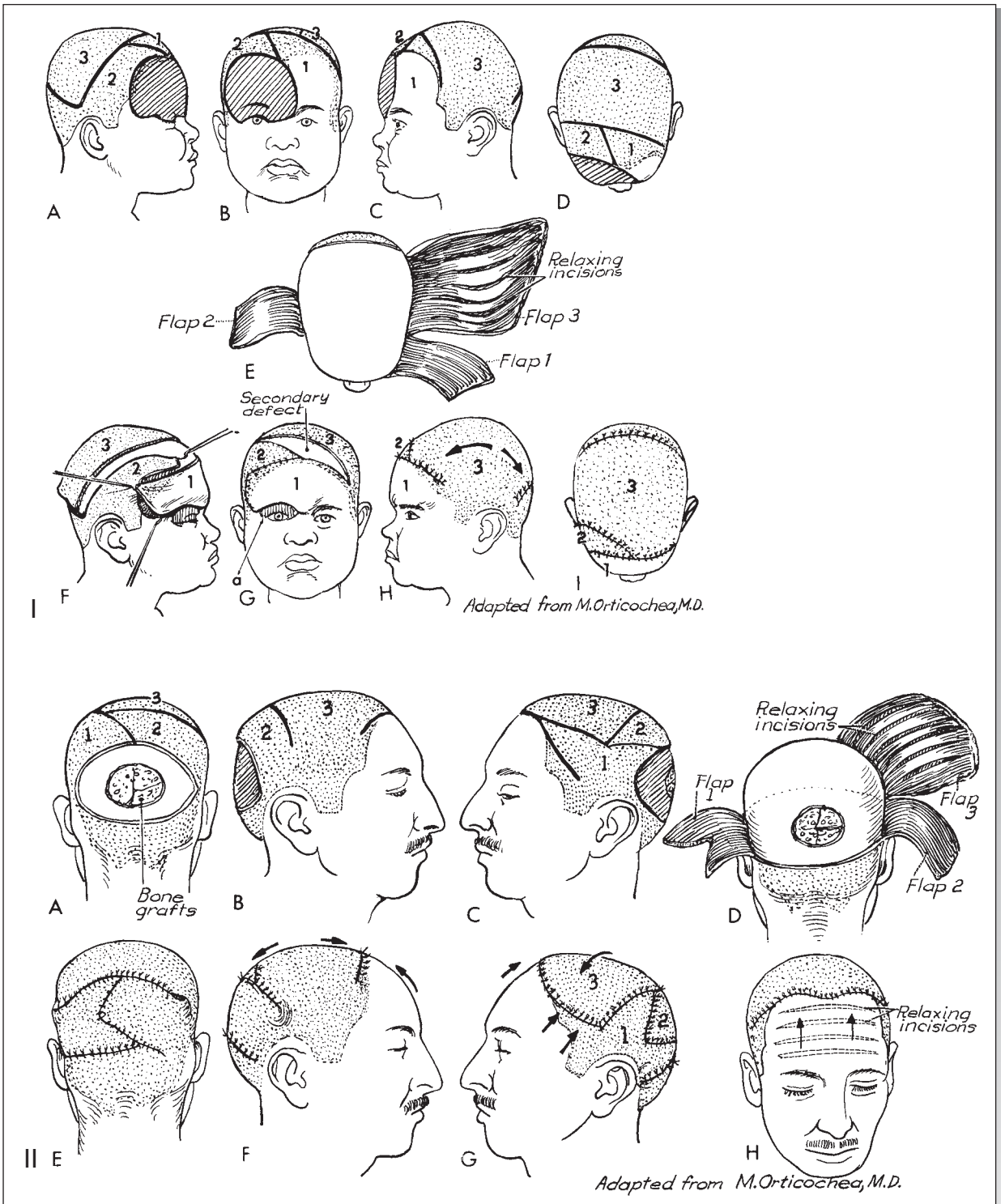


Figure 2 • I, Orticochea's three-flap technique for a frontal defect. **II,** Three-flap technique for a posterior defect. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, WB Saunders, 1990. Modified from Orticochea M: New three-flap scalp reconstruction technique. Br J Plast Surg 24:184, 1971.)

Reconstruction of Congenital and Acquired (Nontraumatic) Scalp and Forehead Defects

Aplasia Cutis Congenita (Cutis Aplasia)

Aplasia cutis congenita can occur over any portion of the body. However, approximately 90% of cases involve the scalp, with 70% to 75% of patients having a single lesion, 20% a double lesion, and 8% a triple lesion. Defects occur at or near the hairline whorl in 80% of small lesions but may also occur anywhere over the vertex between the anterior and posterior fontanelles.

Most cases of cutis aplasia are sporadic, but familial inheritance (autosomal dominant with incomplete penetrance) has been described. Patients with cutis aplasia can have multiple associated anomalies, including limb and cardiac anomalies (Adams-Oliver syndrome). Defects range from absence of hair follicles to large areas of absent scalp, skull, and dura.

Small areas of cutis aplasia are treated with wound care and allowed to heal secondarily. In more severe cases, or those with exposed dura and sagittal sinus, complications such as meningitis or desiccation of the sinus with resultant bleeding or thrombosis can occur. Nonetheless, conservative therapy is often the best option for these patients, especially when the defects are too large for local scalp flap closure. Large defects are treated with silver sulfadiazene (Silvadene) or sterile occlusive dressings to prevent desiccation and allow for epithelialization. After spontaneous closure of the defect, spontaneous, complete bony restoration often occurs.

In the event that coverage of the dura is required to prevent infection or hemorrhage, local scalp flaps are the preferred option. Skin grafts, pericranial flaps, tissue expansion, and microvascular free flaps can also be used.

Congenital Nevi

The rate of malignant degeneration of large pigmented congenital nevi is unclear, with authors citing malignancy rates between 0% and 42%. A recent review of the literature found a statistically significant increased risk for melanoma in large congenital melanocytic nevi, with a 2.8% malignancy rate. Current reconstructive efforts rely on serial excision, local flaps, or tissue expansion.

For smaller lesions, serial excision or local flap coverage is often possible. However, if excision and coverage cannot be performed without increasing the deformity, tissue expansion should be considered.

The incisions for expander placement should be made within the borders of the lesion, with alignment of the expander's long axis parallel to the long axis of the lesion. Closing wounds with permanent suture and leaving the sutures in place throughout the expansion process can decrease the chance of wound complications. Expanders are placed in a subgaleal or subfrontalis plane, and expansion begins 1 to 2 weeks after insertion. After expansion has been completed, the nevus is excised and the expanded tissue can be advanced or transposed into the wound (Fig. 3).

Bauer has developed the following guidelines for treatment of forehead and scalp congenital nevi:

1. Midforehead nevi are treated by expanding bilateral normal forehead segments with medial advancement. Scars are placed along the brow and at or posterior to the hairline.
2. Hemiforehead nevi require serial expansion of the uninvolved forehead to minimize the need for a back cut to release the advancing flap.
3. Supraorbital and temporal forehead nevi are preferentially treated with a transposition flap of a portion of the expanded normal skin medial to the nevus.
4. Temporal scalp nevi, when small, can be treated by advancing the expanded parietal scalp to the hairline. When the nevus involves the temporoparietal region of the scalp, a transposition flap from the central portion of the scalp provides the optimal hair direction for the temporal hairline and allows greater movement of the expanded flap.
5. If the level of the brow is too high after treatment of forehead lesions, forehead skin must be advanced and interposed to lower the brow.
6. In general, always use the largest expander possible, and overexpand if needed.

Reconstruction of Postablative Scalp Defects

Postablative reconstruction should be divided into two major groups: reconstruction of small defects resulting from resection of skin malignancies and reconstruction of larger postablative defects. Small defects resulting from skin malignancy can often be allowed to heal secondarily. Moderate-sized defects can be treated with local flap closure, as described earlier, or with skin grafts.

Reconstruction of large oncologic scalp defects can be challenging. External beam radiation, which is frequently required, damages tissue making local flap coverage difficult or impossible. In addition, many patients have undergone extensive or multiple procedures that result in scarring, cerebrospinal fluid fistulas, and bone or soft tissue devitalization. Standard considerations for the use of scalp flaps in oncologic patients should include the following:

1. Design the flap sufficiently large to cover the entire defect.

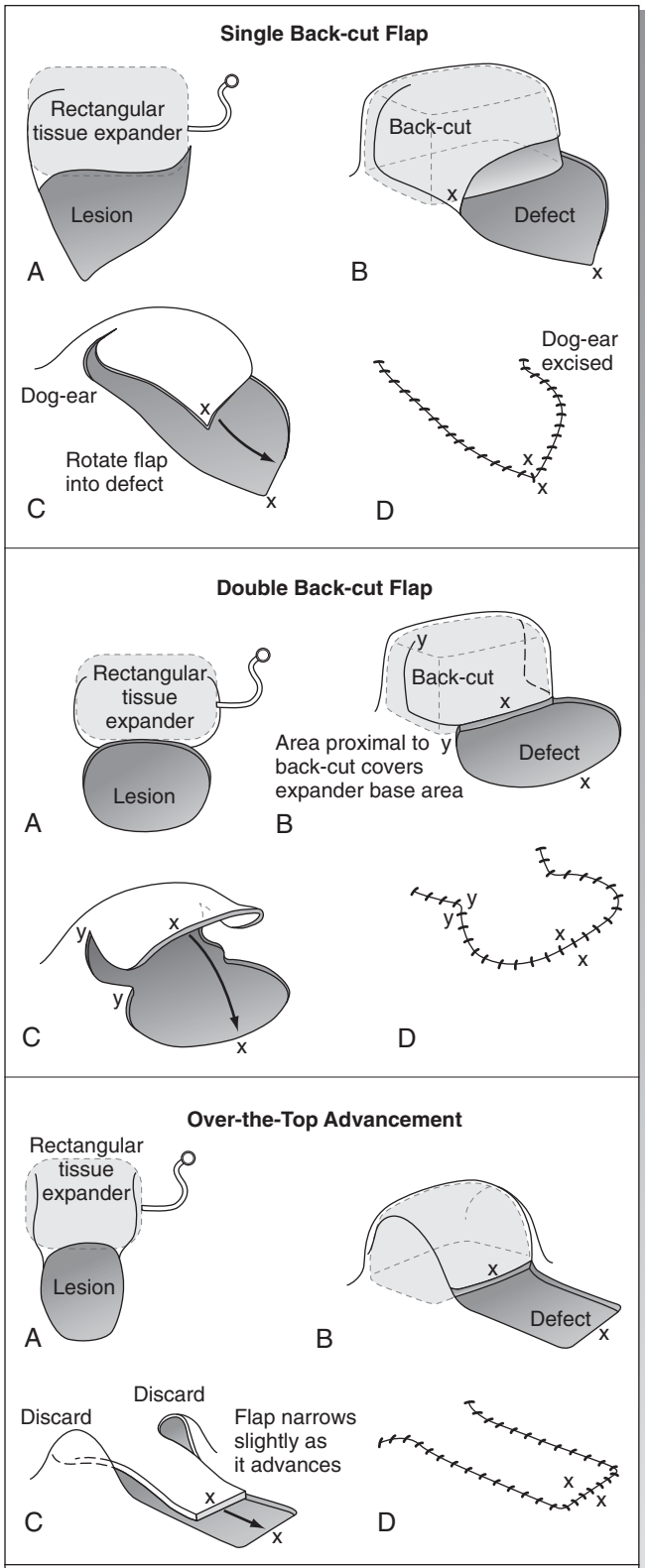


Figure 3 • Three methods of flap design after tissue expansion. **Upper panel,** single back cut. **Middle panel,** double back cut. **Lower panel,** "over-the-top" advancement. (From Zide BM, Karp NS: Maximizing gain from rectangular tissue expanders. Plast Reconstr Surg 90:500-504, discussion 505-506, 1992.)

2 Minimize the number of flaps.
3 Avoid suture lines in critical areas.

Patients who have been treated by radiotherapy or chemotherapy or who have cerebrospinal fluid leaks or anterior defects are at the highest risk for complications. A useful treatment algorithm for scalp defects after ablative surgery has been presented by the Memorial–Sloan Kettering group (Fig. 4). They emphasize using a limited number of reliable free flaps for scalp coverage.

Cranioplasty

The goals of cranioplasty are coverage and protection of the intracranial contents. In addition, frontal and temporal defects are noticeable, and reconstruction should provide restoration of a natural contour. Ideally, cranial defects should be repaired with autogenous bone. However, if the defects are too large for autogenous material, methylmethacrylate or a milled prefabricated prosthesis (alloplast) may be required.

Autogenous Bone

The outer table of the calvarial vault can be removed and used as a bone graft (in situ harvest) and is conveniently within or near the field during cranioplasty. The parietal calvaria posterior to the coronal suture is usually harvested, either in situ or after en bloc removal of both the inner and outer tables. In situ harvest is performed by marking the desired piece of bone for removal and burring through the outer table into the diploic space. Osteotomes are carefully admitted between the inner and outer tables, proceeding circumferentially to avoid cracking the outer table or penetrating the inner table. If the bone graft is harvested en bloc, a formal craniotomy is required. It is split on the back table, and the inner table is replaced (see Chapter 4, "Structural Support: Cartilage and Bone").

Rib grafts are useful in patients who require larger autogenous cranioplasties or in whom the calvaria cannot be split (as in a younger patient with a thin calvaria). The obvious disadvantages are the donor site scar, postoperative pain, and the potential for pneumothorax. An attempt is made to place the incision in the inframammary crease in women and along Langer's lines in male patients. Alternate ribs are harvested subperiosteally. The ribs can be split with an osteotome to increase the amount of bone and the chance of revascularization (split-rib grafts). Because revascularization is hastened by adequate bone-to-bone contact, débriding the recipient site to well-vascularized, bleeding bone should be a priority. The ribs are secured in the defect under tension. Careful placement and contouring of the grafts improve the aesthetic outcome.

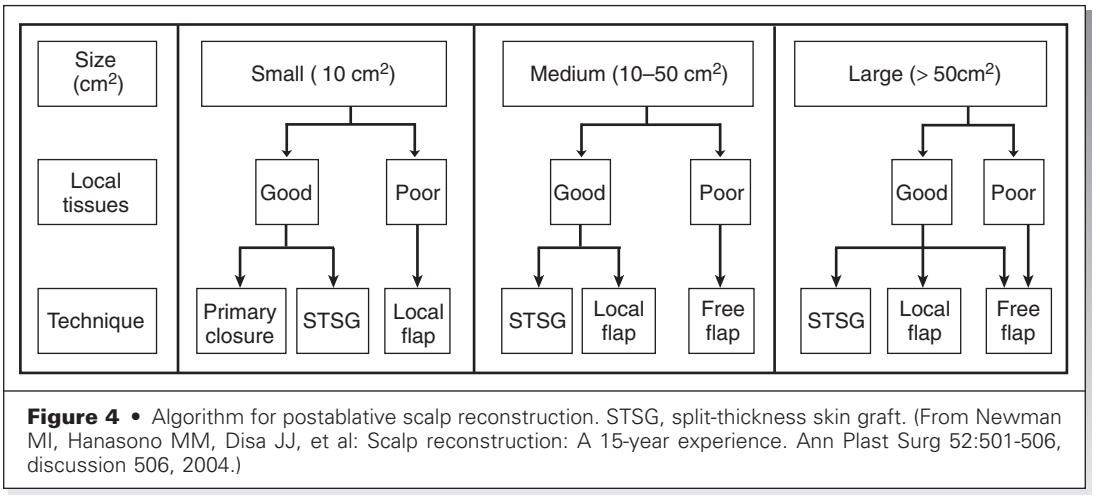


Figure 4 • Algorithm for postablative scalp reconstruction. STSG, split-thickness skin graft. (From Newman MI, Hanasono MM, Disa JJ, et al: Scalp reconstruction: A 15-year experience. Ann Plast Surg 52:501-506, discussion 506, 2004.)

Alloplast

Alloplastic material is useful but can be placed only in a well-vascularized bed that is free of infection. If there is poor soft tissue coverage or the implant is placed in proximity to the frontal sinus, exposure or infection will often occur. In general, alloplastic material should be avoided in growing children.

Multiple alloplastic materials are available for cranioplasty. However, in an adult patient, methylmethacrylate remains the material of choice for the majority of surgeons. The material is easily prepared and can be placed directly over bone or over wire mesh when dura is exposed. Chilled irrigation fluid should be used as the methylmethacrylate solidifies because a considerable amount of heat is produced that can damage the intracranial contents and bone.

Prefabricated prostheses created from computed tomography data can be used for large defects. These implants are placed with relative ease if properly constructed, and they are readily available from commercial sources.

Bone substitutes consisting of hydroxyapatite cement are also available; however, long-term studies have shown the complication rate to be higher with these materials than with autogenous bone or methylmethacrylate.

Pearls and Pitfalls

- Wide elevation with galeal scoring is useful for both primary closure and flap closure of scalp defects.
- Scars following flap repairs and tissue expansion are prone to widening and alopecia. Secondary hair transplatation is useful for camouflage.
- Attention to direction of hair growth is important when designing scalp flaps. Hair growth can hide exposed scars.

SUGGESTED READINGS

Bauer BS, Few JW, Chavez CD, Galiano RD: The role of tissue expansion in the management of large congenital pigmented nevi of the forehead in the pediatric patient. Plast Reconstr Surg 107:668-675, 2001.

Moreira-Gonzalez A, Jackson IT, Miyawaki T, et al: Clinical outcome in cranioplasty: Critical review in long-term follow-up. J Craniofac Surg 14:144-153, 2003.

Newman MI, Hanasono MM, Disa JJ, et al: Scalp reconstruction: A 15-year experience. Ann Plast Surg 52:501-506, discussion 506, 2004.

Orticochea M: New three-flap reconstruction technique. Br J Plast Surg 24:184-188, 1971.

Unlu RE, Tekin F, Sensoz O, Bauer BS: The role of tissue expansion in the management of large congenital pigmented nevi of the forehead in the pediatric patient. Plast Reconstr Surg 110:1191, author reply 1191-1192, 2002.

Zide BM, Karp NS: Maximizing gain from rectangular tissue expanders. Plast Reconstr Surg 90:500-504, discussion 505-506, 1992.

The External Ear

CHARLES H. THORNE

Ear deformities can be categorized as congenital, traumatic, or oncologic. Congenital defects, such as prominent ears and microtia, are the most common. Posttraumatic deformities often affect young adult patients. Cancer defects occur in older age groups.

Diagnostic Studies

Diagnosis of external ear deformities is based solely on clinical examination. Microtia can occur in isolation or may be part of a larger syndrome. Appropriate diagnostic tests must be performed when microtia is syndromic; otherwise, specific tests are not indicated.

Prominent Ears

Pathologic Anatomy

Anatomic abnormalities that contribute to the prominent ear deformity include the following:

1. Incomplete formation of the antihelical fold (Fig. 1)
2. Prominence of the concha (either excessive height of the posterior conchal wall or an increased angle between the conchal floor and the side of the head, or both) (see Fig. 1)
3. Prominence of the ear lobe

Treatment

NONSURGICAL. Patients with prominent ears who are identified shortly after birth can be treated nonsurgically. Such treatment should be initiated as soon as possible but is probably effective up to 3 to 4 months of age. The length of treatment and the modalities used may differ, but the principle remains the same. Splints of either resin or silicone-coated resin are constructed. The ear is taped or compressed around the splints, molding it to the desired shape. The splints should be worn continuously for 2 to 3 months until improvement is no longer seen.

If the results are not optimal, surgery may be undertaken at a later time to achieve the desired results.

SURGICAL. Surgical treatment of prominent ears may be performed at any time from childhood to adulthood, but is usually not performed prior to 4 years of age. Adult patients can be treated under local anesthesia.

Numerous surgical techniques have been described to deal with each of the anatomic abnormalities outlined. The techniques are used in combination to address all aspects of the prominent ear.

Antihelical Fold. Techniques advocated to create a more prominent antihelical fold include the following:

1. The Mustarde technique. Mattress sutures are placed on the medial surface of the ear between the triangular fossa and scapha and between the concha and scapha (Fig. 2).
2. The Stenstrom technique. The lateral surface of the cartilage is abraded to allow the cartilage to bend toward the side with intact perichondrium (i.e., toward the medial surface of the ear). This technique, however, may leave visible irregularities.

The Mustarde technique has proved to be the most powerful tool to create the antihelical fold. The sutures should create a helical rim that is straight when viewed from the rear. They can be used in combination with conchal resection and the Furnas conchal setback technique in patients with prominence of the middle third of the ear (see Fig. 2).

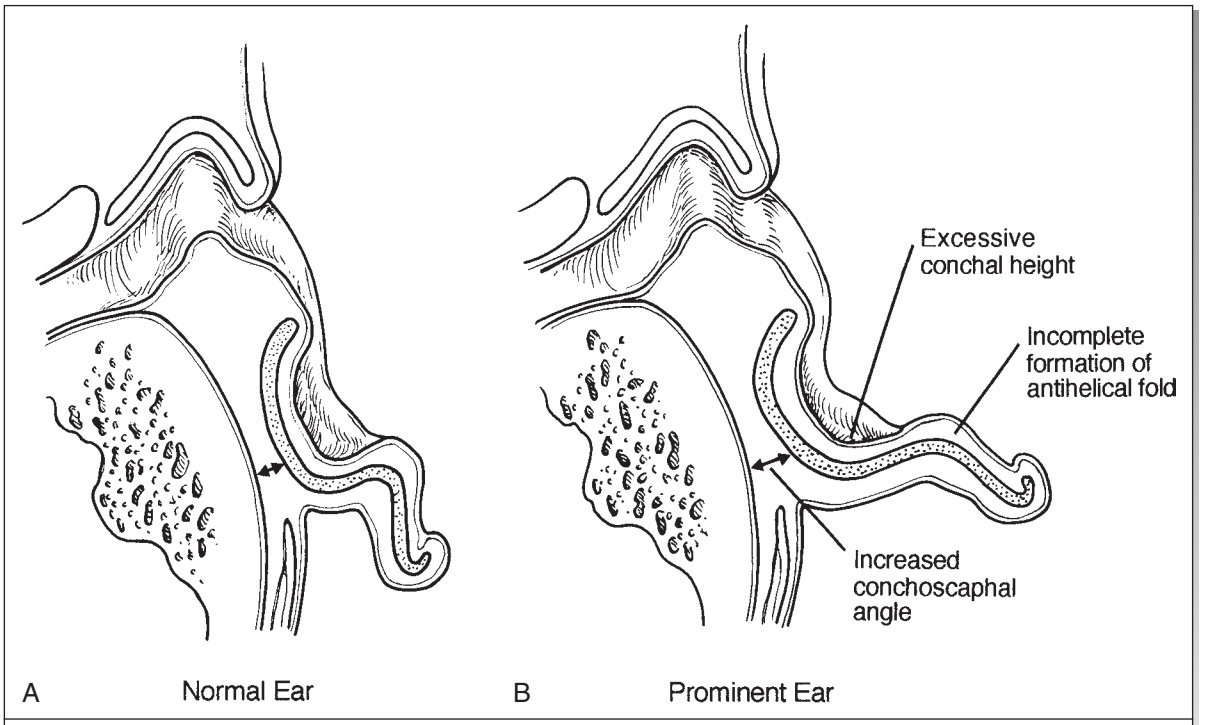


Figure 1 • Comparison of normal and prominent ears. **A,** Normal ear. **B,** The three components of a prominent ear are depicted. (Original artwork rendered by Kevin A. Somerville. Courtesy of Charles H. Thorne, MD.)

Prominence of the Concha. Conchal setback can be achieved in one of three ways:

1. Excision of a crescent of cartilage from the posterior wall of the concha, followed by suture approximation of the cartilaginous margins.
2. The Furnas technique, which involves suture setback of the concha. Soft tissue is removed deep to the concha, and sutures are placed between the conchal cartilage and the mastoid fascia to decrease the angle between the concha and the side of the head.
3. A combination of techniques 1 and 2 (see Fig. 2).

Prominence of the Earlobe. Even if not apparent preoperatively, the lobule often becomes relatively

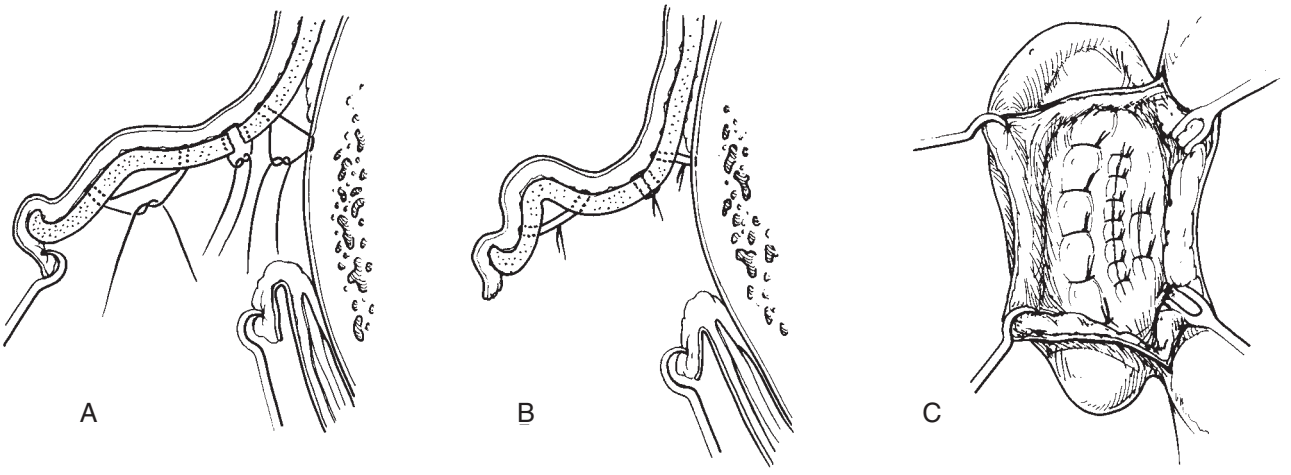


Figure 2 • Otoplasty for prominent ears. The combination of Mustarde scaphoconchal sutures, conchal resection, and conchal-mastoid sutures is shown. **A,** Sutures placed. **B,** Sutures tightened. **C,** Sutures as seen through the retroauricular incision. (Original artwork rendered by Kevin A. Somerville. Courtesy of Charles H. Thorne, MD.)

more prominent after conchal setback and may require surgical repositioning. To correct a prominent earlobe, the tail of the helix can be repositioned by placing a suture between the lobule and the concha. The author, however, has not found this to be a reliable technique. In addition, excessive tension on this suture can cause an abnormal contour in the region of the antitragus. Skin can also be excised from the medial surface of the earlobe.

Repositioning of the helical tail alone may not be consistently effective in repositioning the earlobe. Instead, when helical tail repositioning is not adequate, skin excision and suture repositioning of the earlobe should be performed. One must exercise caution because it is preferable to *avoid* skin excision in otoplasty. In addition, it is critical to preserve sufficient free lobule for a natural appearance and wearing of earrings. Because of the lack of cartilage in this region, earlobe repositioning is the most difficult and overlooked part of an otoplasty.

Pearls and Pitfalls

- *Overcorrection*. One of the most common complications is unnatural, excessively sharp contours. If one portion of the ear is set back excessively relative to other portions of the ear, the helical rim assumes an unnatural shape. For example, if the central portion of the ear is set back excessively, the helical rim assumes a "C-shaped" contour (telephone ear deformity).
- *Suture complications*. The sutures used in otoplasty may protrude either early or years later. This complication can be reduced by using a soft, braided suture rather than a monofilament suture. Relapse is not likely to occur after removal of a protruding suture.
- *Hematoma*. Pain after otoplasty may indicate a hematoma, which should be evacuated promptly.
- *Infection*. An infection after otoplasty can be devastating. Collections should be drained immediately and any infection treated to complete resolution. Untreated chondritis may lead to permanent deformation of the ear.

Partial Defects of the Ear

Partial ear defects must be critically examined to determine the anatomic derangements present. Although some defects may be limited to the helical rim, it is important to accurately determine what is missing. Often, it is useful to conceptualize the different anatomic planes of the ear because reconstruction of multiplanar defects requires more extensive procedures. For example, the helical rim, sulcus, and antihelical fold represent three distinct planes.

Helical Rim Defects

SMALL DEFECTS. Small defects of the helical rim (less than 2.0 cm) can be closed by the Antia-Buch technique of helical advancement (Fig. 3). This

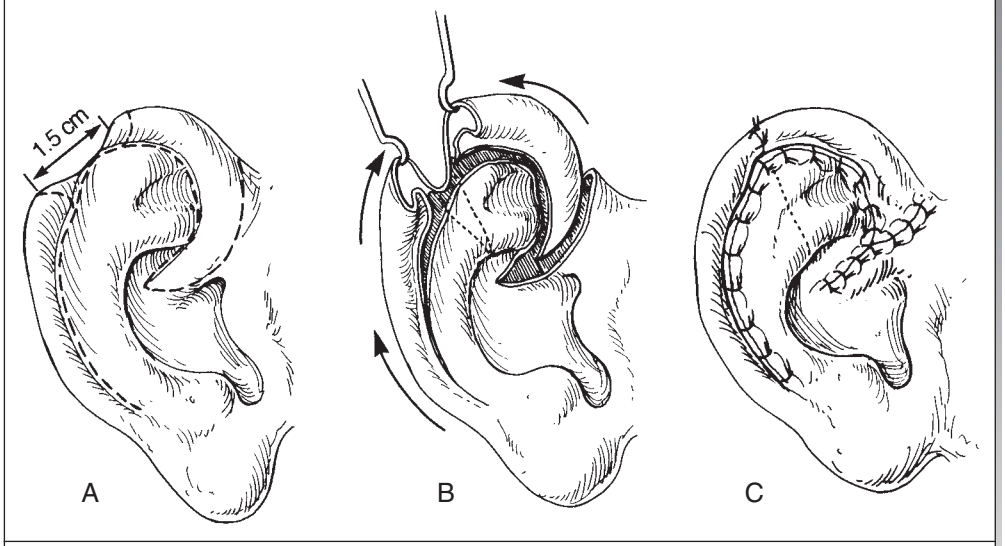


Figure 3 • Antia-Buch helical advancement. **A,** The helical defect and proposed incisions are outlined. **B,** The lateral skin and cartilage have been incised in a manner that leaves the medial skin intact as the two segments of the helical rim are advanced. This technique results in a dog-ear on the medial surface of the ear, which is excised, as shown by the *dotted line*. **C,** Final result showing that the size of the ear has been reduced, the crus of the helix has been advanced out of the concha, and the conchal donor site has been closed primarily. (Original artwork rendered by Kevin A. Somerville. Courtesy of Charles H. Thorne, MD.)

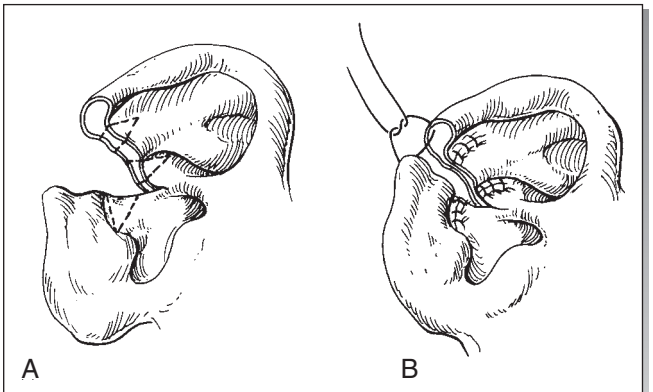


Figure 4 • A and **B,** Primary closure after wedge resection. To avoid excessive cupping and prominence of the auricle caused by the closure and to equalize the length of the sides to be closed, triangular excisions from the scapha and concha are performed. The size of the ear is significantly reduced by this procedure. (Original artwork rendered by Kevin A. Somerville. Courtesy of Charles H. Thorne, MD.)

technique is most applicable to non-melanoma skin cancer and traumatic defects because often only the helical rim is involved. Invasive melanoma, in contrast, is usually treated by wedge excision. Closure of the wedge inevitably causes "cupping" of the ear and a decrease in the size of the auricle. The tendency for "cupping" and the length of mismatch between the two sides of the wedge can be mitigated somewhat by removing accessory triangles in a "star" pattern (Fig. 4).

LARGER DEFECTS. Larger partial defects require cartilaginous support for reconstruction. The following guidelines (Firmin) are used:

1. A conchal cartilage graft can be used for cartilaginous support when the defect involves less than 25% of the helical length and two or fewer anatomic planes.
2. For defects involving greater than 25% of the helical length or smaller defects involving more than two anatomic planes, rib cartilage is required to provide adequate support (Fig. 5). The cartilage is covered with retroauricular skin and elevated at a second stage.

Thermal Injuries

Thermal injuries to the ear cause unique problems because of involvement of the adjacent retroauricular skin and scalp. Several patterns typically occur. In some cases, the helical rim will be lost, with the remaining ear and retroauricular skin left intact and uninjured. In such cases a tubed flap from the retroauricular sulcus can be used for staged reconstruction of the helical rim.

Injuries causing loss of the superior aspect of the helical rim and scapha are common. The best technique, as described earlier, involves the use of costal cartilage to reconstruct the superior part of the ear with cutaneous coverage. If local skin is not available because of the burn injury, the superior aspect of the ear can be reconstructed by transposing a chondrocutaneous flap from the concha. This technique yields a definite improvement in the appearance of the ear but falls far short of the potential results when rib cartilage and uninjured skin are used.

Malignant Melanoma

Melanomas of the ear are excised with the same margins as recommended elsewhere in the body. In the case of malignant melanoma in situ, however, a more conservative approach can be used because there is no reason to perform wedge excisions for intraepithelial malignancies. The skin is removed from the ear with preservation of the delicate perichondrium, and a full-thickness skin graft is placed on the perichondrium. Invasive melanomas less than 1.0 mm in thickness require a 1.0-cm margin and can be managed by wedge resection and primary closure. Deeper melanomas require wider margins, thereby precluding primary closure.

Microtia and Total/Near Total Ear Defects

The two most commonly used techniques for autogenous reconstruction of the auricle are the Brent four-stage technique and the Nagata two-stage technique.

Brent Technique

The Brent technique involves four stages commencing at 6 years of age and separated by an interval of at least 3 months.

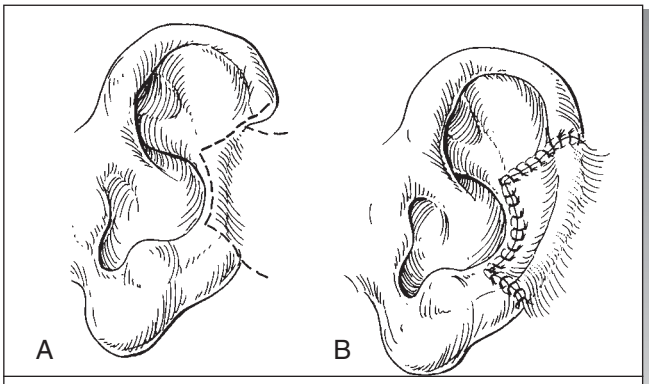


Figure 5 • Reconstruction of an auricular defect by means of a rib cartilage framework. **A,** An incision is made along the previous scar. **B,** The cartilage framework has been inserted and covered with retroauricular skin. A second stage is required for flap division and resurfacing of the medial surface. (Original artwork rendered by Kevin A. Somerville. Courtesy of Charles H. Thorne, MD.)

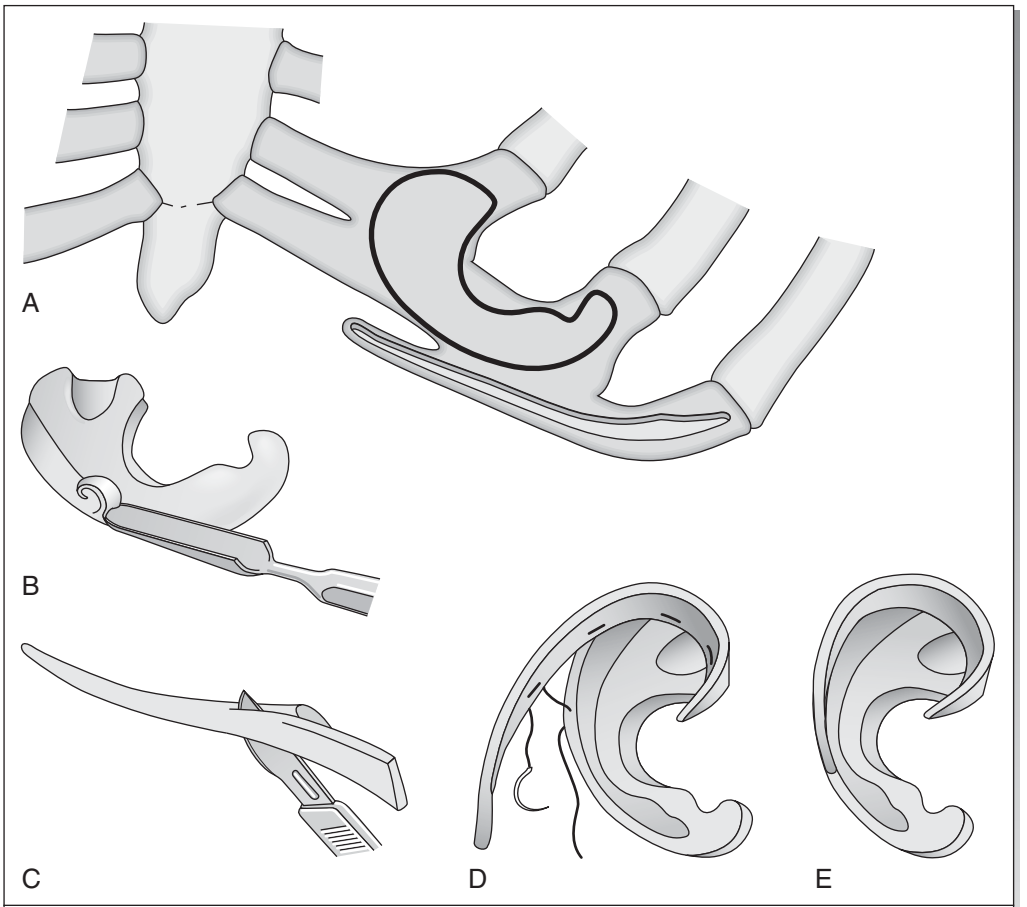


Figure 6 • First-stage auricular reconstruction, Brent technique. **A,** Donor site: contralateral thorax. Two pieces of cartilage are obtained. **B,** Sculpting the base block. **C,** Thinning the second piece to construct the helical rim. **D,** Suturing the helical rim to the base block. **E,** Completed framework. (From Sherrel J, Beaseley RW, Charles HM [eds]): Grabb & Smith's Plastic Surgery. Philadelphia, Lippincott Williams & Wilkins, 1997, p 431.)

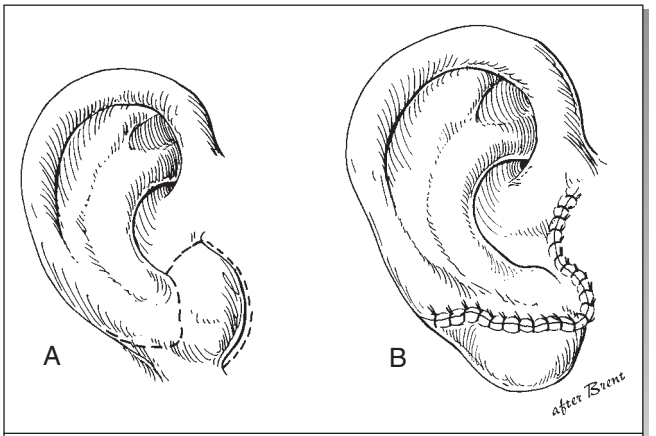


Figure 7 • Second-stage auricular reconstruction, Brent technique. **A,** An incision is made around the vertically malpositioned lobule such that a caudal pedicle is left, and a corresponding transverse incision is made over the caudal aspect of the framework. **B,** The lobule has been transposed and the cartilage framework inserted into the transferred lobule. (Original artwork rendered by Kevin A. Somerville. Courtesy of Charles H. Thorne, MD.)

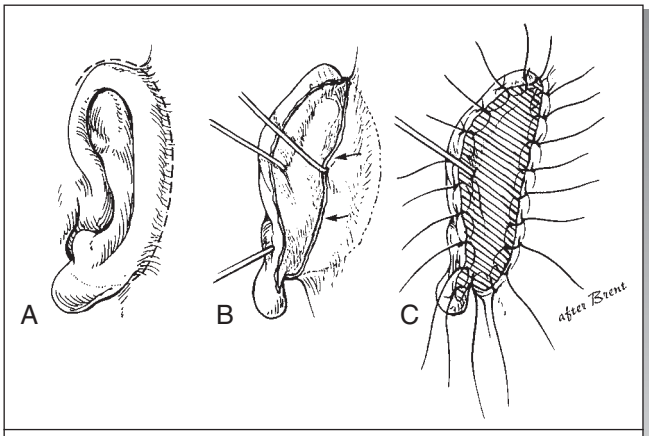


Figure 8 • Third-stage auricular reconstruction, Brent technique. **A,** An incision is made around the posterior 180 degrees of the buried framework. **B,** The retroauricular aspect of the scalp is undermined and advanced into the retroauricular sulcus. The raw surface on the medial aspect of the elevated framework is resurfaced with a skin graft. (Original artwork rendered by Kevin A. Somerville. Courtesy of Charles H. Thorne, MD.)

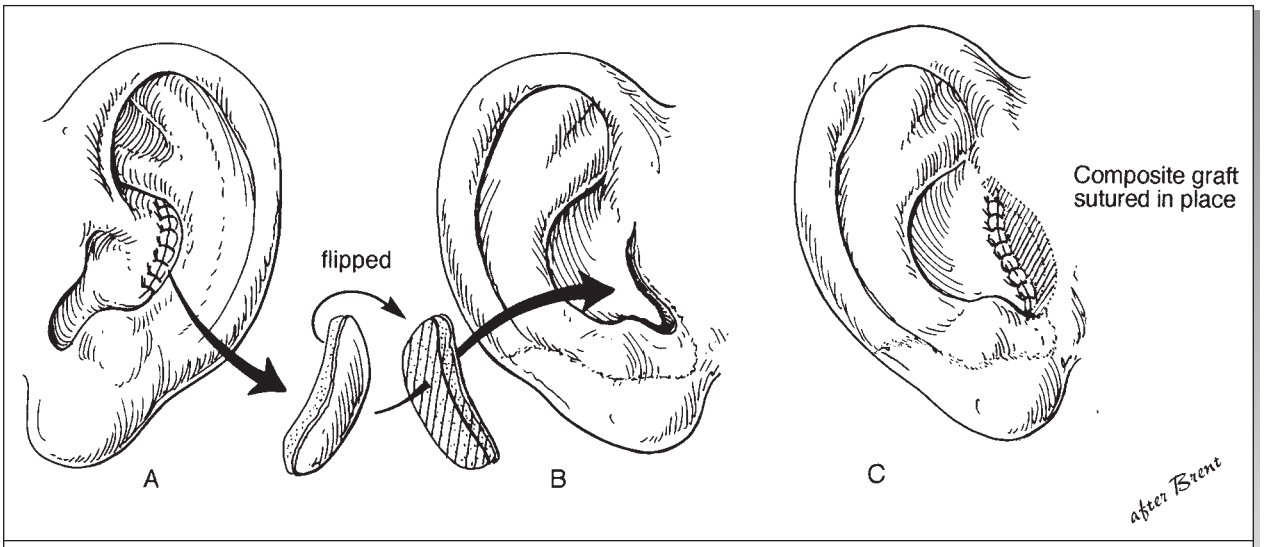


Figure 9 • Fourth-stage auricular reconstruction, Brent technique. **A,** A composite graft consisting of skin and cartilage is obtained from the lateral surface of the normal ear. **B,** An incision corresponding to the desired position of the tragus is made, the tragal flap is undermined, and the composite graft is sutured "upside down" under the tragal flap. **C,** The composite graft has been sutured in place. Not shown is a full-thickness skin graft, which is necessary on the floor of the concha because of elevation of the tragal flap. (Original artwork rendered by Kevin A. Somerville. Courtesy of Charles H. Thorne, MD.)

Stage 1. Dissection of the cutaneous pocket, removal of the cartilaginous vestige, and placement of the costal cartilage framework (Fig. 6)

Stage 2. Rotation of the malpositioned lobule (Fig. 7)

Stage 3. Elevation of the reconstructed framework, advancement of the retroauricular aspect of the scalp into the sulcus, and resurfacing of the medial surface of the elevated framework with a skin graft (Fig. 8)

Stage 4. Deepening of the concha and reconstruction of the tragus with a contralateral chondrocutaneous composite graft (Fig. 9)

In some cases in which the malpositioned earlobe is small, the second and third stages can be combined so that the total reconstruction requires only three stages.

Nagata Technique

The Nagata technique involves two stages separated by an interval of at least 6 months. This technique uses a more complex cartilaginous framework and is not performed before the age of 10.

Stage 1. Dissection of the cutaneous pocket, removal of the cartilaginous vestige, rotation of the malpositioned earlobe, and placement of a complex, cartilaginous framework, including reconstruction of the tragus and ascending crus of the helix (Figs. 10 and 11)

Stage 2. Elevation of the reconstructed auricle, placement of a cartilage graft deep to the framework to provide projection, coverage of the cartilage graft with a flap of temporoparietal fascia, and resurfacing of the defect with a skin graft (Fig. 12)

Pearls and Pitfalls

- The best result obtained from a Nagata-type reconstruction is superior to the best result from a Brent-type reconstruction.

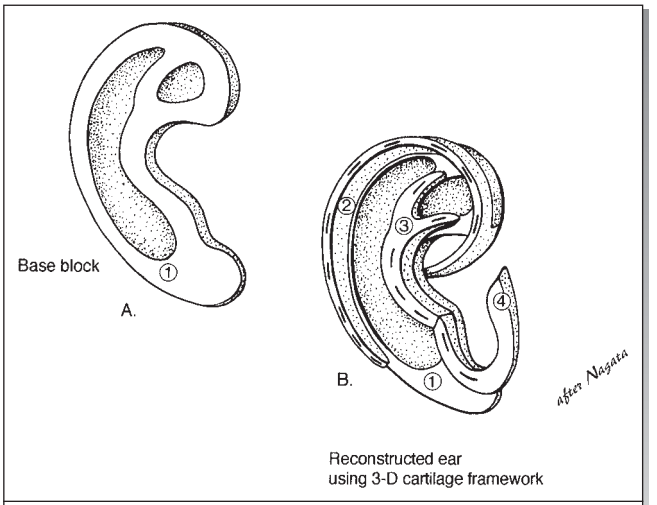


Figure 10 • First-stage auricular reconstruction, Nagata technique, Firmin modification. **A,** Base block. **B,** Four pieces of cartilage are used: the base block, the helical rim, the crura of the triangular fossa and antihelix, and the antitragus/tragus. The four pieces are secured with stainless steel wire tied on the deep surface of the framework. (Original artwork rendered by Kevin A. Somerville. Courtesy of Charles H. Thorne, MD.)

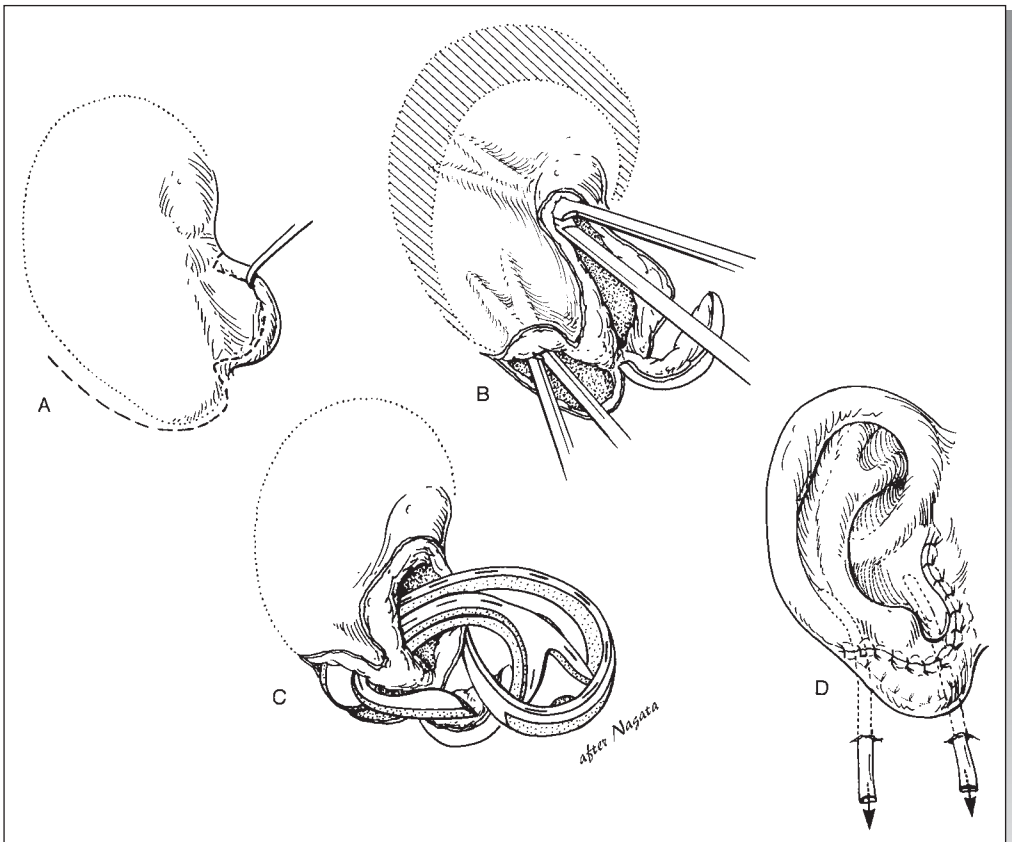


Figure 11 • First-stage auricular reconstruction, Nagata technique. **A,** The skin incision. **B,** The skin flap is elevated. Nagata recommends leaving a small subcutaneous attachment at the caudal tip of the skin flap. **C,** The framework is inserted. **D,** Suction is applied to the skin flap to reveal details of the framework. (Original artwork rendered by Kevin A. Somerville. Courtesy of Charles H. Thorne, MD.)

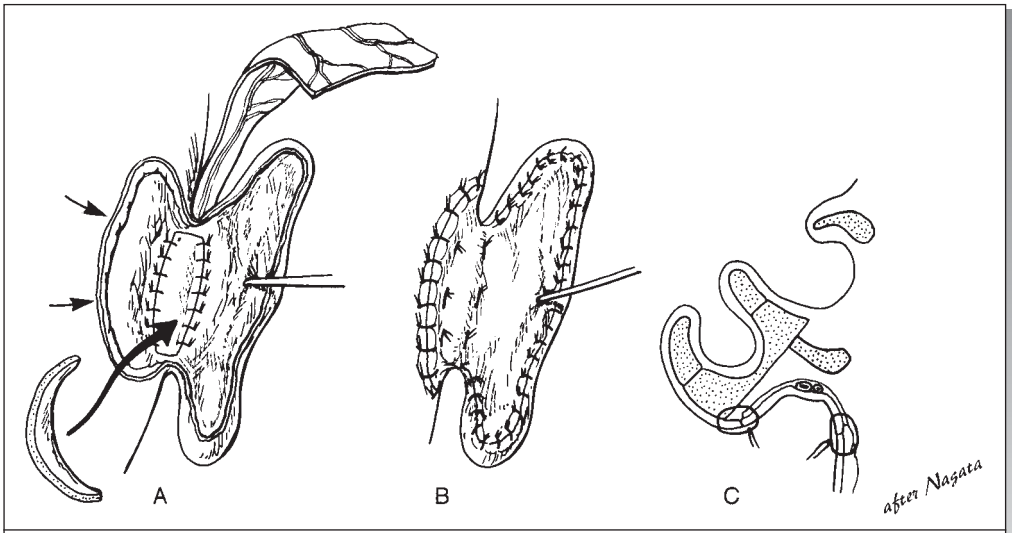


Figure 12 • Second-stage auricular reconstruction, Nagata technique. **A,** The reconstructed framework is elevated, a cartilage wedge is inserted deep to the antihelix for projection, and the medial surface of the ear and the cartilage are covered with a temporoparietal flap and skin graft (**B**). **C,** Cross section of the elevated framework and cartilage wedge showing the position of the temporoparietal flap and skin graft. (Original artwork rendered by Kevin A. Somerville. Courtesy of Charles H. Thorne, MD.)

- The Brent technique is safer. There are more complications associated with the Nagata technique, which demands *much* more of the overlying skin because the framework is more complex and more projected.
- Results are superior when the reconstruction is delayed until after the age of 10.
- Best results are obtained with increasing experience. Therefore, surgeons performing ear reconstruction should obtain maximal experience with the procedure.

SUGGESTED READINGS

Antia NH, Buch MS: Chondrocutaneous advancement flap for the marginal defect of the ear. Plast Reconstr Surg 39:472, 1967.

Brent BD: Technical advances in ear reconstruction with autogenous rib cartilage grafts—personal experience with 1,200 cases. Plast Reconstr Surg 104:319, 1999.

Firmin F: Ear reconstruction in cases of typical microtia. Personal experience based on 352 microtic ear corrections. Scand J Plast Surg 32:35, 1998.

Furnas D: Otoplasty. In Sherrel J, Beaseley RW, Charles HM (eds): Grabb and Smith's Plastic Surgery. Philadelphia, Lippincott Williams & Wilkins, 1997, p 431.

Mustarde JC: Correction of prominent ears using buried mattress sutures. Clin Plast Surg 5:459, 1978.

Nagata S: A new method of total reconstruction of the auricle for microtia. Plast Reconstr Surg 92:187, 1993.

Stenstrom SJ, Heftner J: The Stenstrom otoplasty. Clin Plast Surg 5:465, 1978.

Upper and Lower Eyelid Reconstruction

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Reconstruction of the eyelids must restore proper anatomic relationships, preserve function, achieve a pleasing aesthetic result, and most importantly, protect and preserve the integrity of the underlying globe. Restoration of the upper and lower eyelids requires evaluation of the (1) cause, (2) anatomic defect, and (3) availability and quality of tissues that can be used for reconstruction. Procedures available to the surgeon often overlap; consequently, indications for the type of reconstruction depend on the patient's diagnosis and needs, as well as the surgeon's experience with different techniques, previous outcomes, and personal preferences. Accurate preoperative assessment of these specific details and understanding of the available reconstructive techniques ensure the best result.

Etiopathogenesis

The causes of defects of the eyelids may be congenital, involutional, or posttraumatic. The most common causes are involutional age-related issues or postablative defects following removal of benign or malignant lesions. Congenital absence of eyelid tissue, or lid coloboma, is far less common and usually accompanied by a strong Bell phenomenon with satisfactory tear production.

The upper eyelid provides more than 90% of palpebral closure, producing most of the coverage of the cornea. Reconstructed upper eyelids have decreased mobility, proportional to the size of the initial defect. The resulting inability to fully close the lids usually results from compromised function of the orbicularis oculi muscle, the degree of which is dependent on the initial tissue loss. Elevation of the upper eyelid may also be compromised to varying degrees, depending on the amount of loss of the levator aponeurosis or muscle.

The orbicularis oculi, specifically, the preseptal and pretarsal heads, entwine the lacrimal sac and provide a pumping action that propels tears out of the palpebral aperture and prevents them from overriding the eyelid margin. This relationship is less important when planning reconstruction in patients beyond the sixth decade of life because tear production naturally diminishes each decade thereafter and renders epiphora less of a problem.

Although the lower eyelid moves superiorly only 1 to 2 mm, it does lubricate the inferior corneal pole. Consequently, in cases in which dryness is likely, the lower lid can be elevated to alleviate these symptoms. Function is paramount, and sacrifice of aesthetic appearance to narrow the palpebral aperture may be required.

Pathologic Anatomy

Normal *upper eyelid* anatomy is illustrated in Figure 1. The lid margin measures 30 mm in horizontal length. Three to four rows of cilia are directed up and away from the ocular surface. The anterior lamella of the lid is composed of skin and orbicularis muscle. The middle lamella of the upper eyelid above the lid crease is composed of orbital septum, orbital fat, and the levator complex. The middle lamella of the lower eyelid is composed of orbital septum, orbital fat, and the capsulopalpebral fascia (lower lid retractors). The posterior lamella is composed of the tarsal plate, Müller's muscle, and the conjunctiva. The tarsal plate measures approximately 10 mm in vertical height. The upper lid has two major vascular arcades: the marginal arcade,

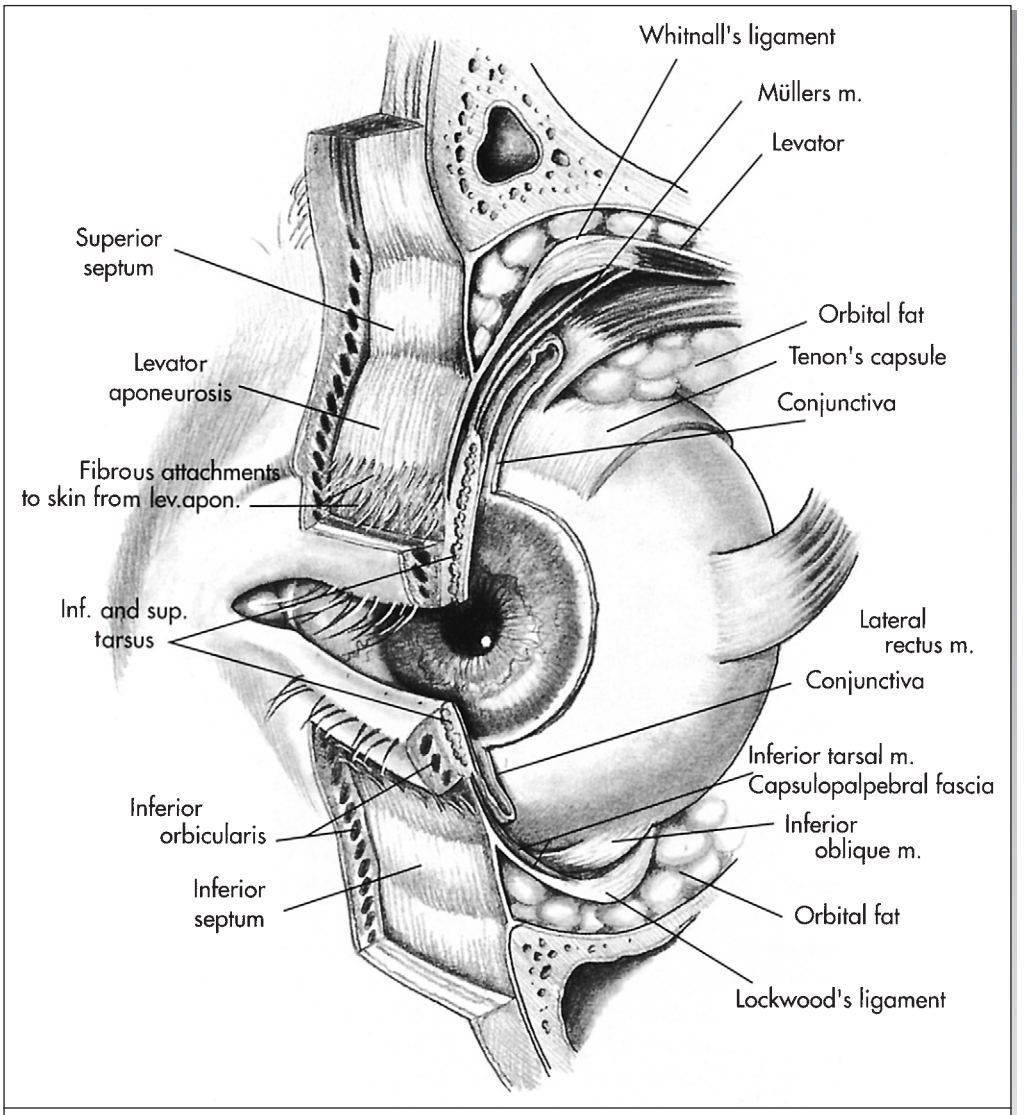

Figure 1 • Oblique section through the upper and lower eyelids. (From Nesi FA, Lisman RD, Levine MR [eds]: Smith's Ophthalmic Plastic and Reconstructive Surgery, 2nd ed. St Louis, Mosby-Yearbook, 1998, p 37.)

which lies just above and parallel to the lid margin beneath the orbicularis muscle, and the peripheral arcade, which lies above the superior tarsal border between the levator and Müller's muscle.

The anatomy of the *lower eyelid* is similar to that of the upper eyelid (see Fig. 1). The margin is approximately 30 mm in horizontal length with a single row of cilia. The anterior lamella is composed of the skin and orbicularis muscle. The posterior lamella consists of the tarsal plate, the lower eyelid retractors, and the conjunctiva. The tarsus of the lower eyelids is composed of dense connective tissue and measures approximately 4 to 5 mm in vertical height. The inferior marginal arcade parallels the lower lid margin and lies between the tarsal plate and the orbicularis muscle.

Moving anterior to posterior in both the upper and lower lids, the eyelid margin is composed of the lash line, the meibomian gland orifices, the gray line, and the mucocutaneous junction. The lash line is defined by the rows of eyelashes. The meibomian gland orifices are seen as a row of tiny pores within the tarsal plate that transmit secretions from the meibomian glands. The gray line defines the juxtaposition of the anterior and posterior lamellae and represents the superior end of Riolan's muscle, or the margin of the pretarsal orbicularis. The mucocutaneous junction separates the keratinized from the nonkeratinized epithelial border of the lid margin. Recognition of these landmarks is extremely helpful in obtaining correct anatomic alignment of the lid margin.

The posterior lamella is lined by the nonkeratinizing surface of the conjunctiva, and options for replacement include the surrounding conjunctiva, as well as buccal, gingival, or hard palate mucosa.

The tarsal plate can be replaced by free tarsal grafts, by advancement or transposition of the remaining tarsus, or by hard palate mucosa. It is difficult to use replacements that do not have a nonkeratinizing surface on one side, such as sclera, auricular cartilage, or synthetic products. If replacement tissue is not lined with a mucosal surface, the resulting granulation on that surface produces some element of cicatrization. The tarsus of almost all upper eyelids measures 10 mm in vertical height. All lid-sharing procedures must leave at least 3 to 4 mm to maintain integrity and stability of the upper eyelid. Therefore, an intact upper eyelid posterior lamella has up to 7 mm to contribute to a lid-sharing procedure for the opposing lower eyelid or for use as a posterior lamellar graft for another eyelid. The vertical tarsal height of a lower eyelid is 4 mm and thus has nothing to offer for donation to another eyelid except in a patient with eyelid laxity, where excess lid can be used as a composite graft.

The middle lamellae (the levator and the orbital septum, in the upper eyelid; Müller's muscle, the capsulopalpebral fascia, and the orbital septum in the lower eyelid) are reconstructed with relatively atonic soft tissue and fill the gap between the skin and conjunctiva, but they are rarely functional or mobile. The skin surface should be light, without bulk to avoid weight with subsequent retraction. Therefore, in a large defect, a skin graft is often preferable. Small to moderate-sized defects can be reconstructed with skin from surrounding or contiguous areas.

Diagnostic Studies

Accurate assessment of the defect is critical for optimal reconstructive results. In cases of eyelid trauma, careful exploration of the wound for foreign bodies should be performed. Medial defects require an evaluation of the lacrimal system to determine injury to the punctum or canaliculus. Such evaluation is best accomplished by probing and irrigation, which can easily be performed in the office for older children and adults but often necessitates examination under anesthesia at the time of surgery for young children or uncooperative patients. In all cases, examination of the underlying globe is recommended in addition to assessment of visual acuity. Evidence of ocular compromise, such as corneal exposure, penetrating injury, or loss of visual acuity, should prompt ophthalmologic consultation.

Reconstructive Goals

The goals of upper and lower eyelid reconstruction are as follows:

1. Seamless realignment of the eyelid margin to attain a pleasing height and contour
2. Placement of surgical incisions in natural skin lines when possible
3. Avoidance of pitfalls that can tether the lid, such as suturing the septum or levator
4. Restoration of lid functionality to preserve the integrity of the globe
5. Use of skin that most closely matches the thin eyelid skin when performing skin grafts
6. Avoidance of any rough surfaces (sutures, keratinized epithelial surfaces, etc.) that can traumatize the ocular surface during posterior lamella reconstruction

Treatment

The eyelids are a privileged, highly vascular site; therefore, composite grafting or resuturing of avulsed tissue is a reasonable first attempt.

Partial-Thickness Defects

NONSURGICAL. Granulation is possible in small defects only if the posterior lamella is intact. If healing by secondary intention is allowed in full-thickness defects, coloboma or lid retraction most likely results. Even if the aesthetic result is satisfactory, the mucocutaneous junction is disrupted and superficial punctuate keratopathy, a foreign body sensation, or trichiasis may result. Small lower eyelid defects, when allowed to heal secondarily, can lead to lower lid retraction. Such retraction may be problematic only if the defect is medial and produces eversion of the punctum. Satisfactory results can be anticipated with secondary healing of small medial canthal defects; however, punctual eversion can be expected with defects encroaching on the punctum. For combined medial canthal and palpebral defects, band or web formation is likely.

SURGICAL. Defects involving the anterior lamella must be assessed in the context of the surrounding soft tissues. In older individuals and patients with anterior lamellar laxity, it may be possible to recruit redundant skin to close the defect primarily or fill the defect with a local myocutaneous advancement flap. In younger individuals or older persons who lack excess skin, it may be necessary to fill the defect with a full-thickness skin graft. Donor sites should be chosen to match the thin skin of the eyelids as closely as possible, and grafts may be harvested from the superior lid fold (as in a blepharoplasty incision), the posterior auricular region, or the supraclavicular skin. Grafts should be thinned. More extensive defects may require larger flaps from the forehead or cheek, the description of which is beyond the scope of this chapter.

Full-Thickness Defects

DEFECTS LESS THAN A THIRD—DIRECT REAPPROXIMATION. Small defects in both the upper and lower eyelid are often reconstructed by directly approximating the edges of the defect. In general, defects measuring less than a fourth of the lid margin can be directly closed, whereas defects between a fourth to a third of the margin often require lateral cantholysis to realign the edges without tension. With the use of 6–0 silk, the edges of the margin are joined to approximate three anatomically similar points: the meibomian gland orifices, the lash line, and the gray line or mucocutaneous junction (Fig. 2). The sutures are cut long and tied away from the surface of the eye. The edges of the tarsal plate are approximated with one to two interrupted 6–0 chromic sutures while taking care to avoid corneal abrasion by ensuring that the suture does not pass through the conjunctiva on the upper lid. The anterior lamellar tissues can then be closed with interrupted or running 6–0 silk sutures, with one of them used to tie the long lengths of marginal sutures inferiorly away from the globe.

If the eyelid margin is tight, tethered, or retracted at the time of closure, some form of lateral canthal release is required. Canthotomy alone allows some advancement of the eyelid, but when combined with release of the superior or inferior crus of the lateral canthal tendon, it can allow for closure of defects that encompass up to 30% of the length of the upper or lower eyelids. If canthotomy is needed, a new, sharp canthal angle is created with both absorbable sutures and a permanent suture between upper and lower eyelids at the site of the desired lateral angle. It is occasionally necessary to support the remaining lateral segment of the eyelid with a canthopexy suture. Canthoplasty is not usually possible because of the degree of lateral eyelid loss.

Careful judgment is required when determining the need for additional tissue transfer with a semicircular flap from the lateral canthus (Tenzel's semicircular flap—see later). It is not possible to perform a Tenzel flap if a canthotomy has been performed. Therefore, in cases in which a canthotomy may not allow the needed advancement, it should be avoided in favor of rotation and advancement of this tissue.

DEFECTS BETWEEN A THIRD TO TWO THIRDS—TENZEL'S SEMICIRCULAR ADVANCEMENT FLAP. Reconstruction of defects in both the upper and lower lids that measure between a third and two thirds of the length of the lid margin requires an advancement flap, which is best accomplished with the use of a Tenzel (semicircular advancement) flap (Fig. 3). Skin and muscle are recruited from the lateral canthus by making an inferiorly directed semicircular incision to reconstruct the lower lid or a superiorly directed semicircular incision to reconstruct the upper eyelid. The lateral canthal dissection should reach the lateral orbital rim. Either a superior or inferior cantholysis is performed when reconstructing the upper or lower lid, respectively. This maneuver enables mobilization of the lateral portion of the remaining lid in a nasal dissection. After widely undermining the semicircular flap, the lateral lid tissue can be mobilized to meet the nasal edge of the defect. The lid margin is realigned as described earlier. The lateral canthus is reconstructed with 4–0 PDS suture to plicate the orbicularis muscle of the advancement flap to the periosteum at the inner contour of the lateral orbital

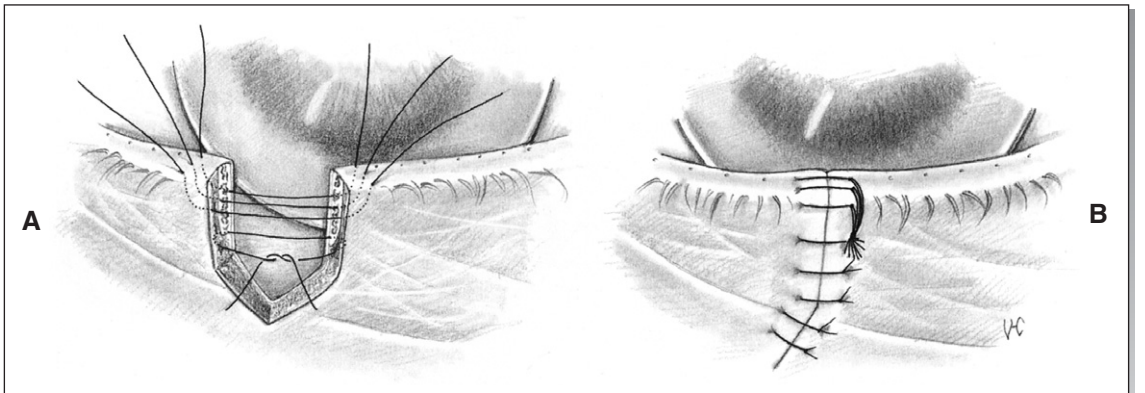

Figure 2 • A, A laceration of the eyelid margin has been freshened in a pentagonal wedge configuration. The first silk suture is passed through the tarsal plate and exits the meibomian gland orifices. The suture is tied, and the remaining tarsal plate is closed with partial-thickness 6–0 Vicryl sutures. Two more 6–0 silk sutures are placed behind the lash line and in front of the lash line. **B,** The marginal silk sutures are left long and incorporated into the first skin suture, and the remaining skin is closed with 6–0 silk sutures. The inferior portion of the wound has been modified in a hockey stick configuration to reduce tissue puckering. (From Nesi FA, Lisman RD, Levine MR [eds]: Smith's Ophthalmic Plastic and Reconstructive Surgery, 2nd ed. St Louis, Mosby-Yearbook, 1998, p 185.)

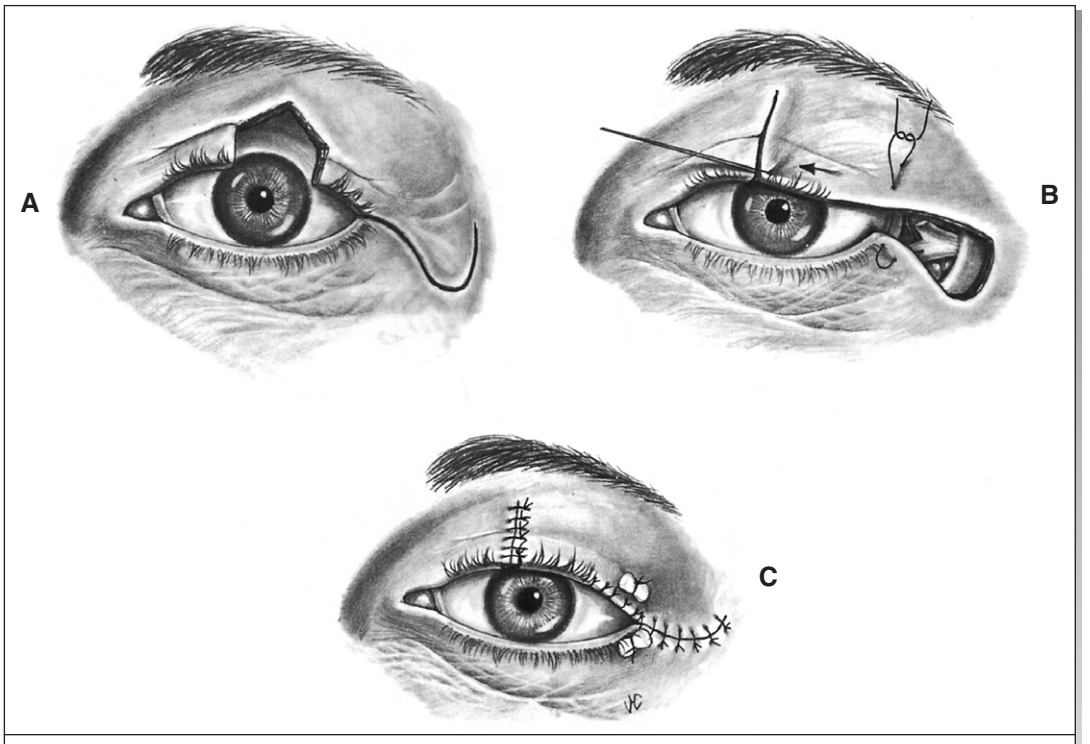

Figure 3 • Tenzel's semicircular flap for closure of an upper lid defect. **A,** Upper lid defect with an outline of the lateral semicircular advancement flap to be developed. **B,** Advancement of a skin-muscle flap to fill the upper lid defect. The flap should be fixated at the lateral canthus and lateral orbital tubercle with a mattress suture that encompasses the lower ramus of the lateral canthal tendon. **C,** Final closure of Tenzel's semicircular flap to correct the upper lid. The lateral canthal area has been reconstructed, and the conjunctiva has been advanced and sutured to the new lid margin. (From Nesi FA, Lisman RD, Levine MR [eds]: Smith's Ophthalmic Plastic and Reconstructive Surgery, 2nd ed. St Louis, Mosby-Yearbook, 1998, p 585.)

rim. The lateral incision is closed with interrupted or running 6–0 silk or nylon suture.

DEFECTS GREATER THAN TWO THIRDS—LID-SHARING FLAPS

Upper Eyelid. The Cutler-Beard bridge flap is used to reconstruct large, full-thickness defects of the upper lid (Fig. 4). Creation of this lid-sharing flap involves the use of a full-thickness lower lid flap of skin, the orbicularis muscle, and the conjunctiva raised from an incision placed 4 to 5 mm below the lower lid margin. In this way, the lower lid incision is placed below the level of the marginal arcade to avoid ischemia of the remaining bridge of the lower lid margin. Flap tension is relieved by making two parallel vertical full-thickness incisions deep into the inferior fornix. The flap is passed beneath the remaining "bridge" of lower lid margin and advanced superiorly to fill the upper lid defect. The advanced conjunctival edge of the flap is sutured to the residual edge of the upper lid conjunctiva. The skin and muscle edges are approximated along the flap-host interface. The flap is divided 6 to 8 weeks later by making a horizontal incision through the flap approximately 2 mm lower than the desired level of the upper lid margin to allow for flap contraction. The flap stretches over time to add length to the reconstructed upper eyelid. The incision is slightly beveled to allow the conjunctival side to be longer than the skin and muscle of the reconstructed upper lid. This maneuver prevents contact of keratinized epithelium with the surface of the eye.

Lower Eyelid. The tarsoconjunctival, or Hughes', flap is the procedure of choice when reconstructing large defects of the lower lid (Fig. 5). The tarsoconjunctival flap is visualized after eversion of the upper eyelid over a Desmarres retractor. Three to 4 mm of tarsus is left undisturbed, and a horizontal incision is placed through the tarsus along either the entire extent of the tarsus or only a segment of the tarsus if a small amount of tissue is required. Dissection is carried out between the tarsus and the orbicularis, up to the upper border of the tarsus. Here, the dissection plane is between the conjunctiva and Müller's muscle. Attempts should be made to avoid injury to Müller's muscle because such injury may result in upper eyelid retraction after the second stage. Flap elevation is extended well up into the superior fornix to allow for significant laxity. The flap is advanced inferiorly to fill the lower lid defect and sutured in tongue-in-groove fashion into the

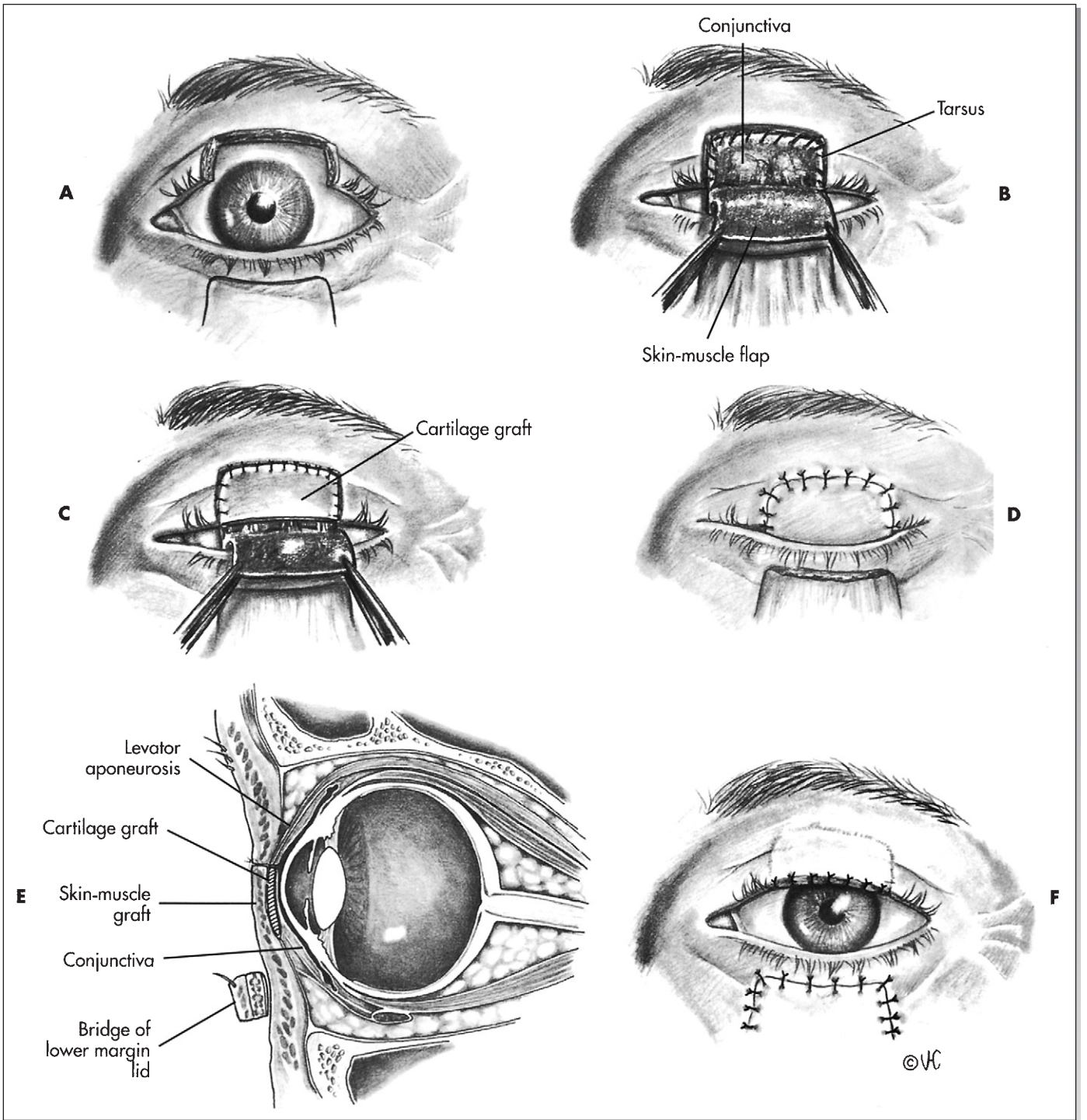

Figure 4 • Cutler-Beard bridge flap. **A,** Rectangular upper lid defect that can be reduced by horizontal stretching so that proper width of lower lid advancement flap can be determined. The lower lid skin-muscle-conjunctival flap should be developed with a horizontal incision not closer than 5 mm to the lid margin. **B,** Advancement of a full-thickness lid flap from the lower lid into the upper lid defect. The inner layer of conjunctiva and capsulopalpebral fascia has been sutured to the conjunctiva of the upper lid. **C,** An autogenous cartilage graft has been sutured in position and attached to remnants of the tarsal edge and levator muscle. This step is often unnecessary. **D,** A skin-muscle flap is sutured into the skin-muscle defect in the upper lid. **E,** Sagittal view of the lower lid flap in place. An autogenous cartilage graft is shown sandwiched between the conjunctiva from the lower lid and the skin-muscle flap. **F,** Appearance after separation of the flap 6 weeks later. It is important when separating the flap to ensure that there is extra conjunctiva to evert around the upper lid margin, as illustrated. Small traction folds must be excised for the lower lid to close properly. (From Nesi FA, Lisman RD, Levine MR [eds]: Smith's Ophthalmic Plastic and Reconstructive Surgery, 2nd ed. St Louis, Mosby-Yearbook, 1998, p 1586.)

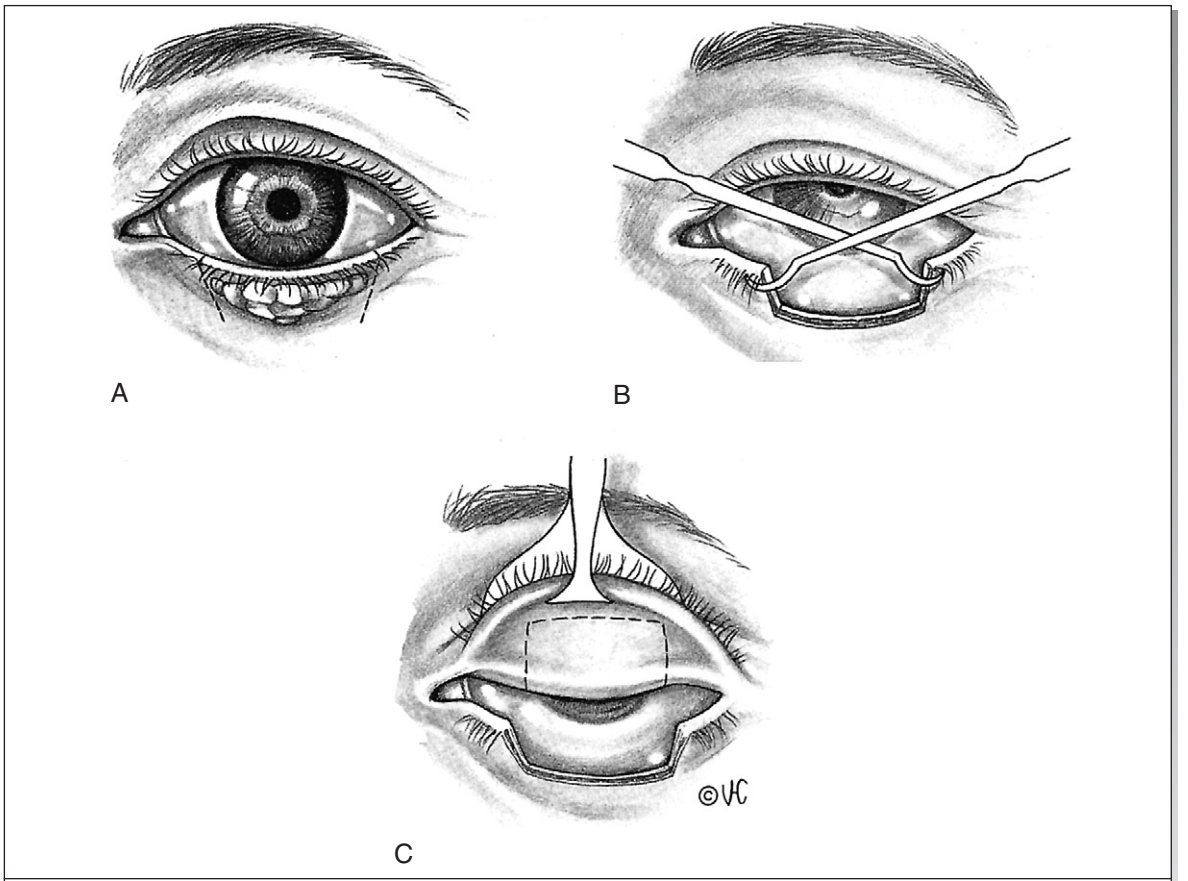

Figure 5 • Tarsoconjunctival flap for repairing a lower lid defect. **A,** Tumor to be excised. **B,** Pulling borders of the defect together to see the minimal amount of tissue necessary for lid reconstruction. **C,** Outline of the tarsoconjunctival flap in the upper lid while staying 3 mm from lid margin. *Continued*

remaining tarsal edges of the lower eyelid. If none exists, the lateral tarsal margins of the flap are sutured to the inner aspect of periosteum at the lateral orbital rim. Medially, it is helpful to suture the flap to a remnant of the canthal tendon. The anterior lamella can be reconstructed by transposition or advancement of surrounding tissue; however, in most situations, the use of a full-thickness skin graft is preferred. Retraction of the newly reconstructed lower eyelid is common, and the use of lighter skin grafts reduces the incidence of contraction. The lower eyelid is subject to involutional changes more than the levator-supported upper eyelid. Even if a thicker flap reconstruction is initially in a satisfactory position, it is subject to laxity over time. For that reason alone, less bulky resurfacing is preferred. If a skin graft is placed, it must be overstretched onto the conjunctival flap because in the second stage, eyelid height is placed at or almost above the inferior limbus.

The flap can be divided as early as 2 weeks, if needed, but most flaps remain in place for 4 to 6 weeks. The flap should be incised straight across to achieve a pleasing height. If it is arched, the eyelid falls further when the patient is upright and it will be too low. If it is incised straight across, the medial and lateral support holds the eyelid extremities, and the central area bows naturally to achieve a pleasing contour.

Postsurgical Care

Corticosteroid/antibiotic combination eye drops and analgesic medications are prescribed for the first postoperative week. Skin sutures are removed within the first postoperative week. Sutures aligning the lid margin are left in place for 10 to 14 days. Eyelid-sharing advancement flaps are divided 4 to 6 weeks postoperatively as described earlier.

Pearls and Pitfalls

- Resuturing of the eyelid margin during a standard horizontal shortening procedure should not be left “overcorrected” with a bump at the

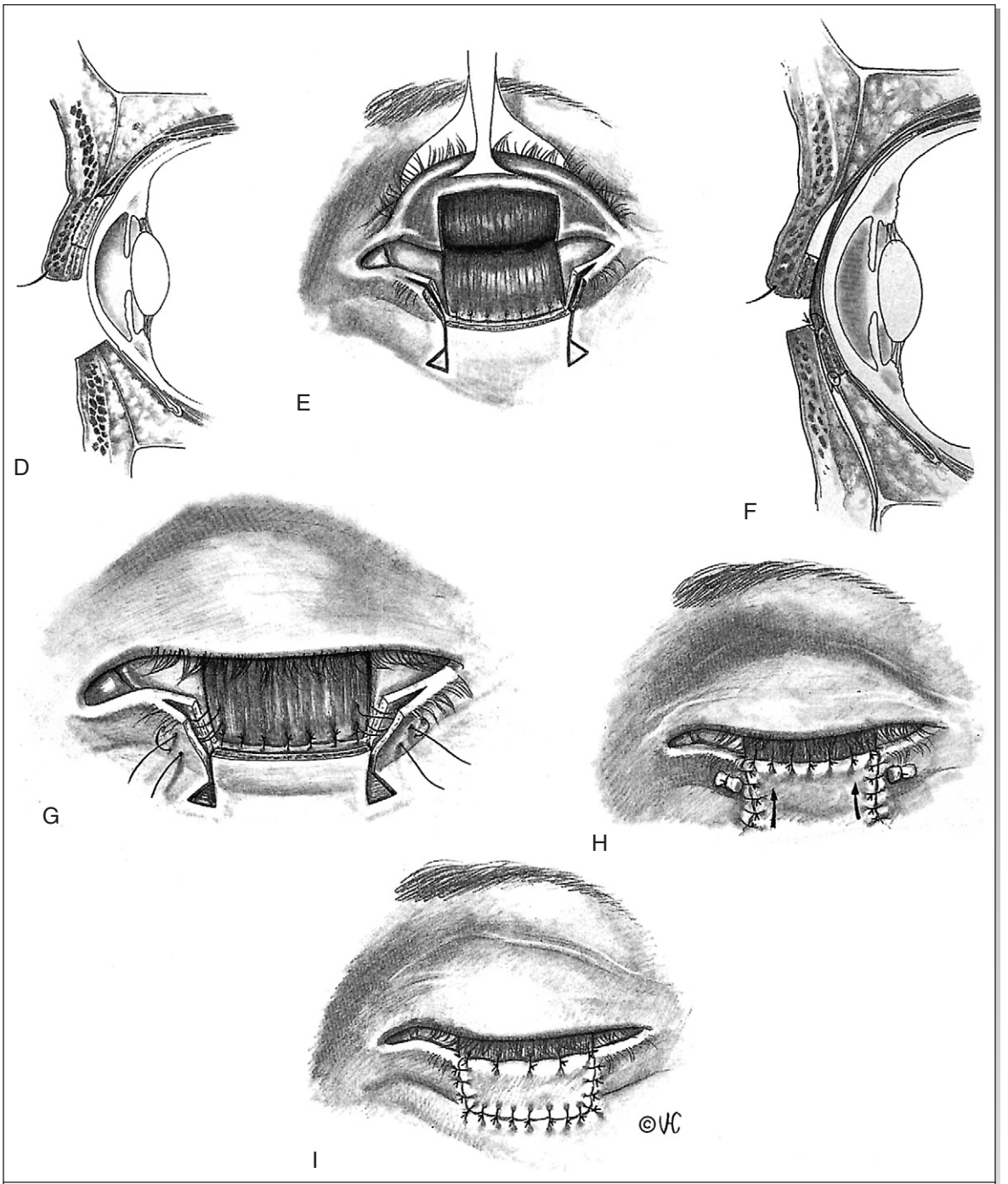

Figure 5, cont'd • D, Sagittal view of the incision. *Dotted lines* indicate planes of dissection. If Müller's muscle is to be included in the conjunctival flap, the dissection plane is between Müller's muscle and the levator aponeurosis. If Müller's muscle is left in place, the dissection plane is between Müller's muscle and the conjunctiva. **E,** The tarsoconjunctival flap is advanced into the lower lid. The inferior margin of the flap is sutured to the conjunctiva and the lower lid retractors with continuous 8–0 chromic suture. **F,** Sagittal view of the tarsoconjunctival flap. **G,** Technique of anchoring the tarsoconjunctival flap to the remaining lid. The lid is split at the gray line for a distance of 2 to 3 mm, and each arm of double-armed 6–0 silk suture enters the skin of the lid, exits the apex of the groove, takes a full-thickness bite in the tarsoconjunctival flap, reenters the apex of the wound, and exits the skin where the suture is tied over a cotton bolster. **H,** The anterior lamella is repaired with an advancement skin flap. **I,** The anterior lamella can also be repaired with a skin graft.

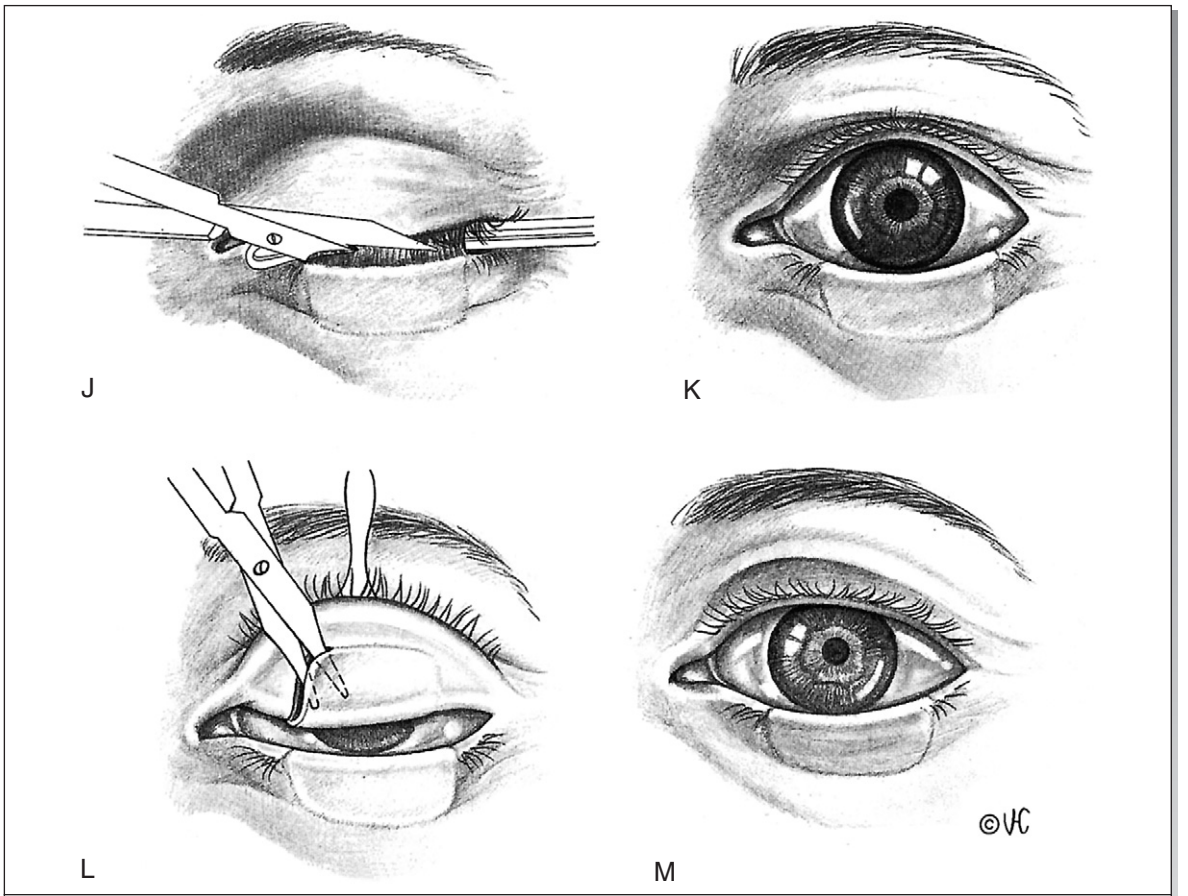

Figure 5, cont'd • J, At the second stage, the flap is incised beneath the lid at the point where it comes from the tarsus. The lower lid is left slightly on the high side, approximately 1 mm, to allow for slight contracture. The remainder of the flap is trimmed. **K,** The patient is asked to open his eyes, and the contour of the upper lid is checked to follow the eye. **L,** If there is elevation or notching of the upper lid, the lid is double everted over a Desmarres retractor and all scar bands to the skin are divided. This technique is especially useful on the medial and lateral corners where the flap was formed. **M,** The procedure is continued until the lid has the exactly desired curvature. (From Della Rocca RC, Nesi FA, Lisman RD [eds]: Smith's Ophthalmic Plastic and Reconstructive Surgery, vol 2. St Louis, Mosby, 1987.)

eyelid margin. If the tarsus is adequately reunited with a long-acting resorbable suture, overcorrections are not required.

- If a semicircular flap or canthotomy has been used, a mild degree of lateral canthal supraplacement is desired.
- Two-stage reconstructions may require treatment for retraction of the upper eyelid. If a large amount of upper eyelid retraction results, one can evert the upper eyelid on a Desmarres retractor and space or gap the granulating wound. It may even be possible to disinsert the levator from the posterior margin of the tarsus to complete the recession of the eyelid as early as 1 to 2 weeks after the second stage.
- If the lower eyelid is too far superior to the lower limbus (too high), it can be lowered by straight resection with sharp-pointed scissors to reduce its height.
- If the newly reconstructed lower eyelid margin granulates with chronic erythema because of chronic keratinization of a conjunctival surface that has migrated too far anteriorly, that area can be reresected at the eyelid margin to a minimal degree and be allowed to "granulate" once again. Any irregularities at the periphery of an eyelid skin graft can be corrected by dermabrasion or the use of a laser to plane the contour or height to achieve a more acceptable shape or contour.

SUGGESTED READINGS

Cutler NL, Beard C: A method for partial and total upper lid reconstruction. Am J Ophthalmol 39:1, 1955.

Hughes W: Reconstruction of the lid. Am J Ophthalmol 28:1203, 1945.

Kwitko GM, Nesi FA: Eyelid and ocular adnexal reconstruction. In Nesi FA, Lisman RD, Levine MR (eds): Smith's Ophthalmic

Plastic and Reconstructive Surgery, 2nd ed. St Louis, CV Mosby, 1998, p 576.
Lemke BN, Lucarelli MJ: Anatomy of the ocular adnexa, orbit, and related facial structures. In Nesi FA, Lisman RD, Levine MR (eds): Smith's Ophthalmic Plastic and Reconstructive Surgery, 2nd ed. St Louis, CV Mosby, 1998, p 3.
McCord CD, Lisman RD: Upper eyelid reconstruction. In Della Rocca RC, Nesi FA, Lisman RD (eds): Smith's Ophthalmic Plastic and Reconstructive Surgery, vol 2. St Louis, CV Mosby, 1987, p 822.
Tenzel RR: Eyelid reconstruction by the semicircle flap technique. Ophthalmology 85:1164-1169, 1978.
Tenzel RR: Lid reconstruction. In Della Rocca RC, Nesi FA, Lisman RD (eds): Smith's Ophthalmic Plastic and Reconstructive Surgery, vol 2. St Louis, CV Mosby, 1987, p 771.

The Nose

FREDERICK J. MENICK

The origins of plastic surgery are rooted in repair of the nose. The nose, as a central facial figure, contributes significantly in telling the world who we are and what we may become.

Etiopathogenesis

In ancient times, noses were commonly amputated in war or as punishment for moral or political transgressions. Today, nasal defects most often follow the ablation of cutaneous skin cancer (basal cell carcinoma, squamous cell carcinoma or, less commonly, melanoma). Human and animal bites may also occur. Destruction of nasal support alone may follow facial fracture, but cartilage destruction without loss of cover more often follows necrosis of the intranasal lining as a result of infectious complications of syphilis, leprosy, noma, or following cocaine use. Inflammation and destruction of nasal lining expose and devascularize the septal cartilage, thereby leading to progressive nasal collapse and lining contraction. Infectious diseases such as noma or meningococcemia may lead to full-thickness nasal destruction. A multiply operated cosmetic rhinoplasty patient may also have scarred, contracted, or excised nasal lining; absent or distorted bone and cartilage framework; and scarred, contracted external skin.

The nose is made of a thin, pliable vascular lining and a sculptured soft and hard tissue framework composed of subcutaneous fat and muscle covering the nasal bones, upper lateral cartilage, septum, and alar cartilage. The framework is covered by a thin conforming skin envelope. When all or part of the nose is missing, the requirements for repair depend on the degree of cover, support, and lining loss, as well as the goals of restoration. Ideally, the repair combines restoration of function (avoiding airway obstruction as a result of soft tissue collapse, excessive bulk, or constricting scar) and restoration of a normal attractive nasal appearance.

Preoperative Evaluation

The first step is to make a diagnosis and formulate a treatment plan. If possible, the patient is examined before excision of a skin cancer. The patient usually has an acute or old nasal defect after Mohs' surgery, trauma, or infection, and the apparent defect may not reflect what is truly missing. Edema, gravity, and skin tension may enlarge the apparent defect, whereas secondary healing and contraction may decrease the apparent defect. Clinical examination combined with evaluation of medical photographs permits formulation of a surgical plan. Radiographic evaluation of large complex defects involving bone or extensive soft tissue by computed tomography or magnetic resonance imaging is useful. Sources of donor material, including the forehead, cheek, intranasal lining, septum, ear, rib, cranium, and ilium, should be evaluated.

Goals of Reconstruction

It is important to evaluate the defect in the context of normal facial aesthetics. The desired three-dimensional reconstruction must be clearly visualized to allow a specific, exact, properly staged plan to be formulated.

Patients must be given a treatment choice. The defect can be allowed to heal by secondary intention, or a skin graft or flap can be used to close the wound. However, most patients are fastidious and wish the missing part to be restored to its original color, texture, contour, and function. The number of surgical stages, morbidity, aesthetic requirements, and financial costs must be discussed.

The external surface of the nose is crossed by ridges and valleys that separate it into slightly convex or concave surfaces or subunits—the tip, dorsum, paired sidewalls, alar lobules, soft triangles, and columella (Fig. 1). The regional units are

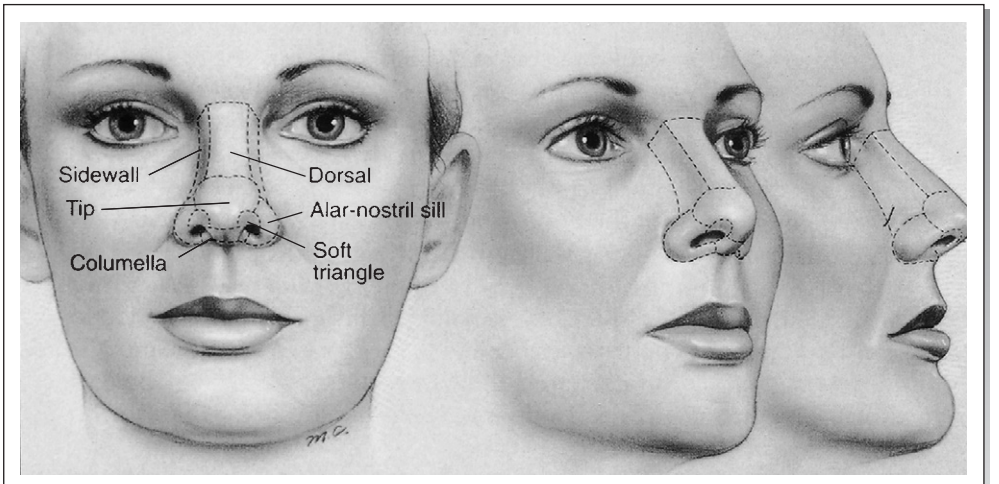

Figure 1 • The nasal subunits are adjacent topographic areas individually characterized by skin quality, border outline, and three-dimensional contour. (From Burget G, Menick F: Aesthetic Reconstruction of the Nose. St Louis, Mosby–Year Book, 1994.)

adjacent topographic areas with characteristic skin quality, border outline, and three-dimensional contour. If a "normal" appearance is the goal, the regional units be restored.

The wound often needs to be altered in site, size, and depth to improve the outcome. Flaps often leave trapdoor scars as a result of fibrotic contraction in the interface between the recipient bed and the donor tissue. The flaps often become raised above the adjacent normal skin (unlike skin grafts, which may atrophy or become unpredictably pigmented). Consequently, it is often preferable, when using a flap, to resurface an entire convex subunit rather than part of a subunit so that the expected wound contraction augments rather than distorts the contour of the final result. The subunit principle follows: if a defect occupies greater than 50% of a subunit, consider discarding adjacent normal tissue within the subunit so that the entire subunit is resurfaced with a single flap. This approach (which is not applicable to skin grafts or to flat or concave subunits) also positions scars in the contour depressions between subunits where they are better camouflaged.

Absent tissue must be replaced exactly, neither underfilling nor overfilling the defect. Flap dimensions are not taken from a fresh gaping wound or an old contracted defect but from the contralateral, unaffected side or a model of an ideal nose.

When repairing composite defects that encompass multiple facial units, tissue settling and scar distortion should be anticipated. If a facial landmark must be built on another (nose on lip and cheek), reconstruct the lip and cheek at the first operation and build the nose only after a stable platform is established.

Distant free flaps are the first choice for large, complicated defects. Distant tissue provides bulk, protects vital structures, revascularizes the wound, and reconstructs a stable platform. At a later stage, facial skin quality, border outline, and three-dimensional contour are restored by using local tissues and traditional nasal reconstructive techniques. Distant tissue transfers are thus used only for lining and support.

Treatment

The important facial landmarks, specifically, the glabellar frown lines, the nasal midline, the nasal subunits, the margin of the defect, old scars, the nasolabial folds, and the philtrum columns and vermilion, should be clearly marked with ink. Complicated cases are performed under general anesthesia because local anesthesia with epinephrine is not routinely used so as to avoid tissue distortion and blanching, findings that may make tissue vascularity more difficult to evaluate. A template of the missing skin defect is fashioned in three dimensions (using the defect itself, the contralateral normal side, or an ideal unit) from an aluminum foil suture pack or from quarter-inch tape strips covered with collodion. Templates are invaluable in designing the size and outline of skin cover, lining, and cartilage requirements.

Nasal Cover

SMALL SUPERFICIAL DEFECTS. Full-thickness preauricular, postauricular, or clavicular skin grafts are used to resurface the thin skin of the upper two thirds of the nose when only a millimeter or two of subcutaneous fat is missing. With the exception of full-thickness skin grafts from the forehead, skin grafts invariably heal with a shiny, atrophic appearance and they are thus avoided within the thick skin zones of the tip and ala.

For defects that extend through the subcutaneous fat to the underlying cartilage framework but are less than 1.5 cm in size, local adjacent tissue can be redistributed as transposition flaps. A single-lobe transposition flap is useful within the upper two thirds of the nose, whereas a bilobed flap is most successful within the tip and ala. Both flaps are contraindicated for larger defects because of insufficient residual adjacent skin to redistribute over the nasal surface.

LARGE DEEP DEFECTS. Isolated alar defects that extend to the lining require primary cartilaginous support and can be reconstructed with a two-stage nasolabial flap. If more than 50% of the alar skin is missing, the residual normal skin within the ala is excised. A three-dimensional foil pattern of the contralateral normal ala is designed to serve as a template for skin cover. Septal or ear cartilage is harvested. An alar cartilage batten is designed to support and shape the alar rim and is sutured to residual or repaired nasal lining. The skin template is positioned along the ipsilateral nasolabial fold, near the commissure, to ensure an adequate arc of rotation to reach the alar defect. It is tapered proximally to avoid an extension onto the nasal sidewall and distally to allow excision of the dog-ear. The flap is incised and elevated distally with 2 to 3 mm of subcutaneous fat. The proximal dissection is carried deeper to include all subcutaneous tissue up to the facial musculature, thus preserving the underlying perforators from the facial and angular artery. The flap is transposed and inset. At 3 weeks, the pedicle is divided flush with the cheek. The flap can be partially reelevated and the underlying subcutaneous tissue and scar sculpted to recreate the normal shape of the ala lobule. It is wrapped inferomedially to restore the alar base. The limited size, vascularity, and arc of rotation restrict use of the nasolabial flap to isolated alar defects that do not extend significantly onto the nasal sidewall.

Because of its ideal skin quality, vascularity, and position, the forehead flap is used for most significant nasal reconstructions. Adjacent normal tissue is excised within the subunit if the defect encompasses more than 50% of that subunit. A template is precisely designed to replace the missing surface skin. In a heminasal defect, the contralateral normal side serves as a guide. In a more complex repair, the ideal nose may be sculpted in clay on a facial moulage of the patient.

The supratrochlear vessels (Fig. 2) are identified with a Doppler probe lateral to the medial frown crease. If the nasal defect involves the lateral part of the nose, an ipsilateral pedicle is used. If the defect occurs in the midline or is bilateral, either pedicle can be used. The template is positioned vertically over the appropriate supratrochlear pedicle extended to the hairline. Its arc of rotation can be increased by placing the columella extension within the hair-bearing scalp or by incising its 1.2-cm pedicle base across the eyebrow toward the medial canthus, thus lowering its pivot point. The flap is incised along its periphery and elevated full thickness distally to proximally. After it is clear that the flap is going to reach the recipient site without tension, a decision is made to transfer it in two or three stages. The forehead is multilaminar and consists of skin, subcutaneous tissue, the frontalis muscle, and a thin areolar layer. It is thicker than normal nasal skin, and the excessive bulk must be excised during reconstruction. Initial thinning of the distal 2.5 cm of the flap at the time of transfer should be limited to nonsmokers with small defects that do not require complicated support or lining replacement (Fig. 3). In complex repairs, especially in smokers (and when a skin graft or a folded forehead flap is planned for lining), the forehead flap should be left full thickness with all its vascular layers. Primary cartilage grafts are placed if vascularized intranasal lining is present or has been restored. The use of cartilage grafts is contraindicated at the first stage if skin grafts or a folded forehead flap is used for lining. The forehead donor site is closed in layers after wide undermining in the subgaleal plane.

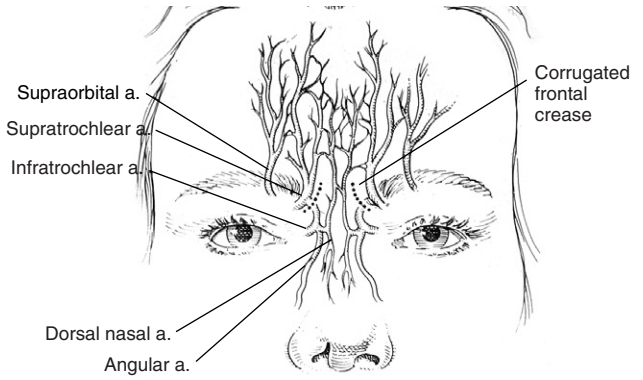

Figure 2 • The vascular supply of the paramedian forehead flap is provided by vertical vessels traveling from the medial aspect of the brow based on the supratrochlear and supraorbital vessels. (From Burget G, Menick F: Aesthetic Reconstruction of the Nose. St Louis, Mosby–Year Book, 1994.)

At 3 weeks, if the distal aspect of the flap had been thinned initially, the pedicle is divided and its proximal component partially reelevated from the inset to allow subcutaneous debulking and completion of the inset. The proximal pedicle is trimmed and inset as a small inverted "V" in the glabella.

If the flap is transferred as a full-thickness flap, it is well healed in the recipient bed and is, in effect, physiologically delayed. Forehead skin with 3 to 4 mm of subcutaneous fat can be elevated completely off the nasal inset except for a few millimeters along the columella. The underlying exposed subcutaneous fat, frontalis muscle, and scar are excised to create a subcutaneous framework. Previously placed primary cartilage grafts are remodeled if required. Delayed cartilage grafts can also be placed if the

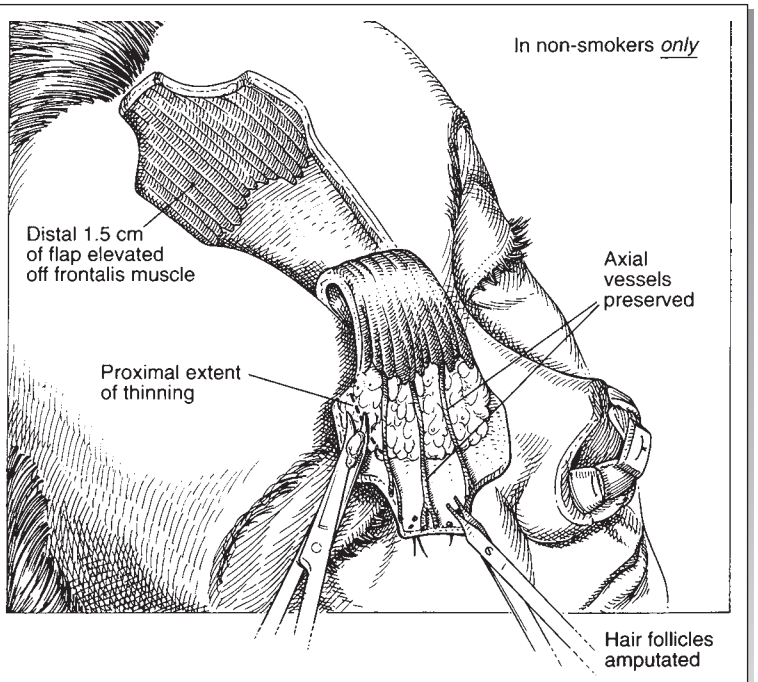

Figure 3 • Because a forehead flap is thicker than nasal skin, its distal 2 cm can be thinned by excising the frontalis muscle before initial inset. It is, however, sometimes preferable to transfer the flap as a full-thickness flap and thin it during an intermediate operation. (From Burget G, Menick F: Aesthetic Reconstruction of the Nose. St Louis, Mosby–Year Book, 1994.)

skin graft or folded forehead flap technique has been used for lining. The sculpted forehead skin flap is replaced on the recipient bed with quilting sutures to close the dead space. At a third stage (3 weeks later or 6 weeks after initial transfer), the pedicle is transected and the inset completed.

Nasal Lining

Missing nasal lining has traditionally been replaced by simply folding the distal end of the forehead flap on itself. However, this technique results in a thick, shapeless rim and precludes accurate placement of a delicate, shaped cartilage support. Often, local adjacent skin is turned over on the periphery of the defect for lining. However, turnover flaps are thick, stiff, and if longer than 1.5 cm, unreliable. Conversely, a forehead flap can be prefabricated by placing a composite skin graft or a full-thickness skin graft and a strip of cartilage under the forehead at a preliminary operation. These techniques require a delay to allow healing to occur before initiating nasal reconstruction, and they also limit the surgeon's ability to mold the lining and support grafts into a nasal shape.

Large amounts of intranasal lining remain after most nasal injuries. Unless injured by previous trauma or nasal surgery, the septum is supplied anterosuperiorly through anterior ethmoid vessels, inferoposteriorly through septal branches of the superior labial artery, and laterally by branches of the facial and angular artery. A bipedicle flap of residual vestibular skin may be incised in the area of the intracartilaginous line and transferred inferiorly to restore the alar rim (Fig. 4). The flap is based medially on the septal branch of the superior labial artery and laterally at the alar base on branches of the angular artery.

The entire ipsilateral septal mucoperichondrium can also be elevated off the underlying septum and transposed laterally on a 1.5-cm pedicle through septal branches of the superior labial artery (see Fig. 4). The contralateral septum can be hinged laterally and based dorsally on the anterior ethmoid vessels.

In fact, the entire septum can be hinged out of the piriform aperture to lie in front of the face by moving bilateral septal mucoperichondrial flaps that sandwich septal cartilage and bone (see Fig. 4). Such intranasal lining flaps require an intact blood supply. They may be poorly tolerated in elderly or medically ill patients because they are associated with intranasal crusting and bleeding. In such cases, it must be remembered that a full-thickness skin graft vascularized on the undersurface of a forehead flap, or conversely, a folded forehead flap can be used.

Small to moderate unilateral lining defects can be repaired with intranasal lining flaps, a skin graft under a forehead flap, or a modified folded forehead flap. If intranasal lining flaps are chosen and resid-

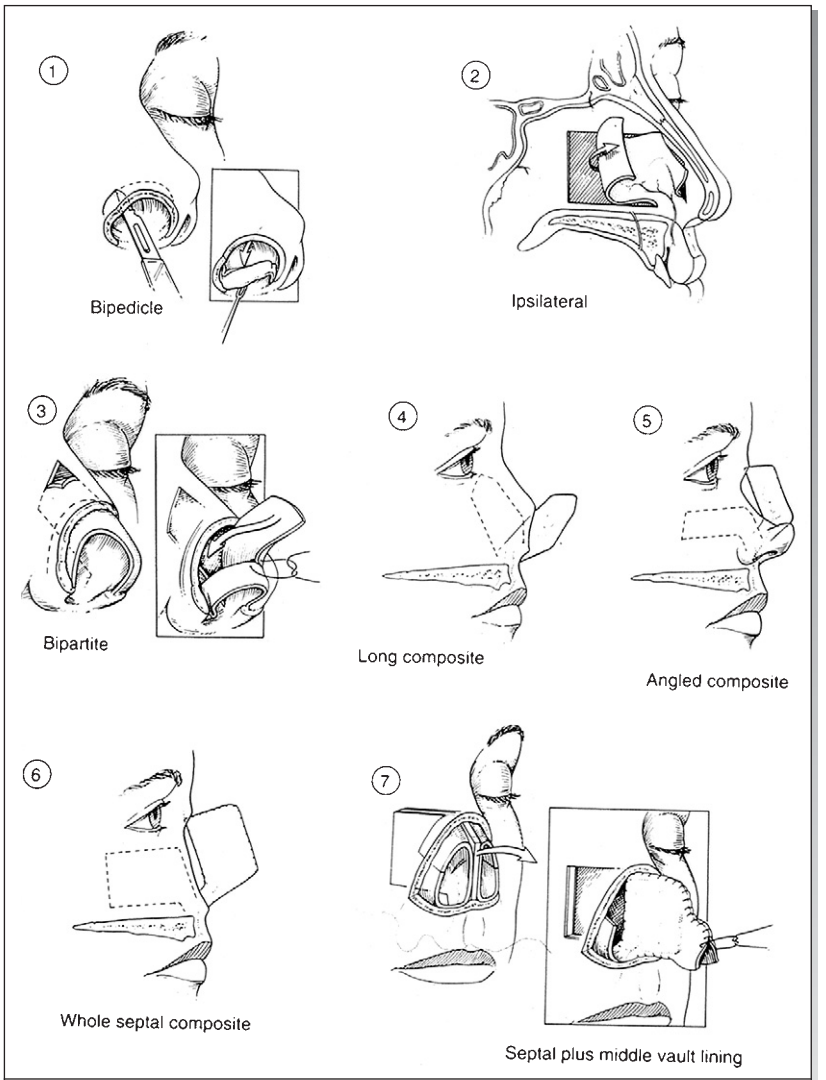

Figure 4 • Residual intranasal lining can be transferred as flaps to restore missing lining in most nasal defects. (From Burget G, Menick F: Aesthetic Reconstruction of the Nose. St Louis, Mosby–Year Book, 1994.)

ual vestibular skin lies above the defect, this skin can be incised in the area of the inner cartilaginous line and transferred inferiorly as a bipedicle flap based medially in the nasal septum and laterally in the alar base to lie at the alar rim. This technique moves the defect from the alar margin to the junction of the ala and nasal sidewall. The superior defect can be repaired with the choice of an ipsilateral septal mucoperichondrial flap, a contralateral mucoperichondrial flap passed through a slit in the intact ipsilateral septal mucoperichondrium after harvesting a cartilage graft, or a full-thickness skin graft. If residual vestibular skin is not available, the ipsilateral septal mucoperichondrial flap can be transposed laterally to line the alar margin. In addition, a dorsally based contralateral mucoperichondrial flap moved through the nasal fistula created by excision of septal cartilage and bone can be transferred to line the superior aspect of the defect.

To avoid intranasal manipulation, a small to moderate unilateral lining defect can also be repaired with a full-thickness postauricular skin graft (or by extension of the forehead flap) that is folded on itself to supply both cover and lining. An exact pattern of the lining defect is made and used to harvest a full-thickness skin graft or design a lining extension on the forehead flap. The full-thickness skin graft is sutured to the lining defect and approximated to the overlying full-thickness forehead flap with temporary absorbable quilting sutures and an intranasal sponge bolus dressing. In the folded technique, the distal end of the forehead flap is sutured to the lining defect and simply folded on itself to create both lining and cover. Both techniques preclude the placement of primary cartilage grafts at this procedure. Three weeks later, at the intermediate operation, the skin graft and the distal extension of the forehead flap are vascularized. The

forehead flap with 2 to 3 mm of subcutaneous fat can be elevated from the underlying skin graft or incised completely along the alar margin to separate the proximal cover aspect of the forehead flap from its distal folded lining extension. Such skin grafts and folded forehead flap lining replacements remain supple and become highly vascularized. Excess subcutaneous tissue and frontalis muscle are excised to create a thin nasal lining on which delayed primary cartilage grafts can be fixed to provide shape and support.

Defects resulting from subtotal amputation are reconstructed by using a composite septal flap based on both the right and left branches of the superior labial artery. The septum is incised deep into the piriform aperture and backcut to leave a 1.5-cm pedicle in the area of the nasal spine. A strip of cartilage must be removed in this area to allow anterior rotation of the septal flap and fixation to the remnants of the upper lateral cartilage or nasal bones. This step can be performed at a preliminary operation to ensure vascularity. Two to 3 weeks later, after the septal flap is healed, the septal lining is separated in the midline and each mucoperichondrium hinged laterally to provide lining of the septal vaults and most of the tip. A small nasolabial flap or a hinged-over remnant of the ala is needed to provide extra lining for the alar base. Primary cartilage grafts can be placed during forehead flap transfer.

Nasal Support

If missing, the supportive framework of bone and cartilage must be replaced to support the soft tissues, create a three-dimensional nasal shape, maintain patent airways, and brace the reconstruction against scar contraction. Cartilage normally does not exist in the ala. If the fibrofatty tissue of the ala is removed, a support graft must be placed. Depending on the defect, a dorsal graft buttress, a sidewall brace, an alar margin batten, and anatomic tip cartilage grafts can be positioned (Fig. 5). They can be placed primarily if the intranasal lining is intact or has been replaced. They can also be placed during the intermediate operation after a skin graft or folded forehead flap has become incorporated into the residual nasal lining. Septal or ear cartilage and rib (bone and cartilage) grafts are most often used, depending on their availability and the reconstructive requirements. Delicate cartilage grafts with a nasal shape are sutured to the nasal lining and to each other. A dorsal bone graft is often fixed with screws or a transnasal wire to the residual nasal bones if a cantilever is needed.

During the intermediate operation, the rigid skeletal framework is exposed after elevating the "thin" forehead flap to permit direct surgical sculpting of the subcutaneous architecture.

Postoperative Care

Except for a forehead flap, most nasal repairs are performed on an outpatient basis under local anesthesia and intravenous sedation. The intranasal bolus dressing, in the case of a skin graft, is removed at 48 hours.

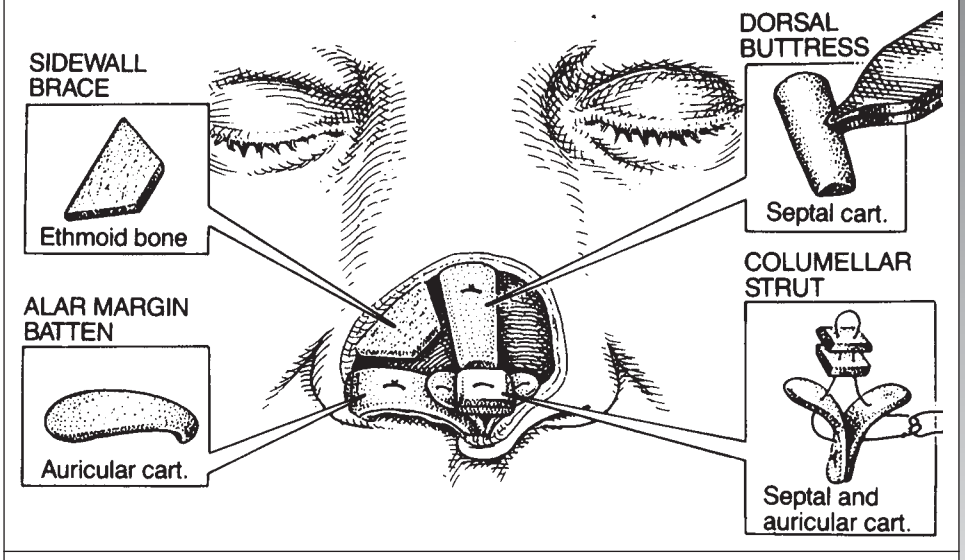

Figure 5 • Primary or delayed grafts of cartilage or bone must be placed before the completion of reconstruction to shape and support the soft tissue against gravity and scar contracture. Support must be present or must be restored to all nasal subunits. (From Burget G, Menick F: Nasal support and lining: The marriage of beauty and blood supply. Plast Reconstr Surg 84:189, 1989.)

Complications

Wound infection is rare, but the surgeon should be alert to the possibility of loss of the internal lining as its cause. If it does occur, primarily placed cartilage grafts are removed, the necrotic lining is excised, and the raw surface of the full-thickness forehead flap is skin grafted. Later, after the skin graft lining is vascularized, the forehead flap is elevated and the delayed primary support grafts replaced.

Necrosis of the covering skin graft or flap is also unusual. A small superficial defect is allowed to heal secondarily. However, in a major nasal reconstruction with underlying cartilage grafts and lining repair, the necrotic flap tissue is excised and the resultant defect resurfaced with a second flap from the cheek or forehead.

Pearls and Pitfalls

- Prepare. Analyze the defect both anatomically and visually. List each available lining, support, and cover option. Think the procedure through mentally and visualize the end result.
- Assume that most patients wish to look normal. Unless precluded by significant medical illness, the patient's appearance and your reputation are better served by a first-class reconstruction.
- If you are unfamiliar or unsure, refer the patient to a more experienced surgeon.
- Be bold. Nasolabial and forehead flaps should not be used as a last resort, but as the first choice for many nasal defects.
- Add the three-stage forehead flap, the skin graft–lining technique, and the folded forehead flap to your armamentarium.
- Beware of tension. Although the flaps are well vascularized, they must transpose easily into the defect without tension.

SUGGESTED READINGS

Burget GC, Menick FJ: Subunit principle in nasal reconstruction. Plast Reconstr Surg 76:239, 1985.

Burget GC, Menick FJ: Nasal reconstruction: Seeking a fourth dimension. Plast Reconstr Surg 78:583, 1986.

Burget GC, Menick FJ: Nasal support and lining: The marriage of beauty and blood supply. Plast Reconstr Surg 84:189, 1989.

Burget GC, Menick FJ: Aesthetic Reconstruction of the Nose. St Louis, Mosby–Year Book, 1994.

Menick FJ: Artistry in facial surgery: Aesthetic perceptions and the subunit principle. Clin Plast Surg 14:723-735, 1987.

Menick FJ: Aesthetic refinements in use of forehead for nasal reconstruction: The paramedian forehead flap. Clin Plast Surg 17:607-622, 1990.

Menick FJ: A ten year experience in nasal reconstruction with the three stage forehead flap. Plast Reconstr Surg 109:1839, 2002.

The Cheek, Malar Area, and Maxilla

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ERIC SANTAMARIA

The midfacial skeleton and the overlying soft tissues are responsible for facial contour. The maxilla is closely related to the globe, nasal airway, and oral cavity (Fig. 1), and it provides support to the muscles of facial expression, mastication, speech, and deglutition. Along with the cheek skin and mucosa, it provides for oral competence and acts as a reservoir for liquids and solids.

Midfacial reconstruction must replace the tissues that are absent. Defects of the cheek that involve only the skin or mucosa may be relatively simple to reconstruct. However, if there are larger defects or if multiple layers of the cheek or adjacent structures such as the eyelid, nose, lips, mouth commissure, and mandible or maxilla are involved, reconstructive procedures become correspondingly more complex.

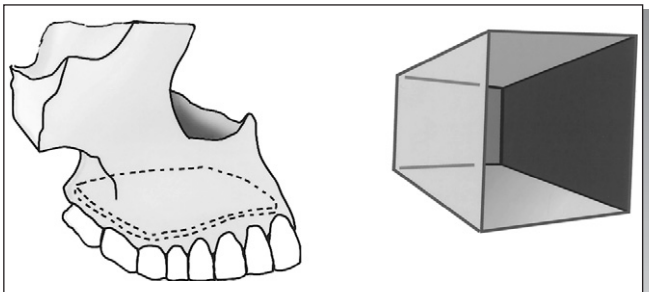

Figure 1 • The maxilla resembles a hexahedron that contains the maxillary antrum. The roof of the maxilla is the floor of the orbit. The floor of the maxilla is the hard palate. The medial wall is part of the nasal cavity and, together with the anterior, posterior, and lateral walls, forms the vertical buttresses.

Treatment of Cheek Defects

The cheek is composed of an external skin layer, muscles of facial expression, fat, and oral mucosa. Most defects of cheek skin may be closed primarily. The best results are usually achieved by hiding the final scar in natural skin lines. Some small and most medium-sized defects require reconstruction with skin grafts or local/regional flaps. For these patients, facial contour, skin color, texture, and appearance are of primary concern. In addition, adherence to the principle of cheek subunits will optimize the aesthetic results.

Split- and full-thickness skin grafts usually undergo contraction and result in a patch-like effect; therefore, they are suitable only for replacement of the lower eyelid or the preauricular/temporal region in the area of cheek flap donor sites. Local flaps provide skin that is similar to that of the face in terms of color and texture, and they are used for medium-sized cheek defects. If a rotation or advancement flap is selected, it should be oriented properly to place the donor scar parallel to the relaxed skin tension lines. Such orientation both minimizes the scar and avoids traction on the lower eyelid or nose. Mobilization of cervicofacial flaps and myocutaneous flaps from the chest is usually reserved for resurfacing larger external defects of the cheek, for intraoral lining, or for full-thickness defects. The disadvantages of regional flaps include their bulk, the need for extensive dissection, a vascular supply that is not always reliable, and the necessity to skin graft the donor site. Furthermore, because the arc of rotation is limited by the vascular pedicle attachments, they are occasionally not able to reach the midface. Although it is possible to use two regional flaps to separately reconstruct the intraoral lining and external skin, the drawbacks of such an approach include the requirement for two or more operations, as well as the creation of a bulky cheek that has poor functional and aesthetic results.

Microvascular free flaps are generally indicated for reconstruction of large defects involving the external skin, intraoral lining, or both. They are essential if resections have also included the maxilla or mandible.

Large external defects require a significant amount of thin pliable skin with minimal underlying soft tissue. The radial forearm fasciocutaneous flap is ideal for these situations because it provides an adequate quantity of skin with minimal bulk. Depending on the amount of soft tissue bulk required, the lateral arm flap, anterolateral thigh flap, and scapular flap are satisfactory options.

Intraoral lining defects that span the maxillary and mandibular sulci require a microvascular free flap. A radial forearm free flap provides thin pliable skin to resurface this area. The flap may also be neurotized to enhance recovery of sensation in this tissue by anastomosing the lateral antebrachial cutaneous nerve to a sensory nerve in the recipient area.

Full-thickness defects of the cheek require at least two skin islands to resurface the inner lining and provide external coverage. The folded radial forearm fasciocutaneous flap is the first choice for small through-and-through defects. Larger resections of the cheek associated with segmental mandibulectomy, as well as partial maxillectomy/orbitectomy, are best reconstructed by using the rectus abdominis free flap with multiple skin islands. If the commissure of the mouth is resected, it should be reconstructed with a local switch procedure from the intact opposite lip and not with a portion of the free flap. The free flap should be reserved for reconstruction of the intraoral and external skin defects.

Treatment of Midface/Maxillary Defects

Defects after resection of the maxilla and adjacent soft tissues may be classified as follows:

Type I defects *(limited maxillectomy)* include resection of one or two walls of the maxilla, excluding the palate. In most patients, the anterior wall is partially removed with either the medial wall or the orbital floor (Fig. 2).

Type II defects *(subtotal maxillectomy)* include resection of the maxillary arch, palate, and anterior and lateral walls (lower five walls), with preservation of the orbital floor (Fig. 3).

Type III defects *(total maxillectomy)* include resection of all six walls of the maxilla. This type of defect is subdivided into *type IIIa*, in which the orbital contents are preserved (Fig. 4), and *type IIIb*, in which the orbital contents are exenterated (Fig. 5).

Type IV defects *(orbitomaxillectomy)* include resection of the orbital contents and the upper five walls of the maxilla, with preservation of the palate (Fig. 6).

This classification assists in reconstruction of maxillectomy and midfacial defects by emphasizing the relationship between volume and surface area requirements. A logical sequence is to address the bony defect first and then thoroughly assess the associated soft tissue, skin, palate, and cheek lining deficits.

Limited maxillectomy defects (type I) have small volume and large surface area requirements. If

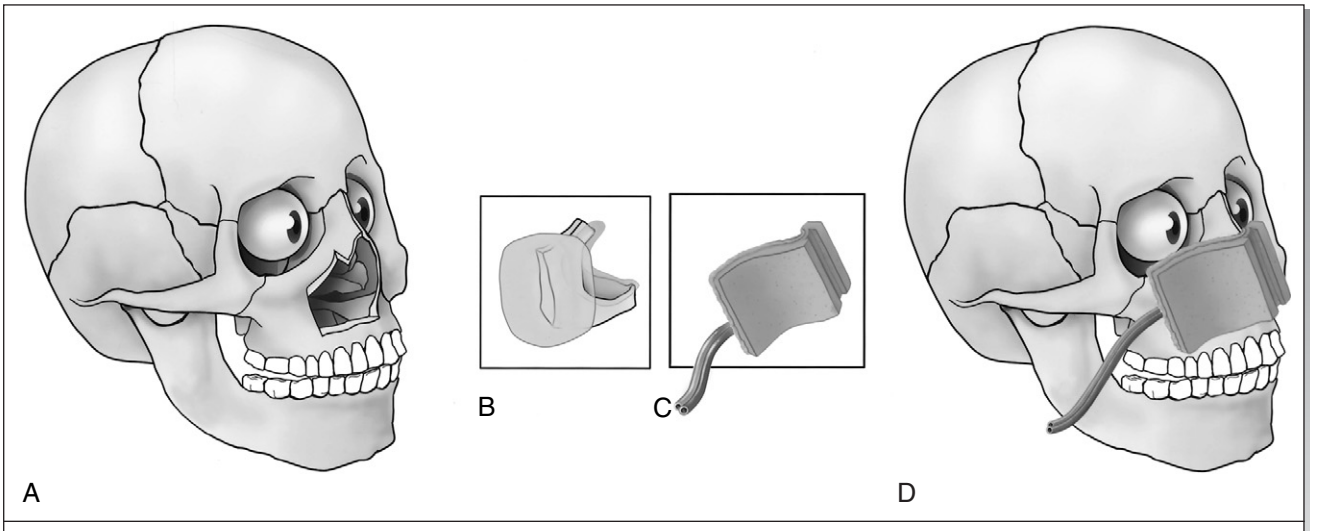

Figure 2 • Type I defect (limited maxillectomy). Resection includes the anterior and medial walls of the maxilla in combination with skin and soft tissue of the cheek (**A** and **B**). Such resection creates a large surface area/low volume defect. The radial forearm fasciocutaneous flap (**C**) provides multiple large skin surface areas with minimal volume to resurface the anterior aspect of the cheek and the medial nasal lining (**D**).

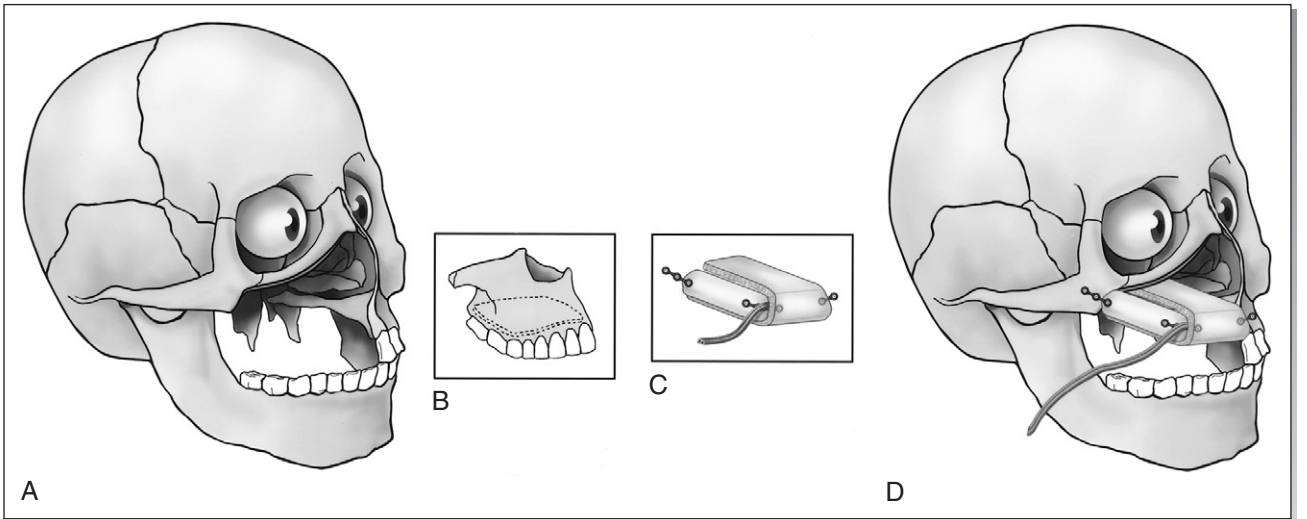

Figure 3 • Type II defect (subtotal maxillectomy). Resection includes the lower five walls of the maxilla, including the palate and nasal lining, but sparing the orbital floor or roof of the maxilla (**A** and **B**). Such resection creates a large surface area/medium volume defect. The radial forearm osteocutaneous "sandwiched" flap provides a large skin surface with vascularized bone and moderate volume (**C**). The strut of vascularized bone is used to reconstruct the anterior maxillary arch, and the two skin islands are sandwiched around the bone to replace palatal and nasal lining (**D**).

critical segments of bone are missing (such as the orbital rim or anterior floor of the orbit), nonvascularized bone grafts provide the needed support. The radial forearm flap provides satisfactory external skin coverage of the cheek and minimal bulk with the availability of multiple skin islands that can be deepithelialized to improve contour, wrap around bone grafts, or supply nasal lining (see Fig. 2).

Subtotal maxillectomy (type II) defects have medium volume and large surface area requirements. For reconstruction of such defects, the osteocutaneous radial forearm flap provides an ample skin segment that is folded over to line the mucosal surface of the palate, as well as the nasal floor. The vascularized bony strut provides support to the upper lip, helps to maintain anterior projection, and also provides adequate bone stock for future dental osteointegration (see Fig. 3). The folded osteocutaneous forearm free flap is an excellent solution for soft tissue maxillary defects or bilateral subtotal maxillectomies, especially in patients who have intact external lip structures.

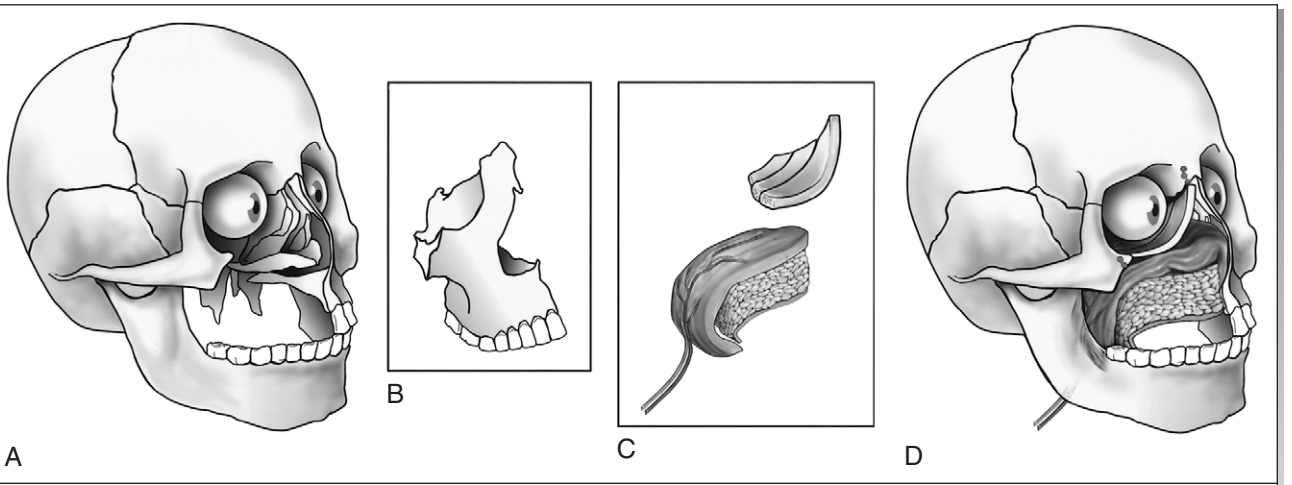

Figure 4 • Type IIIa defect (total maxillectomy with sparing of the orbital contents). Resection of bone in combination with the hard palate and nasal lining creates a medium surface area/medium volume defect (**A** and **B**). A nonvascularized bone graft from rib or cranium is rigidly fixed to reconstruct the floor of orbit and is covered with a single–skin island rectus abdominis myocutaneous flap (**C**). The skin island of the free flap is used to close the palate and provide soft tissue to fill in the midfacial defect and muscle to cover the bone graft (**D**).

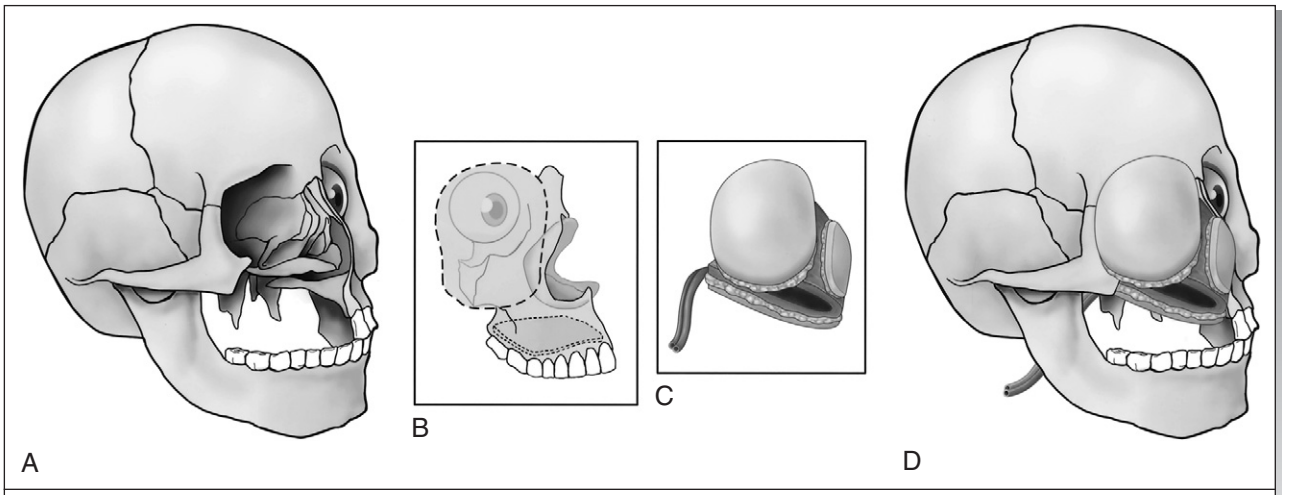

Figure 5 • Type IIIb defect (total maxillectomy with resection of the orbital contents). Resection includes the entire maxilla and palate, in conjunction with the external eyelid, cheek skin, and orbital contents. Such resection creates a large surface area/large volume defect (**A** and **B**). A three–skin island rectus abdominis myocutaneous flap (**C**) is used to resurface the external skin, palatal defect, and nasal lining. Muscle and soft tissue are used to fill in the soft tissue deficit (**D**).

Total maxillectomy defects with sparing of the orbital contents (type IIIa) have medium to large requirements for both volume and surface area. Functional necessities in these defects include support of the orbital contents and reconstruction of the palate. Unless the orbital floor is adequately reconstructed, the orbital contents sink into the cheek, thereby resulting in dystopia, diplopia, and an essentially nonfunctional eye. Reconstruction of the orbital floor can be effectively addressed with nonvascularized bone grafts. The bone graft must be sandwiched between a healthy flap (either a rectus abdominis or temporalis flap) and the orbital contents above. In addition to providing coverage of the bone grafts, the multiple skin islands of the rectus abdominis free flap may be used for palatal closure or external skin/nasal lining (or for both) as needed (see Fig. 4).

Total maxillectomy defects with orbital exenteration (type IIIb) are extensive and have large volume and large surface area requirements (see Fig. 5). The palate needs to be closed, the nasal lining (medial wall of maxilla) must be restored to maintain a patent airway, and the external defect is often extensive and involves the eyelids, cheek, and occasionally the lip. In addition, the anterior cranial

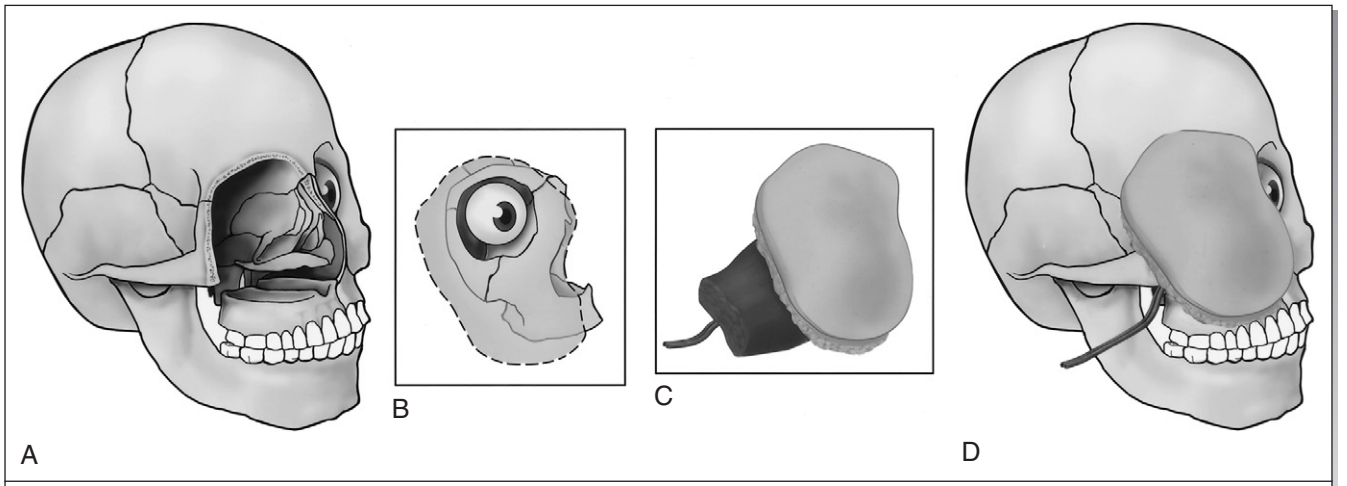

Figure 6 • Type IV defect (orbitomaxillectomy). Bony resection includes the upper five walls of the maxilla along with the orbital contents, eyelids, and cheek skin (but sparing the palate). Such resection creates a large surface area/large volume defect (**A** and **B**). A single–skin island rectus abdominis myocutaneous flap (**C**) is used to resurface the external defect with skin and muscle, and subcutaneous fat is used to fill in the soft tissue deficit (**D**).

base in the area of the sphenoid is often exposed, and coverage of the brain becomes critical. If the external skin of the cheek is intact, a free rectus flap with a skin island is used to close the palate and provide a straightforward solution (see Fig. 5). If the flap is not bulky, a second skin island can be used to restore the lateral nasal wall. A third skin island can provide closure of the external skin deficit (see Fig. 5).

Orbitomaxillectomy defects (type IV) are generally large volume/large surface area defects. Because the palate is usually intact, the reconstructive objectives consist primarily of soft tissue fill and external skin resurfacing (see Fig. 6). The rectus abdominis flap effectively provides all of these features, and the length of the vascular pedicle can be extended up to 18 to 20 cm to reach the donor vessels in the neck without the need for interpositional vein grafts.

Pearls and Pitfalls

- For cheek defects, closure with local tissue (i.e., local flaps or cervicofacial flaps) provides the best aesthetic results. Distant flaps, including pedicle and free flaps, yield a less ideal color and texture match.
- Suspension of flaps by using incorporated fascia, tendon, or bone prevents long-term ptosis and gives more functional results.

SUGGESTED READINGS

Ariyan S: The pectoralis major myocutaneous flap, a versatile flap for reconstruction in the head and neck. Plast Reconstr Surg 63:73, 1979.

Cordeiro PG, Santamaría E: Free flap reconstruction of the cheek. Op Tech Plast Reconstr Surg 6:265, 1999.

Cordeiro PG, Santamaria E: A classification system and algorithm for reconstruction of maxillectomy and midfacial defects. Plast Reconstr Surg 105:2331, 2000.

Cordeiro PG, Bacilious N, Schantz S, Spiro R: The radial forearm osteocutaneous "sandwich" free flap for reconstruction of the bilateral subtotal maxillectomy defect. Ann Plast Surg 40:397, 1998.

Cordeiro PC, Santamaria E, Kraus D, et al: Reconstruction of total maxillectomy defects with preservation of the orbital contents. Plast Reconstr Surg 102:1874, 1998.

Freedman AM, Hidalgo DA: Full-thickness cheek and lip reconstruction with the radial forearm free flap. Ann Plast Surg 25:287-294, 1990.

Juri J, Juri C: Advancement and rotation of a large cervicofacial flap for cheek repair. Plast Reconstr Surg 64:692, 1979.

Kroll SS, Reece GP, Robb G, et al: Deep-plane cervicofacial rotation advancement flap for reconstruction of large cheek defects. Plast Reconstr Surg 94:88, 1994.

Menick FJ: Facial reconstruction in aesthetic units: Reconstructive philosophy and subunit principles. In Kroll SS (ed): Reconstructive Plastic Surgery for Cancer. St Louis, CV Mosby, 1996, pp 166-183.

Niranjan NS, Watson DP: Reconstruction of the cheek using a "suspended" radial forearm free flap. Br J Plast Surg 43:365-366, 1990.

Savant DN, Patel SG, Deshmukh SP, et al: Folded free radial forearm flap for reconstruction of full-thickness defects of the cheek. Head Neck 17:293-296, 1995.

Strasnick L, Calcaterra TC: Reconstruction of full-thickness cheek defects with combined cervicopectoral and pectoralis major myocutaneous flaps. Laryngoscope 99:757-760, 1989.

Lip and Oral Commissure Reconstruction

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BARRY ZIDE

This chapter reviews the tenets of reconstruction of the lips and provides a classification system that can assist in rendering appropriate care. The reconstructive choices represent those that are reliable and are most likely to fulfill the reconstructive and aesthetic goals specific to each defect.

Etiopathogenesis

Most malignant neoplasms of the upper lip are basal cell carcinomas, whereas malignancies of the lower lip tend to be squamous cell carcinomas due to chronic exposure to the sun. The nature of the tumor plays a role in overall therapy. Basal cell carcinomas of the upper lip, unless very large, do not metastasize and thus may not require as large an excisional margin (minimum of 5 mm of normal tissue) as do lower lip tumors, which may metastasize, especially when the commissure is involved.

Reconstruction of the lips and commissure is also indicated after thermal and electrical burns.

Pathologic Anatomy

Knowledge of the topographic landmarks (Fig. 1) and the underlying muscles, vessels, and nerves of the lips and cheeks is essential when planning any reconstructive procedure.

The lips are approximated through the action of the orbicularis oris muscles. The deep elements of the orbicularis constrict the lips to the level of the alveolar arch, whereas more superficial elements (incorporating decussating fibers of the buccinator muscle) purse and protrude the lips. The primary upper lip elevator is the levator labii superioris

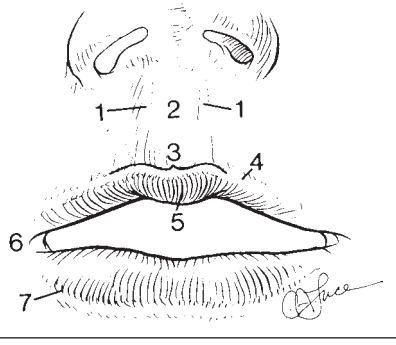

Figure 1 • Surgical topographic landmarks of the lips. 1, philtral columns; 2, philtral dimple; 3, Cupid's bow; 4, white roll; 5, tubercle; 6, commissure; 7, vermilion. (From McCarthy JG [ed]: Plastic Surgery, vol 3. Philadelphia, WB Saunders, 1990, p 2009.)

muscle. The lateral aspect of the lip is elevated by the zygomaticus major and the levator angularis oris muscles, whereas the risorius and buccinator are accessory muscles. The primary lip depressor is the depressor labii inferioris with a secondary effect by the platysma and a somewhat lateral effect provided by the depressor angularis oris. The elevators of the lower lip, as well as the muscle that maintains central lower lip position, are the paired mentalis muscles, which insert into and give bulk to the central chin pad. It should be noted that the sphincteric function of the orbicularis oris muscle is independent of the other accessory muscles of facial expression.

The primary motor nerves of the lips are the buccal and marginal mandibular branches of the facial nerve, and sensory innervation is provided primarily by the infraorbital nerve and the mental rami of the trigeminal nerve.

The arterial supply is via the superior and inferior labial arteries off the external maxillary (facial)

TABLE 1 Treatment of Vermilion and Commissure Defects

Vermilion defects	Mucosal advancement Vermilion flap Facial artery musculomucosal flap Tongue flap Medical tattoo
Commissure defects	Vermilion flap Tongue flap Orbicularis oris muscle flap

artery, which is derived from the external carotid artery. Venous drainage is provided by the anterior facial vein with lymphatic drainage via the submandibular and submental basins.

Goals of Reconstruction

The aims of lip reconstruction are to achieve

- Skin coverage
- Oral lining
- Vermilion
- An adequate stomal diameter
- Sensation
- A competent oral sphincter

Choices for lip reconstruction, in descending order of preference, are the remaining lip, the opposite lip, the adjacent cheek, and distant tissue.

Vermilion

Vermilion defects can be treated with a variety of techniques (Table 1). Small, isolated vermilion defects are best treated with mucosal advancement (lip shave), which can often be accomplished simply with a V-Y advancement. Disadvantages include loss of lip pout and a resulting inward pull at the mucosal-squamous junction. Alternatively, the mucosa can be advanced as a bipedicled flap and the donor site allowed to heal secondarily, a technique that may pull in the lip but usually to a lesser degree. The entire vermilion can also be elevated as a unipedicled flap based on the labial artery and used to close isolated vermilion defects. Bilateral flaps can be used to close central defects.

Pedicled vermilion flaps can also be used as a staged procedure to repair defects of the opposite lip. If adequate vermilion is not available, a mucosal flap is used, but the color match of the mucosa for the neovermilion is less than optimal. A facial artery musculomucosal flap (FAMM) based on the facial artery can also be used and can include mucosa, submucosa, and the buccinator muscle, as well as the facial artery and its venous plexus. It has superior color match with minimal donor site morbidity, but the mucosa tends to be too smooth in texture.

If labial or buccal mucosa from either lip is not available, a pedicled tongue flap is the next source of donor tissue, although its use results in color and texture mismatch. The flap may be based posteriorly or, less commonly, anteriorly. The flap is usually divided after 2 weeks. Other options include full-thickness grafts of palatal mucosa. These grafts are for minor contour and size defects and must be placed well posterior to the wet-dry line because of color mismatch.

For total lip defects, including vermilion defects, medical tattooing of a microvascular free flap is a technique of last resort.

Lower Lip Reconstruction

The lower lip has a less obvious central structure than the philtral columns of the upper lip (although it is usually concave below the vermilion), and therefore it may sustain greater loss without the distortion seen in the upper lip. This anatomic finding permits harvesting of larger amounts of tissue for upper lip reconstruction.

Lip defects must be quantified and systematically approached. Lower lip defects are usually divided into thirds, with total lip reconstruction being a separate subcategory (Table 2).

Defects Less than a Third of the Lower Lip

These defects are usually amenable to primary closure. It is important to realign the essential lip elements to reestablish balance and function. When involved, the muscle should be reapproximated. Surgical excision should follow a straight wedge, an M-plasty base, or a wedge that extends along the labiomental fold laterally. Wedge excision that includes the muscle is sometimes not necessary if some muscle can be salvaged.

Defects between a Third and Two Thirds of the Lower Lip

Larger defects can often be treated with a cross lip flap. As much as 2 cm of the lateral portion of the upper lip may be harvested with little donor site morbidity. With this technique, the philtral columns shift slightly to the donor area. The flap is traditionally designed half as wide as the defect and is based on the labial artery. If the artery is damaged, the flap can be transferred on a 1-cm mucosal

TABLE 2 Treatment of Upper and Lower Lip Defects

	LESS THAN ONE THIRD	BETWEEN ONE THIRD AND TWO THIRDS	GREATER THAN TWO THIRDS	TOTAL LIP RECONSTRUCTION
Lower lip defects	Primary closure	Primary closure Abbé's flap Estlander flap Step reconstruction	Karapandzic's flap Webster's Bernard-Burow cheiloplasty	Nasolabial and mucosal flaps Cervicofacial flap Microvascular free flap
Upper lip defects	Primary closure	Abbé's flap Reverse Estlander flap Cheek advancement, transposition on composite (Yotsuyangi's) flap	Microvascular free flap	

pedicle. Cross lip flaps can be safely divided at 9 to 12 days.

Defects of the commissure (see Table 1) or lateral part of the lower lip are well suited for reconstruction with an Estlander flap. The lateral portion of the upper lip is rotated around the commissure to repair defects of the lower lip (Fig. 2). The technique may be used in conjunction with a lower lip medial advancement for a defect measuring two thirds of the lower lip. However, the commissure is blunted.

A step method of reconstruction can be used for lower lip defects. Each horizontal step measures half the defect so that at least two to three steps are required to close the defect (Fig. 3). When combined with other methods, it can be used for total lip reconstruction, but the incisions are inevitably noticeable.

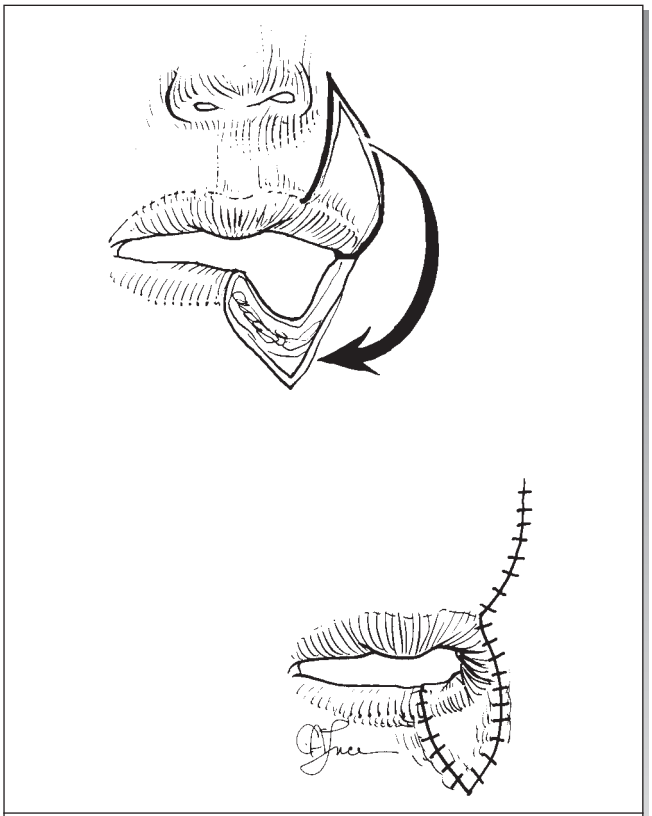

Figure 2 • The Estlander flap is based on the lateral upper lip elements around the commissure to repair defects of the lower lateral aspect of the lip. It may be used in addition to a lower lip medial advancement to close larger defects. (From McCarthy JG [ed]: Plastic Surgery, vol 3. Philadelphia, WB Saunders, 1990, p 2022.)

Defects Greater than Two Thirds of the Lower Lip

The Karapandzic flap is a rotation flap of the upper lip and lateral aspect of the cheek/lip that maintains

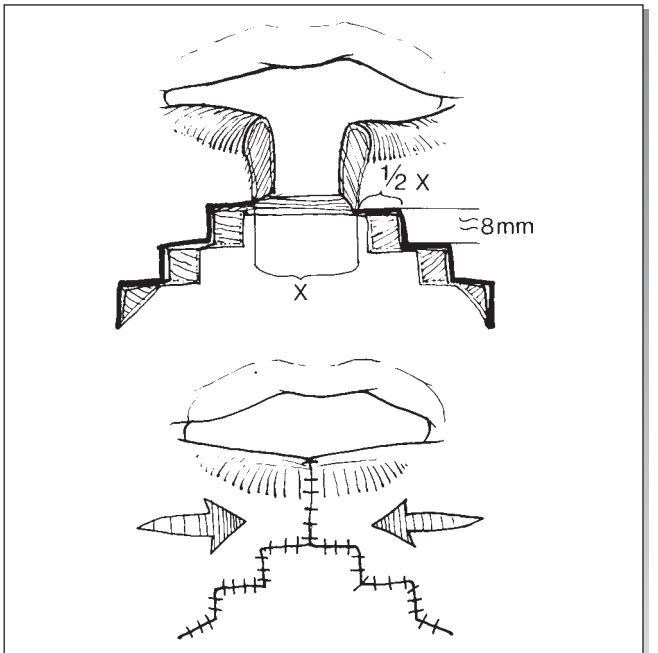

Figure 3 • The step method of reconstruction: each horizontal step measures half the defect; at least two to three steps are required to close the defect. (From McCarthy JG [ed]: Plastic Surgery, vol 3. Philadelphia, WB Saunders, 1990, p 2018.)

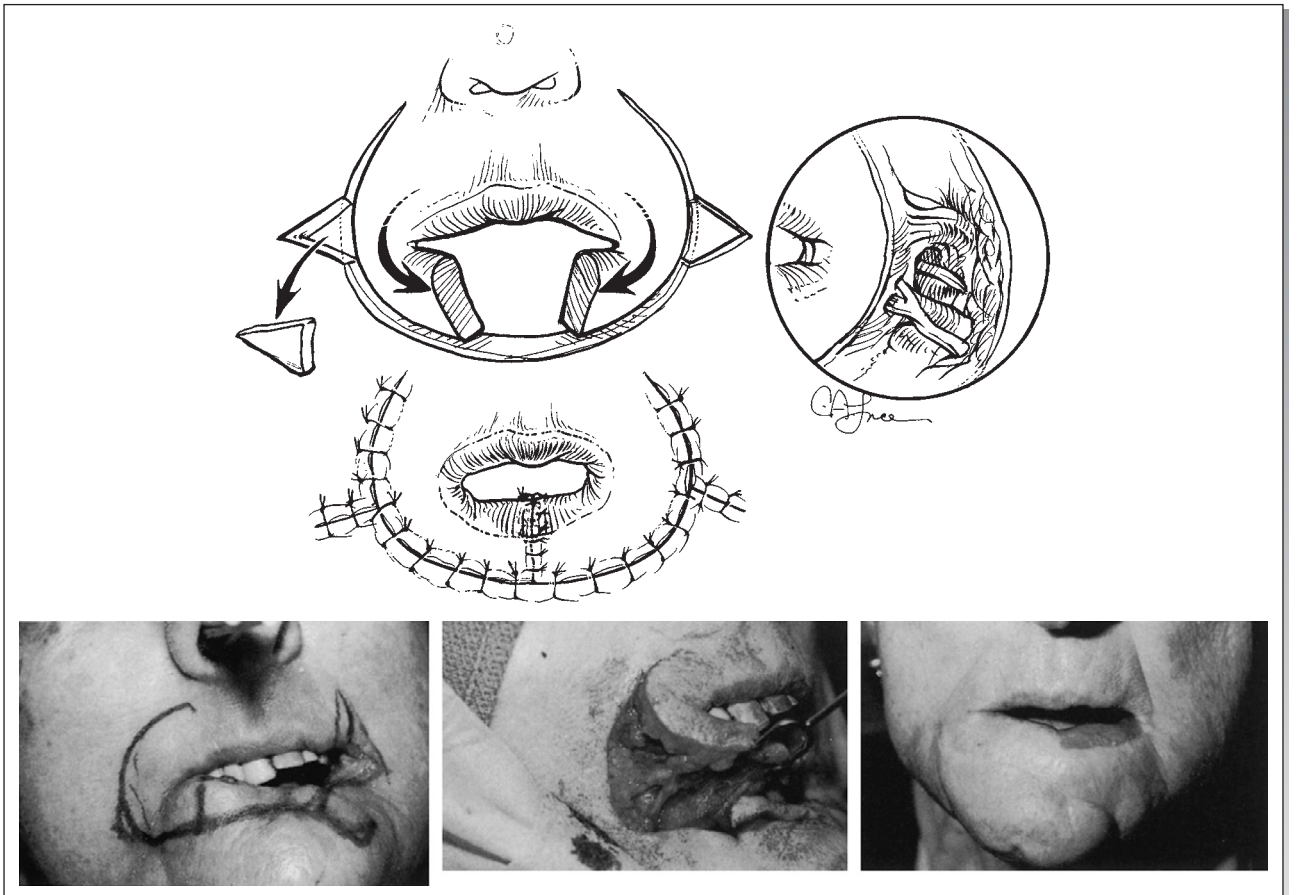

Figure 4 • The Karapandzic flap maintains a neurovascular pedicle to the two rotation flaps to preserve sphincteric function. This method works best with central defects involving less than the total lower lip (80%) and not involving the commissures. (From McCarthy JG [ed]: Plastic Surgery, vol 3. Philadelphia, WB Saunders, 1990, p 2023.)

a neurovascular pedicle to preserve sphincter function (Fig. 4). This technique is best used for central defects involving less than the total lower lip (80%) and sparing the commissure. The main disadvantage of this technique is the potential for microstomia, which requires correction by either a teeth-bone appliance or a commissuroplasty.

The Bernard-Burow cheiloplasty involves full-thickness excision of four triangles, two from the cheek at the level of the alar rim and two at the level of the oral commissure (Fig. 5). The two cheek flaps are advanced and joined at the midline. The mucosa in the labiobuccal sulcus is advanced for reconstruction of the vermilion. Important modifications of the flap include (1) excision of only skin and subcutaneous tissue to maintain innervated flaps, (2) positioning of the superior triangular excisions laterally in the nasolabial fold instead of next to the commissures, and (3) positioning of the inferior triangular incision more medially near the menton. The technique has been further modified by placing the triangular areas of excision at the upper lip vermilion.

Total Lower Lip Reconstruction

Total lower lip defects have historically been managed with local and regional flaps, with mixed results. Microsurgical tissue transfer has, however, emerged as the preferred technique. The most commonly used flap is the radial forearm free flap, a thin pliable flap that can be folded on itself to provide both lip lining and cover. It can be transferred with the palmaris longus tendon, which when suspended to the maxilla or, more commonly, the zygoma, can act as a sling to provide oral competence. Microvascular anastomoses are typically performed to the facial, lingual, or transverse cervical artery, along with the facial or external jugular vein. Secondary refinements include tattooing of the vermilion and hair transplantation to recreate a beard.

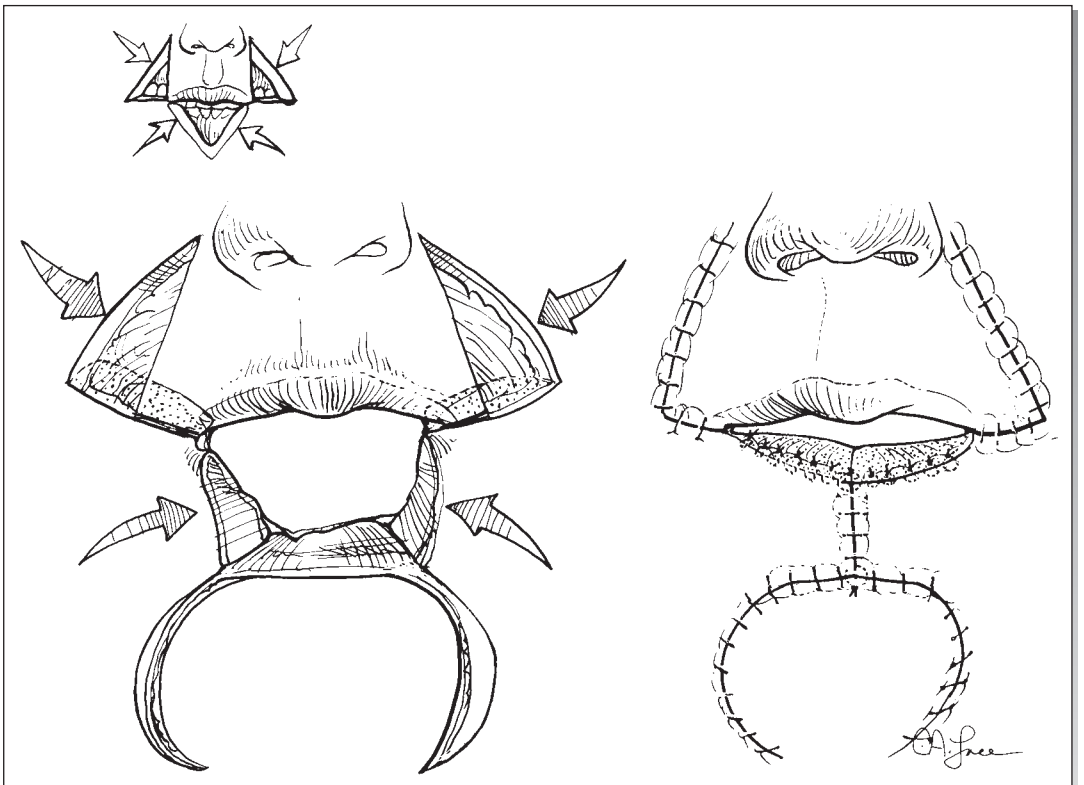

Figure 5 • The Webster modification of the Bernard-Burow cheiloplasty involves full-thickness excision of four triangles, two from the cheek at the level of the alar rim and two at the level of the oral commissure. The two cheek flaps are advanced and joined at the midline. The mucosa is mobilized to form a vermilion. (From McCarthy JG [ed]: Plastic Surgery, vol 3. Philadelphia, WB Saunders, 1990, p 2020.)

The Upper Lip

The concept of the aesthetic subunit principle can be applied to lip reconstruction by subdividing the upper lip into aesthetic units. The lateral subunits are composed of the philtral column, nostril sill, alar base, and the nasolabial crease, whereas each medial subunit consists of half of the philtrum. The most aesthetically pleasing donor tissue for upper lip reconstruction is the lower lip, that is, a cross lip flap, which can often spontaneously reinnervate.

Philtral Defects

Philtral defects are best divided into defects greater than or less than 50%. Defects less than 50% can generally be closed primarily to provide a narrow, albeit satisfactory, philtrum. If the defect is only cutaneous, a full-thickness skin graft can give excellent results.

Philtral defects greater than 50%, especially if the vermilion is involved, are well suited to an Abbé flap reconstruction. Care should be taken to harvest the Abbé flap from the center of the lower lip because it results in a more favorable scar. This lower lip scar often benefits from a small Z-plasty.

Defects Less than a Third of the Upper Lip

Direct primary closure with meticulous attention to anatomic alignment is the procedure of choice (Fig. 6). Care should be taken because the vermilion tapers laterally. Small vermilionplasties may be necessary to equalize the height of the vermilion on each side.

Defects between a Third and Two Thirds of the Upper Lip

For defects involving a third of the upper lip and central in location, an Abbé or cross lip flap remains the best option. A similarly sized defect more laterally situated can be treated with perialar crescentic excisions and cheek advancement (Webster's technique). A larger, superiorly based nasolabial flap can also be used; however, these flaps require secondary debulking.

Defects closer to two thirds of the upper lip and central in location can be treated with an Abbé flap in addition to bilateral cheek advancement. The Abbé flap should be placed centrally to mimic the philtrum. As an alternative, bilateral Yotsuyanagi flaps (1998) based on the angular arteries can reconstruct two-third defects of the upper lip. This flap

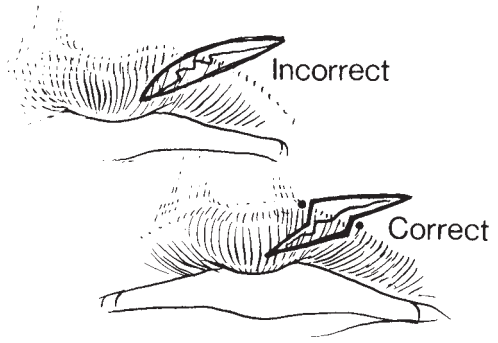

Figure 6 • The white roll should be incised at 90 degrees to prevent postoperative notching. (From McCarthy JG [ed]: Plastic Surgery, vol 3. Philadelphia, WB Saunders, 1990, p 2014.)

can be used to replace the entire lateral subunit of the upper lip. For more lateral defects of the upper lip located near the commissure, reverse Estlander flaps also provide excellent results.

Reconstruction of Hair-Bearing Tissue

In male patients, the results of lip reconstruction can be disappointing because of either absence of hair or, as in the case of cross lip flaps, hair growth in the opposite direction. For patients with absent hair growth, hair transplantation can be a satisfactory option. Even if the hair is shaved, the stubble hides scars and adds natural texture to the skin.

The entire upper lip can be resurfaced with a hair-bearing microvascular free flap that can be based on the superficial temporal vessels. It provides beard and bulk to the upper lip, albeit excessive in volume.

Pearls and Pitfalls

- Lower lip support is essential to prevent drooling. A static or dynamic sling may be created to accomplish this task.
- The upper lip hangs like a window shade and is less important for oral hygiene.
- Vermilion reconstruction must result in a satisfactory color and texture match.

SUGGESTED READINGS

Burget GC, Menick FJ: Aesthetic restoration of one-half the upper lip. Plast Reconstr Surg 78:583, 1986.

Converse JM (ed): Kazanjian and Converse's Surgical treatment of Facial Injuries, vol 2, 3rd ed. Baltimore, Williams & Wilkins, 1974, pp 949-996.

Karapandzic M: Reconstruction of lip defects by local arterial flaps. Br J Plast Surg 27:93, 1974.

Yotsuyanagi T, Yokori K, Urushidate S, Sawada Y: Functional and aesthetic reconstruction using a nasolabial orbicularis oris myocutaneous flap for large defects of the upper lip. Reconstr Surg 101:1624-1629, 1998.

Zide BM: Deformities of the lips and cheeks. In McCarthy JG (ed): Plastic Surgery, vol 3. Philadelphia, Saunders, 1990, pp 2009-2056.

The Mandible

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State-of-the-art mandible reconstruction demands a comprehensive, multidisciplinary approach to address the oncologic, structural, and aesthetic requirements of restoring mandibular function and form. Such reconstruction is facilitated by microvascular free tissue transfer. The application of distant free tissue transfer to this anatomic site enables the extirpation of large defects, the use of aggressive adjunctive therapies, and reconstruction within the hostile environment of the oral cavity in an aesthetically satisfying manner. The ability to satisfy these goals has been made possible by a move away from nonvascularized bone grafts and exotic flaps toward the use of defined, limited, and, therefore, reliable treatment options to reconstruct the vast majority of defects. Future goals include an expansion in the use of osseointegrated implants and perhaps even the use of tissue engineering as an adjunct to guide the creation of prefabricated mandibular replacements.

Etiopathogenesis

Most mandible defects result from the extirpation of an intraoral malignancy. The necessity for partial or total mandibulectomy most often results from the close anatomic proximity of the lesion to the mandible. The mandible is resected either to attain clear margins or to excise areas of involvement of the bone with cancer. The most common tumor type in Western countries is squamous cell carcinoma (SCC) of the oropharynx; other tumors can include ameloblastomas and related odontogenic tumors, osteosarcomas, and fibrous dysplasia. Additional nonmalignant causes include osteoradionecrosis, infections, traumatic injuries, and congenital defects.

A relative indication for mandibular reconstruction is osteoradionecrosis, a potential complication with an incidence as high as 40% after any modality of irradiation. Risk factors for the development of osteoradionecrosis include dental extractions in the peri-irradiation period, poor hygiene, gingival disease, and tobacco use. Osteoradionecrosis characteristically develops after an overt injury or subclinical infection, when the impaired healing capacity of the irradiated bone becomes evident. The mandible is particularly susceptible to radionecrosis because of the bone-weakening effects of tooth decay, the high intraoral bacterial counts, and altered oral environment as a result of radiation-induced xerostomia. The end result is a sequestrum of infected bone, with or without a draining fistula. Although nonoperative therapies such as intensive oral care, fluoride rinses, management of xerostomia, and hyperbaric oxygen may be successful, surgery is occasionally required.

Traumatic defects are commonly seen after high-velocity missile/gunshot injuries. A common cause is a failed suicide attempt with a shotgun placed under the jaw. These patients have traditionally been treated with nonvascularized bone grafts and gap-spanning reconstruction plates. Although short-term outcomes may be adequate, an increased use of microvascular free tissue transfer is associated with fewer secondary complications (osteomyelitis, plate exposure) than observed with the use of reconstruction plates.

Pediatric mandibular diseases are typically benign and do not require the extensive resections that demand major reconstructive efforts as in adult patients. However, malignancies do occur, and many of the same principles seen in adults are also applicable to pediatric mandibular reconstruction. Sarcomas are the most common pediatric mandibular malignancies and include rhabdomyosarcomas and osteosarcomas. Given the concern of delayed or insufficient mandibular growth after mandibular reconstruction, distraction techniques may be more widely available as an option in the future; however,

these techniques may not always be possible in patients who have received radiotherapy.

Congenital causes are covered in another chapter ("Craniofacial Microsomia"). Most of these patients are best treated by distraction, but a certain proportion may require transport of vascularized bone to the mandible. Occasional patients with congenital or acquired temporomandibular joint ankylosis may also require surgical correction of the pathologic arthrodesis.

Pathologic Anatomy

The mandible defines the lower third of the face. It is a massive, curved bone that articulates with the craniofacial skeleton posteriorly at the temporomandibular joint. It is a multifunctional structure, having an impact on speech, chewing, and swallowing. In structural terms, loss of this buttress causes collapse of the soft tissues of the lower part of the face.

The bone consists of thick structural (basilar) buttresses and overlying thinner segments. The alveolar ridge is thick to support the teeth—an important implication for the outcomes of treatment of oral malignancies. In edentulous patients there is typically atrophy of the alveolus. The need for a thick bony ridge to support the teeth is underscored by the necessity for adequate bone stock to support intraosseous dental implants, a requirement that has an impact on the choice of donor tissue. Restoration of the continuity of bone after segmental resection of the mandible is essential to maximize both aesthetic and functional outcomes.

In regard to oncologic scenarios, the mandible has several characteristics that are important in determining how or whether a proximate lesion invades the bone. In general, the presence of teeth is a favorable prognostic indicator because intact dentition, to some degree, impedes tumor invasion. The occlusal surface of the mandible is the main portal of entry of SCC cells into nonirradiated bone, a finding that means that tumor cells must travel a greater distance from the gingiva to reach this area in a tooth-bearing mandible; moreover, the teeth, along with the periodontal ligament, serve to impede invasion. Irradiation alters the protective barrier of the periosteum, thereby facilitating invasion through a broader area than solely the occlusal surface.

The muscles attached to the mandible include the masseter, temporalis, internal and external pterygoids, and the suprahyoid group of muscles. These muscles exert powerful biomechanical stresses that have an impact on the reconstruction plan. For example, the high forces of torsion present in lateral and angle defects may eventually fatigue and fracture reconstruction plates if used in isolation to span a defect; hence, this method of reconstruction has proved to be inadequate in this area.

Other associated structures and tissues of the mandible are of clinical importance. The inferior alveolar nerve runs through the lower border of the mandible and ultimately supplies sensation to the skin of the chin and ipsilateral teeth. The blood supply to the mandible is mostly derived from periosteal perforators originating from the thick muscles inserting on the mandible. The mucosal surfaces of the inner aspect of the mandible are lined with keratinized epithelium. The skin on the face has a rich blood supply, with a thin, but significant, layer of subcutaneous fat that helps soften the edges of the underlying bony framework without masking the contour of the lower jaw. These soft tissues, as well as the bone, may be affected by radiation therapy and are susceptible to problems such as infection, fistula formation, intraoral bone or hardware exposure, and major vessel erosion. In the case of oral lining, scarring or insufficiency results in impairment of tongue mobility and potential obliteration of the sulcus.

Most defects can be categorized as affecting the skin, mucosa, or bone. They can also be described anatomically as anterior or lateral or involving the condyle, with the location of the defect having a varying impact on form and function. Classification systems are useful for comparative and longitudinal outcome studies. The modified Jewer classification system is useful (Fig. 1). Bony reconstruction of the *anterior* defect prevents the "Andy Gump" deformity by supporting the structures of the floor of the mouth, specifically, the hyoid musculature, and minimizes problems with labial/oral competence and swallowing. Loss of a *lateral* or *posterior* mandibular segment results in deviation of the mandible because of the unopposed action of the contralateral masticatory muscles, and in time such loss results in problems with occlusal relationships. These changes are easier to prevent than to correct once established.

The composite requirements vary between patients according to the defect, and therefore the reconstruction needs to be tailored to the specific defect in terms of osseous requirements and soft tissue characteristics. Table 1 lists the characteristics of the major anatomic donor sites available for reconstruction of composite mandibular defects.

Diagnostic Studies

Preoperative evaluation commences with a patient history and physical examination. For the patient with a mandibular malignancy, the original tumor and its extent, previous ablative surgery, radiation therapy and chemotherapy, and comorbid conditions are important. A patient with a history of tobacco use needs to be informed of the resulting higher rate of complications, both local and systemic. Nutritional parameters likewise need to be ascertained.

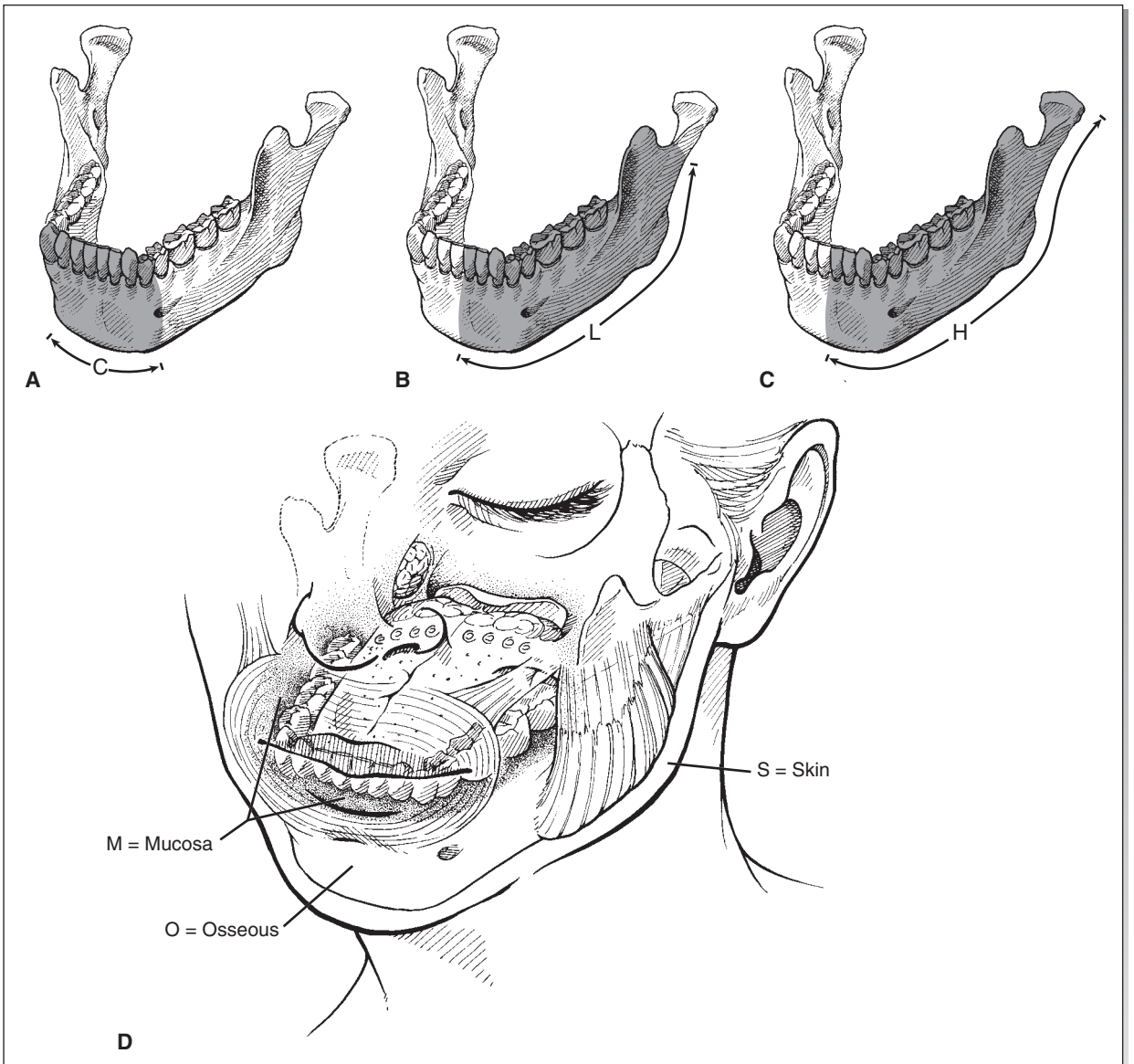

Figure 1 • The Jewer classification system divides mandibular defects as **h**emimandibular (**A**), **l**ateral (**B**), or **c**entral (**C**). This has been modified to document **m**ucosal, **o**sseous, or **s**kin defects. A mandibular defect can therefore be classified as a combination of any of these components (**D**).

Intraoral examination should detect synchronous lesions and evaluate the structures involved. In both posttraumatic and oncologic defects, the condition of the dentition, occlusion, and functional deficits, including sensory or motor loss, should be assessed. Examination of the neck is useful to note any previous neck dissections. Examination of potential donor sites is performed systematically, with emphasis placed on evaluating the presence of pulses in the extremities, as well as the quality and thickness of the skin.

Radiographic Studies

The most useful films are the panoramic series and computed tomography (CT). They are complementary in assisting both the reconstructive surgeon and the extirpative team. Magnetic resonance imaging may be useful to determine soft tissue involvement in patients with oral tumors, but it is of limited practical use for the reconstructive surgeon. Bone scans are occasionally used to determine nonviable segments of mandible in the case of osteoradionecrosis. A panoramic roentgenogram is essential not only for posttraumatic defects but also for segmental resections.

If a fibula free flap is being considered, an angiogram evaluates for a possible peroneus magnus anomaly. However, the authors believe that an angiogram is unnecessary in otherwise healthy candidates without a history of peripheral vascular disease, diabetes, or smoking. An angiogram may be

TABLE 1 Anatomic Characteristics of Major Donor Sites Used for Mandibular Reconstruction

	FIBULA FREE FLAP
Pedicle	Peroneal artery and vein
Pedicle length	15 cm
Innervation possible?	Lateral sural cutaneous nerve
Bone length available	22–27 cm
Suitability for implants	Yes
Skin paddle arc of rotation	Moderate
Skin paddle size (maximum)	15 × 27 cm
Ability to perform osteotomies	Yes
	SCAPULAR FREE FLAP
Pedicle	Circumflex scapular artery and vein
Pedicle length	6 cm, can be extended to 10 cm
Innervation possible?	No
Bone length available	14 cm
Suitability for implants	Marginal
Skin paddle arc of rotation	Moderate
Skin paddle size (maximum)	20 × 7 cm
Ability to perform osteotomies	No
	ILIAC CREST FREE FLAP
Pedicle	Deep circumflex iliac artery and vein
Pedicle length	6–8 cm
Innervation possible?	No
Bone length available	14 cm
Suitability for implants	Yes
Skin paddle arc of rotation	Minimal
Skin paddle size (maximum)	12 × 6 cm
Ability to perform osteotomies	Limited

required in patients with a history of claudication or diminished pulses or in those with aberrant ankle-brachial indices. Promising options to avoid the invasiveness and risks associated with traditional angiography are magnetic resonance angiography and contrast-enhanced CT angiography.

Dental Studies

Dental consultation should be obtained in most cases. If the patient is to receive radiation therapy, preoperative extraction of teeth with significant periodontal disease is advised to prevent postoperative osteoradionecrosis. The oral cavity should be examined and note made of preoperative xerostomia, insensate areas, and the use of dentures and implants.

Reconstructive Goals

Reconstruction of a mandibular defect is an architectural endeavor that needs to satisfy structural, functional, and aesthetic goals. Achieving these goals is possible only when both the bony and soft tissue (skin, muscle, mucosa) components are restored.

The structural goals are accomplished in most instances by restoration of the continuity of the mandibular arch; one exception is a posterolateral defect in an elderly patient without a high load demand on the mandible. If primary closure of the soft tissues can be achieved, the fibrous union and subsequent cicatrization of the surrounding tissues into the defect can suffice as a structural patch to allow adequate function of the mandible. However, in most cases, formal reconstruction is required.

The functional deficits after the varied resections of the mandible are numerous and merit further investigation to quantitate the patterns in loss of specific oral functions resulting from the loss of different segmental areas of the mandible. Historical experience with unreconstructed mandibular defects has starkly exposed the debilitating loss of oral sphincter function, inability to masticate properly, and alterations in speech production. Restoring the continuity of the mandibular arch is therefore critical for preservation of oral function.

Aesthetic restoration of the lower third of the face is as important as restoration of structure and function. The most important advance in mandibular reconstruction has been the achievement of a neomandible that mimics the contour of the patient's native mandible and permits seamless reintegration of the patient into previous routines of daily living.

In addition to the aforementioned objectives, a well-planned reconstruction assists in the oncologic management of patients. The oncologic goals are to permit resection of tumors that historically could not otherwise be resected, allow timely delivery of radiation and other adjuvant treatments, and return patients to their environment in the shortest time possible. The reconstruction must never delay or compromise adjunctive oncologic care.

All effort should be invested in avoiding a delayed reconstruction. Although defects caused by severe trauma and infection are not always amenable to immediate reconstruction, coordinated and timely reconstruction after ablative surgery has been shown to avoid the pitfalls associated with delayed reconstruction, which include cicatrix formation, retraction, and tethering of the surrounding soft tissue. Once present, the secondary sequelae are not fully correctable. Occasionally, a delay in reconstruction may be necessary for reasons of infection, for concerns regarding tissue viability, or when other life-threatening injuries take precedence, as in some gunshot injuries. These cases are salvaged with a reconstruction plate, which assists in healing and

minimizes secondary deformities. However, immediate reconstruction offers the best opportunity to restore optimal aesthetic and functional results and is usually possible in cases of tumor resection and congenital defects.

Lower jaw defects may necessitate osseous reconstruction, soft tissue repair, or both. Although much of the emphasis in mandibular reconstruction is inevitably focused on the bony mandible, reconstruction of the neighboring soft tissues is important, particularly because most reconstructions address composite defects. For example, a neomandible is able to better tolerate postoperative radiation therapy if it is enveloped by well-vascularized muscle. Resection of the intraoral mucosal lining can lead to scarring and immobility of the remaining portion of tongue that can result in significant functional problems. Recreation of an adequate buccal sulcus permits the use of dentures postoperatively and maintains space for the placement of osseointegrated implants. Loss of soft tissue in the submental region and cheek can also lead to a significant aesthetic deformity. In addition, an irradiated bed renders the use of a nonvascularized graft futile. Segmental mandibular resections that include intraoral lining and external skin create the most extensive defects. These patients have major functional and aesthetic deformities, which are best addressed with a composite tissue transfer. However, several factors make reconstruction difficult. First, the aggressive nature of the disease in most patients requiring mandible resection means that most patients have advanced disease; therefore, the reconstructive technique should be single stage and immediate. Second, the site of the defect has often been compromised by oral contamination, previous radiotherapy, or both, thus making the survival of any nonvascularized tissue unlikely. Finally, most tumors are epidermal in origin, and therefore mucosa or skin (or both) needs to be resected and restored; occasionally, two separate flaps are needed. Microvascular techniques address these issues.

Treatment

Optimal care of a patient with a mandibular defect necessitating reconstruction is provided by a multidisciplinary team, including oncologic surgeons, reconstructive surgeons, and dentists working in a center that is dedicated to participating in all of the longitudinal phases of the patient's care.

Timing

Reconstruction is ideally performed immediately after the extirpative procedure. Cases not amenable to immediate reconstruction (e.g., traumatic cases with other serious injuries) are eventually compromised by soft tissue scarring, which makes it difficult to estimate accurate occlusal relationships or tissue deficits, or both.

Adjunctive Procedures

A tracheostomy is often required in patients after cancer resection. Feeding tubes are placed preoperatively to ensure adequate nutritional intake. In most cases, a 2.3- or 2.7-mm locking reconstructive plate molded and predrilled before mandible resection can be used as a template to obviate the need for arch bars, but arch bars are still occasionally required.

Evaluation of Recipient Vessels for Free Tissue Transfer

The face and neck provide a variety of choices for recipient vessels. Unfortunately, these vessels are frequently situated within an irradiated field and are characterized by friability and fibrosis with loss of elasticity, findings which result in a more difficult dissection and a higher risk for postoperative thrombosis. Several guidelines can be followed that permit a reliable anastomosis in this area. The preferred vessel for arterial anastomosis at our institution is the external carotid artery because the vessel is usually spared by the oncologic surgeon. The anastomosis is performed in an end-to-side manner with a 2.3-mm aortic punch used for the arteriotomy. The facial artery or facial-lingual trunk can be used, provided that the contralateral lingual artery is spared to supply the tongue. The superior thyroid artery is not routinely used because of its propensity to spasm. The veins selected should be as large as possible. The authors' preferred recipient vein is the external jugular vein. These larger neck veins act as a sump to increase outflow from the flap and are greatly preferred over other vessels. If necessary, vein grafts can be used to reach into the contralateral vessels in a vessel-depleted neck. If possible, it may be prudent to perform two venous anastomoses to assist in drainage of the flap. Regardless of the vessels selected, care should be taken to prevent kinking, swelling (common in head and neck cases), and infection.

Reconstructive Options

NO BONY RECONSTRUCTION. Not all scenarios call for osseous reconstruction. In particular, limited posterior or lateral defects, especially in edentulous patients, are amenable to direct soft tissue closure. Similarly, a patient in extremis with a grave prognosis may best be served by a minimal reconstructive effort.

LOCAL FLAPS AND SKIN GRAFTS. Local flaps and skin grafts can be used to resurface some defects without resorting to a more complex reconstruction; in these situations, the surgeon can use local flaps

for soft tissue closure. Skin grafts are useful for intraoral lining defects, although the incidence of graft loss is higher in this anatomic area. Their use is limited to non-irradiated beds.

RECONSTRUCTION PLATES WITH OR WITHOUT SOFT TISSUE RECONSTRUCTION. As stated earlier, reconstruction plates are occasionally useful in traumatic situations when other associated injuries delay definitive reconstruction. The plates cannot be used in isolation in mandibles that will be or have been exposed to radiation. If soft tissue is present, reconstruction plates can be of utility in limited lateral (not anterior) defects. However, over the long run, they will likely fail because of the lack of bone reconstitution and subsequent metal fatigue.

NONVASCULARIZED BONE GRAFTS. The modern-day role of nonvascularized bone grafts is limited. Although these grafts, typically harvested from such sites as iliac crest, rib, and calvaria, have historically been used more extensively, their survival rates are low. These rates are worsened by irradiation. This option should therefore be considered only for small (<5 cm) lateral segment bony defects that have not previously been irradiated (for example, in nonmalignant diseases) and in patients who cannot tolerate a lengthy microvascular procedure. In most instances, microvascular transfer is preferable, particularly in cases of irradiation or significant soft tissue loss.

REGIONAL FLAPS. The only role for a regional flap is to resurface cheek or neck skin in full-thickness defects or in occasional salvage situations.

VASCULARIZED OSSEOCUTANEOUS FLAPS. These flaps are the gold standard. Pedicled osseocutaneous flaps provide marginal bone stock for major mandible defects and are no longer widely used. The best option is a microvascular free flap. A benefit of using a vascularized bone flap to reconstruct the mandible is that the shape obtained is typically maintained over the long term. The most common donor options are the fibula, scapula, and deep circumflex iliac artery osseocutaneous flaps, with the fibula flap being the most widely used (see Table 1).

Fibula Free Flap. The fibula microvascular free flap is a versatile tool with many possible indications, including reconstruction of the mandible from angle to angle. The fibula is the flap of choice for most cases requiring mandibular reconstruction. By varying the length of the osseous component and including a skin paddle, it can reconstruct defects in any of the areas of the mandible. The skin paddle is extremely reliable, particularly when the flexor hallucis longus muscle is included, and it can be used to resurface the intraoral surfaces and to act as a sentinel for flap monitoring. When soft tissue bulk is needed, the flexor hallucis longus muscle can be harvested with the flap (Fig. 2).

The flap is harvested with the patient in the supine position. If the width of the skin island exceeds 4 to 5 cm, plans for a skin graft should be made to close the donor site defect. Close cooperation with the ablative team is essential during this part of the operation. As a general rule, the mid-fibula is used for mandibular ramus and angle defects, whereas the distal end of the fibula can be used to carry the long pedicle needed to reach the neck vessels as required for reconstruction of anterior mandibular defects. It is useful to examine the perforators supplying the skin paddle with a hand-held Doppler. A tourniquet is placed on the upper part of the thigh to assist in dissection.

Several key neurovascular structures need to be preserved (see Fig. 2). The dissection proceeds in a lateral-to-medial, distal-to-proximal direction. In general, the safest way to reach the peroneal vessels supplying this flap is to begin the dissection later-

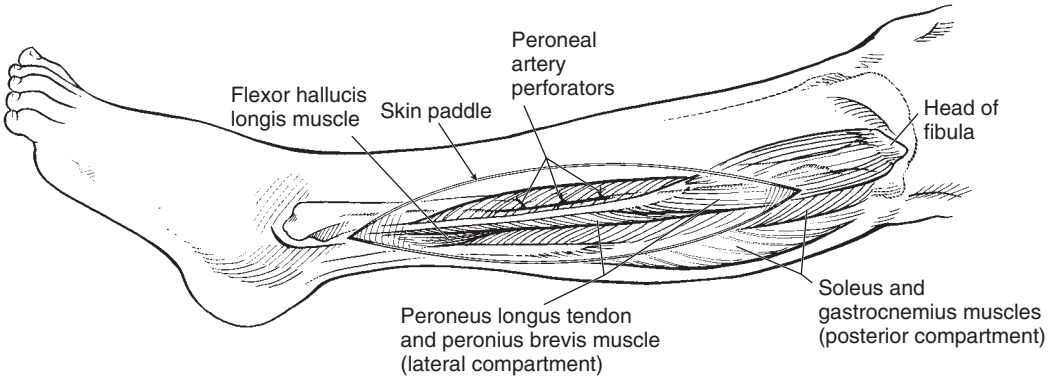

Figure 2 • Schematic of the size of the skin island possible by carefully including the peroneal artery perforators in the dissection of the fibula flap. Also depicted are major anatomic landmarks.

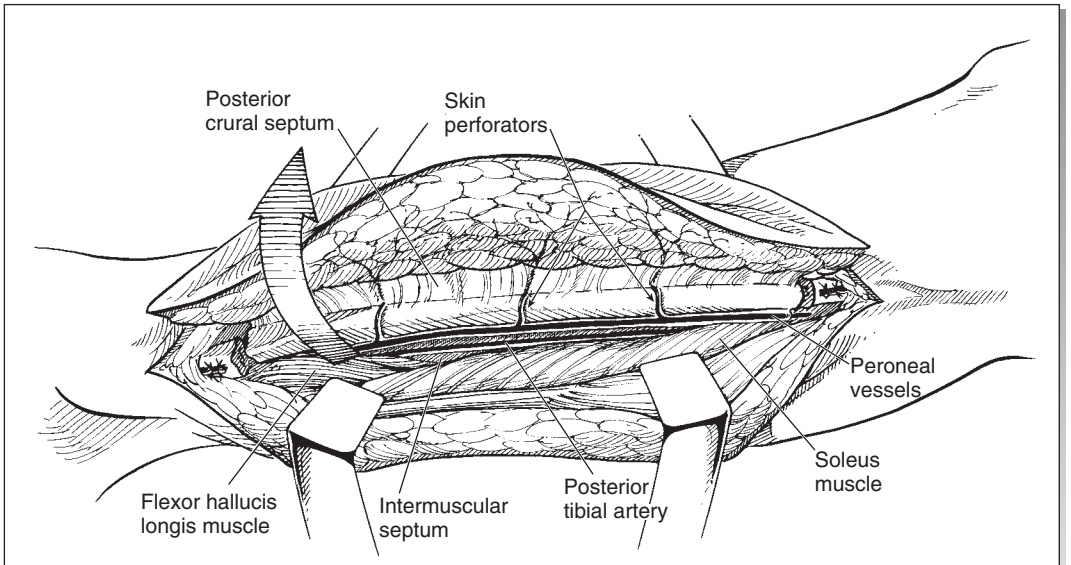

Figure 3 • The fibula flap following elevation by proximal and distal osteotomies. The segmental vascular supply permits multiple osteotomies to be performed to create a mandibular arch.

ally and anteriorly. With care taken to protect the superficial peroneal nerve, the dissection proceeds through the calf tissue while being careful to stay just anterior to the posterolateral intermuscular septum. This plane is followed until the lateral border of the fibula is reached. The muscles attached to the fibula are removed, and anteriorly the anterolateral intermuscular septum is divided, as is the interosseous membrane. The fibula is approached posteriorly; when the soleus muscle is reached, a cuff of this muscle is elevated with the flap to complete exposure of the lateral aspect of the fibula. A saw is used to cut the fibula at the desired points. After these osteotomies, the fibula can be maneuvered to expose the pedicle located medially. This technique permits safe dissection of the peroneal vessels; they can be divided as distally as necessary and dissected proximally up to the trifurcation if necessary (Fig. 3). The perforators supplying the skin paddle are visualized and preserved. In situations in which it is necessary to have a long pedicle, the effective pedicle length can be increased by dissecting the pedicle off the proximal end of the fibula with a periosteal elevator until the nutritive periosteal perforators are seen.

After the osteotomies, the fibula is shaped to the contour of the native mandible by using a premolded reconstruction plate as a template (Fig. 4). The fibula is shaped in situ, and the peroneal vessels are not divided until the ablative team has completed the resection. The fibula is transferred and inset into its new position. The skin paddle is inset first, and the bone is fixed to the mandibular remnants by using the holes made during the molding before resection. After bone fixation, the operating microscope is brought into the field, and the peroneal vessels are anastomosed to the previously prepared vessels, usually in the neck (Fig. 5).

Free Scapular Flap. This flap is mainly used for patients who have a full-thickness bony defect and who lack both external and internal soft tissue surfaces. It is based on the circumflex scapular artery, which emerges from the triangular space bordered by the teres major, teres minor, and the long head of the triceps. Its main advantage is that it is potentially a trisegmented flap with two independently mobile skin paddles and a vascularized segment of bone (the lateral border of the scapula). Because the bone is supplied by a single feeder vessel, it is not possible to perform shaping osteotomies on the scapular bone segment. Furthermore, the flap can be bulky if the cutaneous portion of the flap is used. It is therefore best reserved for situations in which a small segment of bone is needed and both mucosal and cutaneous epithelial cover is missing.

Iliac Crest. The vascularized iliac crest was once considered the "gold standard" of mandibular reconstruction. With the benefit of hindsight, it is now apparent that mandibles re-created with this flap are mostly block-like and bulky and lack the refinement attainable with a fibula free flap. Drawbacks are numerous. For example, the bone lacks a segmental blood supply. Although ample bone is available, its fixed anatomic configuration is a disadvantage, especially in anterior reconstructions. The irregular shape of the ilium can be changed somewhat with osteotomies, but it still lacks the flexibility and precision that can be achieved with the fibula. Furthermore, the skin available with the bone has a tenuous blood supply and tends to be

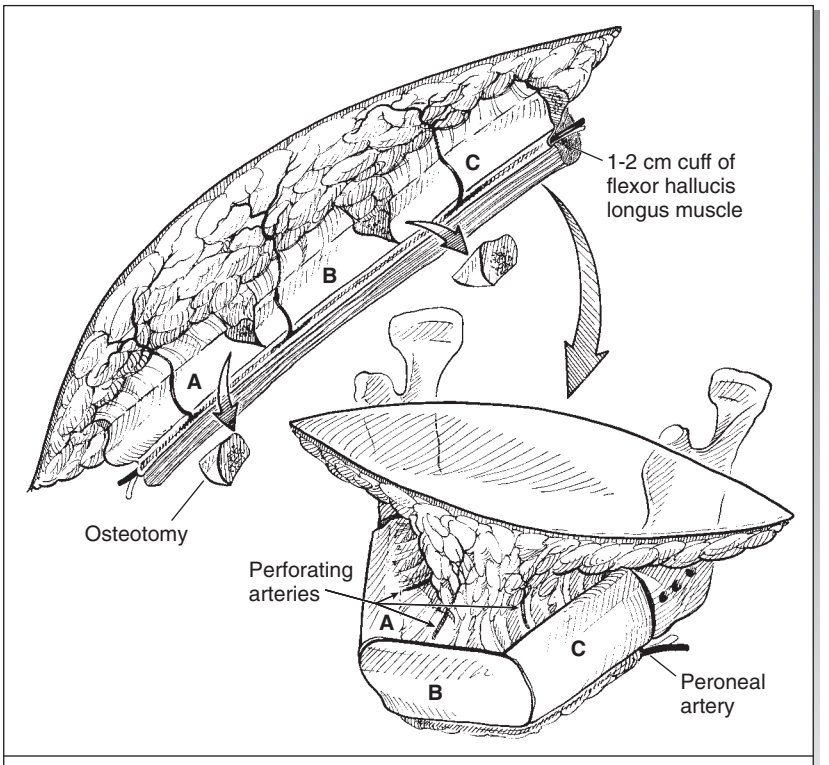

Figure 4 • Precise orientation of the wedge osteotomies facilitates proper insetting of the soft tissue components of the flap.

thick and block-like, thus limiting its ability to be easily inset within the oral cavity. Patients have significant pain postoperatively and the potential for hernia formation. Another disadvantage is that chronic thigh paresthesia can result if the lateral femoral cutaneous nerve is injured. Bone length is limited to 14 cm, so reconstruction of a total mandibulectomy defect may require bilateral iliac crest flaps. Advantages of the iliac crest flap are that the donor scar is well hidden, the pedicle is large

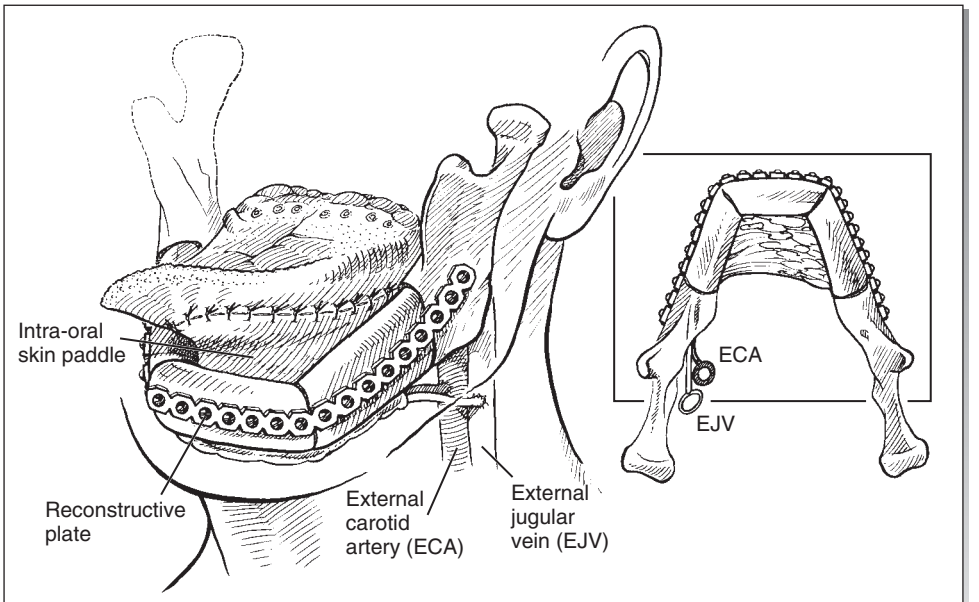

Figure 5 • The final orientation of the osseous, soft tissue, and vascular arrangements of the fibula flap. Mandibular continuity has been restored, the oral defect has been closed, and an anastomosis to large-caliber neck vessels has been performed.

and consistent, and the bony portion is thick and can easily accept osseointegrated dental implants.

The Pediatric Mandible

Microvascular free flap reconstruction of a pediatric mandible is possible and appears to provide the best solution for larger mandibular defects. Despite flap harvest from growing skeletal sites, children typically maintain normal growth at the donor sites, provided that the growth centers are not disturbed. The transferred tissue generally grows with the child to maintain symmetry and reasonable projection. The dynamic status of the pediatric mandible requires conceptualization of the problem in a temporal as well as spatial manner to account for future bone and soft tissue growth. When using microvascular techniques, care should be taken to avoid disruption of the epiphyseal centers in the donor sites. Distraction should also be considered an option in reconstructing pediatric mandibular defects.

Management of the Condyle

Despite a variety of available techniques, the results of condylar reconstruction are generally disappointing. When oncologically sound, the condyle can be disarticulated and reattached to the reconstructed mandible. Fascia or a fat graft may be placed as a spacer. Another option is to use an osteochondral rib graft with a spacer; this option works best in pediatric mandibles. Nonetheless, the outcomes of condylar reconstruction in adults are often disappointing, and patients should be so informed.

Postoperative Care

Free flaps need to be monitored carefully in the postoperative period, ideally in a dedicated unit. Free flap loss usually occurs in the first 24 to 48 hours. If thrombosis develops, the best chance of salvage is immediate exploration and thrombolysis, preferably within 3 hours. Thrombosis is the most feared complication because of its potential to delay radiation therapy and patient discharge. Occasionally, partial flap loss can occur, including partial bone loss. Salvage of these flaps should not be influenced adversely by the complication: if a free flap was the optimal first choice, a second free flap is often still the optimal choice.

Patients with a tracheostomy can usually be decannulated immediately in the postoperative period. Oral feeding is commenced 1 week after surgery if no complications have developed. Patients with gastrostomy tubes can be fed on return of bowel function. Fixation plates can be removed after 6 months, but they are generally left in place unless plate extrusion and exposure occur. The mandible should be monitored with serial Panorex studies, although the clinician needs to be aware that radiographic evidence of healing usually lags behind the clinical picture.

Infections and hematomas develop in approximately 6% of cases but will not usually affect the long-term surgical outcome if properly and vigilantly treated. Plate exposure is most common in the anterior aspect of the mandible because of deficiencies in soft tissue. The main goal is to permit healing and remove the plate when the bone fractures/osteotomies have healed. This is best facilitated by coverage with muscle and the use of low-profile plates. Plate exposure is most common after postoperative adjuvant irradiation. Despite exposure, when a vascularized bone flap is used, bony union rates approach 95%.

Fistulas also occur more commonly in irradiated beds. Although distressing to the surgeon and patient alike, most fistulas usually heal spontaneously.

Malocclusion can result from improper surgical planning, fracture of the fixation wire or plate, or intraoperative failure to position the condyle in the temporomandibular joint. An osteotomy at the junction of the reconstruction flap and remaining native mandible may be required for correction of malocclusion.

Soft tissue atrophy is a significant problem, especially after radiotherapy, and diminishes the aesthetic results. The use of excessively bulky soft tissue paddles, particularly in reconstructions that involve the deep circumflex iliac artery flap, may suffer from the converse problem of interference with functional results, such as speech and swallowing. The paddles can be debulked at a secondary stage.

Osseointegrated implants can be placed 6 months to 1 year after reconstruction. Despite reports in the literature, the volume of the fibula is not always as large as needed to place osseointegrated implants. The use of implants in irradiated mandibles has not been well studied and should be attempted only in the context of a clinical trial. In well-selected patients, osseointegrated implants are, however, of great utility, despite their expense.

When performed properly and attention is paid to the principles outlined previously, reconstruction of the mandible is a safe procedure that maximizes the gains possible from both oncologic surgery and adjuvant care.

Pearls and Pitfalls

- Patients undergoing ablation and mandibular reconstruction should be managed only by an experienced multidisciplinary team.
- When using a fibula free flap, incorporate a skin paddle for postoperative monitoring purposes.

- A buried, continuous Doppler device is useful for monitoring purposes.
- Use the largest recipient vessels within reach of the vascular pedicle when performing a microvascular reconstruction. When possible, the external carotid artery and the internal jugular vein should be the first choice of recipient vessels.
- If a free flap fails, the best solution to salvage the reconstruction is usually another free flap.
- It is crucial to achieve anatomic occlusion at the time of mandibular reconstruction. This is best accomplished by using the resected specimen as a template to accurately place the osteotomies and to shape the fibula free flap. Reconstruction plates are useful for this purpose.

SUGGESTED READINGS

Coleman JJ (ed): Operative Techniques in Plastic and Reconstructive Surgery: Mandible Reconstruction. 1996.

Hidalgo DA, Pusic AL: Free flap mandibular reconstruction: A 10-year follow-up study. Plast Reconstr Surg 110:438-449, 2002.

The Temporomandibular Joint

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Temporomandibular disorder (TMD) is a general term used to describe musculoskeletal dysfunction of the temporomandibular joint (TMJ) and associated structures. Most studies suggest that the prevalence of clinically significant TMD-related jaw pain/dysfunction is at least 5% in the general population, with significantly more frequent and more severe TMD signs and symptoms appearing in women and older adults. This chapter reviews the pathophysiology of TMDs with an emphasis on the most common surgical indications and procedures.

Anatomy

The TMJ is uniquely characterized as a ginglymoarthrodial structure (having rotational and translational movement); its function is dictated by jaw movement and is also dependent on the contralateral joint. Its proximity to many significant structures (e.g., facial nerve, ear, blood vessels) makes surgical access and treatment difficult.

Anatomic components of the TMJ include the condylar head, glenoid fossa, superior and inferior joint spaces, capsule, retrodiscal tissue (or posterior attachment), lateral pterygoid muscle, and disk (meniscus) (Fig. 1). The disk is a biconcave fibrous connective tissue structure composed of anterior and posterior bands surrounding a thin intermediate zone dividing the joint into superior and inferior joint spaces. The first phase of mouth opening (2 to 3 cm) is hinge or rotational, a function of the inferior joint space, followed by a second phase (4 to 6 cm) consisting of a gliding or translational motion, which in turn is a function of the superior joint space. Synovial fluid functions to lubricate and nourish articular cartilage and remove particulate and soluble debris from the joint. The vascular supply of the TMJ is via the superficial temporal artery and branches of the internal maxillary artery. Sensory innervation is via the auriculotemporal nerve, a branch of the posterior division of the mandibular nerve, with contributions from the masseteric and deep posterior temporal nerves.

Pathophysiology

The cause of TMDs remains controversial, and many factors have been implicated. Trauma has been suggested as the most likely cause and is classified as direct (e.g., assault), indirect (e.g., whiplash injury), or secondary to parafunctional habits (e.g., clenching, bruxism). Suggested pathophysiologic mechanisms include direct mechanical injury, hypoxia-reperfusion injury, and neurologic inflammation with modifications in condylar dynamics. Other contributing factors may include dentofacial deformities and psychosocial causes. Numerous studies continue to dispute the significance of occlusal relationships in TMDs. TMJ abnormalities are also associated with systemic diseases such as the arthritides, metabolic disorders (gout), or other syndromes (Marfan's syndrome). In addition, they may reflect disorders in growth and development, as seen in condylar hyperplasia or hypoplasia associated with systemic sclerosis, or iatrogenic causes, including the indiscriminate use of repeated corticosteroid injections.

Classification

The classification of oral and facial disorders is diverse, and it has been estimated that up to a quarter of the adult population experiences

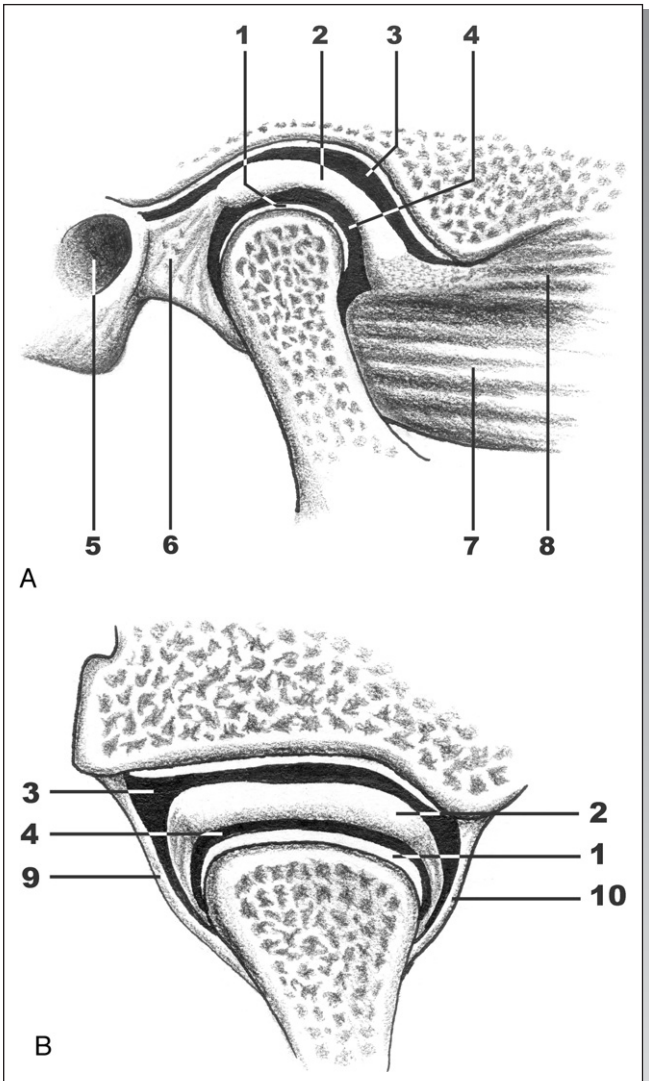

Figure 1 • Temporomandibular joint anatomy. **A,** Midsagittal section. **B,** Coronal section. Structures visualized are the articular cartilage of the condyle (1), articular disk (2), superior joint space (3), inferior joint space (4), external auditory canal (5), retrodiscal attachment (6), inferior belly of the lateral pterygoid muscle (7), superior belly of the lateral pterygoid muscle (8), lateral capsular ligament (9), and medial capsular ligament (10). (Courtesy of Jesse Koskey, www.JesseKoskey.com.)

symptoms. TMDs include internal derangements, myofascial pain and dysfunction syndrome, arthritides, and neoplasia. Disorders that may confound the diagnosis include Eagle's syndrome, vascular anomalies (e.g., temporal arteritis), fibromyalgia, otalgia, paranasal sinus disorders, optic neuritis, headaches, neuralgias, odontogenic infections, and salivary gland disease.

Physical Evaluation

After a comprehensive history, the joint should be palpated preauricularly and intrameatally in the open and closed position to note the presence or absence of tenderness, swelling, and crepitation. The muscles of mastication and the cervical muscles are also examined. Examination of jaw motion should include the amount of active and passive incisal opening, rotational and translational movements, lateral excursions, protrusion, and symmetry of jaw movement. Auscultation should be performed to detect clicking, popping, and crepitus relative to jaw position. Crepitus suggests bone-on-bone contact as a result of surface defects on the disk or articular surfaces. However, joint noises in the absence of pain, functional limitations, or deformity represent an adaptive mechanism of the TMJ and are not necessarily a sign of disease. Oral examination should also focus on evidence of tooth wear and occlusion. Other areas that may be examined include the cervical spine, shoulders, and upper extremities. An auriculotemporal nerve block induced by administering a local anesthetic slightly behind and toward the neck of the condyle may be useful to confirm an arthrogenous cause.

Diagnosis of Temporomandibular Disorders

Myofascial pain and dysfunction are noted in at least 50% of all TMDs. The syndrome manifests as dysfunctional muscle hyperactivity with regional pain, tenderness of the affected muscles, variable amounts of reduced opening, and complaints of malocclusion.

Internal derangements are divided into disk (meniscal) displacement with reduction, disk displacement without reduction (closed lock), hypermobility and dislocation (open lock), and ankylosis.

In *disk displacement with reduction*, the disk is anteriorly displaced in the resting, closed-joint position and reduces on opening (Fig. 2). This derangement is also associated with reduction in lateral excursive movement away from the affected side, variable maximal incisal opening, joint noise that usually occurs at variable positions during opening and closing mandibular movements, variable pain precipitated by joint movement, and mandible deflection (i.e., returns to the midline) on opening, usually during the latter phase of translation.

Anterior disk displacement without reduction (closed lock) is characterized by an irreducible, displaced disk that acts as an obstacle to the sliding condyle (Fig. 3). Patients complain of intermittent self-reducible locking, a sudden onset of limited mouth opening (≤35 mm) associated with cessation of joint sounds, deflection, midline correction on opening, and restricted lateral excursive movements away from the affected side.

Ankylosis of the TMJ is defined as immobility of the condyle and can be classified by tissue type (fibrous, bony), anatomic location (intraarticular,

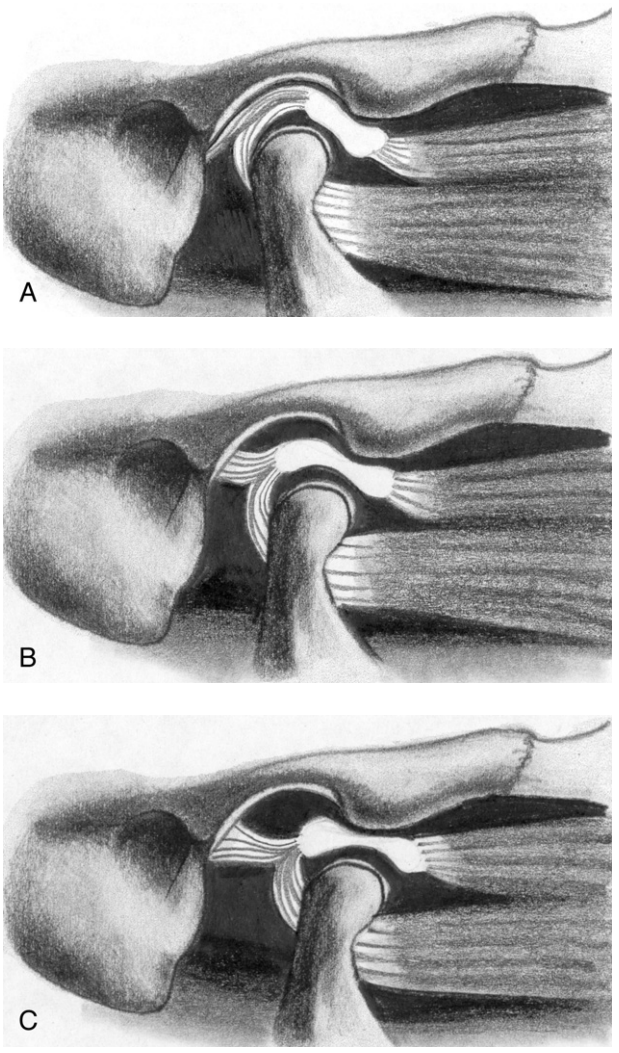

Figure 2 • Disk displacement with reduction. **A,** Disk situated anterior to the articulating surface of the condyle. As the condyle translates forward, it passes over the disk and produces a clicking noise. **B,** The disk remains in an appropriate relationship with the condyle through the remainder of the opening cycle. **C,** Maximum opening position. As the mandible closes, the relationship of the condyle and disk returns to the position shown in **A.** (Courtesy of Jesse Koskey, www.JesseKoskey.com.)

extraarticular), and extent of fusion (complete, incomplete). Joint infection, usually occurring after trauma, accounts for approximately 50% of all TMJ ankylosis cases, but 30% result from aseptic trauma. Fibrotic intraarticular ankylosis is the most common type, especially after trauma-induced hemorrhage (hemarthrosis). Children are more prone to ankylosis because they have greater osteogenic potential and less development of the joint meniscus.

Hypermobility of the TMJ may manifest as dislocation or subluxation, or both. Dislocation describes translation of one or both condyles into an obstructed position anterior to the articular eminence. On clinical examination the jaw is "locked" open. In subluxation, the patient is able to relocate the dislocation with manipulation. Predisposing factors include previous capsule and ligament injury, joint laxity, internal derangements, trauma, drugs associated with extrapyramidal reactions, and seizures.

The *arthritides* are classified as either rheumatoid arthritis or osteoarthritis. In rheumatoid arthritis, patients may complain of pain that is worse in the morning, limited opening, occlusal changes, and preauricular edema and tenderness. Approximately 50% to 75% of patients with rheumatoid arthritis have involvement of the TMJ during the course of the disease. Osteoarthritis, also

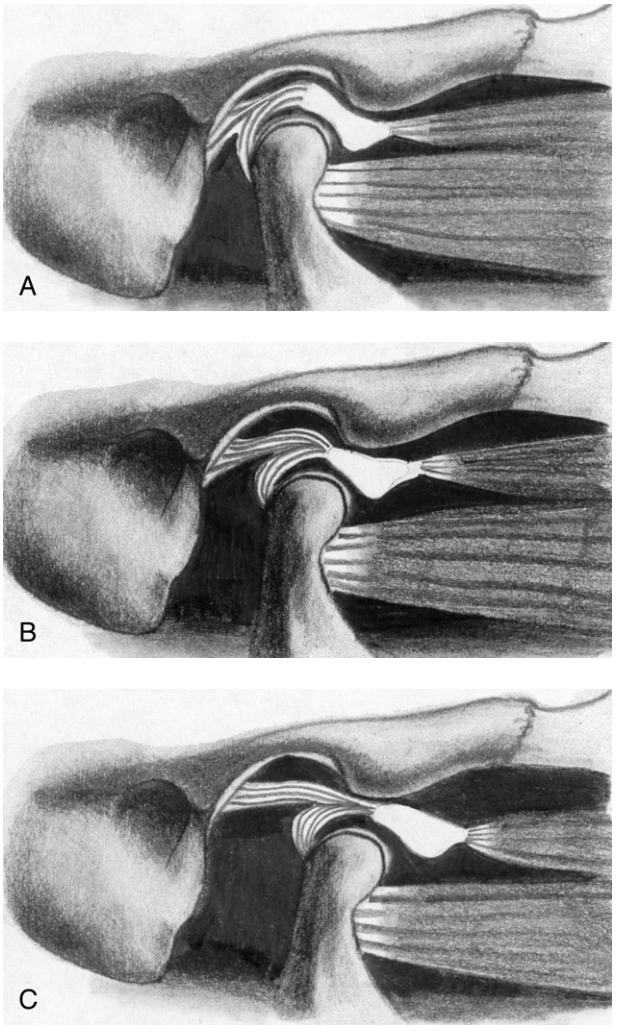

Figure 3 • Disk displacement without reduction (closed lock). **A,** A disk that has been chronically anteriorly displaced has an amorphous shape rather than a distinctive biconcave structure. **B,** As the condyle begins to translate forward, the disk remains anterior to the condyle. **C,** In the maximum open position, the disk continues to remain anterior to the condyle, with posterior attachment tissue interposed between the condyle and fossa. (Courtesy of Jesse Koskey, www.JesseKoskey.com.)

referred to as degenerative joint disease, is the most common disease affecting the TMJ. It is characterized by degenerative changes of the articular cartilage with associated remodeling. Patients may complain of pain that is worse in the evening, limited opening, muscle splinting, and crepitus of the TMJ.

Tumors arising from the TMJ are rare. The most common benign tumors that develop in the condyle include osteochondroma, osteoblastoma, chondroblastoma, and osteoma. Synovial chondromatosis is the most common benign neoplasm of the synovium and is characterized by the development of metaplastic, highly cellular cartilaginous foci in the synovial membrane that result in degenerative changes consistent with osteoarthritis. It is characterized by swelling, pain, and limitation of movement. Radiographic findings are variable and may include the presence of radiopaque bodies in the TMJ, degenerative changes of the articular surfaces, and variable widening or loss of joint space. Osteosarcoma is one of the most frequently occurring malignant bone tumors, is second only to hematopoietic neoplasms, and accounts for about 20% of primary bone cancers. Approximately 6% to 8% of all osteosarcomas occur in the jaw, including the TMJ.

Diagnostic Studies

Radiographic evaluation of patients with TMDs typically includes panoramic or transcranial imaging and is limited to identifying gross osseous changes. Computed tomography is the most accurate modality for identification of osseous abnormalities, and magnetic resonance imaging is mandatory for examining disk position, function, and form and the presence of joint effusions (inflammatory changes). Arthrography may be useful when magnetic resonance imaging is not tolerated and information regarding the position and morphology of the disk is required.

Treatment

Nonsurgical

Therapeutic goals involve improving function (i.e., range of motion) and reducing pain. Treatment typically begins with nonsurgical modalities. The *first phase* of nonsurgical treatment includes pharmacologic therapy, moist heat, physical therapy, a soft mechanical diet, and stretching, jaw coordination exercises. Treatment is indicated for internal derangements, myofascial pain and dysfunction, and the arthritides. Medications include nonsteroidal antiinflammatory drugs, tramadol (Ultram), and narcotics; muscle relaxants and antianxiety agents are used for muscle hypertonicity. Certain patients may benefit from occlusal or orthodontic therapy to correct uneven tooth contact or malocclusion, respectively. If the patient fails to improve or worsens after 1 to 2 months of conservative management, the *second phase* may be initiated: the use of occlusal appliances. The most common is a stabilization appliance (also referred to as a passive splint or flat-plane appliance), which functions to relax the elevator masticatory muscles, protect the dentition, stabilize and protect the TMJs from excessive loading, and provide biofeedback by making patients aware of bruxing habits. The anterior repositioning device functions to protrude the mandible and relieve the load on the disk, thereby allowing repair of damaged retrodiscal tissue. This type of appliance should be carefully monitored because it may cause a posterior open bite. Botulism toxin has recently been advocated to treat severe bruxism by paralyzing the masticatory muscles and thereby reducing inflammation of these muscles and the TMJ.

Surgical

Patients with TMDs, except for myofascial pain and dysfunction syndrome, may be considered surgical candidates if they are refractory to nonsurgical therapy and suffer from pain and functional impairment as a result of interference with TMJ function. Surgical techniques include arthrocentesis and lavage, arthroscopy, condylotomy, and open joint surgery.

Arthrocentesis is a minimally invasive procedure that involves insertion of a 19-gauge needle into the TMJ at the level of the articular glenoid fossa and articular eminence (superior joint space) for irrigation. Lavaging the joint may release adhesions and negative pressure on the disk, thereby reducing surface friction and the viscosity of synovial fluid and decreasing inflammatory constituents and pain mediators in the joint. It is indicated for hypomobility caused by an acute or chronic closed lock in patients complaining of chronic pain with adequate range of motion and failure to improve after other joint surgery. The procedure may be accomplished under local anesthesia with or without intravenous sedation.

Arthroscopy, lysis, and lavage are indicated for hypomobility disorders such as internal derangements with or without pain and intrajoint adhesions, hypermobility disorders resulting in painful subluxation or dislocation, synovitis secondary to rheumatoid arthritis, osteoarthritis, and synovial chondromatosis (early stages). The technique involves insertion of a cannula with an arthroscope into the superior joint space at the maximum concavity of the glenoid fossa and a second 22-gauge outflow needle into the superior joint space anterior to the arthroscope puncture site. This procedure

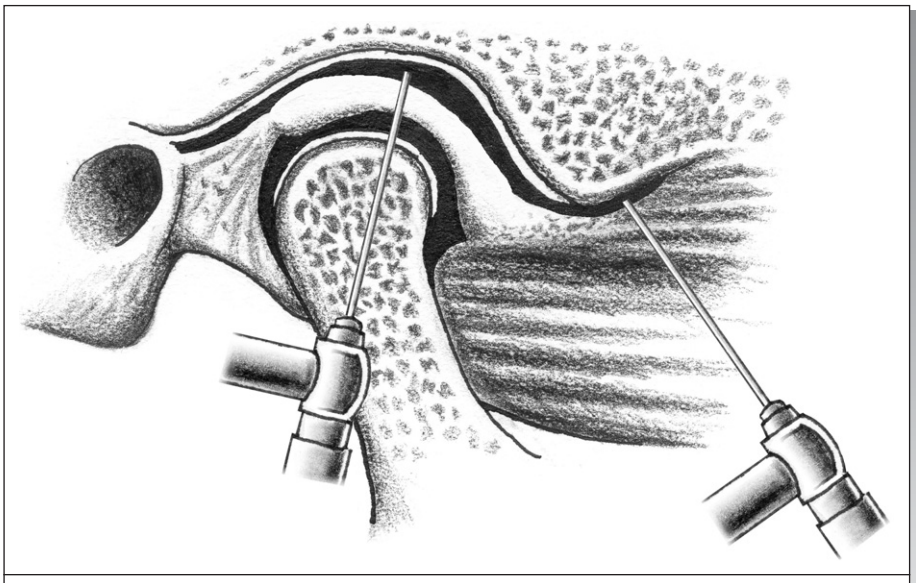

Figure 4 • Techniques of arthroscope insertion into the superior joint space. **Left,** Inferolateral approach to the posterior joint space. **Right,** Anterolateral approach to the anterior recess. (Courtesy of Jesse Koskey, www.JesseKoskey.com.)

allows direct examination of the superior joint space, including the disk, temporal articular surfaces, synovial membrane, and retrodiscal tissues (Fig. 4).

After arthroscopic examination, the irrigation needle can be removed and a second working cannula inserted to facilitate surgical techniques such as lysis of adhesions, synovectomy, and pterygoid myotomy (anterior release of the joint capsule). Other procedures that may be attempted include synovial biopsy and posterior suturing (or plication) of the disk. Steroids are commonly injected into the TMJ to decrease the inflammatory response to surgery, but long-term or excessive use may cause condylar hypoplasia by inhibiting osteoblastic activity. Success rates vary with surgical arthroscopy, and there is a 22% incidence of reoperation for disk displacement without reduction. Complications associated with arthroscopy include vascular injury, hemarthrosis, infection, otologic damage, facial nerve injury, and penetration into the middle cranial fossa.

Open joint surgery (arthrotomy) is a broad term encompassing all procedures that allow direct access for disk repositioning, diskectomy, disk replacement, eminectomy, gap arthroplasty, and joint replacement. Surgical approaches include preauricular, endaural, and postauricular incisions. A *preauricular* incision gives maximum lateral and anterior exposure. An *endaural* incision is a cosmetic modification of the preauricular approach. A *postauricular* incision has an acceptable cosmetic result but is associated with a risk for stenosis of the auditory canal, infection and necrosis of the ear cartilage, and almost complete anesthesia of the auricle. This approach should not be used with associated joint infection or otitis externa.

Disk repositioning (discoplasty) is an attempt to reposition the disk to restore the normal condyle-disk-fossa relationship. The technique is indicated after attempting arthroscopy or arthrocentesis for the treatment of disk displacement with or without reduction. Pterygoid myotomy (releasing the disk from the lateral pterygoid muscle) is occasionally indicated when a nonreducing, anteriorly displaced disk is tethered by the lateral pterygoid muscle. Plication of retrodiscal tissue and excision with reapproximation of the retrodiscal tissue may be indicated for anterior disk displacement secondary to redundant or pathologic (perforated) retrodiscal tissue. Success rates ranging from 80% to 94% have been reported, although long-term evaluation of disk position has not been validated.

Diskectomy involves removal of the disk. Indications include pain or dysfunction (or both) that shows no improvement after nonsurgical treatment, failed procedures, and severe internal derangements precluding repair of the disk. Although reports support an 80% success rate after diskectomy, surgeons continue to debate whether interpositional material (graft or alloplast) should be placed in the joint after diskectomy. *Disk replacement* is indicated for a nonrepairable disk after gap arthroplasty or a costochondral graft when maintenance of a soft tissue interface between the newly articulating surfaces is vital (i.e., ankylosis). Replacement is also indicated to dissipate loading forces, reduce the formation of fibrous or bony ankylosis, decrease pain, improve joint mobility, decrease the incidence or severity (or both) of osteoarthritis, provide a

gliding surface, prevent bone-on-bone contact, and reduce the development of an open bite after diskectomy. A commonly accepted disk replacement tissue is dermis, but other options include temporalis muscle and auricular cartilage. The latter is susceptible to the development of adhesions, is difficult to stabilize, and is likely to perforate.

Eminectomy is performed for recurrent mandibular hypermobility/subluxation and involves removal of the articular eminence of the temporal bone that is blocking the path of the condyle when closing. An alternative strategy involves downfracture of a portion of the zygomatic arch to increase the relative size of the articular eminence.

Gap arthroplasty, often combined with eminectomy, involves recontouring the articular surfaces of the condyle and eminence to permit free unrestricted movement within the joint and relieve compression of the disk. It is indicated for ankylosis, inactive condylar hyperplasia, degenerative joint disease, and arthritis.

TMJ replacement is indicated for patients with ankylosis, degenerated joints, failed autogenous grafts, severe inflammatory disorders (rheumatoid arthritis), a failed Proplast-Teflon graft or alloplastic reconstruction, neoplasm, and condylar aplasia or hypoplasia. Prosthetic devices are classified as stock or custom-made. Several manufacturers design custom-made devices. Relative contraindications to the use of alloplastic reconstruction include uncontrolled systemic disease, advanced age, long-term steroid use, allergy, and infection. Autogenous grafting with costochondral grafts may also be used for reconstruction of the TMJ and has the advantage of biocompatibility and potential appositional remodeling. Unlike a costochondral graft, which depends on the integrity of local soft tissue to provide blood supply, a vascularized second metatarsal-phalangeal joint has recently been recommended as an alternative autogenous graft.

Condylotomy (bilateral subcondylar osteotomy) has remained a controversial surgical technique. It is designed to result in anteromedial displacement of the condyle in an effort to change the condyle-disk-fossa relationship, increase the superior and inferior joint space, and shorten the lateral pterygoid muscle, thereby reducing loading forces on the neurovascular retrodiscal tissues, allowing healing of the articular surfaces, and avoiding progression of the internal derangement. This rationale is somewhat analogous to a mandibular repositioning appliance, except that it is permanent. Condylotomy is considered superior to arthrotomy by virtue of its avoidance of surgical invasion of the TMJ capsule. Indications include painful disk displacement with or without reduction, subluxation, and osteoarthritis. Opponents of condylar osteotomy suggest that there is a lack of long-term follow-up detailing the corrected disk-condyle relationship, inevitable pathologic changes unaffected by condylotomy, and maintenance of an increased joint space. Other considerations include the need for maxillomandibular fixation for 6 weeks postoperatively, secondary skeletal changes that may result in malocclusion, and potential damage to the inferior alveolar nerve.

Pearls and Pitfalls

- Nonsurgical management is the first line of treatment for TMDs. Surgery is reserved for the patient with pain or dysfunction that is refractory to conservative measures.
- Diagnosis of TMDs will guide therapeutic modalities.
- Arthroscopy is a useful diagnostic and therapeutic tool.

SUGGESTED READINGS

Albury CD: Modified condylotomy for chronic nonreducing disk dislocations. Oral Surg Oral Med Oral Pathol 84:234-240, 1997.

Carlsson GE: Epidemiology and treatment need for temporomandibular disorders. J Orofac Pain 13:232-237, 1999.

Dierks EJ, Buehler MJ: Complete replacement of the temporomandibular joint with a microvascular transfer of the second metatarsal-phalangeal joint. Oral Maxillofac Surg Clin North Am 12:139-147, 2000.

MacIntosh RB: The use of autogenous tissues for temporomandibular joint reconstruction. J Oral Maxillofac Surg 58:63-69, 2000.

Mercuri LG: The use of alloplastic prostheses for temporomandibular joint reconstruction. J Oral Maxillofac Surg 58:70-75, 2000.

Seligman DA, Pullinger AG: The role of intercuspal occlusal relationships in temporomandibular disorders: A review. J Craniomandib Disord Facial Oral Pain 5:96-106, 1991.

Zide MJ, Carlton DM, Kent JN: Rheumatic diseases and related arthropathies I: Systemic findings, medical therapy, and peripheral joint surgery. Oral Surg Oral Med Oral Pathol 61:119, 1986.

Orthognathic Surgery

JOSEPH G. MCCARTHY
BARRY H. GRAYSON

The historic roots of plastic surgery lie in orthognathic or maxillofacial surgery. The origin of modern plastic surgery can be traced to the successful reconstruction of maxillomandibular wounds during World War I. Thereafter, surgeons extended their skills to congenital and acquired deformities of the mandible, maxilla, midface, and orbits. Manipulation of the jaws and teeth (i.e., orthognathic surgery) remains one of the most powerful tools in the plastic surgeon's armamentarium. It requires knowledge of dentofacial anatomy and collaboration with an orthodontist for establishing optimal occlusion.

Pathologic Anatomy

Mandible

Pathology of the mandible may manifest in many forms. Hypoplasia of the entire mandible is defined as *micrognathia*; a posteriorly positioned mandible is defined as *retrognathia*. Mandibular excess or forward projection relative to the maxilla is *prognathism*. Asymmetry of the mandible with a lateral crossbite is defined as *laterognathism* and is usually associated with condylar hyperplasia, hemifacial hypertrophy (along with enlarged limbs on the same side), or unilateral craniofacial microsomia.

Mandibular abnormalities may also be isolated to the chin. *Microgenia* describes chin deficiency, whereas *macrogenia* defines chin excess. In each of these conditions, the chin may be abnormal in the sagittal, vertical, or horizontal dimension.

The *dentoalveolar* component of the mandible can also be hypoplastic, especially after loss of the permanent dentition. There can be dental deficits and a crossbite relationship between the mandibular/maxillary components.

Maxilla

The maxilla can be deficient in the anteroposterior or sagittal dimension, as in patients with cleft palate (*maxillary hypoplasia*); *maxillary excess* is unusual but is seen in conditions such as venous malformation and lymphangioma.

Vertical maxillary excess, or the long-face syndrome, is more common. There is usually incisor show at rest with gingival exposure on smiling; the nasolabial angle is obtuse. With *vertical maxillary deficiency*, or the short-face syndrome, there is usually lack of incisor show both at rest and with smiling; the nasolabial angle is acute. Asymmetries of the maxilla in the vertical dimension are observed in conditions such as fibrous dysplasia.

Occlusal Relationships

The Angle classification (Fig. 1) defines the various types of dental relationships. In *class I* (*neutroclusion*), the maxillary and mandibular first molars are in an ideal relationship; that is, the mesiobuccal cusp of the maxillary first molar rests in the mesiobuccal groove of the mandibular first molar. In *class II* (*distoclusion*), the maxilla is positioned anterior to the mandible. More precisely, the mesiobuccal cusp of the maxillary first molar rests anterior to the mesiobuccal grove of the mandibular first molar. It is usually associated with retrognathia or micrognathia. In *class III* (*mesioclusion*), the mesiobuccal groove of the mandibular first molar sits anterior (mesial) to the mesiobuccal cusp of the maxillary first molar.

An *anterior crossbite* refers to an occlusal relationship in which the mandibular anterior teeth project forward of their maxillary counterparts. It is usually associated with mandibular prognathism (Fig. 2). An *overjet* refers to an overly anterior relationship of the maxillary incisal edges relative to their mandibular counterparts in the sagittal dimension (Fig. 3). An *overbite* is vertical overlap of the biting edges. An *anterior open bite* defines a lack of contact between the maxillary and mandibular anterior occlusion.

Abnormalities can also be seen in the buccolingual relationship of the teeth. *In neutral* or *centric*

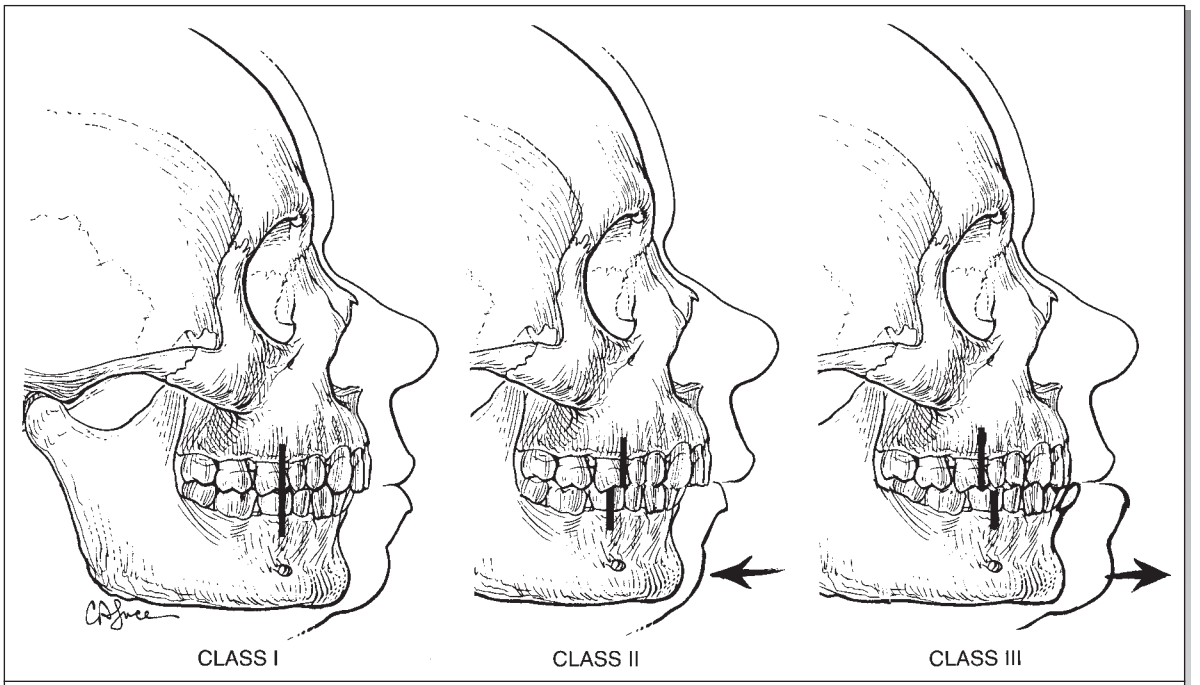

Figure 1 • Angle's classification of malocclusion. The classification is based on the mesiodistal (anteroposterior) relationships of the maxillary and mandibular first permanent molar teeth. The relationship between the position of the jaws and the facial profile is also illustrated. In class I (neutroclusion), the facial profile falls within the normal range. The maxillary and mandibular first molar teeth are in an ideal anteroposterior relation. The mesiobuccal cusp of the maxillary first molar is aligned correctly with the mesiobuccal groove of the mandibular first molar tooth. In class II (distoclusion), the mandible is retrognathic and the lower part of the face appears to be recessed. The mandibular first molar occupies a more posterior (distal) position than normal. In class III (mesioclusion), or the dental occlusion found in mandibular prognathism, the mesiobuccal groove of the mandibular first molar is mesial (anterior) to the mesiobuccal cusp of the maxillary first molar. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990, p 1193.)

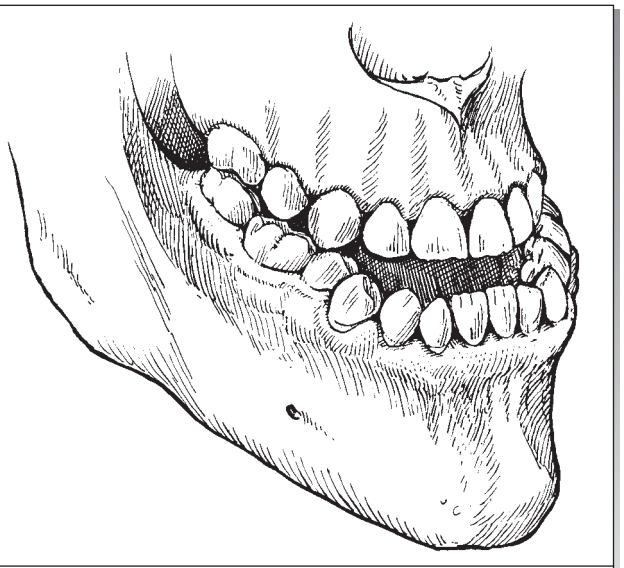

Figure 2 • Mandibular prognathism with an anterior open bite and anterior crossbite. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990, p 1228.)

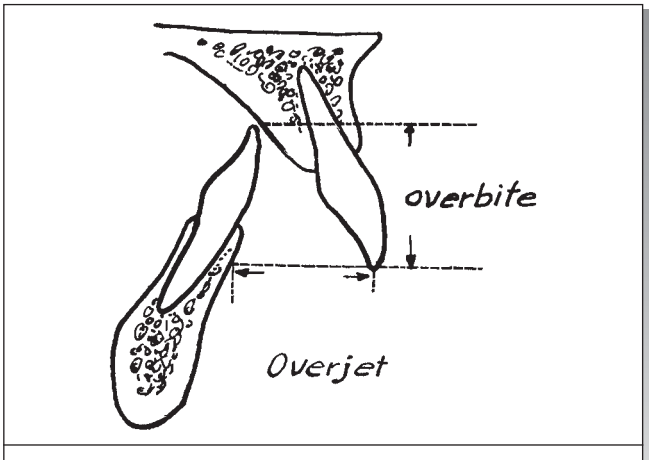

Figure 3 • Relationship of the incisal edges. Overbite is a vertical overlap of the biting edges. Overjet refers to the labiolingual relationship of the incisal edges. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990, p 1194.)

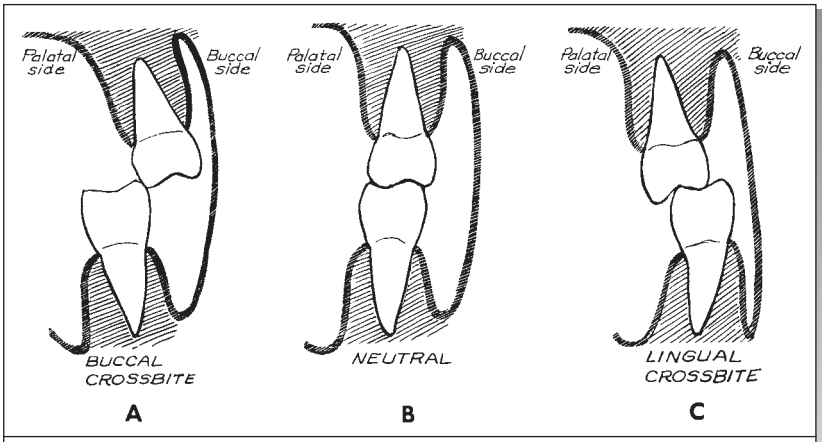

Figure 4 • Buccolingual relationships of the teeth. **A,** Buccal crossbite. Malocclusion is due to tilting of the maxillary tooth toward the cheek. **B,** Neutral (centric) occlusion, or the ideal relationship in which the buccal cusp of the upper tooth overlaps that of the lower tooth. **C,** Lingual crossbite caused by lingual displacement of the upper teeth in relation to the lower teeth. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990, p 1194.)

occlusion, the buccal cusp of the upper tooth overlaps that of the lower tooth (Fig. 4). In a *buccal crossbite*, the malocclusion is represented by tilting of the maxillary tooth toward the cheek, whereas in a *lingual crossbite*, the displacement of the upper tooth in relation to the lower tooth is toward the tongue.

Preoperative Evaluation

The preoperative surgical-orthodontic evaluation is critical and begins with a physical examination of the face both at rest and during animation to evaluate the quality of the soft tissue, the horizontal position of the oral commissure, lip-to-teeth relationships (incisor show at rest or gingival exposure on smiling), and functioning of the facial nerve. It should be determined whether the orbital, alar base, or occlusal planes are parallel or disparate. Various anthropometric measurements and facial planes are available for patient comparison (Fig. 5). Physical examination of the jaws should include mouth opening (mandibular excursion) and chin position. The intraoral dental examination, including oral hygiene, is also documented.

Photographic documentation is helpful and should include frontal, oblique, profile, submental, bird's eye, occlusal, and smiling views.

Dental impressions are taken and models made, except in isolated chin osteotomies. The models are placed in an articulator where simulated osteotomies/model surgery is performed, not only for diagnosis but also for preoperative planning and splint construction.

Cephalograms and *panoramic roentgenograms* are essential to record cephalometric linear and angular data in the vertical and horizontal dimensions (Table 1). The latter can be compared with age- and sex-matched normative standards, which, along with the physical examination, guide planning of the mandibular and maxillary manipulations. Periapical radiographs are occasionally obtained, especially when interdental osteotomies are planned.

Computed tomography (CT) scans are also helpful in planning the more complicated orthognathic surgical procedures. They can be reformatted as a Dentiscan in younger patients to document the position of unerupted teeth and tooth follicles.

Treatment

Nonsurgical Techniques

Before reconstruction, dental hygiene must be established. Dental extractions may be indicated to allow extrusion of the permanent dentition or to facilitate orthodontic movements.

The role of the orthodontist is critical in documenting the pathology and recommending dental extractions. Preoperative orthodontic therapy is usually indicated to coordinate maxillary and mandibular arch width, to level the occlusal plane, to eliminate occlusal interference, to reposition incisors to optimize postsurgical lip position for creation of an anterior crossbite in anticipation of maxillary advancement, and to create interdental spaces for segmental osteotomies. It is not uncommon for the period of preoperative orthodontics to extend 6 months or more.

The orthodontist is also important in jointly planning the procedure with the surgeon and in preparing an intraoperative occlusal split. Moreover, mock

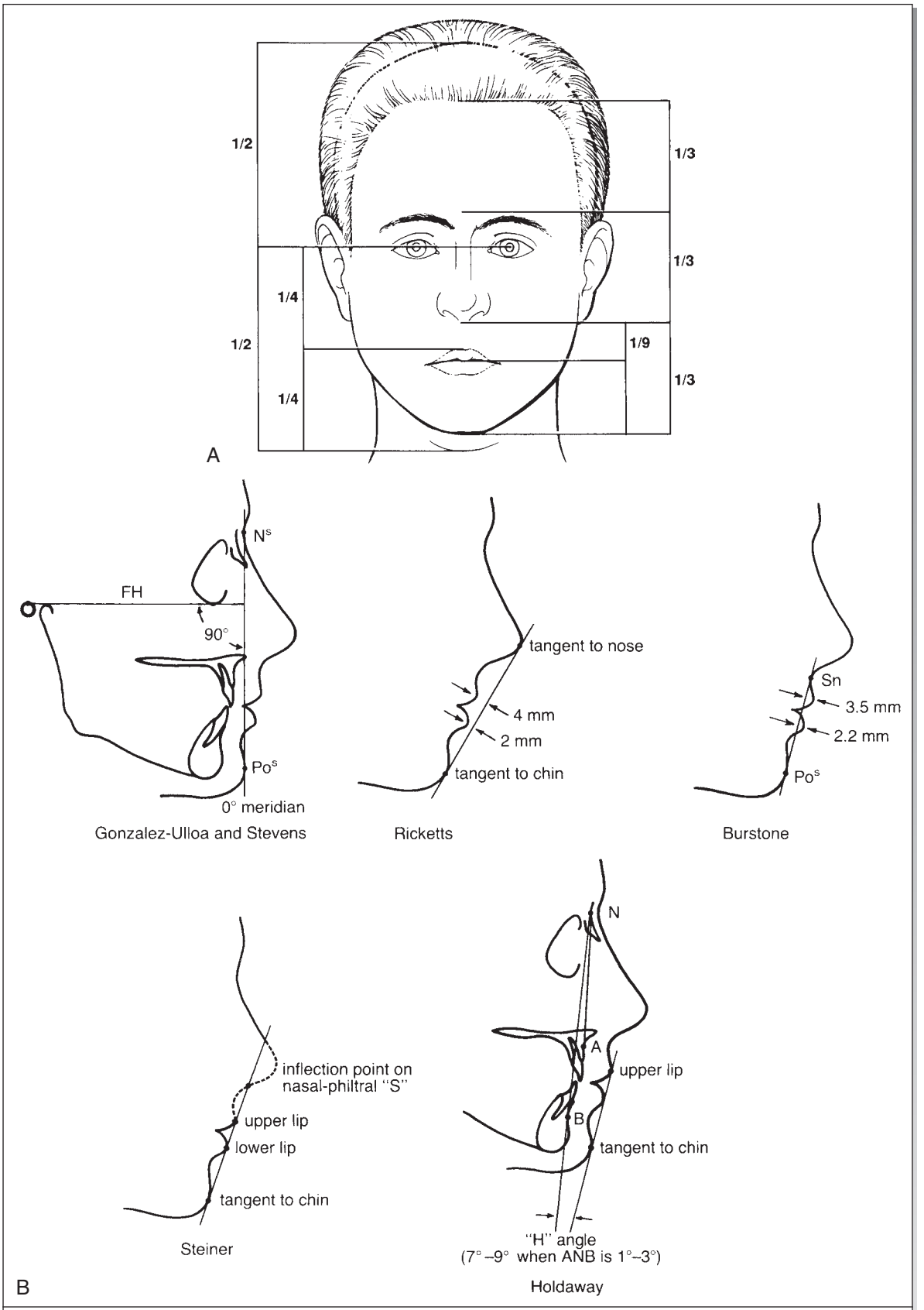

Figure 5 • A, Divisions of the face. The face is divided into thirds by the trichion, nasion, subnasale, and menton. The distance between the vertex of the cranial vault and the midpupillary point defines the upper half, and that between the latter and the hyoid defines the lower half. Additional subdivisions are designated. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, WB Saunders, 1990, p 1191.) **B,** Various "ideal" facial planes. (From McCarthy JG, Ruff G: The chin. Clin Plast Surg 15:125, 1988.)

TABLE 1 Control Cephalometric Data Sheet, Institute of Reconstructive Plastic Surgery, New York University Medical Center

NAME OF PATIENT	DATE OF CEPHALOGRAM									
Age: 16				**Sex: Female**						
Variable	Mea.	–20%	–15%	–10%	–5%	Norm	+5%	+10%	+15%	+20%
Height (cm)	—	130.4	138.6	146.7	154.9	163.0	171.2	179.3	187.5	195.6
Midface Height										
ANS-N (mm)	—	44.6	47.3	50.1	52.9	55.7	58.5	61.3	64.1	66.8
SE-PNS (mm)	—	40.9	43.4	46.0	48.5	51.1	53.7	56.2	58.8	61.3
ANS-SD (mm)	—	14.5	15.4	16.3	17.2	18.1	19.0	19.9	20.8	21.7
SN-ANS-PNS (deg)	—					8.0				
Total Face Height										
Me-N (mm)	—	98.6	104.7	110.9	117.0	123.2	129.4	135.5	141.7	147.8
Me-ANS (mm)	—	55.4	58.9	62.4	65.8	69.3	72.8	76.2	79.7	83.2
ANS-UIE (mm)	—	24.1	25.6	27.1	28.6	30.1	31.6	33.1	34.6	36.1
Me-LIE (mm)	—	33.8	36.0	38.1	40.2	42.3	44.4	46.5	48.6	50.8
Me-ID (mm)	—	25.0	26.6	28.2	29.7	31.3	32.9	34.4	36.0	37.6
Ar-Go (mm)	—	39.7	42.2	44.6	47.1	49.6	52.1	54.6	57.0	59.5
SN/MP (deg)	—					31.2				
6 cusp MP (mm)	—	26.1	27.7	29.3	31.0	32.6	34.2	35.9	37.5	39.1
Dental										
⊥ SN (deg)	—					103.1				
T-MP (deg)	—					92.1				
⊥ T (deg)	—					133.6				
Midface Horizontal										
S-N (mm)	—	61.5	65.4	69.2	73.1	76.9	80.7	84.6	88.4	92.3
SNO (deg)	—	42.8	45.5	48.2	50.8	53.5	56.2	58.9	61.5	64.2
O ⊥ NA (mm)	—	13.1	13.9	14.8	15.6	16.4	17.2	18.0	18.9	19.7
ANS-PNS (mm)	—	45.6	48.5	51.3	54.2	57.0	59.9	62.7	65.6	68.4
PNS-A (mm)	—	41.4	44.0	46.6	49.2	51.8	54.4	57.0	59.6	62.2
PNS-UIE (mm)	—	50.2	53.4	56.5	59.7	62.8	65.9	69.1	72.2	75.4
SNA (deg)	—					81.8				
Lower Face Horizontal										
Ar-Pg (mm)	—	92.2	97.9	103.7	109.4	115.2	121.0	126.7	132.5	138.2
Ar-B (mm)	—	84.5	89.8	95.0	100.3	105.6	110.9	116.2	121.4	126.7
Ar-LIE (mm)	—	79.4	84.3	89.3	94.2	99.2	104.2	109.1	114.1	119.0
Go-Pg (mm)	—	65.2	69.3	73.4	77.4	81.5	85.6	89.7	93.7	97.8
Go-B (mm)	—	61.4	65.2	69.0	72.9	76.7	80.5	84.4	88.2	92.0
Go-LIE (mm)	—	64.3	68.3	72.4	76.4	80.4	84.4	88.4	92.5	96.5
AB-PB (mm)	—	28.5	30.0	32.0	33.8	35.6	37.4	39.2	40.9	42.7
SNB (deg)	—					79.2				
ANB (deg)	—					2.6				
SN-Pg (deg)	—					80.2				
Ar-Go-Me (deg)	—					122.2				

A, A point; ANS, anterior nasal spine; Ar, articulare; Ba, basion; B, B point; Gn, gnathion; Go, gonion; ID, infradentale; LIE, lower incisor edge; Me, menton; MP, mandibular plane; N, nasion; O, orbitale; Pg, pogonion; PNS, posterior nasal spine; SD, supradentale; SE, sphenoethmoidal; SN, sella-nasion plane; UIE, upper incisor edge.

From McCarthy JG, Kawamoto HK, Grayson BH, et al: Surgery of the jaws. In McCarthy JG (ed): Plastic Surgery. Philadelphia, WB Saunders, 1990, p. 1199.

surgery can be performed on the photographic records, the cephalometric tracings, and the dental models. This can be done on any of the commercially available computerized planning programs. For correction of more complex three-dimensional asymmetric deformities, computer-aided surgical planning with cephalometrics and CT-based surgical simulation is possible.

FIXATION. To allow consolidation at the osteotomy sites, maxillomandibular fixation (MMF) was traditionally maintained for a period of 8 weeks, but

often with significant difficulty with feeding and communication. The technique is still used in patients undergoing vertical osteotomy of the mandible for correction of prognathism and in complex two-jaw reconstructions in which bone grafts are used. MMF, however, has been largely replaced by rigid skeletal fixation with plates and screws to allow immediate movement of the mandible, alimentation, and normal communication. With sagittal split osteotomies, lag screws are used to appose the ramal segments after the occlusion has been established and the condyles are anatomically seated in the glenoid fossae.

Surgical Techniques

GENIOPLASTY. Horizontal osteotomy of the anteroinferior border of the mandible (genioplasty) is a workhorse osteotomy for changing chin projection/profile in all dimensions (Fig. 6). It is generally used to advance the chin for correction of microgenia in the setting of normal occlusion. Moreover, a segment of bone can be removed and the vertical dimension of the chin reduced (Fig. 7). Alternatively, an interposition bone graft or alloplastic material can be inserted at the osteotomy site to increase the vertical dimension of the chin (Fig. 8). In a patient with chin asymmetry, an osteotomy can be performed and the chin point moved transversely to the midline (Fig. 9).

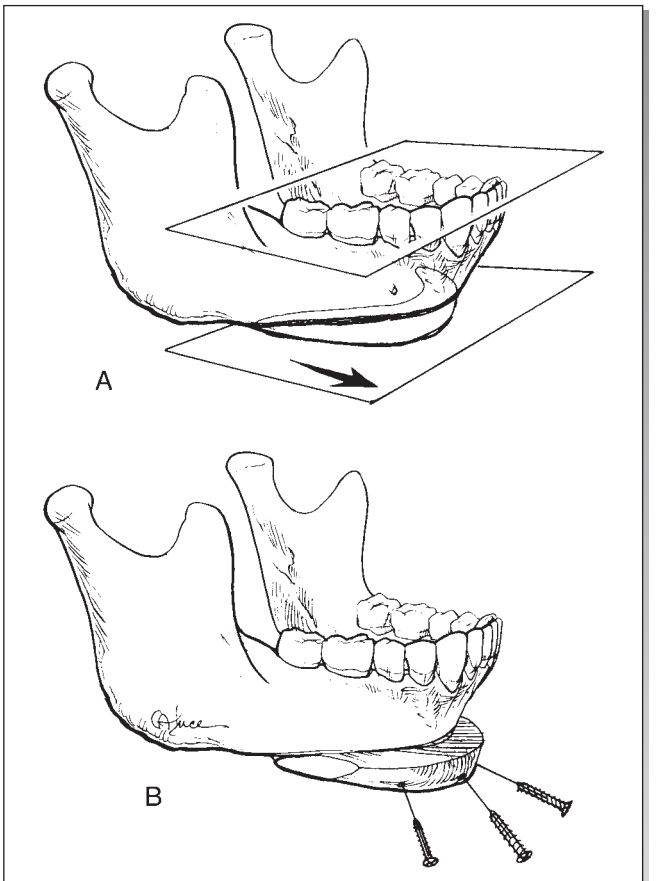

Figure 6 • Sliding genioplasty. **A,** The osteotomy is made in a plane parallel to the occlusal surface; there is no change in the vertical dimension. **B,** Lag screws provide fixation. The poles of the advanced segment have been contoured. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990, p 1315.)

SEGMENTAL DENTOALVEOLAR OSTEOTOMIES. These osteotomies interrupt the dental arch and maxillomandibular relationships. If a segment of dentoalveolar bone is removed, the anteroposterior dimension of the dental arch is reduced, and if a bone graft is placed at the osteotomy site, the dimension is increased, often with a residual interdental space. These procedures are especially indicated when osteotomy/ostectomy can be performed in an existing interdental space.

Although an anterior open bite deformity can be corrected by anterior segmental maxillary or mandibular dentoalveolar osteotomy, the preferred technique is vertical impaction of the posterior maxillary dentoalveolar segment (Fig. 10).

BILATERAL SAGITTAL SPLIT OSTEOTOMY. A bilateral sagittal split osteotomy extends across the lingual aspect of the ramus above the lingula or entrance of the inferior alveolar nerve (Fig. 11). It continues along the oblique line and is extended through the buccal cortex to the inferior border of the mandible. The ramus is split along the sagittal plane, with a wide bony surface left for eventual bony union. The osteotomies, which are performed through intraoral incisions, are versatile and can be used to advance the mandible by moving the tooth-bearing segment forward in patients with micrognathia or moving the segment posteriorly in patients with mandibular prognathism and class III malocclusion. It also permits movement of the tooth-bearing segment in the vertical dimension (three dimensionally), as in patients with craniofacial microsomia. A bilateral sagittal split osteotomy is often combined with a LeFort I osteotomy in "two-jaw" surgery. Although it is a versatile osteotomy, it carries a significant risk of injury to the inferior alveolar nerve and problems associated with improper seating of the condyle in the glenoid fossa.

Rigid skeletal fixation (usually lag screws placed between the ramal segments) usually obviates the need for postoperative MMF.

VERTICAL/OBLIQUE OSTEOTOMIES. Vertical/oblique osteotomies, also performed through intraoral incisions, have traditionally been used for correction of mandibular prognathism (Fig. 12). Bilateral osteotomies extend from the sigmoid notch to the region of the angle of the mandible. The tooth-bearing segment is moved posteriorly to establish normal occlusion. The condylar segments overlap the tooth-bearing segments to provide a site for bony

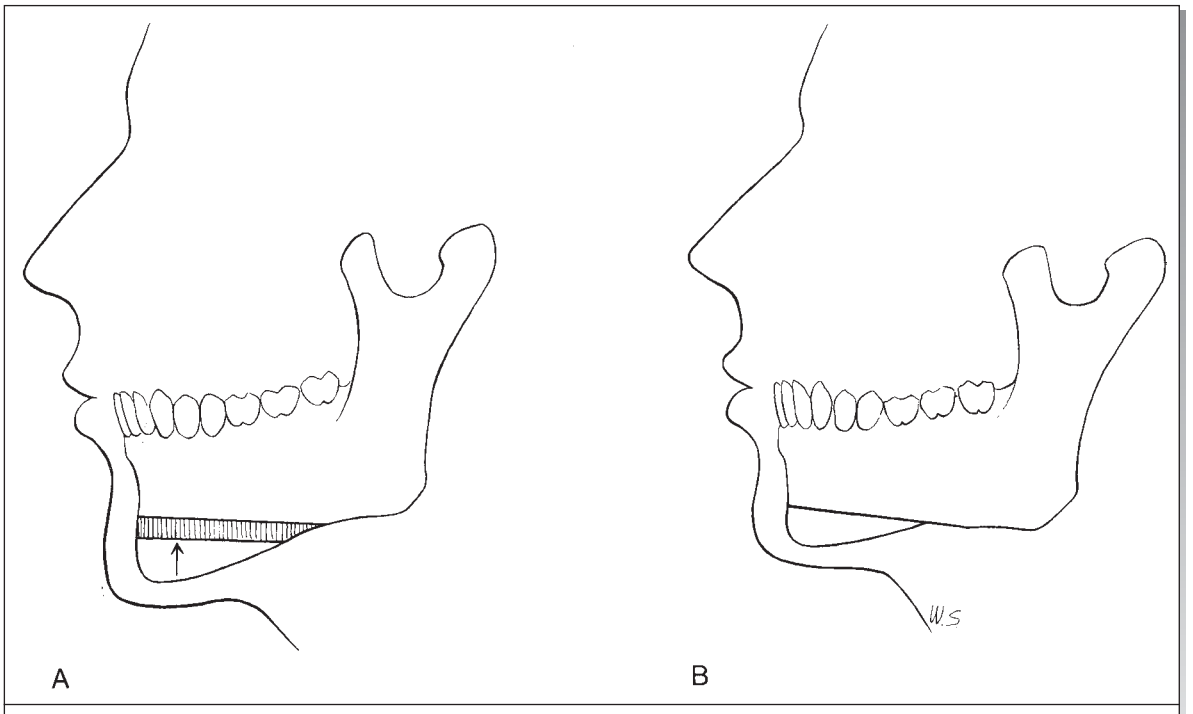

Figure 7 • Vertical shortening of the chin by removal of a wedge of bone. **A,** Area of bone resection. **B,** The lower segment is displaced in a vertical (or anterior) direction and secured by rigid skeletal fixation. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990, p 1320.)

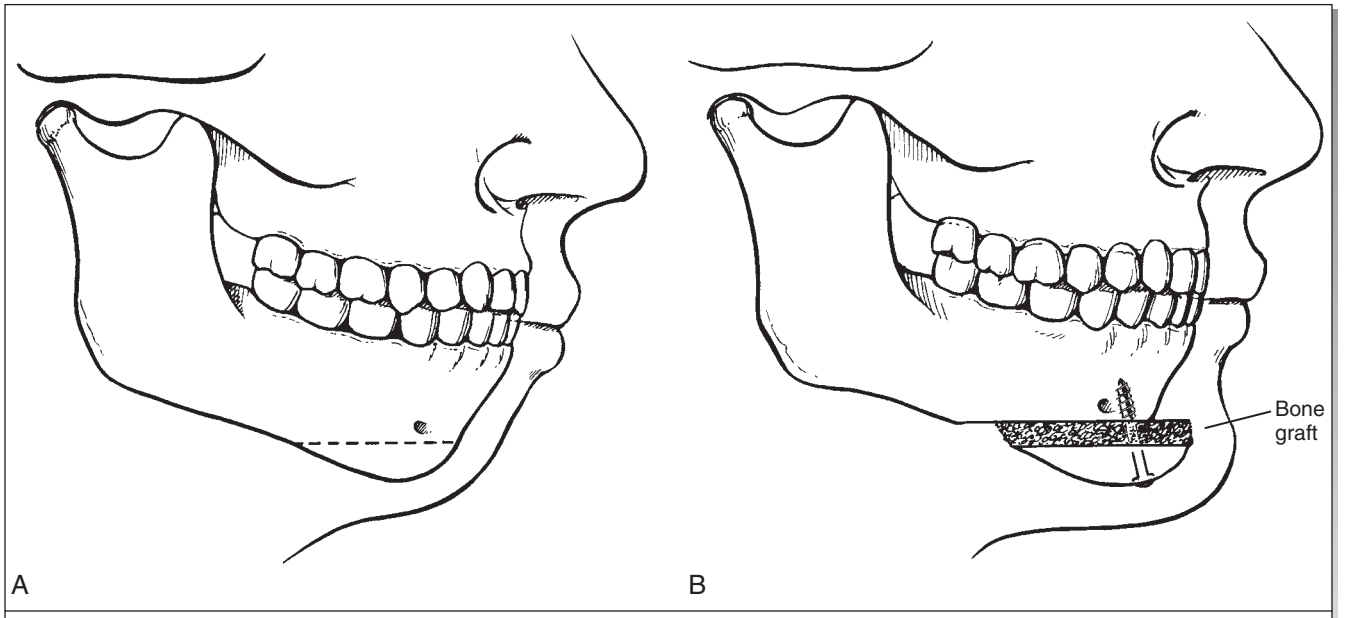

Figure 8 • The sandwich procedure. Horizontal osteotomy is combined with an interposition bone graft or piece of hydroxyapatite for vertical elongation of the anterior portion of the mandible and for increasing the prominence of the chin. **A,** The line of osteotomy. **B,** The bone graft is interposed, in sandwich fashion, between the genioplasty segment and the body of the mandible and is secured with lag screws.

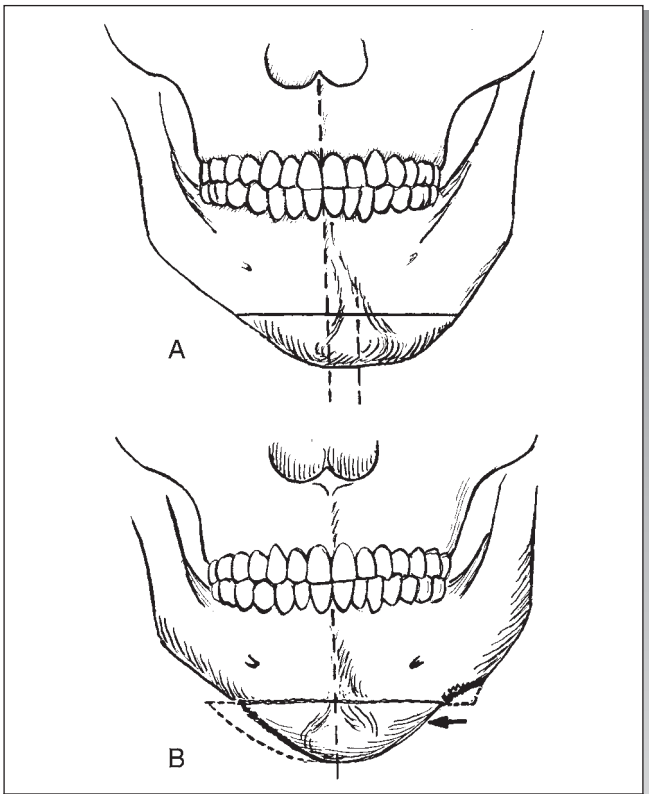

Figure 9 • Correction of lateral deviation of the chin by horizontal osteotomy. **A,** The vertical (midsagittal) *dotted line* shows the amount of deviation of the chin to the left. **B,** After the horizontal osteotomy, the genioplasty segment is displaced toward the right. The *broken line* outlines the protruding bone, which is resected to obtain a smooth contour. (From Converse JM, Wood-Smith D: Horizontal osteotomy of the mandible. Plast Reconstr Surg 34:464, 1974.)

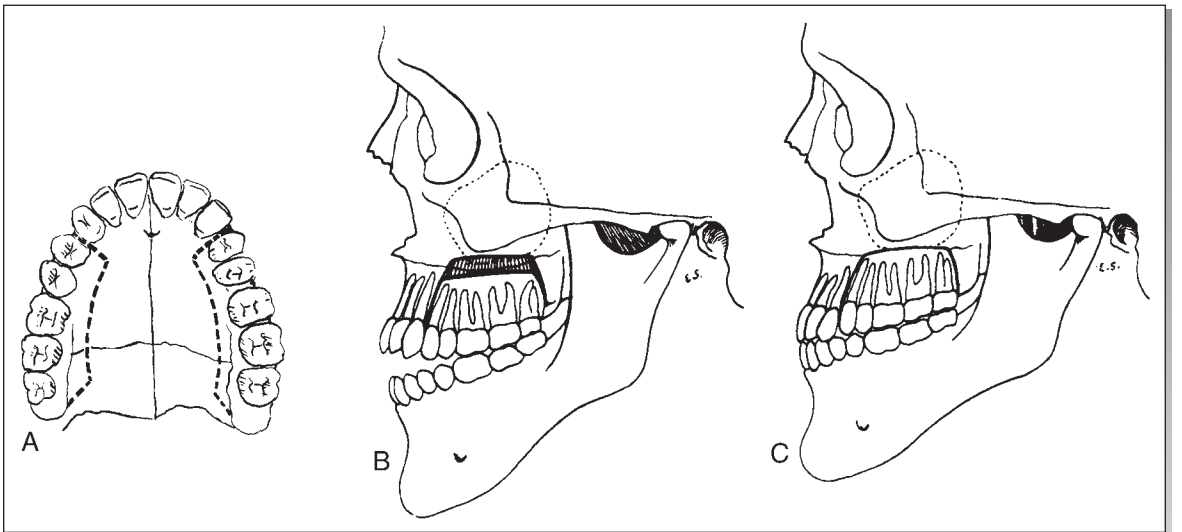

Figure 10 • Schuchardt procedure for closure of an anterior open bite. **A,** In the first stage, bilateral osteotomies of the alveolar processes are performed as outlined. The palatal flap is reapplied to preserve the vascular supply. **B,** Six weeks later, a buccal mucoperiosteal flap is raised, and the lateral aspect of the maxilla is exposed. A segment of the maxilla above the apices of the teeth is resected as indicated by the *shaded area*. **C,** Correction of the open bite achieved after vertical repositioning of the posterior maxillary segments; rigid skeletal fixation is applied. The technique can also be performed without a palatal flap in one stage. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990, p 1358.)

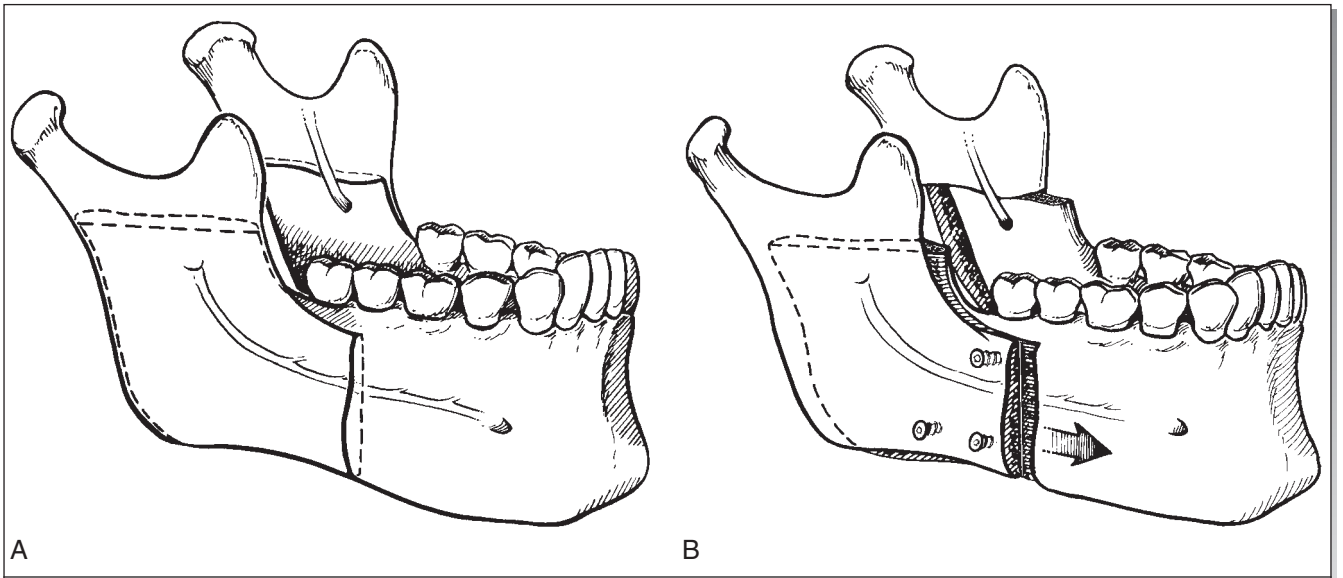

Figure 11 • Bilateral sagittal split osteotomy. **A,** Note the line of osteotomy across the lingual cortex of the ramus, oblique line, and buccal cortex. **B,** Insertion of lag screws after drill holes are made through a percutaneous trocar to secure advancement of the tooth-bearing fragment.

union. This osteotomy can also be used in patients with mandibular deficiency. The tooth-bearing segment is moved anteriorly and interpositional bone grafts placed and rigidly fixated in the resulting defect at the osteotomy site.

The technique suffers the disadvantage that MMF is generally required to permit consolidation at the osteotomy site.

MANDIBULAR DISTRACTION. In recent years, distraction osteogenesis has become more widely used for correction of mandibular deficiencies and asymmetry. The technique includes an osteotomy across which a distraction device is placed and secured with screws or pins (Fig. 13). The *vector* or orientation of the device relative to the maxillary occlusal plane is critical in planning the direction of mandibular movement. For example, a vertical vector is preferred to lengthen the deficient mandibular ramus, whereas a horizontal vector is used to correct deficiency of the body. After a *latency period* of 5 days, *activation* of the device is initiated

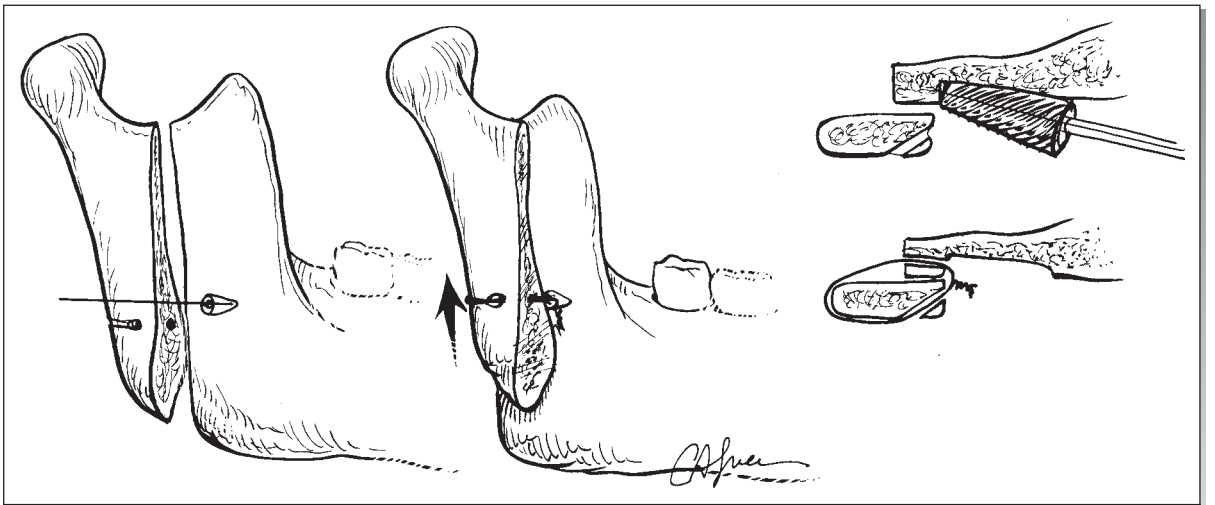

Figure 12 • Setback of the tooth-bearing segment with overlapping of the ramal fragments. Contouring of the condylar fragment may be indicated, and removal of the attached medial pterygoid is necessary to ensure bone-to-bone apposition. Note the technique of interosseous wire fixation of the ramal fragment to seat the condyle in the glenoid fossa. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990, p1238.)

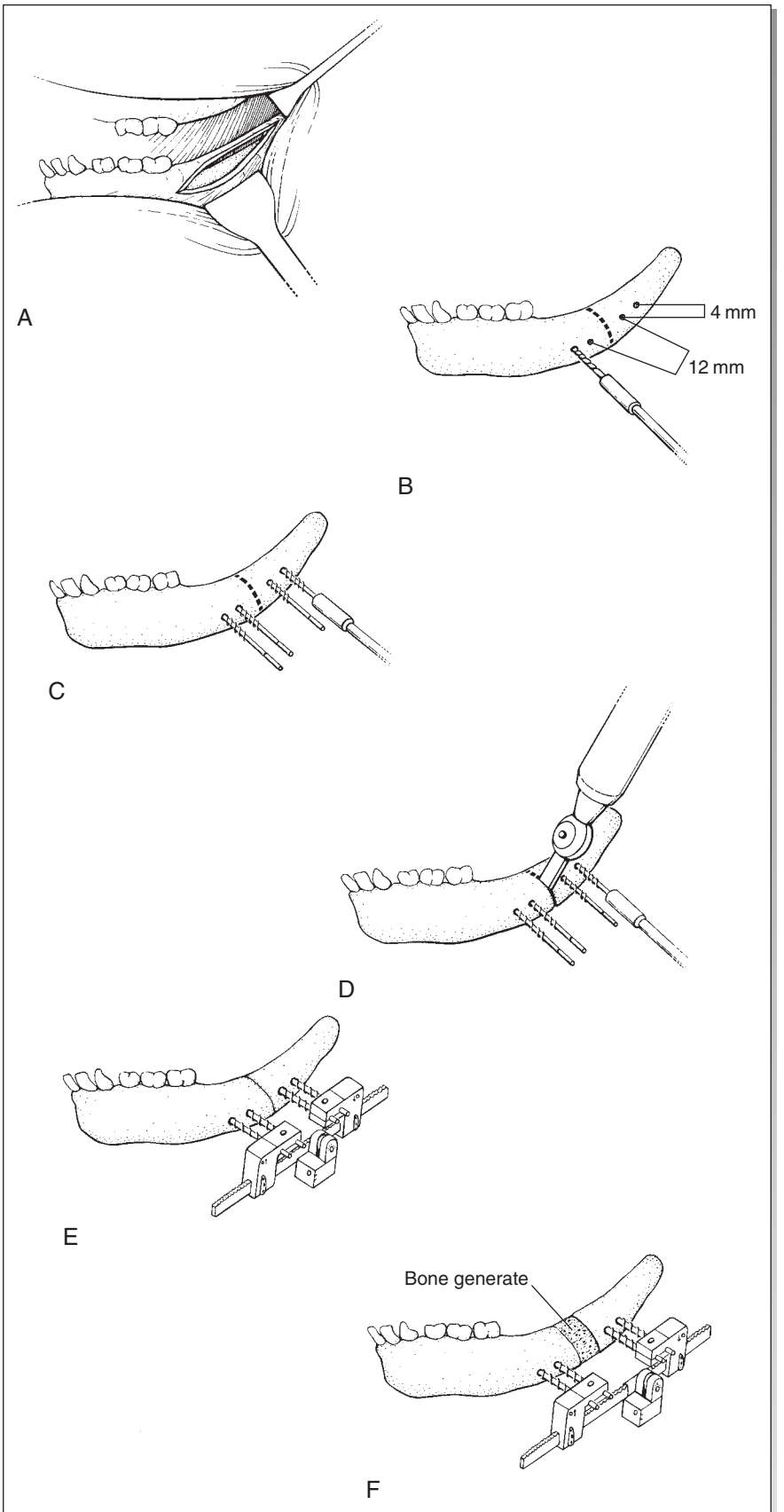

Figure 13 • Technique of mandibular distraction. **A,** Intraoral incision along the ramal remnant. **B,** Proposed line of osteotomy (*interrupted line*) with drill holes. **C,** Insertion of the fourth pin. **D,** Buccal corticotomy with a power saw. **E,** Application of a distraction device on the four pins. **F,** Generation of bone after activation of the device. (From Mathes SJ, Hentz R: Plastic Surgery, 2nd ed. Philadelphia, Elsevier, in press.)

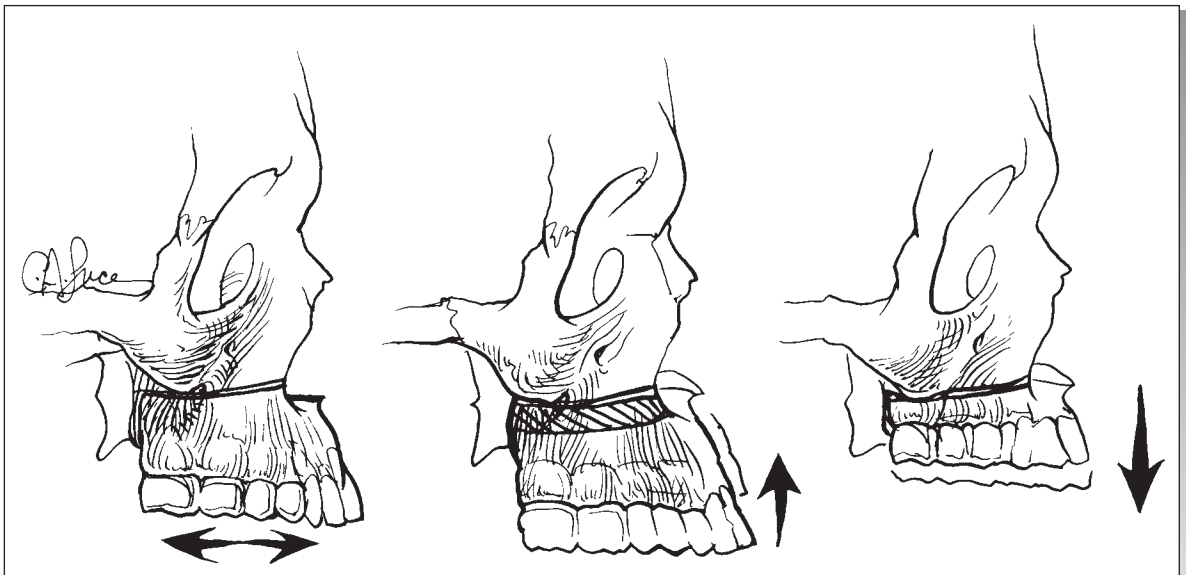

Figure 14 • Versatility of the LeFort I osteotomy. Note the multiple directions in which the tooth-bearing segment can be mobilized. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990, p 1369.)

at the rate of 1 mm/day. After the planned occlusion and mandibular position are achieved, activation is discontinued and the devices are left in place for an additional 8 weeks to allow *consolidation* of the bony generate. The technique has found particular application in patients with unilateral craniofacial microsomia and bilateral mandibular deficiency, especially younger, growing patients with respiratory insufficiency and sleep apnea. It has eliminated the need for MMF, bone grafts, blood transfusions, and prolonged operative times and hospital stays. Because the skeletal elongation is performed gradually (1 mm/day) and because there is also associated elongation of the attached soft tissue, the relapse rate is significantly reduced in comparison to traditional orthognathic surgical techniques.

LEFORT I OSTEOTOMY. A LeFort I osteotomy may be the most useful osteotomy for reconstruction of both simple and complex jaw deformities (Fig. 14). The osteotomy extends across the anterolateral aspect of the maxilla to and through the pterygomaxillary junction. Osteotomies are made through the medial and posterior walls of the maxillary sinus and the base of the septum. The tooth-bearing maxillary (LeFort I) segment, once liberated, can be moved in an anterior direction as in a cleft palate patient, superiorly after removal of a maxillary segment as in a patient with long-face syndrome, inferiorly with interposition of a bone graft as in a patient with short-face syndrome, or in a complex multidimensional movement as in a patient with unilateral craniofacial microsomia. LeFort I osteotomy is often combined with mandibular osteotomy or distraction (two-jaw surgery) (Fig. 15).

The use of rigid skeletal fixation (plates and screws) obviates the need for postoperative MMF.

LEFORT III OSTEOTOMY. A LeFort III osteotomy is performed through a coronal incision with or without eyelid incisions. The osteotomy is made through the nasofrontal junction, medial orbital wall, orbital floor, lateral orbital wall, zygomatic arch, and lateral maxilla to and through the pterygomaxillary fissure (Fig. 16). The septum is sectioned and temporary MMF is established while rigid skeletal fixation is achieved at the nasofrontal junction, lateral orbital regions, and zygomas. Interpositional bone grafts are placed at the buttress locations.

The midface segment can be advanced to expand orbital volume (and correct exorbitism), increase zygomatic projection, and advance the maxillary dentoalveolus (and correct a class III malocclusion).

MAXILLARY-MIDFACE DISTRACTION. In recent years, the distraction technique has become an alternative to classic midface advancement osteotomies and bone grafts. It is the preferred technique for midface advancement in patients whose growth is not completed. Internal and external devices have been used (Fig. 17). The LeFort III osteotomy is performed as described earlier, the devices are applied, and after a latency period of 5 days, activation is initiated at the rate of 1 mm/day until the desired occlusion and craniofacial form are achieved.

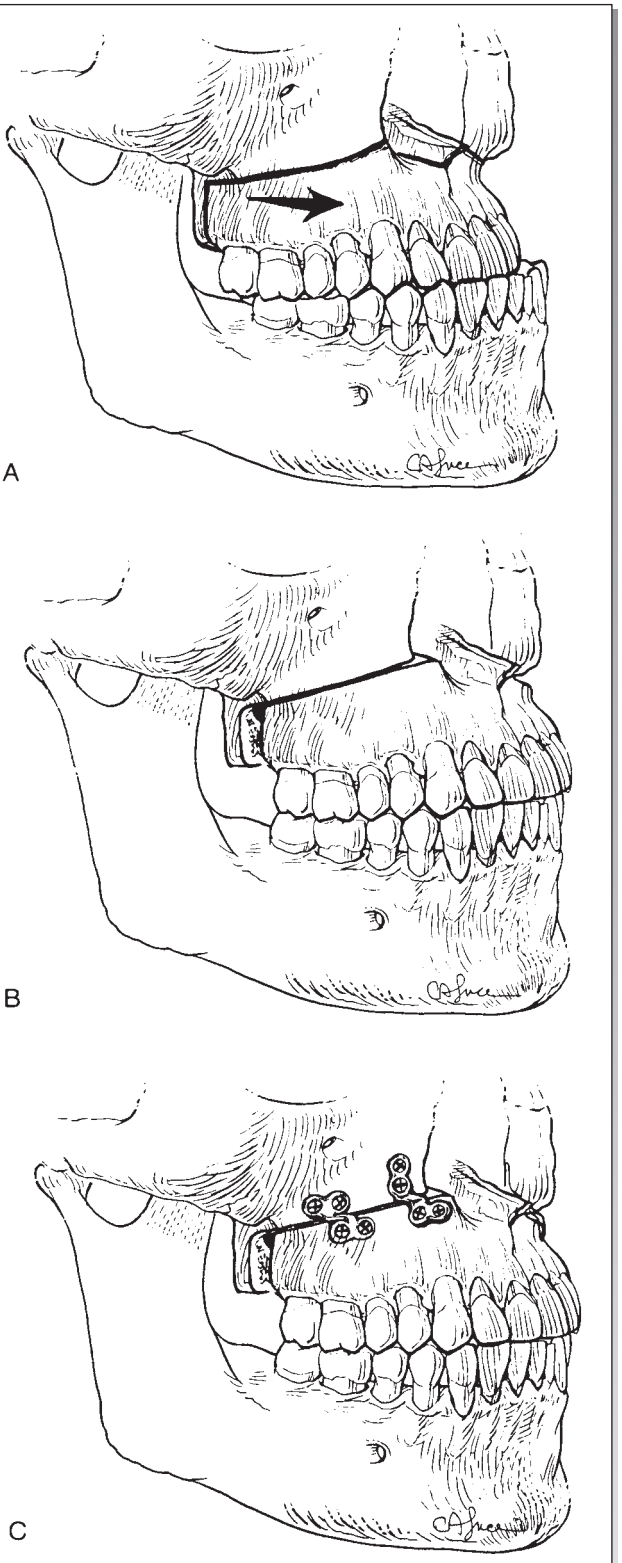

Figure 15 • LeFort I advancement osteotomy. **A,** Lines of osteotomy and direction of the planned movement. **B,** Anterior mobilization of the maxillary segment with restoration of the occlusion. **C,** Miniplate fixation. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990, p 1370.)

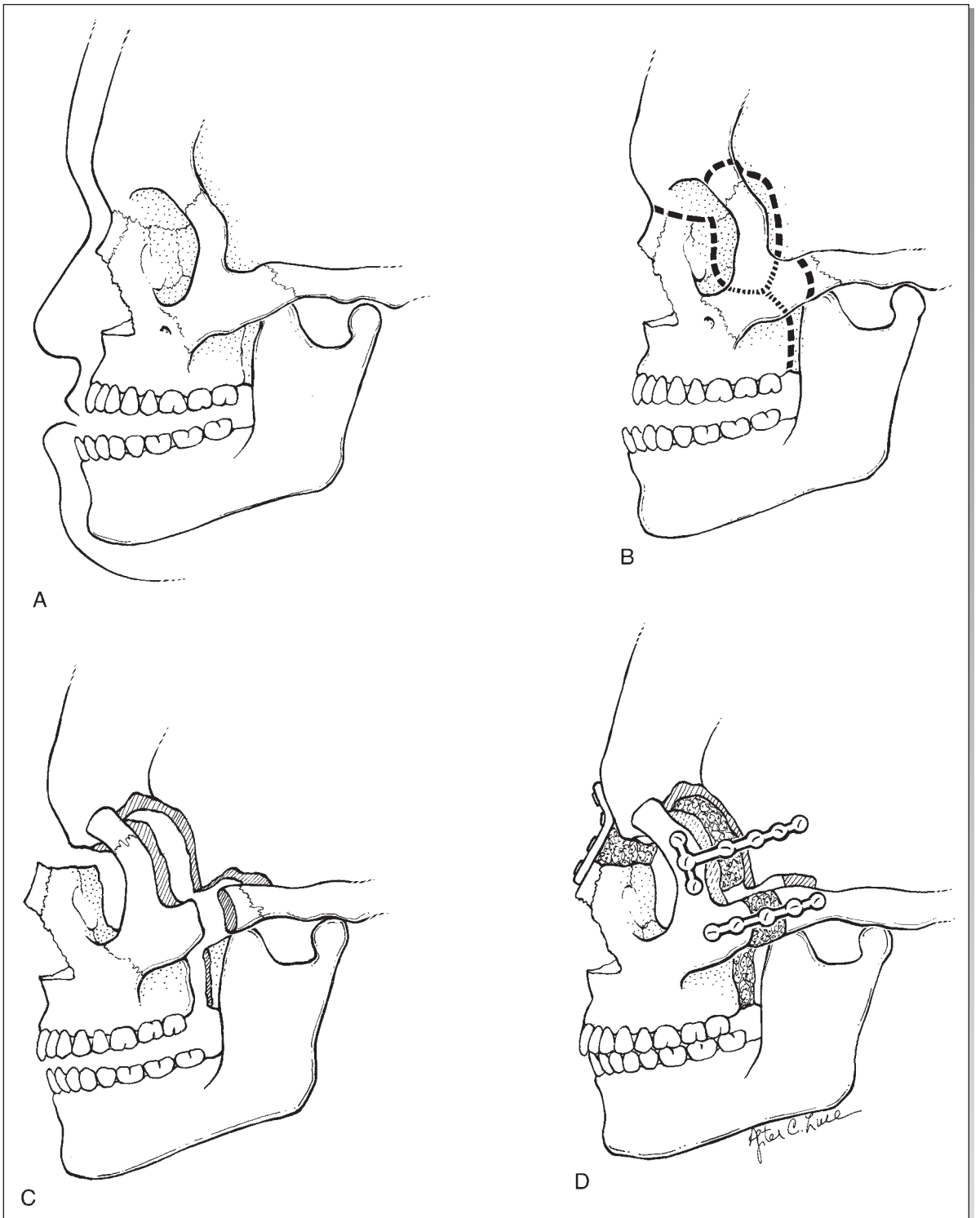

Figure 16 • Subcranial LeFort III advancement osteotomy with bone grafting. **A,** Lateral view demonstrating the pathology. Note the midface hypoplasia, anterior crossbite, and anterior open bite. **B,** Lines of osteotomy: across the nasofrontal junction and through the medial orbital wall, floor of the orbit, full thickness of the lateral orbital wall, zygomatic arch and retromaxillary area, and pterygomaxillary junction. **C,** Mobilization of the osteotomized segment and the resulting skeletal defects. **D,** Autogenous bone grafts are placed in the nasofrontal junction, lateral orbital wall, zygomatic arch, and pterygomaxillary fissure defects. Fixation is obtained by miniplates. (Adapted from Luce C in Mathes SJ, Hentz R: Plastic Surgery, 2nd ed. Philadelphia, Elsevier, in press.)

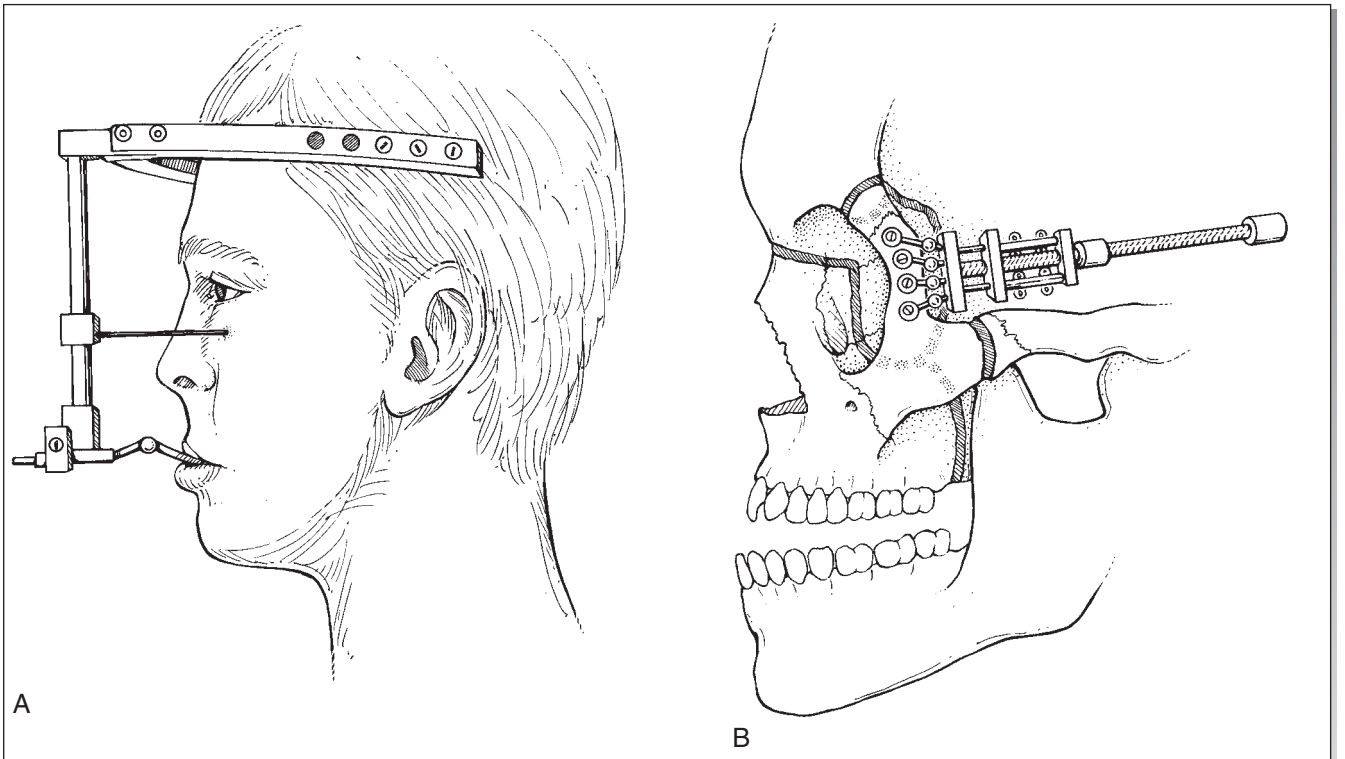

Figure 17 • A, The external halo midface distraction device. The halo is secured to the cranium with large screws, to the infraorbital-zygomatic area with wires to skeletal screws, and to the maxillary occlusion with wires to a dental splint. **B,** A buried midface distraction device. The lines of osteotomy (subcranial LeFort III) are designated. The device is secured with multiple screws attached to the device on either side of the osteotomy. The activating component is passed through a separate incision in the scalp to allow easy access. (From Mathes SJ, Hentz RL: Plastic Surgery, 2nd ed. Philadelphia, Elsevier, in press.)

Pearls and Pitfalls

- The patient must understand that he or she is embarking on a treatment program involving not only a surgical procedure but also prolonged periods of preoperative and postoperative orthodontic therapy.
- Detailed preoperative planning by the orthodontist and surgeon is critical for the success of the treatment program.
- An anesthesiologist experienced in orthognathic surgery is required for maintaining the airway.
- Bony union at the osteotomy sites is not possible without secure fixation techniques.

SUGGESTED READINGS

Bell WH, Proffit WR, White RP: Surgical Correction of Dentofacial Deformities. Philadelphia, WB Saunders, 1980.

McCarthy JG: Distraction of the Craniofacial Skeleton. New York, Springer-Verlag, 1999.

McCarthy JG, Kawamoto HK, Grayson BH, et al: Surgery of the jaws. In McCarthy JG (ed): Plastic Surgery. Philadelphia, WB Saunders, 1990.

The Tongue and Oral Cavity

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FIRAS R. KARMO

Cancer of the tongue and oral cavity is relatively uncommon, but it can have a major impact on quality of life. It often affects self-perception, interaction with others, and the basic functions of speech, deglutition, and breathing. It is vitally important for the surgeon to detect the tumor early, determine the extent of lymphatic involvement, and establish a safe and logical treatment plan. This chapter provides an overview of the anatomy of the oral cavity, the varieties of tumors in this site, their metastatic patterns of spread, ablative techniques, adjunctive treatments, and the potential need for reconstruction. Neck anatomy, neck dissection techniques, and adjunctive treatments are discussed in the chapter "Principles of Head and Neck Oncology".

Etiopathogenesis

Between 30% and 40% of the 60,000 head and neck malignancies in the United States involve the oral cavity. Approximately 9500 people die annually of cancer of the oral cavity; about 45% of these deaths are due to local and regional disease, with the remainder being due to metastatic disease. Many of these deaths are preventable, as early detection and appropriate treatment are associated with a high cure rate.

Alcohol and tobacco (which appear to act synergistically in these patients) act as local irritants of the mucosa and are associated with most cancers of the lips, oral cavity, and oropharynx in the United States. Ethanol is believed to serve as a direct mucosal irritant (promoter or cocarcinogen), whereas tobacco carcinogens play the initiator role. Areas of anatomic dependency—the gingivobuccal and gingivolingual sulci and the floor of the mouth—concentrate these substances, a finding that may explain the higher frequency of tumors in these areas through a prolonged direct contact mechanism. Smokers have an increased relative risk of oral cancer of approximately six times that of nonsmokers. The combination of alcohol and tobacco consumption synergistically (not simply additively) increases the relative risk of oral cavity malignancy to 15 times that of individuals with neither habit. Other factors that can contribute to oral malignancy include poor oral hygiene, poorly fitting dental appliances, viral infection, chronic mouthwash use, nutritional deficiency, and occupational exposures.

It has also been suggested that oral malignancies develop as a result of immune incompetence or failure of host defense mechanisms. Anecdotal evidence for such pathogenesis is found in relatively young patients (<30 years of age) and elderly women (>70 years of age) who have never smoked or ingested alcohol, yet in whom the same tumors develop as in those who have abused these substances. It is also commonly known that individuals who have one cancer of the aerodigestive tract (head and neck, lung, esophagus) are at greater risk for a second cancer, particularly if they continue the social habits that produced the first one.

Pathologic Anatomy (Fig. 1)

Premalignant Lesions

Leukoplakia, or white patch, is frequently a cause for great concern; however, it undergoes malignant change in only 5% of cases. Once observed, leukoplakia should be monitored by both the clinician and the patient, but it should undergo biopsy only if it changes in character, seems to be invasive, or becomes painful. Biopsy usually shows epithelial hyperplasia, but lesions with dysplasia tend to have a high rate of malignant transformation.

Erythroplakia, in contrast, is less common and much more aggressive, with a propensity for the development of malignancy in more than 90% of patients. Carcinoma in situ is often found at the

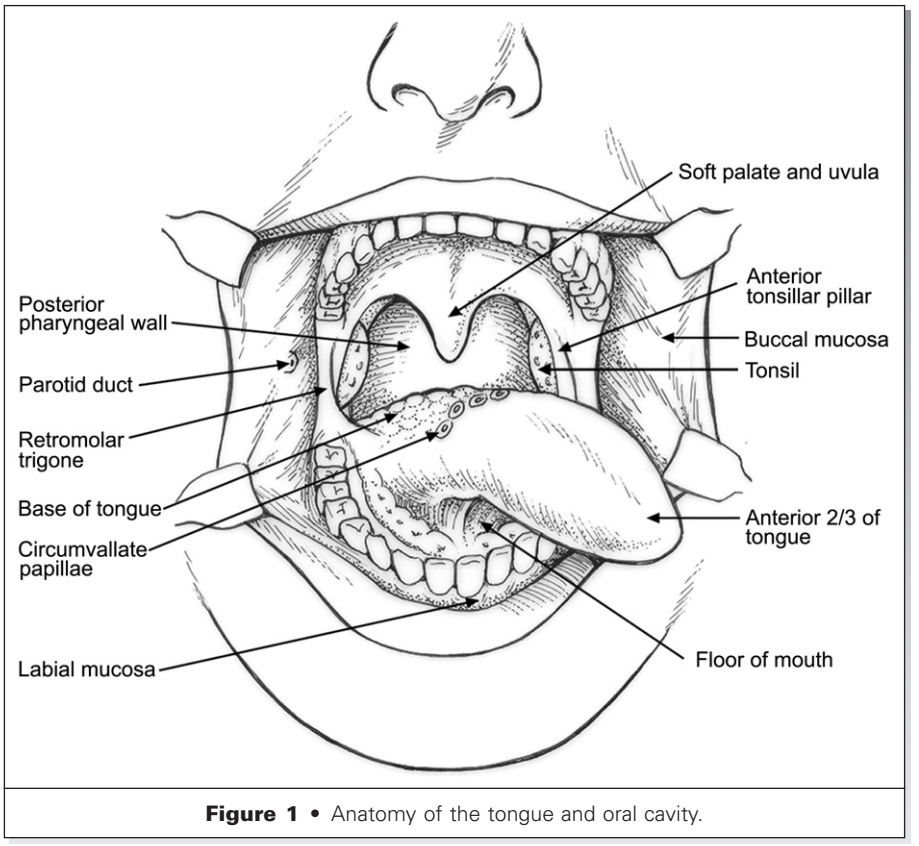

Figure 1 • Anatomy of the tongue and oral cavity.

initial diagnosis of erythroplakia. As with leukoplakia, there is rarely an apparent etiologic factor associated with its appearance. Biopsy is indicated at initial evaluation.

Lichen planus rarely undergoes malignant change. Patients with this disease, particularly the erosive form, should be managed by regular long-term follow-up.

Staging of Oropharyngeal and Oral Cavity Tumors

Staging and 5-year survival rates are presented in Table 1.

Malignant Lesions

SQUAMOUS CELL CARCINOMA. Squamous cell carcinoma (SCC), the most common type of cancer of the tongue and oral cavity, accounts for more than 90% of cancers in these areas. SCC grows along mucosal surfaces and infiltrates deeper structures in a fairly predictable pattern. Although other histologic types seen in the head and neck include primary tumors of the minor salivary glands, lymphoma, sarcoma, and melanoma, these tumors are so uncommon that they are mentioned only for the sake of completeness.

Most SCCs of the upper aerodigestive tract manifest as an ulcer that does not heal, and they frequently produce blood-tinged sputum. The lesion is rarely associated with local pain. Once SCC is established, its growth is steady and unresponsive to local medicinal measures (e.g., antibiotics, mouthwash). As the tumor grows, it can spread along tissue planes of muscle, fascia, and nerves, all of which produce symptoms that can potentially divert the clinician's attention from the tumor (e.g., ear pain, headache, toothache).

VERRUCOUS CARCINOMA. Pathologically considered a low-grade variant of SCC, verrucous carcinoma has unique characteristics that deserve special mention, including the fact that it classically occurs in the buccal mucosa of older women who do not have a history of smoking or alcohol ingestion. Verrucous carcinoma is exophytic and nonaggressive and never metastasizes to lymph nodes or other sites. Once the proper diagnosis is made, the condition should be treated by surgery, as verrucous carcinoma is relatively radioresistant. Because there are no preventable predisposing factors, it often recurs in the same patient, not as a local failure, but as a second or even a third primary tumor. The key to appropriate treatment of this disease is accurate diagnosis by adequate biopsy. It is essential to take serial sections of the primary tumor to make sure that there are no areas of more infiltrative squamous disease. These patients require frequent follow-up, just as those with more invasive disease.

TABLE 1 Staging for Cancer of the Oropharynx and Oral Cavity (Including the Lip), with 5-Year Survival Rates by Stage

PRIMARY TUMOR (T)

TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
Tis	Carcinoma in situ
T1	Tumor 2 cm or less in greatest dimension
T2	Tumor more than 2 cm but not more than 4 cm in greatest dimension
T3	Tumor more than 4 cm in greatest dimension
T4	Lip: Tumor invades adjacent structures (e.g., through cortical bone, tongue, skin of the neck)
T4	Oropharynx and oral cavity: Tumor invades adjacent structures (e.g., through cortical bone, soft tissues of the neck, deep [extrinsic] muscle of the tongue)

REGIONAL LYMPH NODES (N)

NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in a single ipsilateral lymph node, 3 cm or less in greatest dimension
N2	Metastasis in a single ipsilateral lymph node, more than 3 cm but not more than 6 cm in greatest dimension; or in multiple ipsilateral lymph nodes, none more than 6 cm in greatest dimension; or in bilateral or contralateral lymph nodes, none more than 6 cm in greatest dimension
N2a	Metastasis in a single ipsilateral lymph node, more than 3 cm but not more than 6 cm in greatest dimension
N2b	Metastasis in multiple ipsilateral lymph nodes, none more than 6 cm in greatest dimension
N2c	Metastasis in bilateral or contralateral lymph nodes, none more than 6 cm in greatest dimension
N3	Metastasis in a lymph node, more than 6 cm in greatest dimension

DISTANT METASTASIS (M)

MX	Presence of distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis

STAGE	GROUPING	APPROXIMATE 5-YEAR SURVIVAL RATE (%)
0	Tis, N0, N0	99%
I	T1, N0, M0	90%-92%
II	T2, N0, M0	75%-85%
III	T3, N0, M0	
	T1, N1, M0	
	T2, N1, M0	50%
	T3, N1, M0	
IV	T4, N0, M0	
	T4, N1, M0	
	Any T, N2, M0	25%-35%
	Any T, N3, M0	
	Any T, any N, M1	

(Used with the permission of the American Joint Committee on Cancer (AJCC), Chicago, Illinois. The original source for this material is the *AJCC Cancer Staging Manual*, 6th ed [2002], published by Springer-Verlag, New York, www.springer-ny.com.)

MINOR SALIVARY GLAND NEOPLASMS. Malignant tumors occur in the minor salivary glands of both the hard and soft palate and the lips. These glands are ubiquitous throughout the oral cavity. When a tumor does develop in a minor salivary gland, it is usually a firm, painless, submucosal mass. Differentiation between benign and malignant tumors cannot be made clinically, but more than two thirds of these tumors are malignant. The most common malignant gland tumor within the oral cavity is adenoid cystic carcinoma, followed by mucoepidermoid carcinoma and adenocarcinoma. Definitive treatment of these tumors is individualized according to the aggressiveness of the tumor and its size, location, and previous treatment.

Mechanisms of Tumor Spread

Local Extension

SCC can usually be characterized in one of three ways:

1. Superficial, exophytic
2. Infiltrating
3. Ulcerating/fungating

The exophytic variant tends to be superficial and spreads along the mucosal surface, whereas the ulcerating variety invades early and produces an "iceberg" effect. Infiltrating, fungating tumors metastasize earlier than exophytic tumors do. The degree of local extension is directly related to the size and location of the tumor.

Regional Metastasis

Lymphatic spread of the tumor usually takes place after the primary tumor is readily identifiable. The time frame of occurrence of pathologically suspicious lymph nodes in the neck is frequently related to the aggressiveness of the primary tumor, its size, and differentiation, but tumor size is not necessarily directly related to regional metastases. The most obvious example of this is a patient with biopsy-proven SCC in the neck and no apparent primary tumor ("unknown primary"). The pattern of regional dissemination via the lymphatics from oral and oropharyngeal cancer has been well documented.

Oral cavity cancers initially drain into the submental and supraomohyoid lymph nodes (zone I) and then to the upper deep jugular chain (zones II and III; see Fig. 3 in the chapter "Principles of Head and Neck Oncology"). Bilateral nodal involvement is commonly seen in patients with very large tumors that cross the midline and those originating from the midline (e.g., floor of the mouth). Although the incidence of cervical metastases in T1 and T2 tumors is between 10% and 20%, as the tumor increases in size and aggressiveness (T3 and T4),

the incidence of disease in the nodes becomes greater than 50%. When neck nodes are involved with disease, the survival rate plunges to half of what it would otherwise be. To some extent, the location of the primary tumor dictates the specific treatment of the neck. For example, an invasive T1 or T2 tumor of the mobile tongue produces metastases to the neck nodes much earlier than a similarly staged tumor of the lip does. The predilection for nodal disease is discussed in more detail with each anatomic site.

Distant Metastasis

It is highly unusual for a metastatic lesion below the clavicle to arise from a primary, untreated cancer of the lip or oral cavity. In fact, when such a lesion is suspected in the initial workup, the chances are great that it is in fact a second primary cancer. The most common site for development of a second primary cancer is the lung. This "distant disease" finding requires a workup as though it were a primary tumor because such is commonly the case. The importance of distinguishing between the two cannot be overstated. For example, treatment of two synchronous primary tumors carries a better prognosis than does treatment of a primary tumor with a distant metastasis.

As treatment of the primary and regional disease becomes more predictable with higher cure rates, there is a greater incidence of distant disease that is fatal in most patients. Interestingly, over the past 50 years the percentage of people with cancer of the oral cavity who have died of the disease has remained the same. However, rather than dying of recurrent local and regional disease, they have died of distant disease occurring below the clavicles.

After a primary tumor has failed some type of definitive treatment (e.g., surgery or surgery plus radiation therapy), the chance for distant disease increases significantly. Therefore, a suspicious lesion of the lung, brain, bone, or liver, although requiring biopsy to confirm the diagnosis, may be assumed to be a metastatic lesion rather than a second primary lesion. The reason for this difference is thought to be related to the alteration in lymphatic drainage that occurs after surgery and radiation therapy. Distant metastasis carries a grave prognosis.

Diagnostic Studies

History, physical examination, and biopsy of the lesion are essential, as are questions regarding tobacco and alcohol use, pain, weight loss, dysphagia, odynophagia, otalgia, hemoptysis, hoarseness, articulation difficulties, bleeding, presence of ulcers, hypoesthesia, loose teeth or ill-fitting dentures, trismus, throat irritation or pain, drooling, and voice change or loss. A blood count and liver function tests are required because these patients are prone to chronic anemia and liver disease related to alcohol ingestion.

Tissue biopsy is critical, and specimens may be obtained with punch forceps. The biopsy sample should include a portion of the tumor that does not contain an excessive amount of necrosis. Fine-needle aspiration is a valuable tool for evaluation of questionable neck masses. It does not alter later therapy and is usually quite accurate.

The following are the major objectives of imaging studies:

1. Assess the size and extent of the primary cancer.
2. Determine the presence of bony invasion or perineural spread.
3. Assess the extent of regional spread and distant metastasis.
4. Rule out a second primary.

A chest radiograph is usually sufficient radiologic workup for cancers of the tongue and oral cavity. It is ordered primarily to look for a second primary tumor in the lung because most of these patients have a significant history of tobacco abuse. Computed tomography (CT) is the primary modality of imaging tumors of the oral cavity and the neck. Magnetic resonance imaging, which gives excellent definition of the extent of cancer involving the tongue, is reserved for situations in which CT is not useful. Such situations include contrast allergy, excessive dental artifacts, lesions that are not visible on CT, and the possibility of perineural spread. Positron emission tomography (PET) is an emerging technology that appears to have promise in detecting recurrent or metastatic disease, or both, in the neck. Panoramic radiographic films can be used for adjunctive assessment of invasion of the mandible by cancer.

Reconstructive Goals

The basic principles of reconstruction of the oral cavity include the following:

1. Repair or reconstruction should not compromise or limit ablative surgery.
2. The reconstruction should not add to the morbidity of ablative surgery.
3. Immediate reconstruction is preferred.
4. An attempt to replace "like tissue with like tissue" should be made in all cases by using the patient's own soft tissue and bone.
5. Adapt the reconstruction to the needs of the patient.

A small intraoral or tongue surgical defect less than 2 cm in size can usually be closed primarily.

Larger defects may be reconstructed with a skin graft, regional flap, or free tissue transfer. The goal of reconstruction is to replicate the function and appearance of the resected tissue. Split-thickness skin grafts are often used for reconstruction. They are usually the most expedient and efficacious reconstruction for small defects because they minimize morbidity without hindering swallowing or speech.

Larger defects may require a local or regional flap. Small intraoral defects can be reconstructed effectively with palatal, tongue, and buccal mucosal flaps, but usually at the cost of decreased function. Regional flaps used in reconstruction of the oral cavity include the pectoralis major, trapezius, and latissimus dorsi flaps. The most commonly used regional flap for the oral cavity is the pectoralis major flap. This flap is reliable, with a failure rate of 3% to 15%. Its disadvantages are lack of bone and the limited extent to which it can reconstruct the oral cavity as a result of tethering by its pedicle. Because of the bulk of the flap, oral function is often poor.

The benefits of free tissue transfer include:

1. Superior blood supply
2. Greater range of tethering by the vascular pedicle
3. Greater variety and versatility of donor sites

Disadvantages of free tissue transfer include the complexity of the technique and the increased surgical time. In many situations, free flaps offer the best reconstructive option. These situations include:

1. Bony defects, particularly of the anterior portion of the mandible
2. Defects that require considerable bulk and soft tissue
3. Defects that are not amenable to regional flaps because of location

The free tissue transfers most commonly used in the oral cavity are the rectus abdominis and the radial forearm flaps. Free flaps also hold the potential for creation of a sensate flap through a neural anastomosis.

Treatment

A number of options are available to treat cancer of the oral cavity: surgery alone, radiation therapy alone, or surgery combined with postoperative radiation therapy. The modality used clearly depends on the stage of the tumor, the physiologic and emotional status of the patient, and the patient's preference. In the hands of most head and neck surgeons, most cancers of the oral cavity and tongue should be treated with surgery. Postoperative irradiation is indicated as an adjunct when there are poor prognostic indicators.

Resection of the floor of the mouth, buccal mucosa, tongue, and mandible can be performed via one of four approaches: transoral, mandible sparing (pull through), mandibulotomy, or composite resection (including the mandible and associated neck dissection). Factors that might influence the approach include the size, location, and invasive characteristics of the tumor; mandible invasion (or suspicion thereof); need for neck dissection; and previous treatment (surgery or radiation therapy, or both).

Transoral Resection

Transoral resection is reserved for smaller lesions that are easily accessible. Tumors removed via the transoral approach are usually anterior, superficial, and well-circumscribed lesions of the floor of the mouth, mobile (anterior two thirds) tongue, the buccal mucosa, or the palate. Included would be all T1 and early, exophytic T2 cancers. If the tumor involves the submandibular duct, reconstruction is not usually attempted, nor is it necessary. In these early lesions, discontinuous neck dissection is easily accomplished if dictated by the primary tumor. Defects resulting from resection of small tumors of the floor of the mouth can be left to heal secondarily, with little morbidity. If the wound is larger, a split-thickness skin graft suffices. Defects affecting less than a third of the tongue can be closed primarily, with little long-term morbidity in speech or swallowing.

A variation of transoral resection is resection of the upper half of the mandible as a marginal resection. Such surgery is usually performed when there is no obvious clinical tumor involvement of the mandible. Reconstruction is accomplished with a split-thickness skin graft placed on the cancellous bony surface of the remaining mandible.

Pull Through Procedure

The pull through procedure is ideal for moderate-sized cancers of the anterior and lateral floor of the mouth areas, particularly if the mandible is not involved or part of the mandible has been removed as a deep margin. This approach has the distinct advantage of maintaining the contour of the mandible while excising the primary tumor in continuity with the neck specimen. Reconstruction is usually accomplished with a free tissue transfer.

Mandibulotomy

Patients with a large tumor of the posterior portion of the oral cavity that does not involve the mandible are best suited for mandibulotomy. Also called a "mandibular swing," mandibulotomy divides the mandible just lateral to the midline (anterior to the mental foramen), thereby preserving sensation to the lip. Once the tumor is resected and reconstruction is performed (e.g., skin graft or

musculocutaneous, regional, or free flap), the mandible is returned to its original position and plated. This approach can be used for patients regardless of dentition. Another advantage of this method is that the portion of the mandible that has been divided is out of the field of postoperative radiation therapy. Mandibulotomy is the most common approach for resection of tumors of the retromolar trigone, base of the tongue, tonsils, and lateral pharyngeal wall.

Mandibulectomy

Tumors that approximate the gingiva should be resected with the gingiva and periosteum as an additional deep margin, and those that appear to involve the periosteum should be resected with an additional deep margin of bone. This last procedure is termed a marginal mandibulectomy. Depending on the extent of tumor involvement, resection of a bicortical rim of bone at the upper aspect of the alveolus (rim mandibulectomy) or selective removal of the inner cortex with the use of a vertical or oblique resection (sagittal mandibulectomy) may be necessary. Surgeons should leave at least a 1-cm-thick segment of bone inferiorly after rim mandibulectomy to reduce the risk for pathologic fractures. If this segment is preserved, rim mandibulectomy has been reported to be more resistant than sagittal mandibulectomy to fracture.

Composite Resection with Mandibulectomy

If the mandible is involved with cancer, it must be removed as a full-thickness, segmental resection. When such late-stage disease is treated, there is almost always a soft tissue deficit, and restoration of both bone and soft tissue is necessary. Reconstruction may often require a free tissue transfer (and at times two transfers), to be performed immediately after ablative surgery. A composite resection is generally used when the tumor invades the lateral or anterior arch of the mandible. In the latter setting, immediate reconstruction is required, and in the former, it is highly desirable. The indicated neck dissection can be carried out at the same time as resection of the primary tumor.

Cancers of the retromolar trigone may already be advanced at first examination because only a thin layer of soft tissue overlies the bone in this region and invasion of the underlying bone may occur early. In addition, there are multiple pathways for spread from this site, including the buccal mucosa, tonsillar fossa, glossopharyngeal sulcus, floor of the mouth, base of the tongue, hard and soft palate, masticator space, and maxillary tuberosity. Because patients tend to have advanced disease of the retromolar trigone at initial evaluation, many patients are also found to have regional metastases at that time.

Postoperative Care

Intraoperative Complications

Intraoperative complications include injury to the neurovascular structures or the thoracic duct. Bleeding from the internal jugular vein is especially risky because of the potential for an air embolus. The risk for air embolism is highest when the head of the patient's bed is elevated and the patient is breathing spontaneously.

Postoperative Complications

In addition to the universal potential postoperative complications of pneumonia, deep venous thrombosis, fluid overload, and other cardiopulmonary problems, surgical procedures involving the oral cavity have unique postoperative complications related to the site of the wound. Surgical complications may include infection, orocutaneous or pharyngocutaneous fistulas, and carotid artery rupture.

Site-Specific Treatment Sequelae

1. Patients with cancer of the tongue, floor of the mouth, alveolar ridge, and retromolar trigone often experience problems with deglutition and speech as a result of either surgery or irradiation.
2. Resection of the anterior arch of the mandible results in the "Andy Gump" deformity, which causes poor aesthetic, swallowing, and speech outcomes. As stated previously, these patients should always be reconstructed immediately.
3. Fractures of the mandible can occur after marginal mandibulectomy.
4. Exposure of the mandible can occur when insufficient soft tissue coverage is provided.
5. Surgical procedures of the palate and retromolar trigone can result in hypernasality.

Prognosis

Five-year locoregional control rates of 91% and survival rates of 55% to 62% are achievable for early-stage SCC of the tongue, depending on nodal involvement. Advanced-stage disease is usually managed with multimodality therapy, including surgery, irradiation, and sometimes chemotherapy. These patients have a 3-year survival rate of 40% to 50%. Locoregional control remains a challenge in the treatment of cancer of the floor of the mouth. It is the major source of treatment failure for these patients. Distant metastasis is uncommon (10%) but occurs after local failure. Five-year survival rates for patients with cancer of the floor of the mouth range

from 64% to 80% in the case of stage I disease, 61% to 84% for stage II disease, 28% to 68% for stage III disease, and 6% to 36% for stage IV disease.

Physiologic tests of speech and swallowing can provide a standardized, reproducible way to assess outcome in patients with cancer of the oral cavity. These measures are often integrated with psychometric assessment.

Pearls and Pitfalls

- SCC is by far the most common primary tumor of the tongue and oral cavity.
- The preferred method of treatment of cancer of the tongue and oral cavity is surgery followed by irradiation, when indicated. Primary radiation therapy is rarely indicated.
- A complete workup of patients with cancer of the tongue and oral cavity includes a thorough history and physical examination (for staging purposes), a chest x-ray, and appropriate medical evaluation.
- After a patient has survived 24 months without recurrence of cancer of the tongue and oral cavity, the chance of being cured is 90%.
- All cancers of the tongue and oral cavity should undergo immediate reconstruction.
- Although regional spread of cancer is predictable before treatment, recurrence after therapeutic irradiation is not.

SUGGESTED READINGS

Barttelbort SW, Ariyan S: Mandible preservation with oral cavity carcinoma: Rim mandibulectomy versus sagittal mandibulectomy. Am J Surg 166:411-415, 1993.

Christopoulos E, Carrau R, Segas J, et al: Transmandibular approaches to the oral cavity and oropharynx. Arch Otolaryngol Head Neck Surg 118:1164-1167, 1992.

Taneja C, Allen H, Koness RJ, et al: Changing patterns of failure of head and neck cancer. Arch Otolaryngol Head Neck Surg 128:324-327, 2002.

Urken ML, Moscoso JG, Lawson W, Biller HF: A systematic approach to functional reconstruction of the oral cavity following partial and total glossectomy. Arch Otolaryngol Head Neck Surg 120:589-601, 1994.

The Pharyngoesophageal Region

GRANT W. CARLSON

Resection of the larynx, hypopharynx, and cervical esophagus for advanced malignancy or for stricture repair results in severe functional impairment. Loss of speech and disruption of deglutition severely handicap patients who are already nutritionally depleted. The ideal reconstructive method would be a single-stage procedure with low morbidity that provides rapid restoration of function. Reconstructive methods have evolved from the use of skin flaps alone to reconstruction with musculocutaneous flaps, visceral transposition, and microsurgical free tissue transfer.

Anatomy and Physiology

The hypopharynx is a muscular, funnel-shaped tube composed of five paired striated muscles with a narrow lower end that becomes continuous with the esophagus at the level of the sixth cervical vertebra (Fig. 1). The superior, middle, and inferior constrictors compose the outer circular musculature. They overlay along the posterior wall of the pharynx and insert on the central raphe. Successive contractions of the constrictor muscles propel food into the esophagus. The inner, longitudinal musculature is composed of the stylopharyngeus and the palatopharyngeus, which insert on the posterior border of the thyroid cartilage. They act to elevate the larynx and pharynx during swallowing. The cricopharyngeal muscle originates from the cricoid cartilage looping around the exit of the pharynx. It acts as a physiologic sphincter to prevent entry of air into the esophagus between acts of swallowing; it is continuous without an insertion into the central raphe.

Swallowing is a complex function requiring coordination of numerous rapidly occurring processes. During the oral phase, the tongue moves food from the front of the oral cavity to the anterior faucial arches, where the swallowing reflex is initiated.

Coordinated motor stimulation results in four sequential stages of pharyngeal swallowing: (1) closure of the soft palate, (2) peristaltic contraction of the pharyngeal constrictors, (3) elevation and closure of the larynx, and (4) relaxation of the cricopharyngeus muscle.

The tongue is the main force for propelling food through the pharynx. It functions as a piston, with the pharyngeal walls acting as a chamber for the pump. Manofluorometry has been used to analyze patients after both gastric transposition and free jejunal flap reconstruction of the hypopharynx and cervical esophagus. The reconstructed segment was found to function as a passive conduit. Dysphagia can therefore occur as a result of either impaired lingual coordination or functional obstruction. In addition, excision of the wall of the oropharynx or hypopharynx interferes with the sensory innervation responsible for triggering the swallowing reflex. Impaired lingual propulsion also results in loss of bolus control, as well as decreased driving force. Moreover, stricture formation is most often seen at the distal anastomosis, but anatomic obstruction can also occur as a result of flap redundancy or excessive bulk, as previously noted in patients who have undergone reconstruction with a pectoralis major musculocutaneous flap. Dysphagia can occur even though large dilators can pass through the reconstructed segment. A long reconstructed segment also results in greater impedance to the driving force of the tongue.

Jejunal peristalsis was originally though to aid in food passage; however, the jejunum is not a rapid-transit organ and has a predominance of type I, or mixing, peristaltic waves. Its segmental bowel contraction serves a mixing function that can impede or delay the food bolus. Thus, functional hypopharyngeal reconstruction is more dependent on the extent

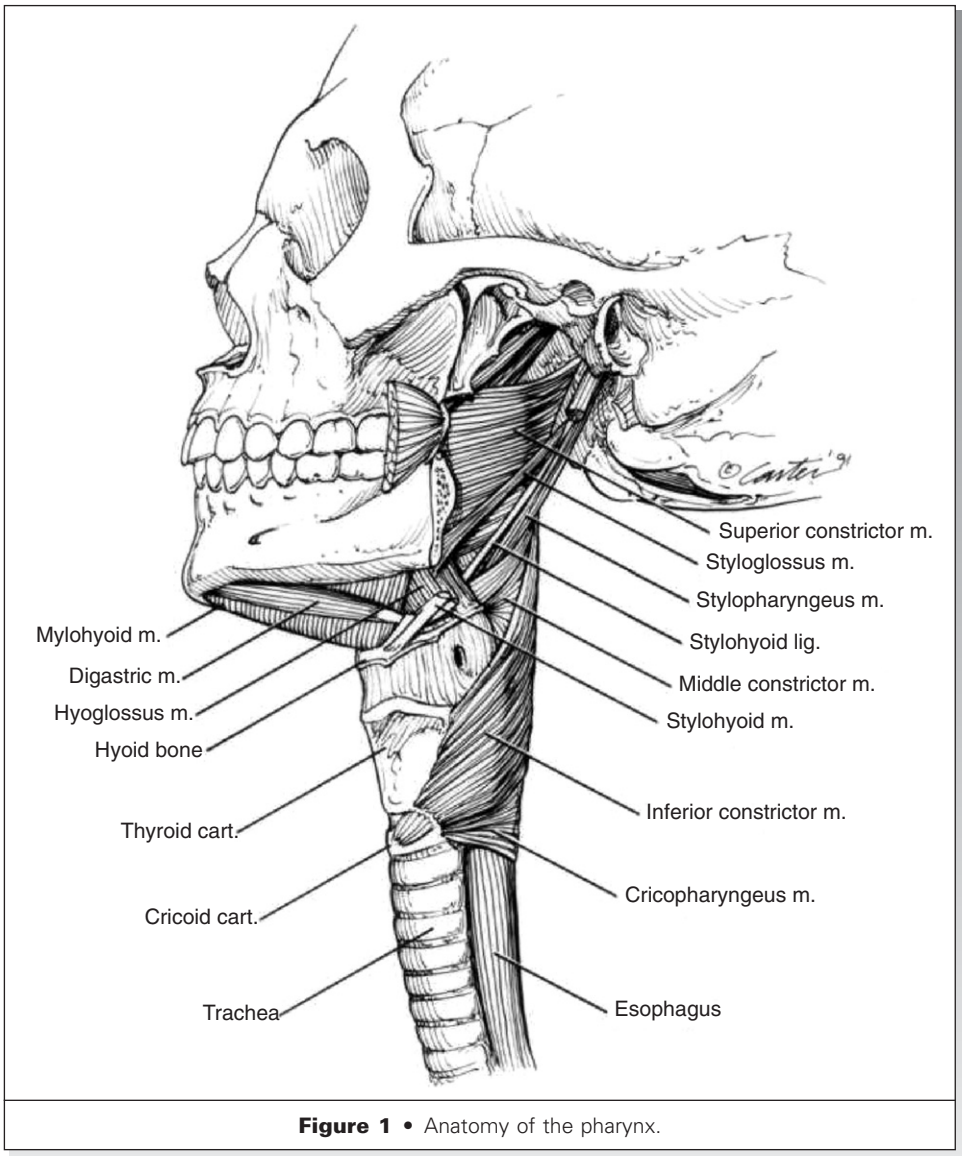

Figure 1 • Anatomy of the pharynx.

of the extirpative defect and less on the method used to reconstitute gastrointestinal continuity.

Reconstructive Goals

The two goals of pharyngoesophageal reconstruction are to:

1. Restore the ability to swallow safely
2. Provide an anatomic conduit that permits esophageal speech

Diagnostic Studies

Examination under anesthesia is essential to determine the extent and location of tumor invasion or pharyngeal stricture. A length of 20 cm can be reconstructed with a free jejunal flap or radial forearm flap. Resection extending into the superior mediastinum would entail total esophagectomy, which requires gastric transposition to restore gastrointestinal continuity.

Treatment

Most esophageal defects can be successfully managed by gastric pull-up performed by a general or thoracic surgeon without plastic surgical consultation. Defects of the upper cervical esophagus or hypopharynx, however, require plastic surgical techniques, as this is beyond the safe reach of a gastric pull-up procedure. Although the radial forearm free flap has a role in pharyngoesophageal reconstruction (specifically, for large segmental defects), the

free jejunal flap has emerged as the option with the greatest versatility and flexibility for a wide range of defects. The free jejunal flap has therefore become the preferred technique for reconstruction in this area.

Circumferential defects of the hypopharynx and cervical esophagus can be closed by a variety of methods. Because of its lower morbidity and higher success rate, the free jejunal flap has supplanted the deltopectoral and pectoralis major flaps. The radial forearm flap is useful for patients in whom laparotomy is contraindicated. It is associated with a high rate of fistula formation but may allow better restoration of voice than possible with the jejunal flap.

Free Jejunal Transfer

This method has become the standard technique for reconstructing the hypopharynx and cervical esophagus. Thorough preparation is key to a successful outcome. Optimal nutritional repletion is necessary in these patients, who are often emaciated and have suffered substantial weight loss. A two-team approach is planned. Hydration and warmth of the patient and avoidance of vasoconstrictors are critical for success.

It is helpful to reverse the operating table so that the surgeons have unencumbered access to the patient's head and neck. During the extirpative procedure, it is important that the surgeon preserve a useful proximal recipient vein (e.g., external jugular) and artery (facial or superior thyroid), as long as therapeutic resection is not compromised.

Toward the latter portion of the extirpation, when the extent of the resection becomes defined, a second surgical team begins the abdominal portion of the procedure. An upper midline incision is made and a segment of jejunum about 40 cm distal to the ligament of Treitz is isolated. The length of jejunum for cervical esophageal reconstruction averages 8 to 10 cm, but it depends on the resectional defect. A reliable radial vascular arcade is identified within the mesentery, and a suture is used to mark the proximal end of the bowel to ensure isoperistaltic placement. The vessel arcade is isolated (back lighting and vessel palpation are helpful), and a wedge of supporting mesentery is divided. Meticulous technique prevents mesenteric hematoma. The bowel is isolated, divided between clamps, wrapped in a moistened laparotomy pad, and allowed to perfuse. Routine bowel anastomosis is performed and the mesenteric defect closed. A conventional tube gastrostomy is performed. The postablative defect is measured, and the appropriate vessels are identified and prepared in the neck. Arterial inflow is usually provided by the superior thyroid or facial artery. Venous outflow is generally provided by the external jugular, facial, or internal jugular vein.

The jejunal segment is checked for viability. The mesenteric vessels are divided, and the segment is transferred to the head and neck and placed in an isoperistaltic position. The proximal oropharyngeal jejunal anastomosis is performed. The proximal portion of the bowel can be opened along the antimesenteric border and tailored to fit the defect from the tongue to the posterior pharynx if necessary (Fig. 2). At the completion of the pharyngeal-jejunal anastomosis, the operating microscope is used to check the vessels for torsion and contour, and the outflow of the artery is checked for pulsatile flow. The deeper vessel anastomosis is completed first.

The jejunal-esophageal anastomosis is performed after completion of the microvascular anastomoses. The neck is flexed and the length of jejunal segment needed is determined after a period of flap perfusion. The flap should be under slight tension to avoid redundancy, which can result in functional obstruction to food passage. A small distal piece of vascularized bowel and mesentery is brought through the neck incision to serve as a monitor.

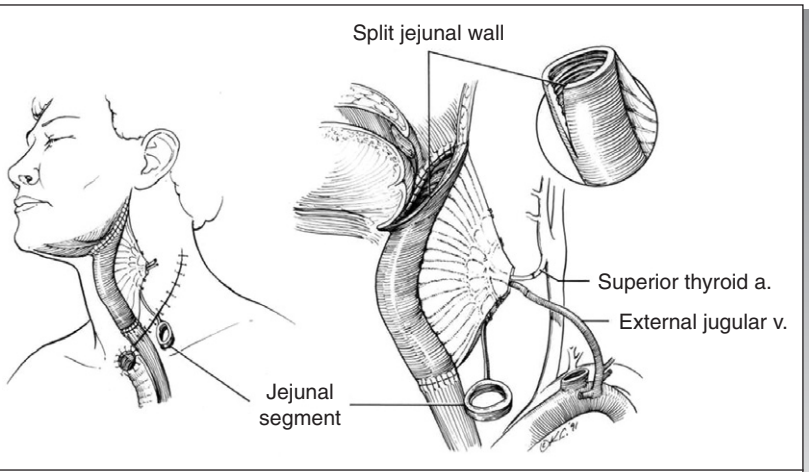

Figure 2 • A free jejunal flap can be split to reconstruct defects extending to the base of the tongue. A small jejunal segment is brought through the skin to serve as a monitor.

Postsurgical Care

After the free jejunal flap reconstruction, the patient spends at least 1 day in the intensive care unit to optimize pulmonary management and permit flap monitoring. A gastrostomy tube with gravity drainage is used for 24 to 48 hours until bowel function returns. Enteric feeding is started via the gastrostomy tube, and the patient takes nothing by mouth for at least 7 days. At that time a Gastrografin swallow is performed to detect occult leaks or stricture. After a normal contrast study, oral intake is commenced under the guidance of a speech and swallowing specialist.

Pearls and Pitfalls

- The jejunal flap has to be placed in an isoperistaltic position under slight tension. Jejunal redundancy and a reversed segment can result in functional obstruction to food passage.
- Anastomosis of the proximal bowel to the tongue base is performed before the microvascular anastomoses to facilitate surgical exposure, especially when the resection extends high on the pharyngeal wall.
- The jejunal-esophageal anastomosis is performed after completion of the microvascular anastomoses. The neck is flexed and the length of the jejunal segment determined after a period of flap perfusion.
- The incidence of flap necrosis should be less than 10%.
- Fistulas form in approximately 20% of patients, usually at the proximal anastomosis, but the majority close without operative intervention in nonirradiated fields.

SUGGESTED READINGS

Anthony JP, Singer MI, Deschler DG, et al: Long-term functional results after pharyngoesophageal reconstruction with the radial forearm free flap. Am J Surg 168:441-445, 1994.

Carlson GW, Coleman JJ, Jurkiewicz MJ: Reconstruction of the hypopharynx and cervical esophagus. Curr Probl Surg 30:427-472, 1993.

Carlson GW, Schusterman MA, Guillamondegui OM: Total reconstruction of the hypopharynx and cervical esophagus: A 20-year experience. Ann Plast Surg 29:408-412, 1992.

Chang DW, Hussussian C, Lewin JS, et al: Analysis of pharyngocutaneous fistula following free jejunal transfer for total laryngopharyngectomy. Plast Reconstr Surg 109:1522-1527, 2002.

Coleman JJ 3rd: Reconstruction of the pharynx and cervical esophagus. Semin Surg Oncol 11:208-220, 1995.

The Skull Base

HRAYR SHAHINIAN

One of the most complex regions of the human anatomy is the skull base. It is a difficult area of the body to navigate given the vital blood vessels and major cranial nerves that pass through it (Fig. 1).

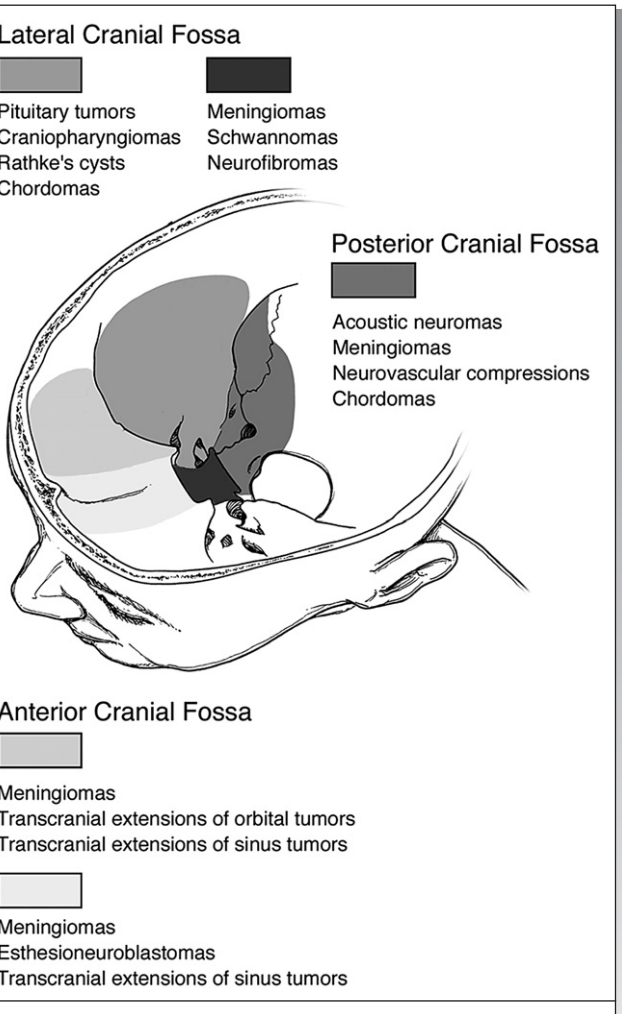

Figure 1 • Anatomic topography of the skull base.

Many skull base tumors, although benign, can have devastating effects because of their proximity to these vital structures. Damage to vessels and nerves can result in blindness, deafness, inability to speak or swallow, paralysis, and death. In the 1950s, Tessier of France and Fish of Switzerland discovered innovative techniques to approach and remove lesions of the skull base.

The introduction of computed tomography and magnetic resonance imaging, combined with the availability of more precise microsurgical instruments, microscopic magnification, and illumination, opened a new frontier in skull base surgery. As in many other surgical subspecialities, the field of skull base surgery started with radical exposures such as the transfacial approaches to the anterior skull base and the infratemporal or transcochlear approaches to the lateral skull base; however, there is an evolving trend toward more functional and less invasive techniques. These minimally invasive approaches benefit from fiberoptic and endoscopic technology.

Surgical Anatomy

The Nasal Cavities

Skull base surgeons must learn to navigate the neurovascular anatomy of the anterior and middle cranial fossae in transcranial pituitary surgery, as well as the length of the nasal septum in the transseptal transsphenoidal approach. The introduction of transnasal endoscopic pituitary surgery has further underscored the necessity for surgeons to become familiar with the relationships of the sella and pituitary gland to the bony, cartilaginous, and mucosal architecture of the nasal cavities and the paranasal sinuses. During embryonic development, bilateral invaginations of ectoderm located superior to the opening of the mouth pass posteriorly through the mesoderm of the head region to form the left and right nasal pits. These invaginations meet and fuse

with the endoderm of the most cranial extensions of the primitive foregut and create a passageway from the external environment to the gut lumen. Thus connected, the nasal pits are referred to as the right and left nasal cavities, and the part of the involved foregut is called the nasopharynx. The common medial wall of each nasal cavity is the embryonic nasal septum, and the floors of these cavities form the primary palate. Cartilage forms in the mesoderm of the roof of each nasal cavity and extends into the adjacent upper regions of the lateral walls and the septum. Connective tissue develops in the inferior lateral walls, inferior septum, and cavity floors. Bony deposition begins in ossification centers located throughout the cartilaginous and membranous portions of the nasal capsule. The posterior and inferior parts of the nasal septum become ossified to form the perpendicular plate of the ethmoid and the vomer, respectively. The crista galli, the superior extension of the perpendicular plate of the ethmoid, is also formed in this process. The anterior extent of the septum remains cartilaginous. The roof of the nasal cavities ossifies around nerve fibers that communicate superiorly with the olfactory bulbs. The cribriform plate of the ethmoid is thereby formed.

A "gap" in the deposition of cartilage in the lateral nasal cavity walls results in formation of the hiatus semilunaris. In its mature form, it is identified as a depression in the lateral nasal wall at the level of the middle meatus. Cartilage formation resumes at the inferior aspect of this hiatus in the uncinate process, which invaginates to create the inferior turbinate. The remainder of the cartilage of the lateral wall ossifies to generate the ethmoid labyrinth. The labyrinth is polarized into lateral and medial walls; the former makes up the medial wall of the orbit, whereas the latter generates two additional invaginating bony processes, the precursors of the middle and inferior turbinates. The superior turbinate sits near the roof of the nasal cavity. The recesses created by the curvatures of the turbinates are referred to as meati and are the site of origin of the paranasal sinuses.

The mucous membrane lining the lateral nasal walls invaginates into the skeleton of the midface to create air pockets surrounded by bone. Posterior to the oral cavity, the mucous membrane covering the anterior surface of the sphenoid bone pushes back to create the sphenoid sinus. The sinus is divided into left and right components by the formation of a thin bony septum. The sphenoid sinus communicates with the nasal cavities via ostia located behind the superior turbinates.

During endoscopic transnasal pituitary surgery, endoscopes are advanced into the vestibule to identify the inferior turbinate and the anterior portion of the septum. The inferior and middle turbinates span almost the entire depth of the nasal cavity. Therefore, with slight superior and posterior advancement of the endoscope, the anterior limit of the middle turbinate comes into view. With further caudal progress, the remainder of the middle turbinate and the superior turbinate are appreciated. The goal of the intranasal portion of endoscopic pituitary surgery is to create a wide passage from the exterior to the sphenoid rostrum. The space of the middle meatus is therefore obliterated as the middle turbinate is outfractured. The sphenoid ostia may or may not be readily apparent until mucosa is dissected from the anterior surface of the body of the sphenoid. The superior and middle turbinates serve as valuable landmarks for identifying the approximate locations of the ostia until they are directly visualized.

The Sella Turcica and Pituitary Gland

The hypophysial fossa of the sella turcica contains the pituitary gland. It represents a rounded excavation of the sphenoid bone that is flanked by critical structures. The dorsum sellae is the uppermost extension of the clivus and forms the posterior wall of the sella. It has lateral protuberances that are referred to as the posterior clinoid processes. The anterior boundary of the sella is marked by the tuberculum sellae, a raised prominence on the superior surface of the sphenoid bone immediately in front of the hypophysial fossa. The curving projections of the lesser wings of the sphenoid terminate medially into the anterior clinoid processes, which rest above and posterolateral to the tuberculum. Anterior to the tuberculum sellae is a depression in the sphenoid called the prechiasmatic groove, on either side of which lie the intracranial openings of the optic canals.

The hypophysial stalk serves as a direct conveyance of hormones to the posterior pituitary and as a conduit for hormone-releasing signals to the anterior pituitary via its portal vessels. It descends from the median eminence of the hypothalamus and passes through a central hiatus in the diaphragma sellae, a dural reflection between the anterior and posterior clinoid processes that covers the hypophysial fossa. Above the diaphragma, the hypophysial stalk is related to the optic chiasm. The chiasm usually overlies the sella and pituitary. This anatomic arrangement accounts for the visual symptoms, most notably temporal hemianopia, seen in patients with pituitary tumors that have suprasellar extension. The optic nerves emerge from the optic canals anteromedial to the tips of the anterior clinoid processes to pass posteromedial toward the chiasm.

The parasellar vascular anatomy must also be appreciated to minimize the chance of intraoperative vascular injury. Subfrontal approaches to the sella expose the carotid arteries and the anterior arc of the circle of Willis, as well as any perforating branches from these major vessels. Vascular structures in the suprasellar area may be displaced by superior extensions of tumors such that normal anatomic relationships are distorted and manipulation of these vessels becomes extremely dangerous.

The carotid artery emerges from the roof of the cavernous sinus beneath the optic nerve and immediately gives off the ophthalmic branch, which enters the optic canal on the underside of the nerve. The carotid turns back toward the posterior clinoid process, where it meets the posterior communicating artery and gives off the anterior cerebral artery. This artery courses over the superior surface of the optic chiasm and gives off an anterior communicating branch to its contralateral counterpart. Surgeons using these approaches must carefully work around the optic nerves and chiasm and associated neurovascular structures while removing a tumor from the sella and sphenoid. The carotid arteries are also at risk during transsphenoidal approaches to the sella. Their tortuous transcranial course carries them alongside the lateral margins of the sphenoid sinus; they are at risk when the anterior wall and the mucosa of the sinus are dissected. The intracavernous segments of the arteries are vulnerable to overly aggressive curettage of tumor from within the sphenoid or sella.

The Anterior, Middle, and Posterior Cranial Cavities

The embryologic development of the anterior, middle, and posterior cranial fossae is dependent on the formation of ossification centers within the chondral template of the developing skull base. This sheet of cartilage provides the framework for the base of the cranium, as well as the subjacent parts of the midface and nose. Deposition of bone within the cartilage gives rise to most of the occipital, temporal, sphenoid, and ethmoid bones and determines their ultimate shape. The frontal bone defines the rostral limit of the anterior cranial cavity. Mature bone represents the fusion of separate ossification centers within the embryonic membranous neurocranium that articulates in the midline at the metopic suture. An osseous projection called the frontal crest extends posteriorly from the inner surface of the frontal bone along the floor of the cavity. The frontal crest points to the cribriform plate and is separated from it by the foramen cecum, through which passes an emissary vein. The cribriform plate is a punctate bony surface that forms the roof of the nasal cavities bilaterally. Its perforate nature is derived from its formation around the differentiated nerves of the upper nasal passages; these nerves pass into the anterior fossa and synapse on the olfactory bulbs. The cribriform plate is bisected by the falx cerebri and crista galli, which represents the intracranial extension of the perpendicular plate of the ethmoid bone. The remainder of the floor of the anterior fossa is made up of the thin orbital plates of the frontal bone that form the roofs of the orbits and support the frontal lobes. The middle cranial fossa houses the temporal lobe and the pituitary gland; most of the floor is made up of the sphenoid bone. Various foramina in the floor of the middle fossa allow passage of neurovascular structures into and out of the cranium: the carotid artery, middle meningeal artery and vein, and branches of the trigeminal nerve. A gap at the medial junction of the greater and lesser sphenoid wings, the superior orbital fissure, is occupied by the first branch of the trigeminal nerve; by the oculomotor, trochlear, and abducens nerves; and by the ophthalmic veins. The bony optic canal provides passage of the optic nerve into the apex of the orbit.

The posterior cranial fossa houses the cerebellum and the pons, which passes through the foramen magnum. The floor of the posterior fossa is made up of the clivus anteriorly. The superior surface of the clivus is formed by the sphenoid bone, whereas the lower portion is formed by the occipital bone. Between the clivus anteriorly and the pons posteriorly lies the basilar artery. Along its lateral aspect, the posterior fossa is made up of the petrous portion of the temporal bone, and the remainder of the floor of the posterior fossa is composed of the occipital bone. The roof of the posterior fossa is made up of the tentorium cerebelli.

Other foramina in the floor of the posterior fossa allow passage of other neurovascular structures into and out of the cranium, for example, cranial nerves IV to XII. The trochlear nerve (IV) is seen transiting through the posterior fossa, traveling below the tentorium cerebelli medially, and entering the middle cranial fossa by traveling through the cavernous sinus and subsequently entering the orbit through the superior orbital fissure. The trigeminal nerve exits the pons and has a variable cisternal portion before its larger sensory root and its smaller motor branch enter Meckel's cave to form the gasserian ganglion within the cavernous sinus in the middle cranial fossa. The trigeminal nerve divides into its ophthalmic, maxillary, and mandibular branches.

The ophthalmic (V1) branch enters the orbit through the superior orbital fissure, the maxillary (V2) branch travels through foramen rotundum within the floor of the middle cranial fossa, and the mandibular (V3) branch passes through foramen ovale within the floor of the middle cranial fossa. The abducens nerve emerges from the pontomedullary area, has a long cisternal segment, and exits the posterior fossa through Dorello's canal within the occipital bone. It subsequently transits through the middle fossa by traveling through the cavernous sinus and enters the orbit via the superior orbital fissure.

The acousticofacial bundle is composed of the facial nerve (VII) and the vestibulocochlear nerve (VIII); the bundle is made up of four separate nerves as they exit the brainstem together and travel to exit the cranium through the internal auditory canal. They enter the internal auditory canal within the petrosal portion of the temporal bone, where they separate. The cochlear nerve provides hearing and vestibular nerve balance. The facial nerve traverses the temporal bone, exits the cranium through the

stylomastoid foramen, and divides within the parotid gland into its five branches.

The lower cranial nerves (IX, X, XI) exit the posterior cranial fossa along with the jugular vein through the jugular foramen. The hypoglossal nerve (XII) exits the medulla as two separate rootlets and exits the posterior cranial fossa through the hypoglossal canal within the occipital bone. The clinically significant blood vessels within the posterior cranial fossa include the transverse sinus, which becomes the sigmoid sinus; the latter travels within the sulcus of the sigmoid sinus at the junction of the petrous and occipital bones and finally becomes the jugular bulb, which transmits the jugular vein through the jugular foramen. The arteries include the superior cerebellar artery, the anterior inferior cerebellar artery, and the posterior inferior cerebellar artery, all of which are branches of the vertebrobasilar system. These arteries are clinically significant because they are frequently responsible for neurovascular compression syndromes such as trigeminal neuralgia and hemifacial spasm.

Goals of Surgery

Skull base surgery has evolved over the past 10 years and has followed a path similar to that of other surgical disciplines. Radical techniques such as the transfacial approaches, which required translocation of large segments of the craniofacial skeleton, have yielded to more functional approaches that use natural openings such as the nostrils or minimally invasive keyhole (1 to 1.5 cm) openings through hidden incisions. In the posterior fossa, invasive approaches such as suboccipital or translabyrinthine craniotomies have been superseded by keyhole endoscopic approaches that provide a panoramic view of the posterior fossa and its contents. The common principle in this evolution has been a combination of customized and specially designed microinstruments, fiberoptic technology adapted to microendoscopes measuring 2.7 to 4 mm in diameter, and better illumination with brighter light sources such as halogen or xenon. These minimally invasive techniques translate into a reduced hospital stay, a shorter operative time, and overall superior outcomes.

Treatment

Endoscopic Supraorbital Approach

Through an incision within the eyebrow, a 1- to 1.5-cm keyhole supraorbital craniotomy is performed lateral to the frontal sinus (Fig. 2). If the frontal sinus is breached, cranialization of the frontal sinus can be performed by removing the posterior wall, stripping the mucosa, and occluding the frontonasal duct. The dura overlying the frontal lobe is incised, and cerebrospinal fluid (CSF) is drained from the anterior fossa. After adequate relaxation of the ipsilateral frontal lobe, a 0-degree endoscope is introduced into the anterior cranial fossa. The endoscope is advanced between the floor of the anterior fossa inferiorly and the inferior surface of the frontal lobe superiorly. A panoramic view of the anterior cranial fossa is obtained. The falx cerebri is visualized, and as the endoscope is angled further posteriorly, the ipsilateral olfactory bulb is clearly visualized along with the olfactory nerve. The contralateral frontal lobe and olfactory tract are usually visualized, and the endoscope can be advanced toward the contralateral side. By angling the endoscope further posteriorly, both optic nerves and the optic chiasm can be visualized. The anterior communicating artery is often seen without manipulation or retraction. By sweeping the endoscope laterally and reaching the posterior aspect of the anterior cranial fossa, the contents of the middle cranial fossa and the ipsilateral temporal lobe are visualized. The medial aspect of the middle cranial fossa between the cavernous sinus medially and the medial surface of the temporal lobe laterally is readily accessible. The

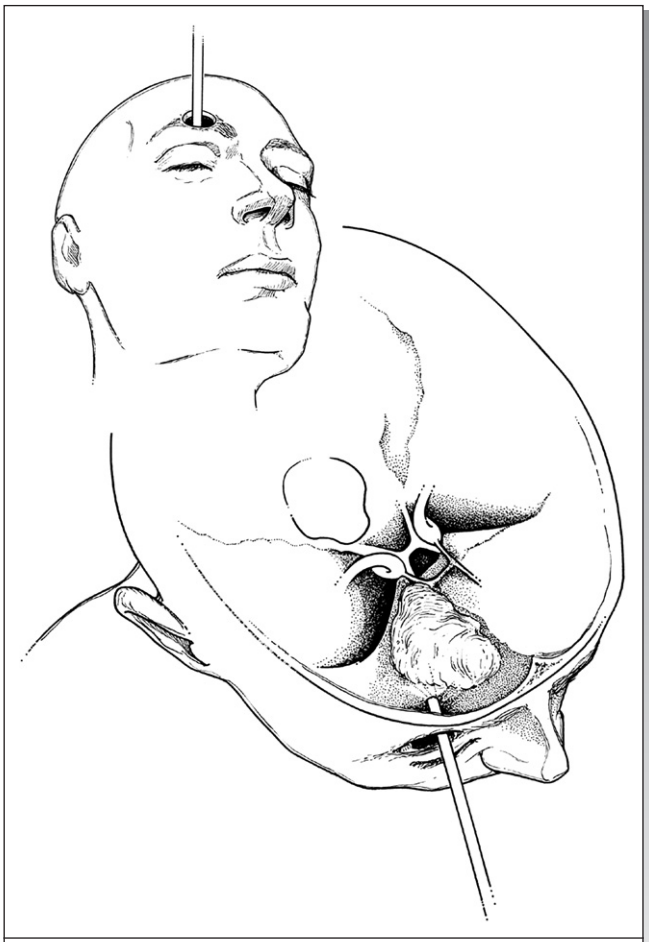

Figure 2 • Endoscopic supraorbital "keyhole" approach.

supraclinoid internal carotid arteries are visualized. This approach allows visualization and control of the contralateral supraclinoid internal carotid artery. The endoscopic supraorbital approach is ideal for the treatment of ipsilateral olfactory bulb and anterior cranial fossa meningiomas, suprasellar extensions of pituitary tumors, and Rathke's cysts or craniopharyngiomas. The approach has also been used for middle cranial fossa lesions such as sphenoid wing meningiomas and vascular lesions such as anterior communicating artery aneurysms.

Endoscopic Transglabellar Approach

Through an incision within the skin crease at the bridge of the nose, a 1- to 1.5-cm keyhole opening is made in the anterior wall of the frontal sinus; the bone flap is removed and preserved (Fig. 3). After using a diamond bur, the entire mucosa of the frontal sinuses is removed and both frontonasal ducts are identified. A small bur hole is made in the posterior wall of the frontal sinus and a 1- to 1.5-cm keyhole craniotomy is performed. The bone flap is removed and the bone is broken into small bone chips, which are used to obliterate both frontonasal ducts. Thus, an endoscopic cranialization of both frontal sinuses is completed. The dura overlying both frontal lobes is incised in a curvilinear fashion and CSF is drained from the anterior cranial fossae. After adequate relaxation of both frontal lobes, the falx cerebri is incised with modified dura scissors. The cribriform plate is visualized, fractured, and removed. A 0-degree endoscope is introduced into the midline anterior cranial fossa. The endoscope is gently advanced between the floor of the anterior cranial fossa inferiorly and both frontal lobes laterally and superiorly. A panoramic view of the midline anterior cranial fossa is appreciated. Both olfactory bulbs and olfactory tracts are visualized. The endoscope is gradually advanced between the olfactory tracts along the floor of the anterior cranial fossa.

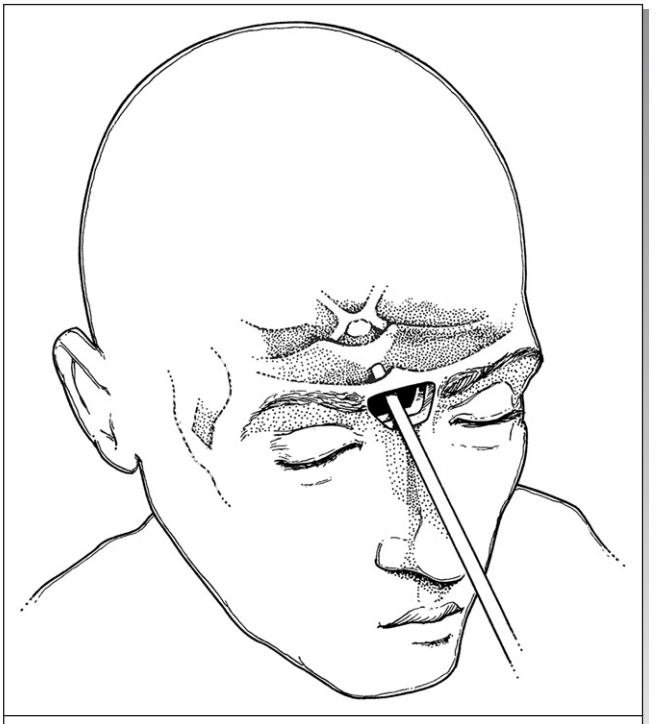

Figure 3 • Endoscopic frontal or transglabellar approach.

Midline lesions of the anterior cranial fossa such as olfactory bulb meningiomas are clearly visualized at this point. As the endoscope is advanced toward the end of the floor of the anterior cranial fossa, a suprasellar lesion such as a craniopharyngioma or a large suprasellar pituitary adenoma can be assessed. The endoscopic frontal approach allows resection of midline olfactory bulb meningiomas with direct visualization of the contralateral olfactory bulb and olfactory tract to enhance the possibility of preservation of olfaction. The approach allows resection of suprasellar lesions such as craniopharyngiomas, Rathke's cysts, optic gliomas, or large pituitary adenomas that may compress or adhere to the optic chiasm or optic nerves.

Endoscopic Transnasal Approach

A 0-degree endoscope is introduced through the right nostril; the middle turbinate is identified and outfractured (Fig. 4). The endoscope is advanced to the confluence of the middle turbinate and the nasal septum. The keel of the vomer is removed and the sphenoid sinus is entered. The mucosa of the sphenoid sinus is stripped and the septations are removed by using a combination of pituitary and Decker rongeurs. The posterior wall of the sphenoid sinus (i.e., the sella turcica) is identified. A small opening is made, and the opening in the sella turcica is enlarged from carotid canal to carotid canal laterally and from the top of the clivus inferiorly to the planum sphenoidale superiorly. The dura of the sella is electrocoagulated and incised. It is the approach of choice for the treatment of pituitary tumors and is also ideal for craniopharyngiomas and Rathke's cysts that are completely intrasellar. As the tumor is gradually resected, the medial wall of both cavernous sinuses is visualized laterally and the normal pituitary gland is similarly visualized. This approach also provides access to the suprasellar infrachiasmatic space. The introduction of a 30-degree endoscope provides improved visualization of the right cavernous sinus (on rotating the endoscope in clockwise fashion) and the left cavernous sinus (on rotating the endoscope in counterclockwise fashion). Better but limited visualization of the suprasellar space and the optic chiasm is achieved with the 30-degree endoscope. Other indications for this approach include resection of tumors in the upper third of the clivus, such as chordomas, and biopsy of metastatic and lymphoproliferative lesions of the sphenoid sinus, the clivus, and the posterior pituitary gland.

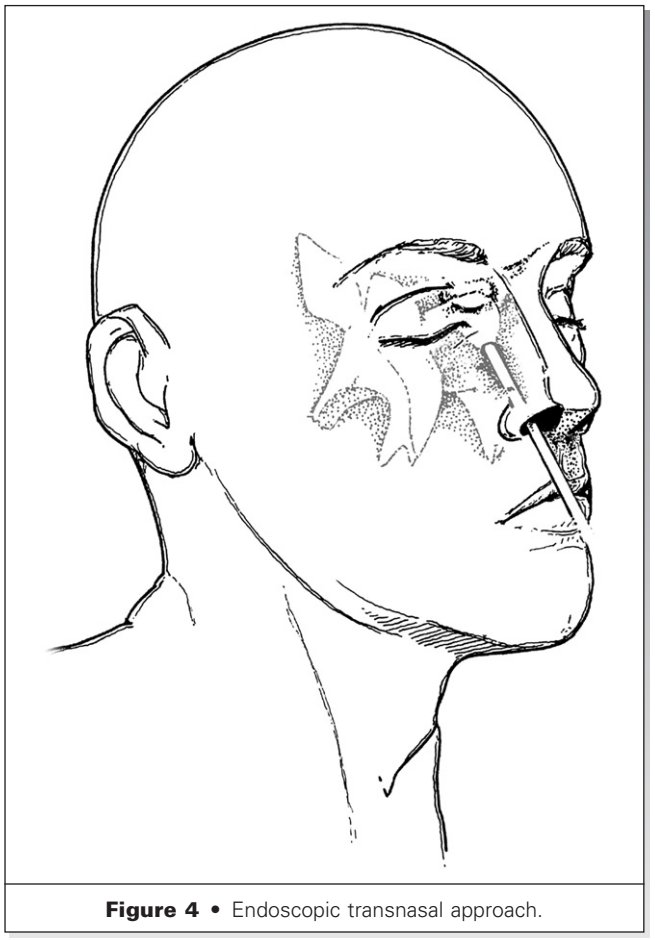

Figure 4 • Endoscopic transnasal approach.

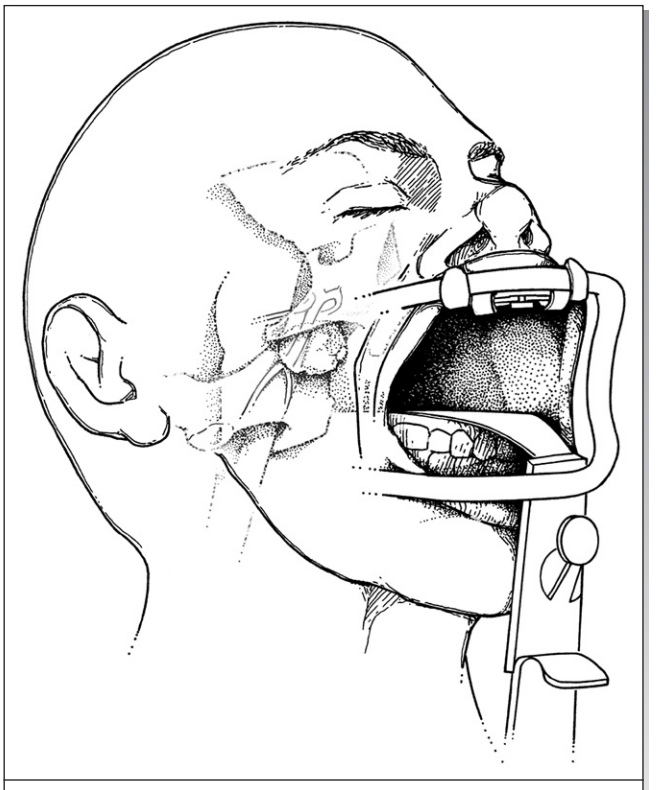

Figure 5 • Transoral/transmaxillary approaches.

Transoral Approach

For lesions extending from the lower third of the clivus through the arch of C2, the approach can be performed by making a midline incision in the soft palate, incising the mucosa of the nasopharynx in the midline, and drilling the bone of the lower third of the clivus and the arches of C1 and C2 (Fig. 5). Access is gained to midline extradural, transdural, and intradural lesions. This approach is ideal for small midline chordomas of the lower third of the clivus and for odontoid pannus in patients with rheumatoid arthritis. For more extensive lesions extending throughout the length of the clivus, either a simple LeFort I downfracture or an extended LeFort I downfracture with a midline split is necessary. The transoral approach provides excellent access to retroclival posterior fossa lesions extending throughout the length of the clivus (in the extradural and intradural space) and to malignant lesions such as chordomas and large epidermoids.

Endoscopic Retromastoid Approach

Through a 3-cm incision within the hairline behind the ear, a 1-cm retromastoid bur hole is created (Fig. 6). The dura of the posterior fossa is incised and CSF

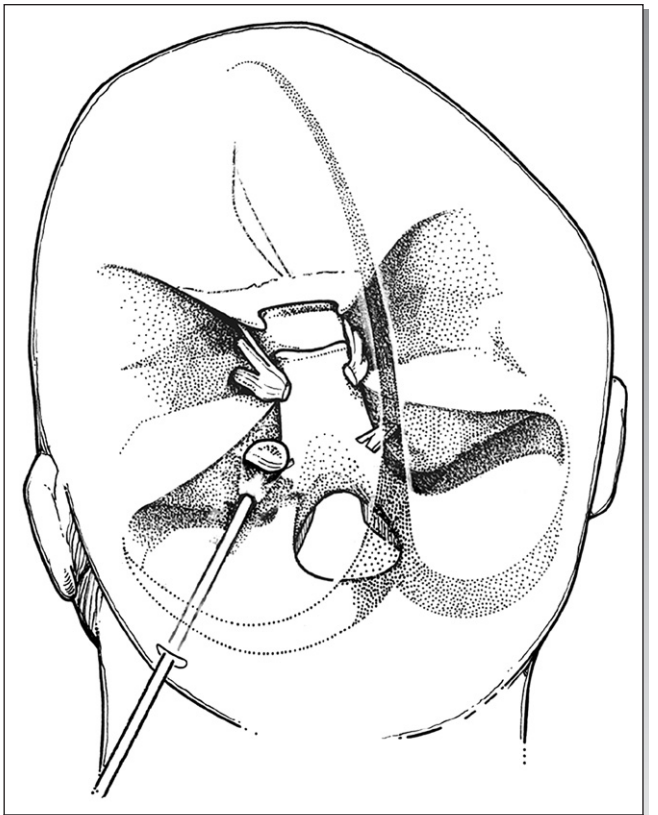

Figure 6 • Endoscopic retromastoid approach for tumors.

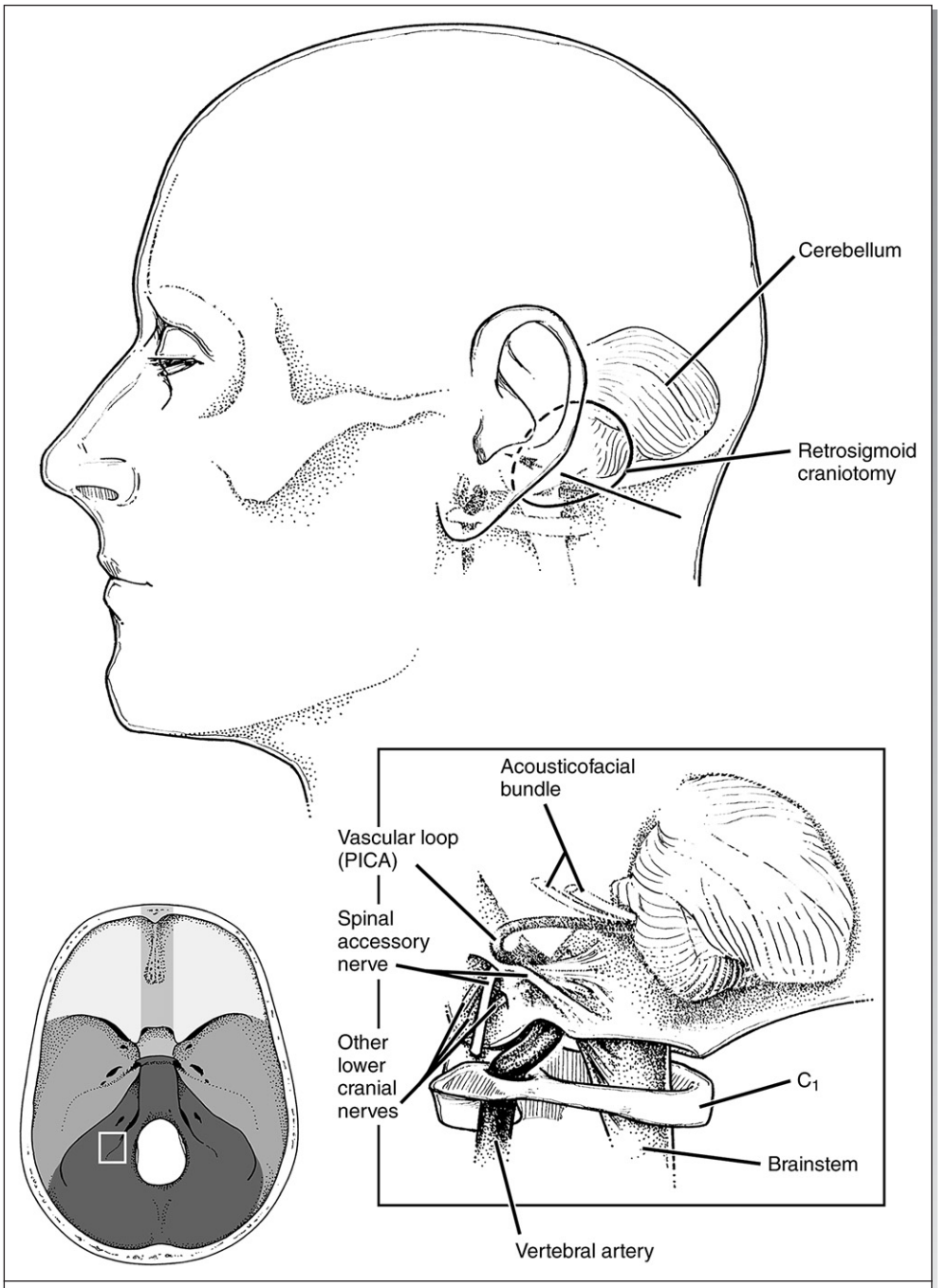

Figure 7 • Endoscopic retromastoid approach.

is drained from the posterior fossa. After adequate relaxation of the cerebellum, a 0-degree endoscope is introduced into the posterior fossa and gently advanced to the cerebellopontine angle. A panoramic view of the posterior fossa that extends from the tentorium cerebelli superiorly to the foramen magnum inferiorly is achieved. The course of cranial nerves IV, V, VI, VII, VIII, IX, X, XI, and XII within the posterior fossa is clearly visualized. The ipsilateral cerebellum and brainstem are also fully visualized.

The endoscopic retromastoid approach is the method of choice for posterior fossa skull base tumors such as acoustic neuromas, meningiomas, petrous apex cholesteatomas, and cholesterol granulomas and for neurovascular compression syndromes such as trigeminal neuralgia and hemifacial spasm (Fig. 7). The approach provides excellent visualization of the neurovascular complex at the root entry zone of the facial nerve for hemifacial spasm and the trigeminal nerve for trigeminal

neuralgia. The approach has been used in performing vestibular neurectomies for debilitating vertigo in patients with Meniere's disease. Other lesions that have been accessed through this approach are selected vertebral artery and posterior inferior cerebellar artery aneurysms.

Pearls and Pitfalls

- Advances in fiberoptic technology, microinstrumentation, and minimally invasive techniques have revolutionized the field of skull base surgery.
- Minimally invasive endoscopic approaches have resulted in shorter surgical times, shorter hospitalizations, and overall better outcomes.

SUGGESTED READINGS

Fisch U, Mattox D: Microsurgery of the Skull Base. New York, Thieme, 1988.

Jarrahy R, Shahinian HK: Surgical management of pituitary tumors. In DeGroot LJ, Jameson JL (eds): Endocrinology, 4th ed. Philadelphia, WB Saunders, 2000, pp 343-353.

Jarrahy R, Eby J, Shahinian HK: Fully endoscopic decompression of the trigeminal nerve. Minim Invasive Neurosurg 45:32-35, 2002.

Magnan J, Sanna M: Endoscopy in Neuro-otology. New York, Thieme, 1999.

Shahinian HK: Skull base surgery. In Aston S, Beasley R, Thorne C (eds): Grabb and Smith's Plastic Surgery. Boston, Little, Brown, 1995.

Facial Burn Treatment Principles

MATTHIAS B. DONELAN

Facial burn deformities are unique and complex reconstructive challenges for the plastic surgeon. Successful treatment requires sound surgical judgment and technical expertise. The surgeon must also have a complete understanding of the burn wound and its evolution. Patients and their families alike must be committed to the reconstructive process. Other disciplines must be available, including nursing, occupational and physical therapy, and psychological and social support systems. The surgeon should also have familiarity with ancillary treatment modalities such as pressure therapy, steroids, and lasers. Perhaps the most important factor for successful treatment outcomes is that both patients and surgeons clearly understand what is possible and what is not possible after facial burn injuries. Restoration of the preburn condition is not possible after serious facial burns. Facial burns of any significance result in scarring, and scars cannot be removed. Scar "excision" has always been and will always remain an "oxymoron." A scar can only be modified or traded for a scar or scars of a different variety—an important concept to emphasize to patients and to keep in mind when planning surgical intervention.

Burns constrict and deform the face by altering proportion, features, and expression. The goal of the reconstructive surgeon is to restore "normalcy" to the face as much as possible. Burns also alter the surface of the facial mask by causing changes in texture and pigmentation. However, these changes are much less important than changes in proportion, features, and expression. Removal of scars should not be the primary goal of facial burn reconstruction. A normal-looking face with scars is always preferable to a grotesque-looking face with fewer scars. In many circumstances, mature scars that result from burn injury are less conspicuous than surgically created scars or surgically transferred skin, usually because of the subtle and gradual transition between unburned skin and burn scar. The irregular borders of a burned area are often an excellent example of nature's camouflage. The overriding goal of facial burn reconstruction should be the restoration, as much as possible, of a pleasing and tension-free facial appearance with appropriate animation and expression. If this goal is kept in mind, the amount of improvement that can be achieved after devastating facial burn injuries can be truly remarkable. Failure to keep this concept in mind is probably the most common cause of iatrogenic catastrophes after facial burn reconstruction.

General Concepts

Although the main focus of this chapter is reconstruction of "late" facial burn deformities, an understanding of the acute care of facial burn victims is essential for an accurate perspective in dealing with these patients. Excision plus grafting of deep second-degree and full-thickness burns has become the standard of care. It is still controversial, however, whether such treatment is optimum for severe facial burns. Early excision with grafting is problematic because of the difficulty in diagnosing the depth of the facial burn and accurately predicting long-term prognosis from both a functional and aesthetic standpoint. The vast majority of facial burns treated conservatively with topical antibiotics heal within 3 weeks. Burns that are clearly full thickness are best treated by early excision and grafting within 7 to 10 days. Problem cases are those that have not healed within 2 to 4 weeks. Advocates of early tangential excision and grafting believe that the latter patients have more favorable healing with less secondary contracture if excision plus grafting is carried out at that time. Proponents of more conservative therapy argue that excision and grafting

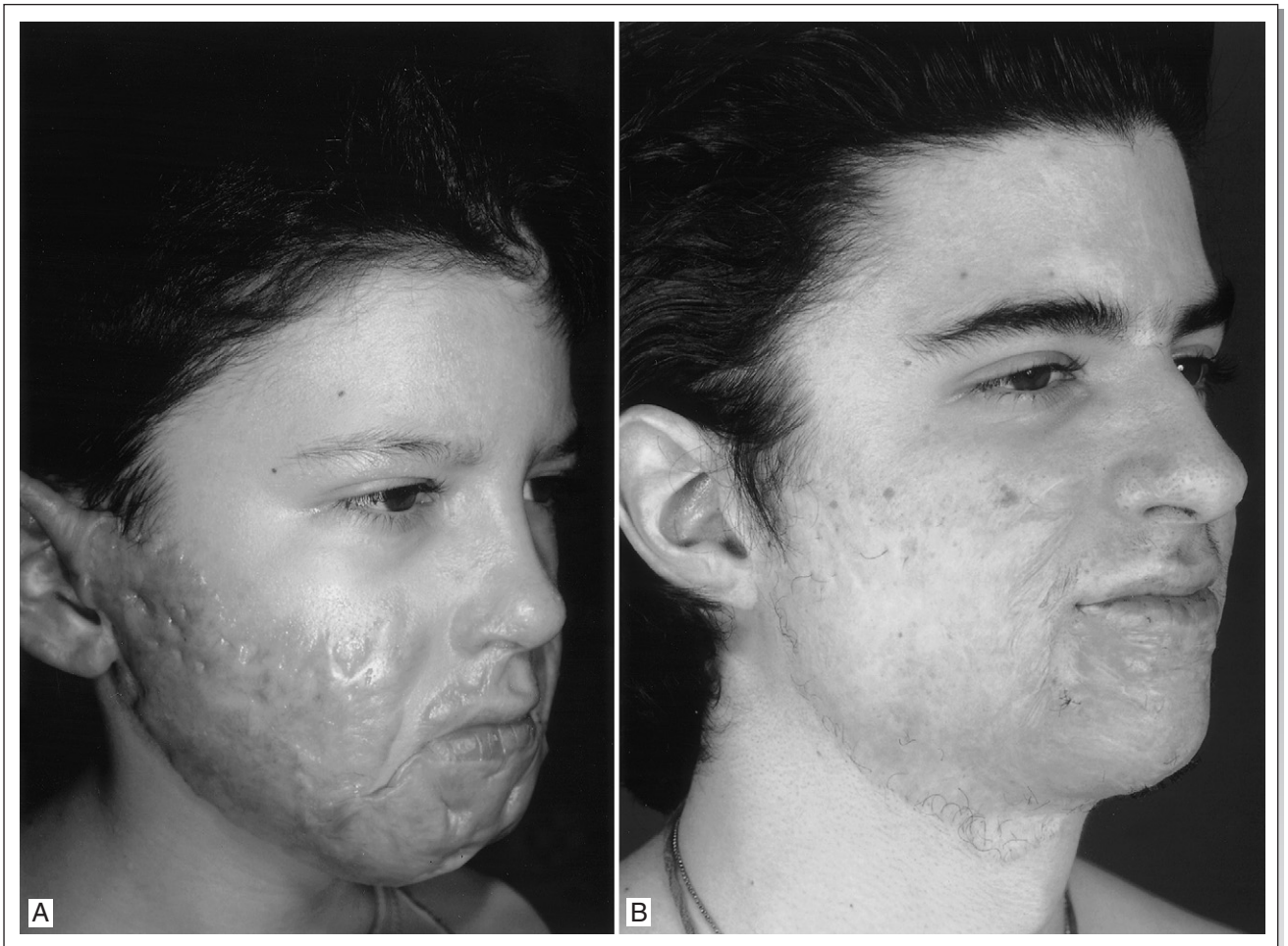

Figure 1 • A, Nine-year-old boy with diffuse hypertrophic scarring 6 months after a deep second-degree burn. **B,** Ten years later after pressure therapy, steroid injections, and multiple Z-plasties within the scars. No tissue was excised. The depth of the burn is demonstrated by the lack of beard growth.

at such an early period commit the patient to a grafted face that might otherwise have healed favorably by epithelialization alone, especially if complemented with treatment such as pressure, silicone, computer-generated facemasks, topical and intralesional steroids, and the pulsed dye laser (Fig. 1). At the present time, the majority of acute facial burns are treated expectantly in most burn centers, with early excision and grafting limited to cases in which it is clear that a full-thickness burn injury has occurred. It must be reemphasized that early excision and grafting tend to produce a more mask-like facies than when healing occurs spontaneously with favorable scar maturation.

Superficial second-degree facial burns usually heal spontaneously without scarring or pigmentary changes. Medium-thickness second-degree burns, which epithelialize in 10 to 14 days, often heal without scarring, although long-term alterations in skin pigmentation and texture are frequent. Medium to deep second-degree burns, which epithelialize in 14 to 28 days or longer, must be carefully monitored because they are prone to the development of late hypertrophic scarring. All ancillary modalities (see earlier) and judiciously timed surgery should be considered. Pressure garments are effective in suppressing and reversing hypertrophic scarring. The addition of silicone to pressure therapy has also been a significant advance. Computer-generated masks for facial pressure, although expensive, have significantly advanced the technology of pressure therapy. Use of the pulsed dye laser has been helpful in decreasing the persistent erythema of burn scars and may favorably influence maturation.

Timing of Reconstructive Surgery

Patients with facial burn deformities typically present to the plastic surgeon in one of three different circumstances. The ideal situation is one in which the plastic surgeon is involved in the patient's care from the time of the acute injury, as in a spe-

cialized burn center. The involvement may be either as the treating surgeon or, more commonly, as a consultant and occasional participant in the patient's acute care. A close relationship with the acute burn surgeons allows the plastic surgeon to have input from the beginning and can help in formulating a long-range plan involving conservation of donor sites and timing of surgery. The second situation is the patient who has received acute burn care at another facility and is referred to the plastic surgeon for reconstructive surgery. The third circumstance is the patient with established facial burn deformities many years after injury.

The timing of plastic surgical intervention falls into three separate phases: *acute*, *intermediate*, and *late*. Acute reconstructive intervention occurs during the early months after burn injury, when urgent procedures are required to facilitate patient care or prevent acute contractures from causing permanent secondary damage.

Acute Phase

Severe eyelid contractures can lead to corneal ulceration and opacification. This condition can be treated temporarily with eye ointments and scleral lenses. The use of tarsorrhaphy sutures to maintain eyelid closure is not usually effective and can lead to secondary eyelid damage. Acute release and grafting of eyelid contractures can be successfully carried out even in the presence of open wounds and should be expeditiously performed to prevent irreversible injury to the globe. Split-thickness skin grafts should be used with full-thickness skin grafts reserved for definitive reconstruction.

Perioral deformities can result in either microstomia or macrostomia. Microstomia occurs as a result of circumferential scarring at the junction between the lips and cheek. Microstomia can compromise alimentation, airway access, and oral hygiene. Oral commissure releases should be performed to correct this problem. When the contracture is localized or moderate, Z-plasties or local flaps may suffice. When the contracture is extensive, skin grafts may be required. Care must be taken to avoid iatrogenic deformities across the cheek aesthetic unit by limiting the releasing incisions.

Macrostomia is caused by the rapid contraction of open wounds or grafts in the adjacent cheek and lip and results in eversion of the upper and lower lips and lateral displacement of the oral commissures. Loss of an effective oral sphincter compromises feeding and hygiene and also causes secondary damage to the teeth and gingiva. Acute intervention with release of the contracted scars and split-thickness skin grafting of the lower or upper lips (or both) should be performed expeditiously, even in the presence of open wounds. Definitive reconstruction can be achieved at a later period.

Severe cervical contracture with anterior flexion deformities or even cervicomental adhesion can compromise airway access and cause severe tension on the facial soft tissues. Acute release and skin grafting are indicated to facilitate patient care and minimize the effect of excessive tension on facial hypertrophic scarring.

Intermediate Phase

The intermediate phase of plastic surgical intervention is best described as scar manipulation designed to influence the healing process favorably. Treatment in this phase is evolving rapidly and holds much hope for the future. It is usually recommended that definitive reconstructive surgery be performed only after facial scars and skin grafts are mature, soft, and supple, which takes at least a year and frequently many years. However, well-timed and well-conceived surgery to favorably influence the scar maturation process can be beneficial. After facial wounds are closed by either epithelialization or grafting, the maturation process is influenced by multiple factors. Perhaps the most important factor is the amount of tension present in the healing soft tissues of the face. Surgical intervention to decrease tension and favorably alter the orientation of scars can improve facial scars during the first year or two after injury. Surgery to relieve tension may consist of either Z-plasties or appropriately located releases and skin grafts. Split-thickness skin grafts should be used when the location of the graft renders it inconspicuous or large amounts of skin are required to decrease tension. Such treatment is frequently indicated in patients with neck contractures. Tension from the neck must be eliminated to allow for favorable maturation of facial scars. The use of full-thickness skin grafts during the intermediate period should be limited to situations in which definitive repair is being carried out. Steroids can be helpful but must be used sparingly to avoid atrophy and irreversible skin changes. As noted previously, the pulsed dye laser can be used to reduce erythema.

Late Phase

Late-phase reconstructive surgery includes all other circumstances in which the deformities are essentially stable; the scars and grafts are mature, soft, and supple; and elective, staged interventions can be planned and executed.

The transition from an acute facial burn with open wounds to an established deformity is prolonged and varies greatly from patient to patient. After the acute phase has passed, the patient and family often press for reconstruction. This is usually the wrong thing to do. As Gilles said, "time is our most faithful ally," and in probably no circumstance is this more true than in the evolution of facial burn deformities. Hypertrophic scars that are problematic at a year or longer may become flat and soft, particularly with appropriate manipulation and, because of the subtle transition from unburned to

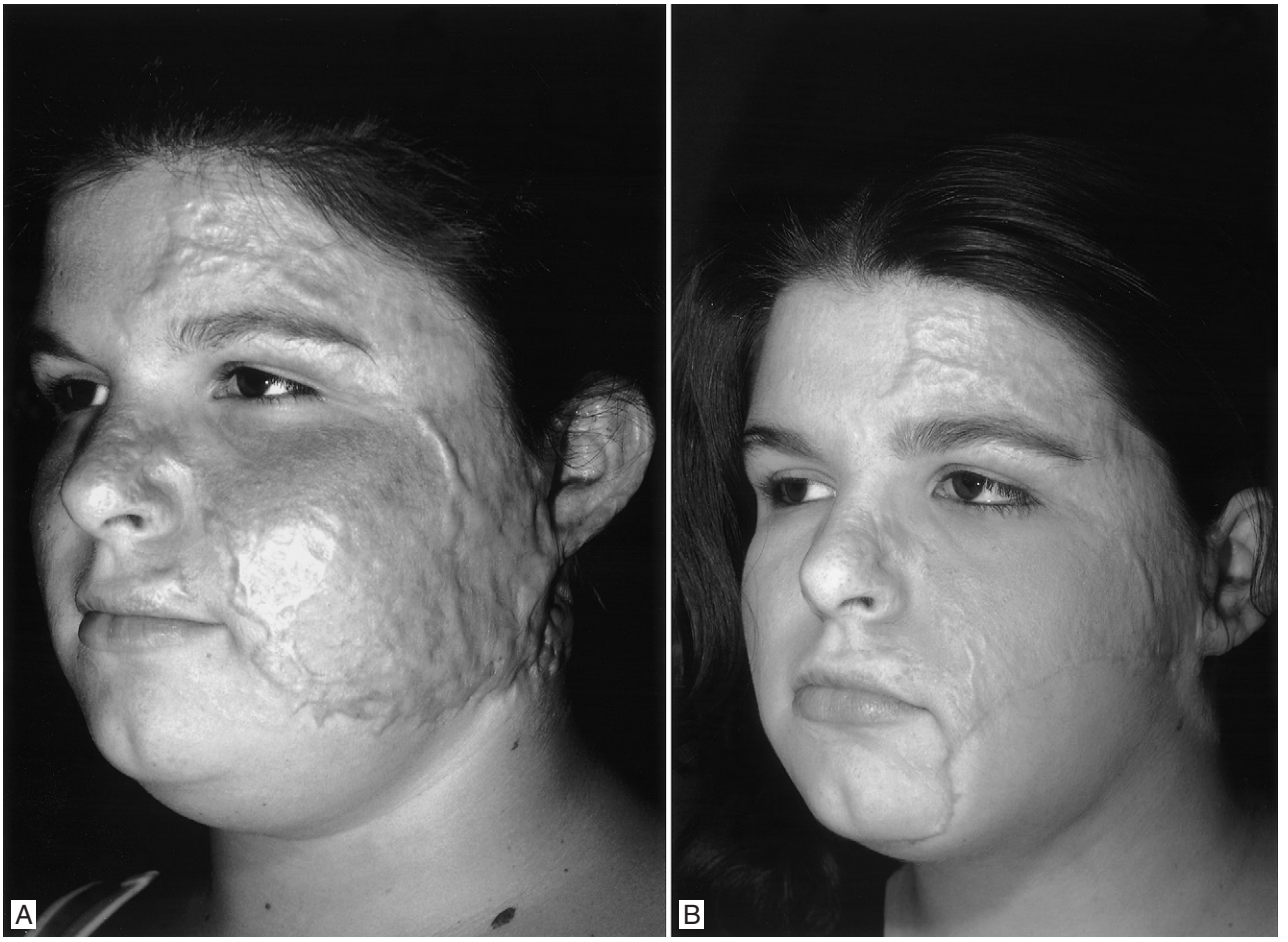

Figure 2 • A, Fourteen-year-old girl 9 months after a second-degree burn with mild hypertrophic scarring of the cheek. **B,** Partial excision and skin flap advancement created lower eyelid ectropion, lower lip dystopia, and a scar with a dog-ear at the oral commissure. Note the excellent appearance of the superior burn scar, which was allowed to mature without surgical intervention.

burned tissue, they may become less conspicuous. The surgeon must resist the desire of the patient and family to "remove the scars" as soon as possible. Succumbing to the patient's requests for surgery may lead to unfortunate outcomes. In the example illustrated in Figure 2, a second-degree burn that was healed at 21 days became mildly hypertrophic over a 9-month period. The lower half of the burn was partially excised and a cervical flap elevated. The upper half of the burn scar healed extremely well despite increased tension. The iatrogenic deformities from the flap procedure included lower eyelid ectropion, distortion of the lips, loss of definition of the jaw line, and a dog-ear deformity at the oral commissure.

During the intermediate period when scars and grafts are maturing, a prospective plan for reconstructive surgery should be outlined with the patient and family. Genuine enthusiasm on the part of the surgeon and other members of the team regarding successful reconstruction is quickly sensed by patients and can make them significantly more optimistic. The actual sequence of surgery, however, frequently deviates from the original plan. Attitudes and circumstances may change as the reconstructive process unfolds. As the patient improves, enthusiasm for further surgery may decrease, whereas some patients become committed to operations for many years. It is important that the patient-surgeon relationship be strong and supportive. When the patient knows that the surgeon is available and committed to the best possible final outcome, it can help in reaching the endpoint of the reconstruction.

Fundamental Principles

PROPORTION. The importance of precise diagnosis in the treatment of facial burn deformities cannot be overemphasized. As Goethe said, "The most difficult thing of all to see is that which lies directly in front of your eyes." Analyzing a facial burn requires doc-

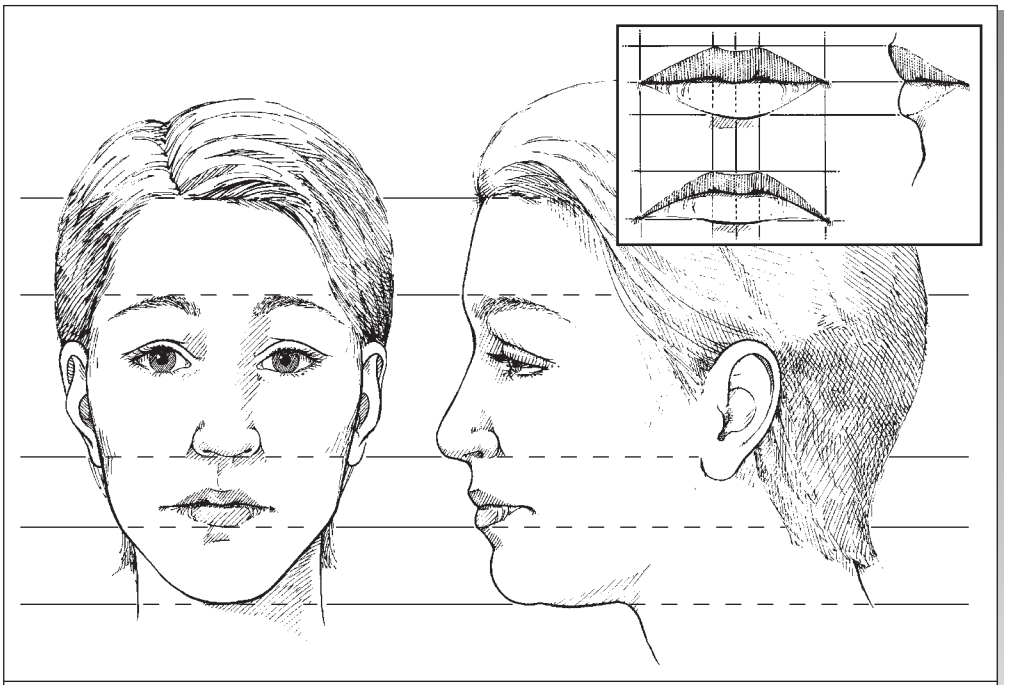

Figure 3 • The face is roughly divided in thirds (see text). The vermilion-cutaneous junction of the lower lip lies halfway between the nose and lower part of the chin. The upper lip is wider than the lower and protrudes beyond the lower in lateral view. Restoration of normal proportion is essential for successful facial burn reconstruction. (Redrawn from Tolleth H: Concepts for the plastic surgeon from art and sculpture. Clin Plast Surg 14:585-598, 1987.)

umentation of facial proportions (Fig. 3). A normal face is divided into three segments: anterior hairline to the glabella, glabella to the base of the columella, and base of the columella to the menton. The length of the nose fills the middle third vertically, and the lower third is divided in half by the vermilion-cutaneous junction of the lower lip. In anterior view, the upper lip is wider than the lower lip. In lateral view, the upper lip precedes the lower lip. The lower border of the face is defined by the jaw line. These basic relationships can be profoundly altered by burn injury (Fig. 4). Unless these proportions are restored in the reconstruction, the face will be distorted regardless of all other factors.

CONTRACTURES. Burns cause tissue loss, wounds heal with contraction, and contractures result. Contractures can be of either the intrinsic or extrinsic variety. *Intrinsic* contractures are those that result from loss of tissue in the area of the contracture with subsequent distortion of the part. In *extrinsic* contractures, the loss of tissue is at a distance from the affected area, but the distorted structures, such as eyelids, lips, and so forth, are not themselves injured. Corrective measures must be directed at the cause of the contracture. It is helpful to minimize the amount of skin and scar that are excised when dealing with facial contractures. Because of the beneficial effects of releasing skin tension, many scars mature favorably after a release has been carried out. Healed second-degree burns under tension may be unattractive but, when restored to a tension-free state, are often superior in appearance to any replacement tissue. Minimizing excision also decreases the amount of new skin that must be provided.

TENSION. All facial burns of medium second-degree depth or deeper result in tightening of the facial skin even if there is only minimal associated scarring. Gilles and Cuthbert referred to "the invisible skin loss" after burns on the dorsum of the hand, and a similar situation exists in the face after burn injury. This type of tension may be evident as deforming contractures of mobile soft tissues such as eyelids or lips, but it may be only subtly apparent as compression of the soft tissue contours. When facial burn injury results in diffuse loss of skin, reconstructive operations that excise large areas of scar or skin are potentially adding to the underlying clinical problem. *Tight faces are never attractive; relaxed scars are happy scars.*

DONOR SITES. A major reason for the difficulty in reconstructing facial burns is that they rarely occur in isolation and are often associated with extensive burns over the entire body, particularly the cervicopectoral area. Intelligent selection of procedures along with judicious use of graft or flap donor sites is critical.

AESTHETIC UNITS. The concept of facial aesthetic units, originally conceived as the ideal approach for resurfacing the face after burn injury, is emphasized

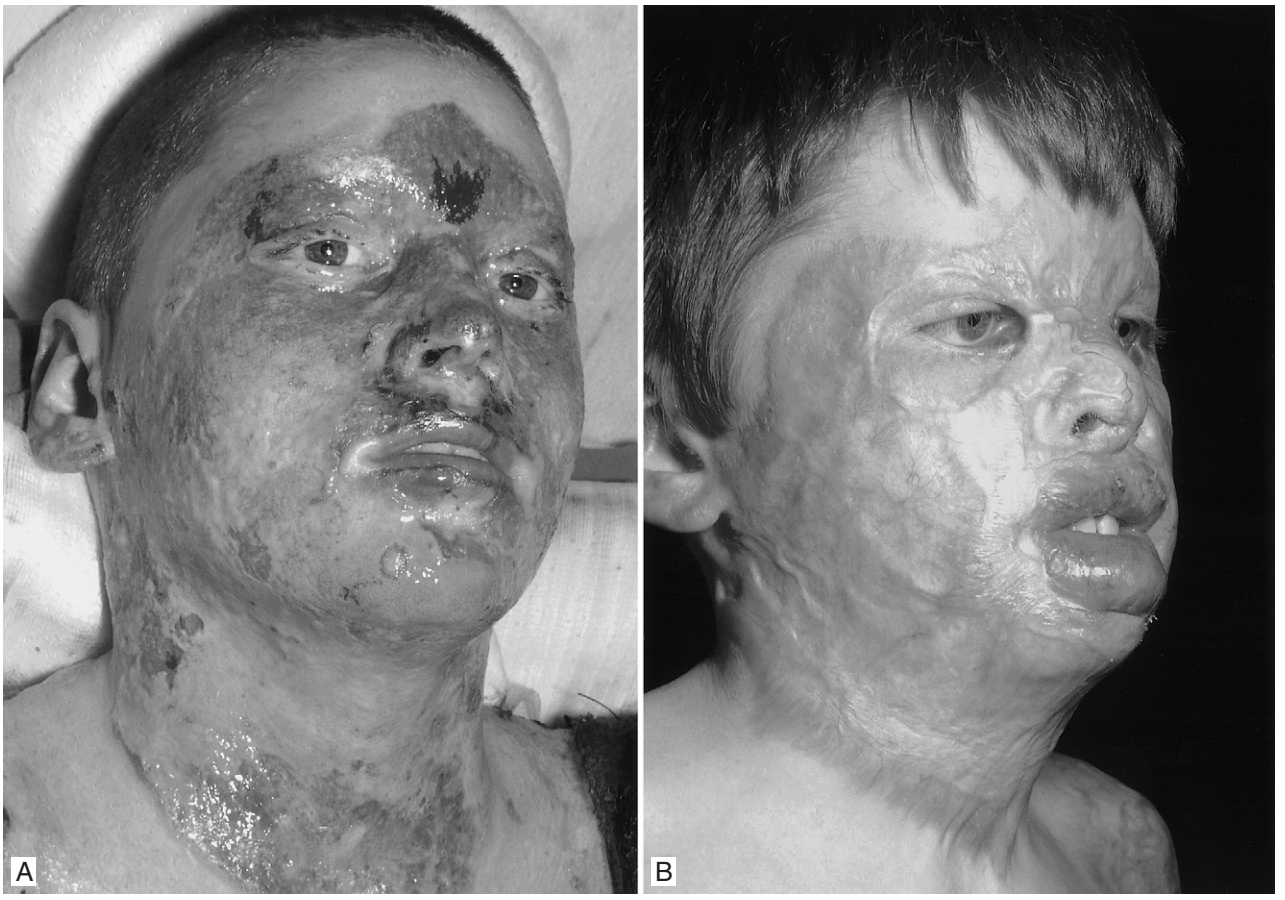

Figure 4 • A, Three weeks after a deep second-degree burn, the facial features and proportion are normal. **B,** Six months after the burn, contraction has created the stigmata of facial burn injury and disturbed facial proportions.

in all writings about facial burns; however, it should not supersede common sense. When small, unburned islands of skin are in an aesthetic unit that requires resurfacing, they should be sacrificed. Otherwise, excision of normal skin is rarely indicated. All burned faces are, of necessity, a mosaic. Scar revision with Z-plasties can do wonders to camouflage scars in a burned face. Mosaic faces that are proportional, well contoured without tension, and expressive appear better in daily life than in photographic images. Relentless pursuit of the elimination of burn scars is an elusive goal that can lead to unfortunate outcomes.

Z-PLASTY. Z-plasty is an important part of the surgeon's armamentarium in dealing with facial burn deformities. Z-plasty lengthens linear scars by recruiting relatively lax adjacent tissue. Z-plasty, however, is much more than a simple geometric exercise in lengthening a scar. If executed properly, it causes a salubrious alteration in the physiology of contracted and hypertrophic scars when carried out within the "scar-infiltrated tissues" rather than after excision of the scars. This phenomenon is related to the immediate and continuing breakdown of collagen that occurs in hypertrophic scars after the relief of tension. The improvement in the appearance of the scars is often dramatic (see Fig. 1).

SKIN GRAFTS. Skin grafts are pivotal in facial burn reconstruction. Decisions regarding donor site selection, thickness (split thickness versus full thickness), timing of intervention, and postoperative management often determine the success or failure of facial burn reconstruction. A few generalizations may be helpful. Split-thickness skin grafts contract more, have a greater propensity to wrinkle, and remain shiny with a "glossy" look. Their application should be restricted in the central, prominent areas of the face unless limited availability of donor sites requires use of these grafts. Split-thickness skin grafts are excellent for upper eyelid release and resurfacing and for lower eyelid release and grafting when the contractures are severe. Split-thickness skin grafts are best used for releasing contractures around the periphery of the face to relieve tension and enhance scar maturation in the more important central areas. Hyperpigmentation is a frequent problem in dark-skinned patients, particularly those of African descent.

The full-thickness skin graft is a reliable workhorse in facial burn reconstruction. When resurfacing the broad, conspicuous areas of the face such as the cheeks, lips, and dorsum of the nose, consideration should always be given to using full-thickness skin grafts. The missing or damaged skin parts in the vast majority of severe thermal burns are the epidermis and the dermis—components provided by full-thickness skin grafts. The subcutaneous fat and deeper structures may be compressed or distorted, but it is rare that they are injured. Facial burn contractures "eat skin." One must be sure to provide adequate donor tissue when performing a definitive operation with full-thickness grafts. Contractures must be overcorrected and large grafts used. Postoperative management with conformers and pressure is essential.

FLAPS. Flaps, with or without tissue expansion, can be useful for facial burn reconstruction. The thickness of skin flaps from all donor sites is greater than the thickness of normal facial skin, and it tends to mask normal facial movement and expression. Because facial skin is tight after burn injury and because flaps that are thick tend to contract when transferred, they can compress or obscure the underlying facial contours. The size of flaps required to relieve contractures can be underestimated. Transposing or advancing flaps from the neck and chest to the face can create extrinsic contractures that adversely affect facial appearance. Expanded flaps are even more dangerous in this regard. Contractures with a downward vector create a "sad" facial appearance that is distressing to patients. Cervicopectoral flaps have the best match in color and texture to facial skin. Distant flaps, whether transferred by traditional techniques or microsurgery, share the common deficiency of poor color and texture match. Probably the most appropriate use of flap tissue for facial burn reconstruction is when one side of the face is severely burned and the contralateral side is completely unburned. Despite these potential pitfalls, flaps can be helpful, particularly in resurfacing the large flat aesthetic unit of the cheek. When transferred with tension, however, the results can be disastrous (see Fig. 2).

The principles of facial burn reconstruction are outlined in Table 1.

TABLE 1 Principles of Facial Burn Reconstruction

Time improves scars
Scar excision is an oxymoron
Scars are the surgeon's friends
Relaxed scars are happy scars
Tight is never right
A normal-looking face with scars is better looking than a grotesque face with fewer scars

TABLE 2 Stigmata of Facial Burns

Lower eyelid ectropion
Foreshortened nose with alar flaring
Short, retruded upper lip
Lower lip eversion
Lower lip inferiorly displaced
Flat facial features
Loss of jaw line definition

Evaluation and Treatment

Successful facial burn reconstruction is based primarily on the overall strategy and an understanding of the problem, *not* specific surgical techniques. The best reconstructive plan is usually a judicious combination of skin grafts and flaps, followed by appropriate scar revision.

Deep second-degree and third-degree burns heal by contraction and epithelialization. The deeper the injury, the more the healing takes place by contraction. The changes in facial appearance after a deep burn injury are dramatically demonstrated in Figure 4. Three weeks after a deep second-degree burn, the patient's face remains essentially normal in appearance before contraction. Six months later, contraction has deformed the face in a pattern common to all severe facial burns. These changes can be referred to as the stigmata of facial burn injury and are listed in Table 2. The eyelids are distorted with ectropion and the nose is foreshortened with alar flaring. The upper lip is shortened and retruded with loss of philtral contour, and the lower lip is everted, inferiorly displaced, and wider than the upper lip in frontal view. The soft tissues of the face and neck are drawn into the same plane, with resultant loss of jaw line definition. The severity of the changes is proportional to the severity of the injury and may be focal or diffuse.

It is helpful to think of patients with facial burn deformities as falling into two fundamentally different categories (Table 3). Type I deformities consist of essentially normal faces that have focal or diffuse burn scarring with or without associated contractures. Type II deformities make up a much smaller number of patients who have "panfacial" burn deformities with some or all of the facial burn stigmata. These categories are not rigidly defined, and some patients clearly do not fit neatly into one or the other. Nonetheless, understanding the fundamental

TABLE 3 Facial Burn Categories

Type I: Essentially normal face with focal or diffuse burn scarring with or without contractures
Type II: Panfacial burn deformities with some or all of the stigmata of facial burns

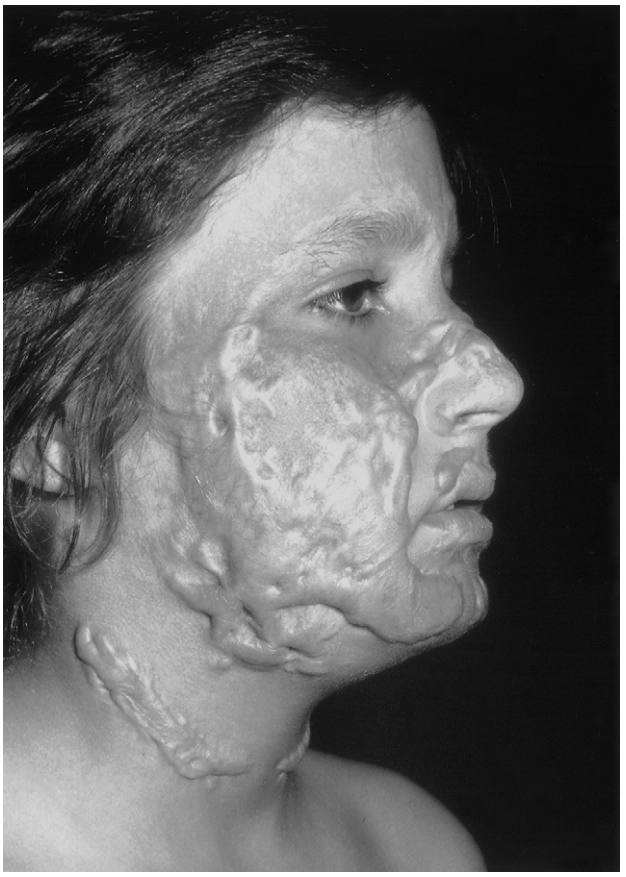

Figure 5 • Patient with type I facial burn deformities (see text).

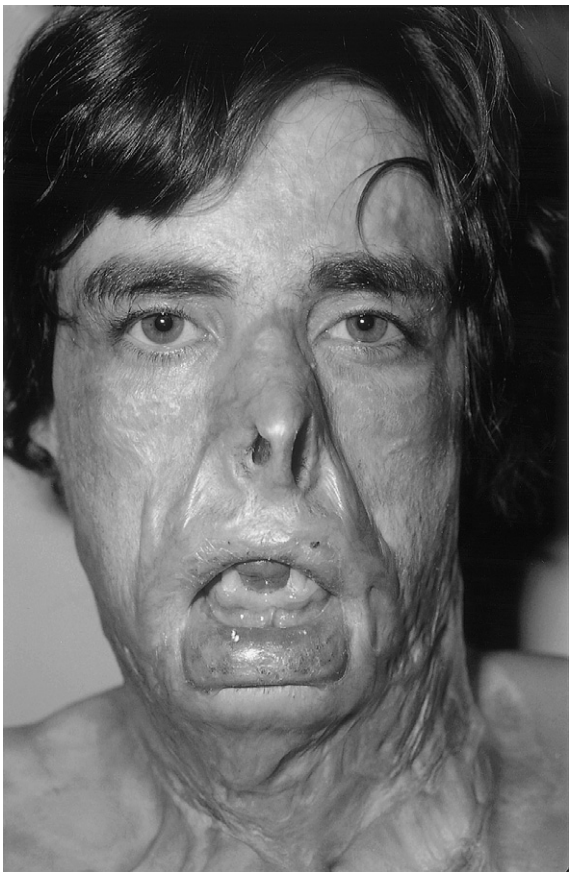

Figure 6 • Patient with type II or panfacial burn deformities (see text).

differences between the two groups of patients can help to define the treatment goals and aid in selecting the most appropriate methods of reconstructive surgery.

TABLE 4 Treatment Principles for Patients with Facial Burns

TYPE I DEFORMITIES: BASICS

Do no harm!
Do not sacrifice facial appearance to "excise scars"
Do not trade deformities
Recognize that "cure" is defined as "a satisfactory outcome"
Remember that when tension from skin loss is the problem, excision is often *not* the best solution

TYPE II DEFORMITIES: BASICS

Restore facial proportion
Restore the position and shape of facial features
Provide adequate soft tissue to accomplish restoration
Do not be a slave to aesthetic units
Mosaic faces look much better in real life than in photos

Figure 5 shows a patient with facial burn scarring who falls into the type I category. The surgical goals when treating type I deformities are very different from those appropriate for treating type II deformities (Table 4). Because type I patients have essentially normal faces, one must be certain that surgical intervention does not adversely affect overall facial appearance. *The surgeon must not fall into the trap of sacrificing overall facial appearance to "excise scars."* One should not trade postburn deformities for iatrogenic deformities (see Fig. 2). Surgery should be performed only when it is reasonably certain that it will improve facial appearance. Recognizing that "complete cure" after a facial burn injury is not possible is a helpful moderating influence on both patient expectations and surgeons' desire for heroic outcomes. It is important to keep in mind that patients who have sustained facial burn injury have an underlying tension associated with their skin coverage that is not always obvious. Scar excision and primary closure may cause more secondary changes than expected. Adequate time must pass to allow full scar maturation to occur. Scar revision with Z-plasties and local flaps is often the best option for type I patients. The pulsed dye laser is helpful in decreasing erythema. Full-thick-

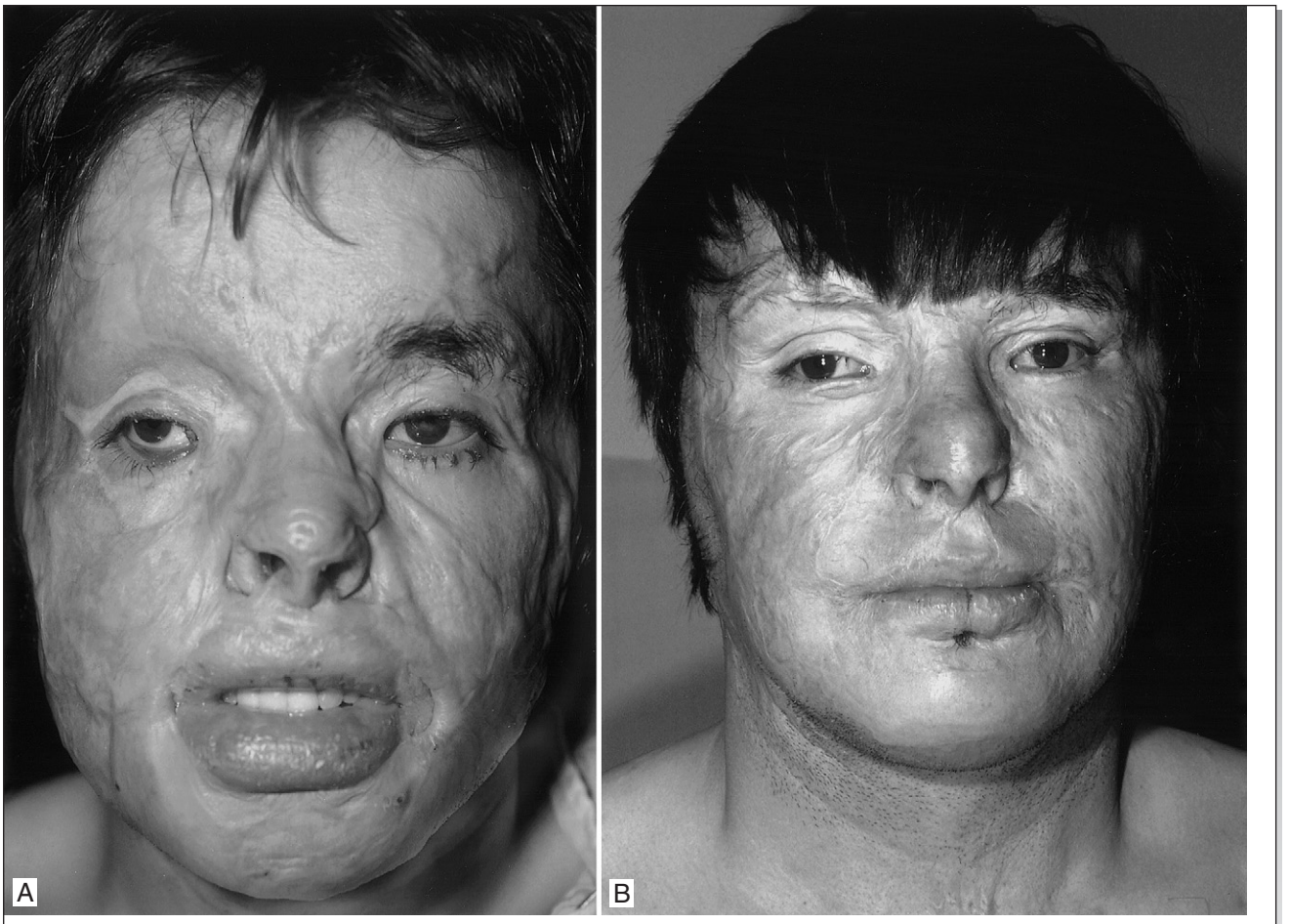

Figure 7 • A, Panfacial burn deformity in a 26-year-old firefighter. **B,** Seven years later. The reconstructive surgical sequence consisted of lower eyelid release and the application of split-thickness skin grafts, upper eyelid release and split-thickness skin grafts, lower lip–chin full-thickness skin grafts with a chin implant, full-thickness skin graft in the upper lip, a triangular fossa graft to the philtrum, and right eyebrow reconstruction with composite scalp grafts.

ness skin grafts are excellent for correction of focal contractures. Resurfacing of aesthetic units and major flap transpositions are rarely indicated.

Type II deformities present a completely different clinical situation (Fig. 6). The surgical goals are primarily restoration of normal facial proportion and, as much as possible, restoration of the position and shape of facial features (see Table 4). Correction of intrinsic and extrinsic contractures requires large amounts of skin and should be carried out in staged fashion. The sequence of operations is usually as follows: eyelids, lower lip-chin complex, upper lip, cheeks, nose, and whatever else remaining needs attention. As each deformed area is improved, the accompanying relief of tension has beneficial effects on adjacent areas. Excision of normal skin or elastic second-degree burned skin is rarely indicated. The final stage of reconstruction is scar revision to recontour and blend junctional scars. When normal facial proportion has been restored and the facial features have been returned to a more normal location and shape without tension, significant improvement in facial appearance can be achieved (Fig. 7).

All faces are a mosaic of colors, textures, wrinkles, and irregularities despite the imaginary ideal of perfect skin color and texture. In a face that has sustained major burn injury, the mosaic of scars, grafts, wrinkles, pigmentary abnormalities, and other flaws can be aesthetically acceptable provided that the facial features are normally shaped, well positioned, and sufficiently mobile for appropriate facial expression (Fig. 8). The use of cosmetics can be helpful in camouflaging areas of disparate pigment and texture, especially in females.

Pearls and Pitfalls

- In patients with facial burns without distortion of features, it is best to avoid large resurfacing procedures.

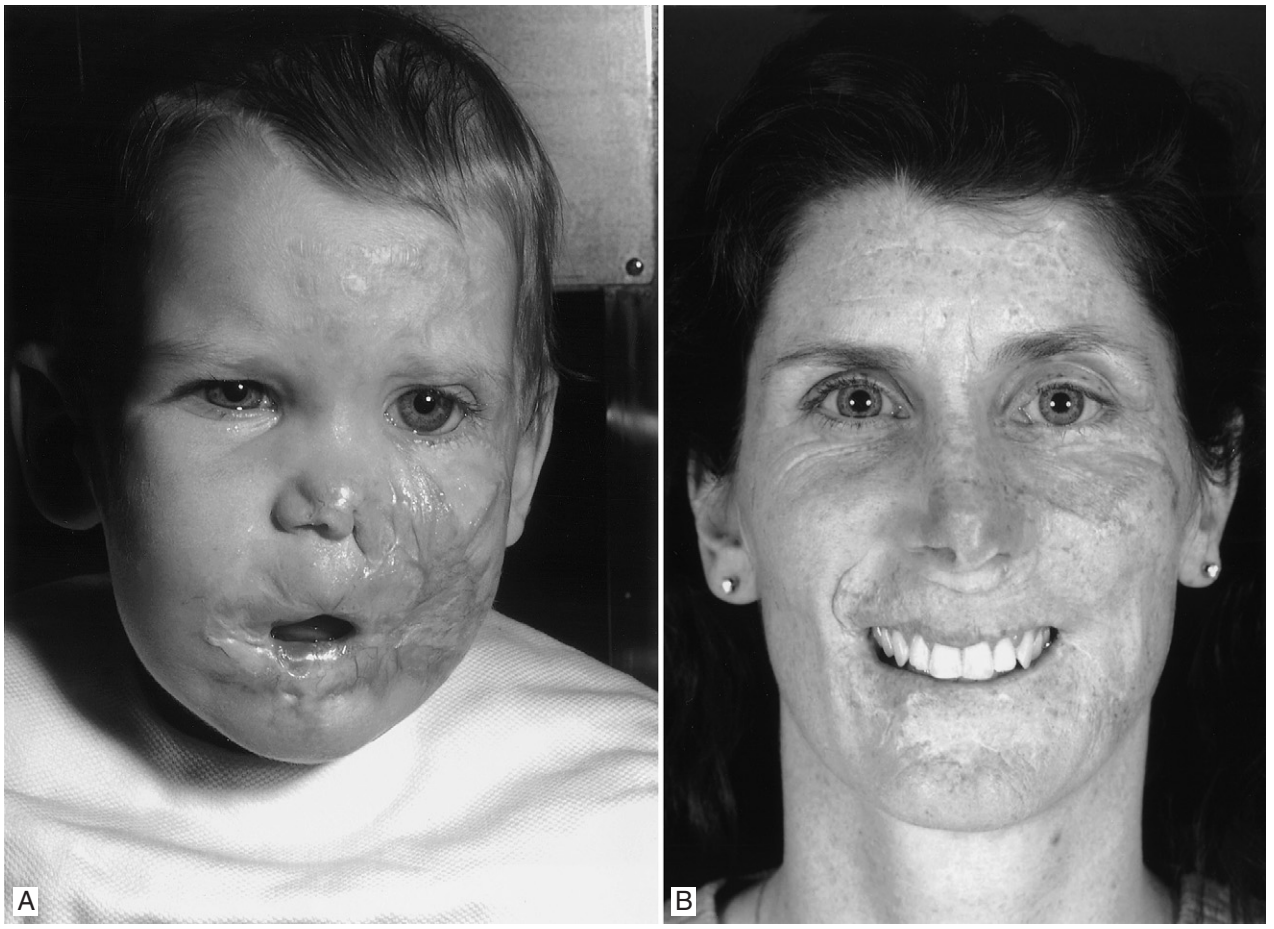

Figure 8 • A, Nineteen-month-old girl after a severe chemical burn injury with complete loss of the lips and left lower eyelid ectropion previously treated with excision of scar and application of skin grafts. **B,** Appearance 26 years later after full-thickness skin grafts applied to the left lower eyelid, left cheek, and upper and lower lips. Note that her face is attractive, proportional, and normally expressive.

- If the surgical plan achieves normal facial proportion, eliminates tension, and restores the shape and location of facial features, satisfactory results can be obtained.
- The development of burn centers has concentrated expertise and created a clinical experience that yields improved results.

SUGGESTED READINGS

Cole J, Engrav LH, Heimbach DM, et al: Early excision and grafting of face and neck burns in patients over 20 years. Plast Reconstr Surg 109:1266-1273, 2002.

Davis J: The relaxation of scar contractures by means of the Z-, or reversed Z-type incision: Stressing the use of scar infiltrated tissues. Ann Surg 94:871-884, 1931.

Donelan M, Silverman RP: Full-thickness skin grafts for elective facial burn reconstruction; review of 237 consecutive cases. J Burn Care Rehabil 23(2):S68, 2002.

Engrav L, Donelan MB: Face burns: Acute care and reconstruction. Op Tech Plast Reconstr Surg 4:53-85, 1997.

Engrav LH, Heimbach DM, Walkishaw MD, Marvin JA: Excision of burns of the face. Plast Reconstr Surg 77:744, 1986.

Feldman J: Facial burns. In McCarthy JG (ed): Plastic Surgery. Philadelphia, WB Saunders, 1990, pp 2153-2236.

Gilles H, Cuthbert JB: Plastic surgery of burns of the hand. Med Annu 61:259, 1943.

Longacre J, Berry HK, Basom CR, Townsend SF: The effects of Z-plasty on hypertrophic scars. Scand J Plast Reconstr Surg 10:113–128, 1976.

McIndoe A: Total facial reconstruction following burns. Postgrad Med 6:187, 1949.

Facial Paralysis

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Facial paralysis refers to a condition in which all or portions of the facial nerve are paralyzed. It is a devastating condition that has functional, aesthetic, and psychosocial consequences. The facial nerve controls the mimetic muscles that support the face at rest and provide movement to the forehead, eyelids, cheeks, mouth, and neck. In unilateral cases, there is asymmetry at rest that increases with facial expression. In bilateral cases, the expressionless face carries no emotion, and patients are often treated as mentally rather than physically challenged. The muscles that frequently require reconstructive management are the frontalis, orbicularis oculi, zygomaticus major, levator labii superioris, orbicularis oris, and depressor labii inferioris.

Etiopathogenesis

Facial paralysis can be classified as congenital or acquired. Congenital causes may be genetic, developmental (craniofacial microsomia), or intrapartum from birth canal or forceps compression. One of the most common causes of congenital paralysis is Möbius' syndrome, often a combined bilateral 6th and 7th cranial nerve paralysis. However, Möbius' syndrome can be unilateral and is frequently associated with dysfunction of the 9th, 10th, and 12th cranial nerves. In addition, cases of isolated nonsyndromic paralysis occur.

Causes of acquired paralysis include trauma, tumor, inflammatory disease, and neuromuscular disease. The most common cause of facial paralysis is inflammatory—Bell's palsy, probably related to a viral infection. Approximately 80% to 90% of persons with Bell's palsy recover from their paralysis without a residual deficit.

In each case, the paralysis may affect all branches of the facial nerve (panfacial) or only individual branches. In any particular region of the face, the paralysis may be partial or complete. In addition, the paralysis may be bilateral. The most common causes of bilateral paralysis are Möbius' syndrome, brainstem tumors, bilateral acoustic neuromas, and bilateral temporal bone fractures.

Anatomy

Muscles of Facial Expression (Fig. 1, Table 1)

The frontalis muscle is a broad sheet-like muscle that originates from the galea, passes forward over the forehead, and inserts into the orbicularis oculi, procerus, and corrugator supercilii. It elevates the brow and maintains the position of the eyebrows. When frontalis function is lost, the eyebrow descends and loses its ability to convey emotions. Ptosis of the eyebrow is problematic in older patients because it leads to marked asymmetry of the eyebrows and may obstruct upward gaze. At rest, a person with a depressed eyebrow appears unhappy—an appearance that is accentuated with animation.

The orbicularis oculi is a thin muscle that acts as a sphincter for the eyelids. The pretarsal and preseptal portions close the eye both during volition and with blinking. Eye closure protects the eye from trauma and desiccation. Blinking spreads the tear film over the cornea and maintains a healthy corneal epithelium. With paralysis, the patient has dryness, conjunctivitis, and tearing. The eyelid margins are wider than those of the opposite eye, and there is no movement with facial expression. Older patients with loss of lower lid support have ectropion with scleral show.

The musculature of the cheeks and lips controls lower facial expression and is important in eating, drinking, and speaking. The two groups of muscles are the lip retractors, including the zygomaticus

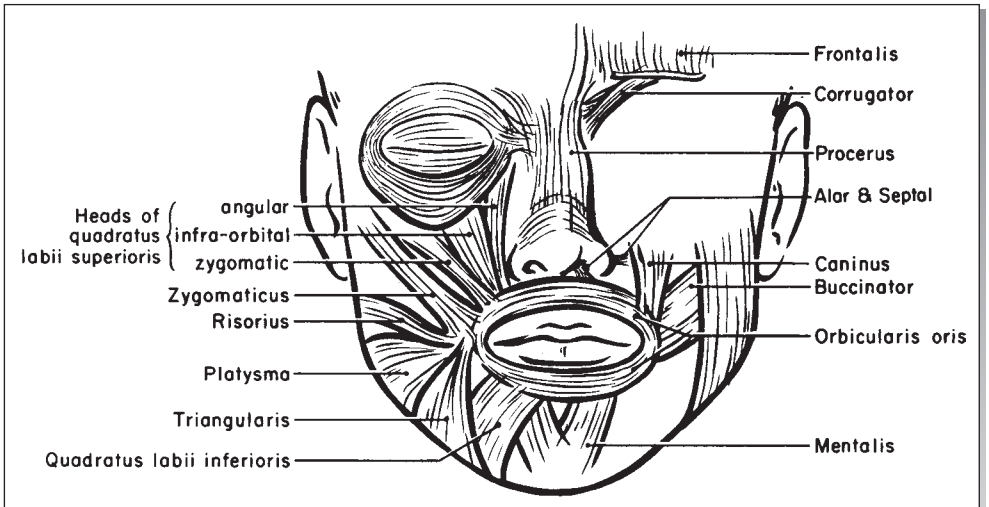

Figure 1 • Muscles of facial expression. The deeper mimetic muscles are on the right side, and the more superficial are on the left. (This drawing does not show the interweaving fibers of the various muscles to form the orbicularis oris, but regards them as individual muscles seemingly attached to a central raphe. Close examination at the time of surgery shows that the muscles interweave to form the orbicularis oris, which is actually a composite muscle.) (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990.)

TABLE 1 Muscles of Facial Expression

Scalp

Frontalis: raises brows and wrinkles forehead as in surprise
Occipitalis: with frontalis, in raising eyebrows (insignificant physiologic function)

Ear

Anterior auricular: draws ear up and forward
Superior auricular: draws ear up
Posterior auricular: draws ear back

Eye

Orbicularis oculi: sphincter of eye, closes lids, compresses lacrimal sac
Corrugator: draws eyebrows downward and medially as in frowning or suffering

Nose

Procerus: draws medial angel of eyebrow downward
Anterior and posterior dilator naris: enlarge nares in hard breathing and anger
Depressor septi: constricts nares
Nasalis: draws alar wings toward septum and depresses cartilage

Mouth

Levator labii superioris: raises upper lip
Levator labii superioris alaeque nasi: raises lip and dilates nares
Zygomaticus minor: with the 2 levator labii muscles, forms nasolabial furrow; deepened when in sorrow
Levator anguli oris: with the 2 levator labii muscles and the zygomaticus minor, expresses contempt or disdain
Zygomaticus major: draws angle of mouth up and back as in laughing
Risorius: retracts angle of mouth
Depressor labii inferioris: draws lip down and back as in irony
Depressor anguli oris: depresses angle of mouth
Mentalis: elevates and protrudes lower lip
Platysma: retracts and depresses angle of mouth
Orbicularis oris: a complex muscle with layers, some parts intrinsic to the lips, the others derived from the following facial muscles: buccinator, levator anguli oris, depressor anguli oris, and zygomaticus major and minor; closes lips, protrudes lips, and presses lips to teeth
Buccinator: compresses cheek, holds food under teeth in mastication, important in blowing when the cheeks are distended with air

Adapted from Pansky B, House EL: Review of Gross Anatomy. New York, Macmillan, 1964, p 2246.

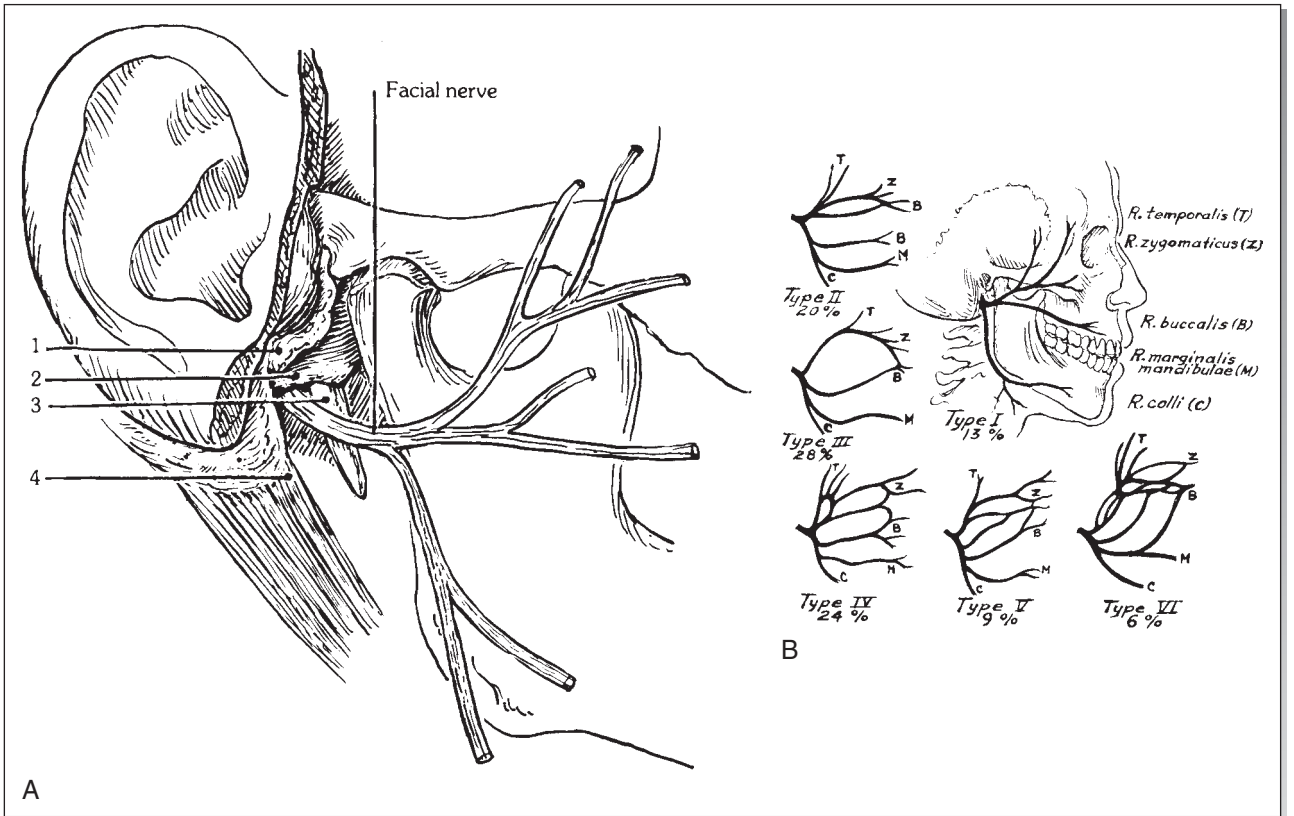

Figure 2 • A, The critical landmarks in identifying the facial nerve's main trunk are as follows: 1, the "conchal cartilaginous pointer"; 2, the tympanomastoid sulcus; 3, the styloid process; and 4, the superior border of the digastric muscle's posterior belly. These structures should be identified before searching for the main trunk of the seventh cranial nerve. (From Baker DC: Facial reanimation by hypoglossal-facial nerve anastomosis. In Brent B [ed]: The Artistry of Reconstructive Surgery. St. Louis, Mosby, 1987, p 298.) **B,** Various types of branching of the facial nerve. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, WB Saunders, 1990.)

major and minor, levator labii superioris, levator anguli oris, depressor anguli oris, and depressor labii inferioris, and the lip closers, the orbicularis oris. The orbicularis oris forms the bulk of the lips.

The main complaint in lower facial paralysis is an inability to smile. In addition, patients may have difficulty with speech and conveyance of ideas and emotion. The most important muscles in smiling are the zygomaticus major and the levator labii superioris. If the individual's smile shows the lower teeth, the depressor labii is also important. The orbicularis oris is important in maintaining lip competence for both drinking and speech. The *B*, *P*, and *M* sounds require buildup of oral pressure, and compensatory articulations often develop in a patient with paralysis.

The Facial Nerve

The facial nerve exits the skull base at the stylomastoid foramen (Fig. 2). It becomes more superficial as it divides the parotid gland into superficial and deep lobes. In the parotid gland the nerve usually divides into two trunks, each of which divides further within the substance of the gland. On leaving the parotid, there are between 8 and 15 branches making up the five divisions of the facial nerve: frontal-temporal, zygomatic, buccal, marginal mandibular, and cervical. The buccal and zygomatic regions have significant overlap because both of these groups of nerves can supply the eye and the mouth. Two divisions, the frontal-temporal and the marginal mandibular, are of particular significance because of their potential for injury (see Fig. 2).

The temporal division passes superiorly from the parotid gland deep to the temporoparietal fascia and crosses the zygomatic arch approximately 3 to 5 cm from the lateral orbital margin. Some branches pass under the superior part of the orbicularis oculi before entering the frontalis. The upper branches enter the frontalis laterally, just above the supraorbital ridge. There is little adipose tissue in this region, and the nerves are prone to injury and laceration when performing many surgical procedures, including rhytidectomy.

The fourth division or group of nerve branches is the marginal mandibular. One to three branches pass below the ramus of the mandible and turn superiorly to cross the mandible at the midbody. This division supplies the depressor anguli oris,

depressor labii inferioris, mentalis, and occasionally the lower orbicularis oris.

Diagnostic Studies and Patient Evaluation

Facial paralysis does not produce the same effect in each person. In some, the effect on appearance is the most distressful aspect of their paralysis, and with others, the main complaint is a specific dysfunction such as a dry eye or drooling mouth. Thus, it is crucial to identify the patient's chief complaint. The cause of the paralysis is important because it affects the potential for spontaneous recovery and nerve regeneration.

Clinical examination usually pinpoints the site of facial nerve injury. If the injury is within the bony canal or more proximal, there may be taste alterations and hyperacusis in addition to the facial weakness. Examination must be systematic and detail each portion of the face, both at rest and with movement. The examination reveals the extent of tone in each muscle or muscle group and the amount of movement. Movement of key areas such as the oral commissure and midportion of the upper lip should be recorded with a measurement technique. Synkinesis is the contraction of one or more muscles when the individual's intent is to contract a different muscle, such as contraction of the orbicularis oculi when attempting to smile. Synkinesis frequently occurs with partial recovery of facial paralysis.

All of the cranial facial nerves should be evaluated as part of the assessment. In addition, magnetic resonance imaging and computed tomography may be useful in cases in which the diagnosis is uncertain because these imaging modalities may reveal tumors, acoustic neuromas, or other lesions such as malaligned fractures or aneurysms.

Reconstructive Goals

For the eye, the aim of treatment is to protect the globe and to treat symptoms such as burning, discomfort, tearing, and redness. The aesthetic goal is to provide symmetry at rest and some movement to the lids. The ultimate goal of reconstruction of the eye should be to enable it to express emotion.

The goals for the mouth are to correct asymmetry, provide oral continence, provide movement that simulates a smile, and improve speech. Only rarely can all goals can be completely accomplished. The patient should be counseled regarding the attainable expected results of surgery. Patients are more likely to be satisfied with the results of surgery if the expectations are realistic.

Treatment

Nonsurgical Management

Most patients with facial paralysis require some type of nonsurgical treatment to protect the eye. Such management may consist of lubricating the eye to prevent drying; taping the lid closed, particularly while sleeping; and wearing soft contact lenses or moisture chambers that are taped to the skin around the orbit at night. Many patients wear protective eyeglasses, sometimes with a lateral shield for windy or dusty conditions.

A number of eye preparations are commercially available; the most fluid preparations are either hydroxypropyl methylcellulose or polyvinyl alcohol. The function of these agents is to prevent the cornea from drying; however, they must be used repeatedly throughout the day. Thicker gel-based agents usually have a longer-lasting effect but blur vision. Ointments containing petrolatum are retained the longest and are generally applied at night. Use of the aforementioned agents must be instituted immediately when paralysis occurs to prevent corneal damage. The most dangerous clinical situation is a combined fifth and seventh cranial nerve paralysis. Because of the sensory loss in the eye, the patient is unaware of the extent of damage that is taking place, and the likelihood of corneal ulceration is high.

Patients with partial paralysis can frequently be improved with neuromuscular retraining supervised by an experienced therapist. By practicing exercises in front of a mirror and using biofeedback, patients are often able to strengthen some facial movements and improve the symmetry of their expressions.

Surgical Management

FOREHEAD RECONSTRUCTION. A brow lift is a particularly useful procedure for improving an individual's symmetry. All brow lifts are static, and currently there are no useful procedures that can provide dynamic forehead movement. Unilateral paralysis may result in a difference in brow height of up to 12 mm. Such disproportion produces marked asymmetry in facial appearance, and correction of brow height is a relatively easy approach to improving the patient's appearance. For such a large discrepancy, a direct brow lift through a supraciliary approach is most effective, albeit with the possibility of a visible scar. The incision should be placed just inside the first hair follicles on the upper aspect of the eyebrow. The supraorbital nerve is at risk and must be carefully preserved. Nonetheless, even with careful nerve preservation, patients often complain of numbness or hypoesthesia.

A brow lift through a coronal incision, with or without a fascial sling, can suspend the brow; however, the scar, which is said to be concealed, is

usually visible on top of the head, and there are often paresthesias posterior to the incision. It is not possible to achieve as much lift through a coronal incision as through a direct brow lift.

Endoscopically assisted brow lifts may be quite useful for small amounts of brow ptosis, but they are not satisfactory for an older individual with significant ptosis. An alternative is to weaken the contralateral normal frontalis muscle by frontal nerve division, resection of strips of muscle, or injection of botulinum toxin (Botox). These techniques are useful in a person who has marked wrinkling of the forehead resulting from and a very active brow.

EYELID RECONSTRUCTION. With loss of orbicularis function, the upper lid is held open by the unopposed action of the levator palpebrae superioris. Two options for dynamic closure of the upper lid are a gold prosthesis, which places additional weight in the upper eyelid, or a temporalis transfer. Gold weights are the simplest and thus the first approach to treatment. For the best result, they should be placed at least 4 mm above the lid margin (to decrease visibility) and secured to the tarsal plate with a permanent suture. The weight should allow for partial, but not complete corneal coverage. Insertion of a weight that is sufficiently heavy to produce complete eyelid closure often results in the weight drifting, eroding through the skin, and having to be removed. The most commonly selected weights are 1.0 and 1.2 g. Care must be taken to avoid damage to the levator insertion during dissection for placement of the gold weight because ptosis may occur. After placement of the gold weight, eyelid closure is a slow rather than a rapid blink. It is important that the patient realizes this and learns to close and hold for a short moment to allow the eyelid to descend.

Complications of gold weights include extrusion, capsule formation (causing a visible lump), and irritation of the cornea. In any of these situations, the weight can be easily removed, replaced, or repositioned.

Temporalis transfer can be used for eye closure. A 1.5-cm-wide strip of temporalis muscle is detached superiorly, turned anteriorly, and extended with a piece of tendon or fascia. Tendon is preferable because it does not stretch. It is placed only in the upper lid; a static correction for the lower lid is created separately with a tendon sling technique. This procedure is more dynamic than placement of a gold weight. It is activated each time that the individual closes the jaw. It does result in a bulge over the lateral orbital margin, a slit-like appearance to the palpebral aperture when closed, lateral movement of the eyelid skin if it is adherent to the tendon and, occasionally, prevention of upper eyelid movement by adhesions of the underlying tendon.

Another alternative is a spring made of stainless steel wire with two arms. One arm is placed along the lid margin and the second is attached to the inner aspect of the orbital margin. The tension in the spring closes the eyelid when the levator labii superioris is relaxed. Therefore, it is not gravity dependent. There is a difficult learning curve to place the spring correctly. Problems with the spring technique include malpositioning, erosion, breakage or weakening, and an unnatural curving of the upper eyelid margin.

A microvascular neuromuscular transfer involving a cross facial nerve graft can also be used for eyelid closure. The platysma muscle is the most suitable muscle for a free microvascular neuromuscular transfer for reconstruction of the orbicularis oculi. The technique is tedious and complex, but it should be available for patients when other techniques have failed. Reinnervation of the free flap may not occur, and the muscle may produce undesirable thickening of the eyelids.

Lateral tarsorrhaphy remains a mainstay of treatment of a paralyzed eyelid. The eyelashes should not be removed to minimize the appearance of a shortened eye. The main indication is in a patient who has an anesthetic cornea or severe corneal exposure with failure of the previously mentioned techniques. It is especially indicated in older patients with concomitant senile ectropion.

The lower eyelid can be managed separately or as part of a temporalis transfer. With normal eyelid closure, the lower eyelid moves only 1 to 2 mm superiorly. However, with paralysis, the lower lid tends to stretch and sag, which results in scleral show, tearing, and long-term ectropion. The lower eyelid should be repositioned at the level of the limbus with the punctum in apposition to the globe. The best result is obtained by supporting the lower lid with a strip of tendon. Proper placement of the tendon is difficult, but crucial because a properly placed tendon strip does not deform the eyelid. Canthoplasties alone, in our experience, are not permanent, tend to stretch, and produce unsatisfactory long-term results.

Overall, the first-line management of the eye in facial paralysis is the use of both the gold weight and the lower eyelid sling in the majority of patients.

NASAL RECONSTRUCTION. Because of paralysis of the nasalis and levator labii alaeque nasi, along with the drooping of the paralyzed cheek and deviation of the paralyzed lower part of the face to the normal side, there is often loss of support for the nostril and collapse of the ala. Correction is best accomplished by elevating and laterally supporting the alar base with a sling of tendon passed through the alar base and fixed to the zygoma or inferior orbital rim.

UPPER LIP AND CHEEK (SMILE) RECONSTRUCTION. The most common complaints of patients with facial paralysis are the inability to smile, drooping of the paralyzed side of the face, drooling, impaired chewing, and speech difficulty. Options for

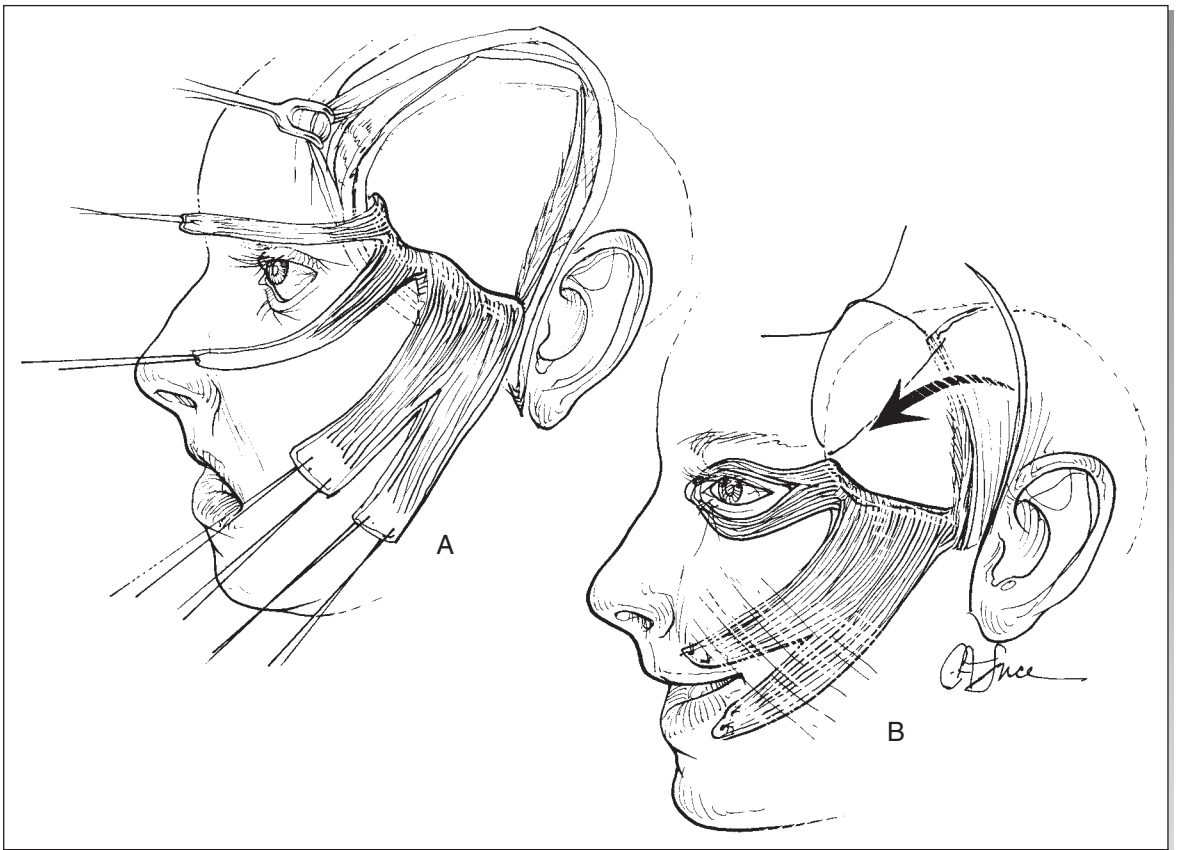

Figure 3 • Technique of temporalis transposition. **A,** Preauricular incision with extension into the scalp. The entire muscle is elevated, and four or five incisions are made for insertion of the slips. **B,** Muscle slips are transposed to the upper and lower eyelids, upper lip and melolabial fold, lower lip, and commissure. Overcorrection is essential. The muscle should be sutured medial to the melolabial fold and interdigitated with the orbicularis oris muscle in the upper lip. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990.)

management are regional muscle transfers or free microneurovascular muscle transfers.

Regional Muscle Transfers. For regional muscle transfers, either a portion of the temporalis, the masseter muscle, or both are transferred. The temporalis muscle is detached from the temporal fossa, turned over the zygomatic arch, and extended to the oral commissure with fascia (Fig. 3). It is important to overcorrect the commissure of the mouth because it does stretch in the immediate postoperative period. An implant is required in the temporal region to correct the residual hollow that is usually present at the muscle donor site. The temporalis also produces a significant muscle bulge where it is redraped over the zygoma. Alternatively, the temporalis muscle can be detached at the coronoid process and lengthened with fascia grafts to reach the mouth. This technique does not result in hollowing in the temporal fossa, nor does it create a bulge over the cheek. The problem with temporalis transfer is that the amount of movement obtained is not sufficient to satisfy most patients. However, the position of the commissure at rest is often satisfactory.

Microneurovascular Muscle Transfers. A microvascular neuromuscular transfer uses a portion of muscle, usually the gracilis, latissimus dorsi, or pectoralis minor, for facial motion. Innervation is achieved with either the ipsilateral or contralateral facial nerve. Occasionally, in patients with bilateral paralysis the masseter motor nerve from the paralyzed side can be used. The transferred muscle is attached to the mouth at the insertion of the levator labii and the zygomaticus muscles into the orbicularis oris. The muscle can be placed at an angle that simulates the vector of movement similar to that on the opposite side of the face. The amount of muscle that is used determines, to some extent, the amount of movement ultimately achieved. The surgeon is limited in the amount of muscle that can be inserted because an excessive amount of muscle produces a contour deformity on the side of the face.

It is important that the individual's smile on the normal side be assessed. The commissure and upper lip are evaluated for direction of movement, strength of movement, and the amount of movement of the upper lip relative to the commissure. The shape of the lips, the nasolabial fold, and the presence of a labiomental fold are also

assessed. The surgeon can use this analysis to plan for muscle insertion on the paralyzed side in such a way that it closely simulates movement on the normal side.

The classic approach to a microneurovascular muscle transfer is to innervate it with a cross facial nerve graft. First, facial nerve mapping with a microbipolar nerve stimulator that has variable frequency and voltage control is used to identify the function of each individual facial nerve branch. The disposable nerve stimulators that are frequently used in peripheral nerve surgery are not suitable. The muscles that contract can be identified by visual examination. The zygomaticobuccal nerve branches that produce a normal smile are identified. The most common error is falsely interpreting upper lip orbicularis oris contraction as being due to zygomaticus major movement. If a nerve that produces puckering of the lips is used for smile reconstruction, movement of the paralyzed muscle does not occur during smiling. It is important to evaluate all branches that produce a smile on the normal side before dividing branches for the nerve graft. It is possible to insert a cross facial nerve graft without seriously downgrading the smile on the normal side; however, in rare situations, sufficient branches are not available.

A short nerve graft, approximately 10 cm in length, is passed from the normal side to the contralateral buccal sulcus. A short nerve graft requires a waiting period (approximately 6 months) between the nerve graft and the muscle transfer. Short nerve grafts also achieve stronger muscle contraction.

The gracilis muscle is the muscle of choice for reconstruction. The pedicle is a good match for the facial artery and vein, the muscle can be sacrificed without any functional loss, and the scar is in an inconspicuous location. A segment of muscle based on the dominant pedicle is cut and should be of sufficient length to reconstruct the smile. The amount of muscle varies between 12 and 40 g, depending on the size of the patient and the patient's facial requirements. The muscle is divided longitudinally, and approximately 30% to 70% of the cross section of the muscle is harvested. The correct tension on the muscle is difficult to ascertain because the eventual appearance of the face is determined by a combination of mechanical tension within the muscle and the degree of tone that the muscle develops.

After a microvascular neuromuscular transfer, movement does not occur for 6 to 8 months and gradually increases in strength for an additional year. Disadvantages of the cross facial nerve graft and muscle transfer technique are the complexity of the procedure and the length of time required for eventual recovery. Furthermore, there is a great deal of variability in the amount of movement that occurs from patient to patient. It is not uncommon for the patient to request a third procedure either to tighten or to loosen the muscle and to remove bulk from the cheek (Fig. 4).

Bilateral Paralysis: Microneurovascular Muscle Transfers. Microvascular neuromuscular transplantation is especially useful in patients with bilateral paralysis, such as Möbius' syndrome. The nerves that have been used to innervate the muscle include cranial nerves V, XI, and XII. Cranial nerve XII provides innervation, but movement of the muscle is sometimes weak, and a patient with Möbius' syndrome may also have a degree of 12th nerve paralysis. If the 12th nerve is used, it is done as a hemiinnervated nerve graft so that only half the hypoglossal nerve is divided. The masseteric nerve of cranial nerve V is the motor nerve of choice. It lies on the deep surface of the masseter muscle and is identified by dividing a portion of the muscle origin. The use of this nerve bilaterally does not produce functional deficits with respect to chewing. Innervation is reliable and occurs at approximately 2 to 3 months. Smile movement does not occur spontaneously and must be learned. Patients first practice smiling in front of a mirror with family and friends and finally with strangers. With time, many patients are able to smile without conscious effort and without any evidence of jaw movement. With this technique, the position of the lower lip is improved, there is less of a tendency to drool, and speech may be improved.

Static Slings. In older patients, the amount of facial drooping at rest can be significant and is frequently a main concern. If the only goal is to produce a symmetric face at rest, static slings are useful. A tendon sling is used, either the palmaris longus, plantaris, or extensor digitorum longus. The tendon is woven back and forth between the body of the zygoma and the upper lip and commissure, with slight overcorrection at the time of surgery.

LOWER LIP RECONSTRUCTION. Problems associated with the lower lip are usually due to a lack of function of the marginal mandibular nerve. As a result, when smiling and showing teeth, the lower lip on the paralyzed side assumes a slanted inclination rather than a pull down to show the teeth. In the normal resting position and with a closed lip smile in which the teeth are not showing, the deformity is not usually noticeable. Most patients are primarily concerned with the asymmetric appearance of the lower lip during open lip smiling. Although many techniques have been described for reconstruction of the depressor muscle, including transfer of the digastric muscle, sometimes combined with cross facial nerve grafts, the procedures are complicated and it is difficult to achieve the desired amount of motion. The authors' approach to depressor paralysis has been to perform selective myectomy of the depressor labii inferioris on the nonparalyzed side. This procedure is relatively simple in most patients, although in some it is difficult to remove all the muscle. The procedure is usually preceded by injection of a local anesthetic or botulinum toxin (or both) into the depressor labii

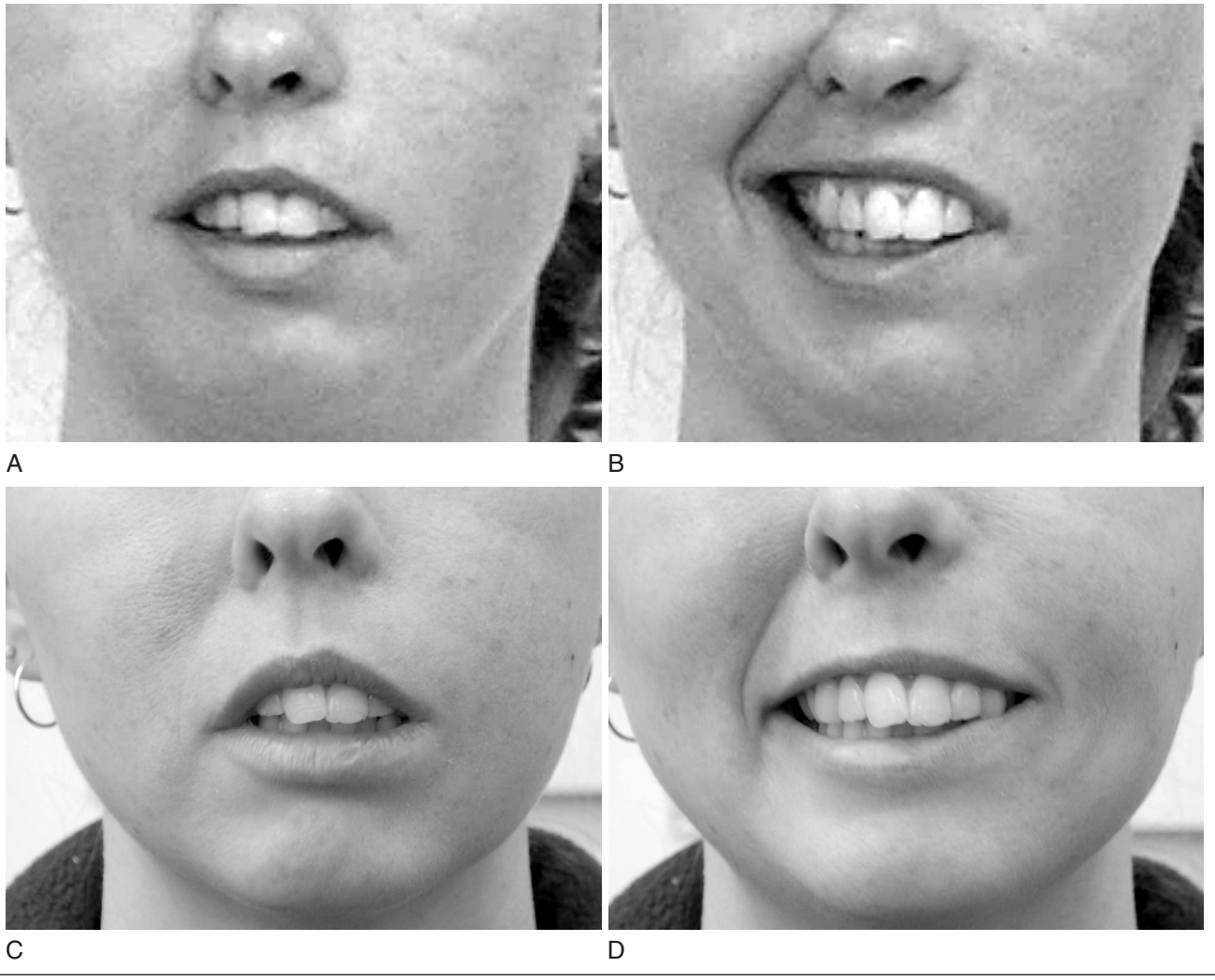

Figure 4 • A, Preoperative appearance of a patient with left-sided partial facial paralysis at rest. **B,** With smiling the patient had only 4 to 5 mm of movement at the left commissure. **C,** After a cross facial nerve graft and microneurovascular muscle transfer the patient has symmetry at rest. **D,** With smiling the patient achieves 10 mm of spontaneous movement at the left commissure.

inferioris so that patients can decide whether they wish to undergo muscle resection.

Multispecialty Approach to Management

The skills of the ophthalmologist and otolaryngologist are frequently necessary to aid in eye and nasal functional problems. In early childhood, feeding may be a problem for a patient with Möbius' syndrome who is unable to effectively suck. The psychosocial aspects of facial paralysis are enormous, and psychosocial support may be necessary for patients who are unable to cope with their paralysis. Social workers, clinical psychologists, and developmental psychologists are valued members of the clinical team.

Pearls and Pitfalls

- A wide variety of reconstructive techniques is required for optimal rehabilitation in a patient with facial paralysis.
- Microsurgical free muscle flap reconstruction of the smile produces excellent results.
- Intraoperative identification of the appropriate donor nerve is fundamental to obtain an uncon-cious smile in patients with unilateral facial paralysis.
- Canthopexy alone yields poor results on lower eyelid position. Tendon grafts are useful to set and maintain the lower lid height.
- Botox is a useful adjunct for reducing asymmetry in a paralyzed face, especially in the lower lip.

SUGGESTED READINGS

Carraway JH, Manktelow RT: Static sling reconstruction of the lower eyelid. Op Tech Plast Reconstr Surg 6:163-166, 1999.

Deutinger M, Freilinger G: Transfer of the temporal muscle for lagophthalmos according to Gillies. Scand J Plast Reconstr Surg Hand Surg 25:151-154, 1991.

Diels HJ, Combs D: Neuromuscular retraining for facial paralysis. Otolaryngol Clin North Am 30:727-743, 1997.

Edgerton MT, Wolfort FG: The dermal-flap canthal lift for lower eyelid support. A technique of value in the surgical treatment of facial palsy. Plast Reconstr Surg 43:42-51, 1969.

Levine RE: The enhanced palpebral spring. Op Tech Plast Reconstr Surg 6:152-156, 1999.

Manktelow RT: Use of the gold weight for lagophthalmos. Op Tech Plast Reconstr Surg 6:157-158, 1999.

Terzis JK, Kalantarian B: Microsurgical strategies in 74 patients for restoration of dynamic depressor muscle mechanism: A neglected target in facial reanimation. Plast Reconstr Surg 105:1917-1931, 2000.

Restoration of Craniofacial Soft Tissue Contour

ALEXES HAZEN
GEOFFREY C. GURTNER

Many methods have been used to attempt to restore facial contour and facial harmony. The advent of microsurgical tissue transfer has revolutionized the treatment of facial soft tissue contour defects, becoming the "gold standard" for severe defects. Microsurgery had its origins in reconstruction after trauma and cancer ablation, with a focus also on functional restoration. Microsurgical reconstruction for aesthetic purposes has become more common as techniques have improved. Minimization of donor site defects has allowed for a role for microsurgical reconstruction of aesthetic defects. The ideal reconstruction of a facial defect will reestablish a harmonious and symmetric appearance. Frequently, these goals are expeditiously and reliably achieved by a microsurgical approach.

Although many techniques are used, experience has been that free tissue transfer yields the longest lasting and most predictable results. Fasciocutaneous flaps specifically have produced the most superior and predictable aesthetic results because they can be sculpted and shaped with more precision.

Etiopathogenesis

Contour deformities can result from a variety of causes, can be unilateral or bilateral, and can range in severity from mild to severe. The most common cause of a facial contour abnormality is Romberg's disease, also known as progressive hemifacial atrophy of unknown cause. This clinical phenomenon was first described by Parry in 1825 and by Romberg in 1846. The incidence is not known, and clinical signs are usually first noted in the initial 2 decades of life. The atrophy is progressive and usually affects the skin and subcutaneous tissue. In severe cases, with childhood onset, the underlying muscles and bones are also affected. The cause of Romberg's disease has not been elucidated, and although distinct from scleroderma, it may represent a localized form of scleroderma. The end result is progressive atrophy of one side of the face followed by a stable "burned out," atrophic appearance.

Lupus erythematosus and scleroderma are the most common connective tissue disorders resulting in soft tissue atrophy with facial involvement. Lupus erythematosus is an inflammatory connective tissue disorder of unknown cause that occurs predominantly in women (90% of cases). It can have a variable clinical course. Patients are often positive for antinuclear antibodies in their serum, including anti-DNA antibodies (more specific for systemic lupus). Various forms of lupus can result in telangiectasia and skin atrophy. Scleroderma, which occurs four times more often in women, causes diffuse fibrosis of the skin, blood vessels, synovia, and vital organs such as the kidneys. The pathogenesis is not clear, although a combination of vascular abnormalities, excess collagen deposition, and extracellular matrix production is implied. The skin is involved in more than 95% of cases.

Additional causes include connective tissue disorders, nerve palsy, burns, trauma, cancer, HIV-associated lipodystrophy, craniofacial microsomia, and other congenital abnormalities. Microsurgical techniques have also been used to restore contour in cases in which silicone or other injectable material had an adverse effect on the skin and underlying soft tissue.

Pathologic Anatomy

The specific abnormality in facial structure depends on causality. To enable the most complete aesthetic restoration, it is important to define the full extent of the defect, which can be usually accomplished by a complete physical examination followed by radiographic evaluation of the bony structures. Such evaluation is important because patients with scleroderma or Romberg's disease may have defects encompassing not only the skin and underlying soft tissue but also the underlying bone. The functional status of the facial nerve branches should be assessed before embarking on any surgical reconstruction.

Diagnostic Studies

If a bony abnormality is suspected, a facial computed tomographic scan should be obtained. If the orbit is evaluated, 1.5-mm cuts through the orbits are necessary. In facial trauma cases, if the mandible is involved, a Panorex view may be helpful. If no bony abnormalities are noted on physical examination, radiographic studies are not indicated. If nerve involvement is present, it may be helpful to obtain an electromyogram (EMG).

Surgical Indications and Options

Patients with Romberg's disease or other connective tissue disorders resulting in facial atrophy may have a deformity that often includes all components of the facial form: skin, subcutaneous tissue, muscle, and bone. Treatment options for correction of facial deformities range from synthetic materials, including liquid silicone and alloplastic implants, to fat, bone, cartilage grafts, dermis-fat grafts, local flaps, and free tissue transfers. The results of facial contouring with silicone injections (not approved by the U.S. Food and Drug Administration) have generally been unsatisfactory, with the possibility of delayed scar and contracture formation, as well as progressive skin breakdown. Removal of free silicone can be a difficult task and can render future reconstructive efforts more challenging. For mild defects, injection of synthetic material appears to be a "quick fix" and is of little cost to the patient and little time to the surgeon. Belying the initial promise of injectable and prosthetic materials, the clinical results are disappointing. Although these materials are easy to use, the results are often dissatisfying to patient and surgeon alike.

Autologous fat injections have the obvious advantage of being cost-efficient and relatively simple to perform with no risk of "rejection" or allergic reaction. Large defects are less suitable for fat injection. However, injectable materials such as fat, collagen, and Cymetra have the drawback of being resorbed over time. More permanent (alloplastic) materials, in contrast, are prone to infection, seroma, and capsule formation and have unpredictable results.

Although other authors have claimed that not all patients are candidates for free flap surgery, in our experience, the following approach is applicable to patients with both severe and mild abnormalities alike. Muscle or myocutaneous flaps are often too bulky for the purpose of facial contouring and thus their use has largely been discarded. The temporoparietal flap is thin and does not usually provide adequate volumetric augmentation. Omentum has been used for facial recontouring but has many disadvantages, including obligate intraabdominal harvesting and difficulty in long-term flap fixation (with no dermal or fascial attachments to fix the tissue to the desired location). The groin flap and superficial inferior epigastric flap are among other options for fasciocutaneous free tissue transfer. The superficial inferior epigastric flap is useful when a large amount of skin coverage is required. However, this flap limits the possibility of incorporating thin, pliable fascia beyond the width of the skin paddle design. Therefore, this flap would not allow optimal management of subtle midline-associated deformities, as often seen in hemifacial atrophy. Flaps based on the circumflex scapular pedicle appear to be the most versatile flaps for restoration of facial contour. This chapter focuses primarily on techniques for harvesting and using this flap.

Timing of the Procedure

Because many of the underlying causes of facial atrophy have a progressive nature, it is important to ensure that the deformity has reached a stable stage before embarking on surgical correction. For posttraumatic defects, all other medical/surgical issues should be resolved. If bony abnormalities exist, they should be corrected before soft tissue correction. A minimum of 18 to 24 months should elapse after evidence of cessation of progressive atrophy. In most cases the superficial temporal vessels are of adequate size for microvascular anastomoses. In bilateral cases, the free tissue transfers are scheduled at least a month apart; revisionary surgery is performed on both sides 6 months after the second free tissue transfer.

Surgical Technique: Parascapular Flap

The areas of facial deformity are marked with the patient in the upright position. Attention is paid to

the three-dimensional nature of the asymmetry. A two-dimensional template can be used with special marking for the areas in need of more soft tissue augmentation (dermis, fat, and fascia) and areas where the edges of the flap will be tapered and intertwined with the normal tissue to avoid unsightly step offs.

The ipsilateral donor site is preferred to allow a two-team approach to the recipient and donor site. The horizontal and vertical branches of the circumflex scapular artery can be outlined on the skin with the help of a hand-held Doppler. The required soft tissue flap is marked on the skin in a vertical-to-oblique orientation, depending on the length of flap required (Fig. 1). The patient is placed in the supine position with a roll behind the ipsilateral shoulder to allow simultaneous exposure of the scapular and facial areas. After closure of the donor site, the roll is removed so that the face can be assessed more easily for symmetry. The ipsilateral upper extremity is prepared and draped along with the remaining operative site. The upper part of the arm and shoulder can be easily positioned during harvesting for better visualization of the circumflex scapular vessels.

The table is positioned in the operating room in a manner that allows the surgeon access to both sides of the head, as well as the ipsilateral side of the patient. The anesthesia equipment is located along the contralateral side of the table. A crossbar placed at the level of the shoulders and draped into the field can be used to stabilize the ipsilateral upper extremity during harvesting.

The face is dissected through a preauricular incision. The facial dissection is performed in a subcutaneous plane to release all of the tethered soft tissue and is extended beyond the borders of the atrophic tissue to allow adequate setting of the flap. Dissection deep to the alar base at the piriform aperture may be required to allow repositioning of the alar base to a more symmetric position.

The superficial temporal artery and vein are most commonly used as recipient vessels and are visualized through the preauricular incision. After an adequate skin envelope is elevated, meticulous hemostasis is achieved.

The parascapular flap can be harvested simultaneously (see Fig. 1). The flap commonly has an ellipsoid design, which simplifies primary closure of the donor site. The circumflex scapular pedicle is

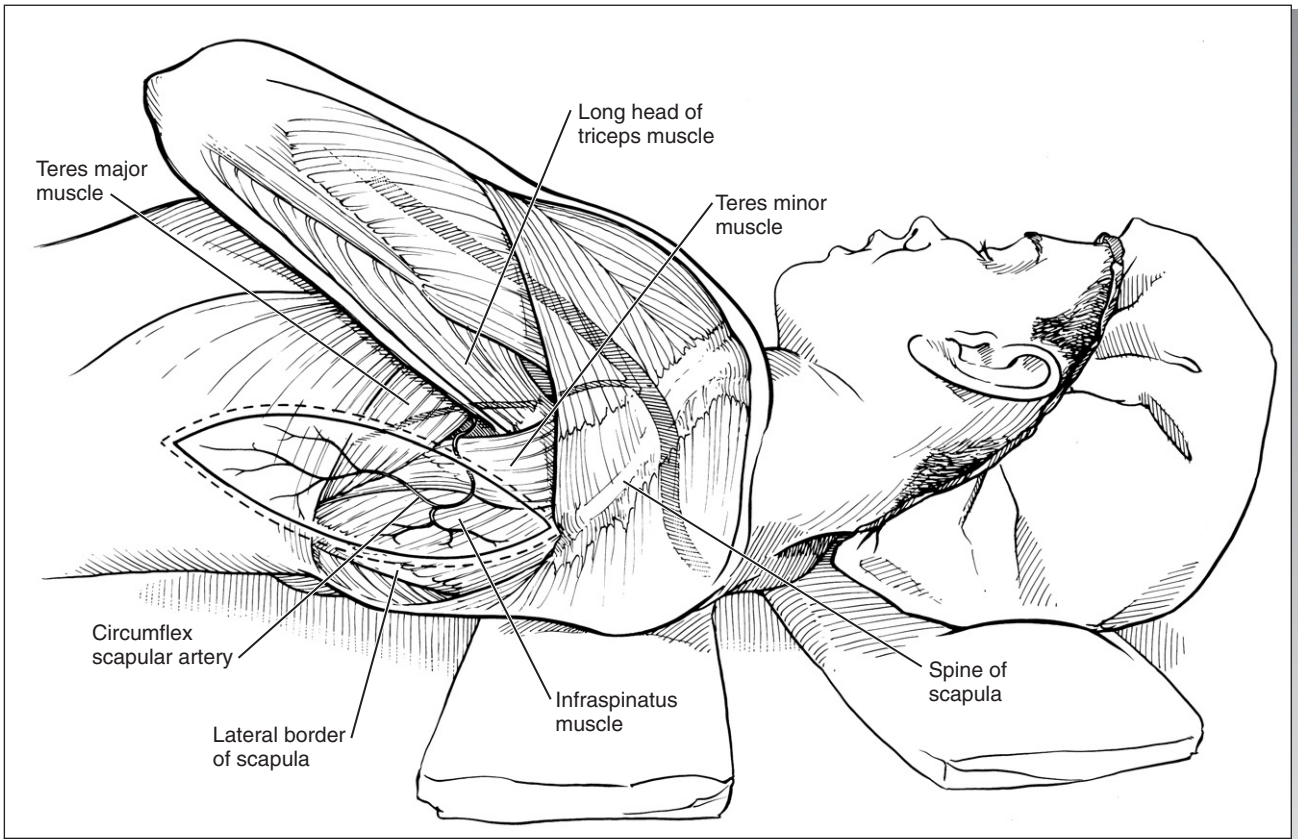

Figure 1 • Positioning of the patient for simultaneous flap harvest and preparation of the donor site. The flap is harvested according to the size of the defect. Fascia and fat can be harvested beyond the borders of the skin harvest. The circumflex scapular vessels exit the triangular space and are approached inferiorly. After dissecting to the level of the teres major muscle, care is taken to identify and isolate the pedicle.

identified within the triangular space bordered by the teres major muscle inferiorly, the teres minor muscle superiorly, and the long head of the triceps muscle laterally. The flap is designed in a fashion to capture the vascular pedicle. An important consideration regarding flap design is the location of the tissue requirements on the face in relation to the proposed location of the anastomoses to the superficial temporal vessels. If tissue augmentation is necessary both superior and inferior to the future anastomosis below the zygomatic arch, the donor flap will extend onto the shoulder as well as into the inframammary fold. The amount of skin harvested with the flap can be limited to the areas of the face with the most severe degree of atrophy and need for greatest augmentation. The fascial extensions can be folded into two or more layers, a maneuver that gives the surgeon a tool to correct variable deficits with the same flap.

The skin and subcutaneous tissue are incised and the flap is elevated in a subfascial plane in retrograde fashion. The vascular pedicle can be followed to the level of the thoracodorsal vessels. If additional bulk is needed, portions of regional muscles, such as the teres major muscle, can be harvested. The wound is closed in layers over a suction drain.

The anastomoses to the superficial temporal vessels are performed in an end-to-end fashion, the

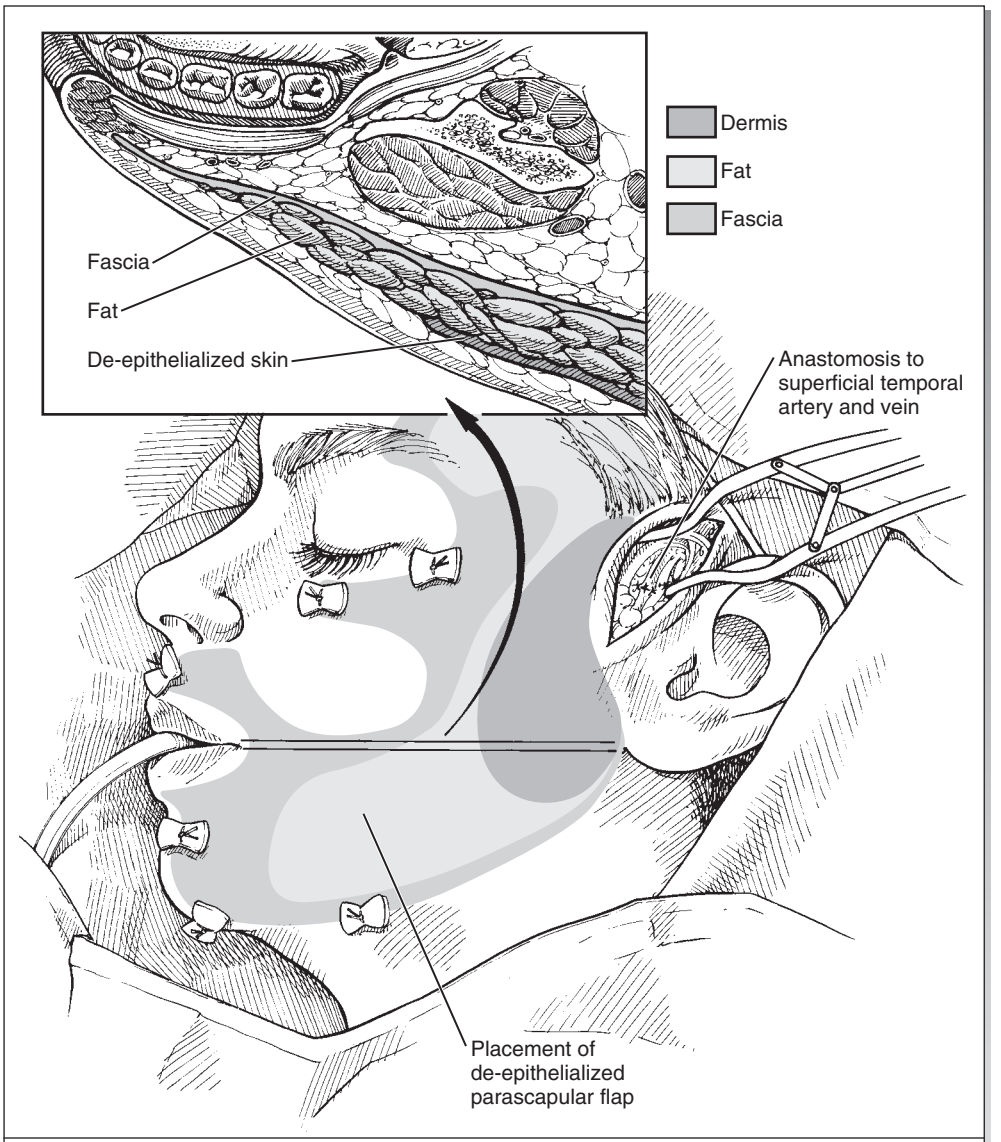

Figure 2 • The deficit should be estimated preoperatively and the face marked accordingly. Areas with the most need will be reconstructed with deepithelialized skin, fat, and fascia, whereas areas of less need include only fascia. Inset shows a cross section extending from the oral commisure to the ear. Note how the flap in tapered such that only fascia is placed at the level of the commisure, whereas closer to the ear the flap has more thickness. Note the through-and-through pledget sutures.

vein followed by the artery. The flap is draped in its final orientation on the face. The skin is deepithelialized and flap thickness tailored to meet the requirements of the various regions of the recipient site. The transition between thicker and thinner areas is tapered to achieve a more natural facial contour. It is almost impossible to overresect or overthin the flap during the first operation. Optimal aesthetic reconstruction is achieved in the majority of patients with a second procedure to revise the flap and finalize facial contouring.

The flap is secured in the area close to the anastomoses and also sutured to the periosteum of the infraorbital rim and zygoma with nonabsorbable material to prevent caudal shifting. The head of the bed is raised to assist in natural contouring of the flap. The fascial extensions of the flap are secured in the desired position with nylon sutures passed through the skin and tied over Vaseline gauze (Fig. 2). It is important to stretch the flap to its original dimensions to avoid sagging or redundancy.

Postoperative Care

No dressing is used on the face. Patients receive one aspirin tablet (rectally) per day starting in the recovery room, and this is continued orally for 1 month. Doppler signals in the artery and vein are monitored for 2 days. Patients leave the hospital between 3 and 5 days postoperatively. The facial drain is usually removed before discharge. The donor site drain and preauricular and facial sutures are removed on postoperative day 7.

Revisions are planned beginning at 5 months after free tissue transfer to allow time for complete resolution of edema.

Most patients will require surgical revision, if only to debulk the flap. Revisions are best scheduled at least 5 months after surgery. This period will allow time for adequate reduction of swelling and maturation of the flap and surrounding bed vascularity. If debulking is all that is required, the initial preauricular incision may be used, and either liposuction or direct excision of fat with scissors may be performed. Some patients may require other procedures, with the overall goal being restoration of facial harmony. Ancillary procedures such as face lift, blepharoplasty, lateral canthoplasty, genioplasty, and rhinoplasty can be performed at the time of revision surgery to optimize the final result.

Pearls and Pitfalls

- The optimal procedure for restoration of facial contour is free tissue transfer.
- With increasing clinical experience, the indications for free tissue transfer have been extended to any subtle contour deformity not amenable to correction with local or regional tissue transfer.
- A limited preauricular incision minimizes the stigmata of facial surgery by eliminating an unsightly neck incision.
- The overall goal of facial harmony may include ancillary procedures and revisions.
- It is more difficult to achieve symmetry in unilateral cases, particularly when changes in skin texture are present.

SUGGESTED READINGS

Mordick TG 2nd, Larossa D, Whitaker L: Soft-tissue reconstruction of the face: A comparison of dermal-fat grafting and vascularized tissue transfer. Ann Plast Surg 29:390-396, 1992.

Pensler JM, Murphy GF, Mulliken JB: Clinical and ultrastructural studies of Romberg's hemifacial atrophy. Plast Reconstr Surg 85:669-674, discussion 675-676, 1990.

Siebert JW, Longaker MT: Aesthetic facial contour reconstruction with microvascular free flaps. Clin Plast Surg 28:361-366, ix, 2001.

Siebert JW, Longaker MT, Angrigiani C: The inframammary extended circumflex scapular flap: An aesthetic improvement of the parascapular flap. Plast Reconstr Surg 99:70-77, 1997.

Tan EM, Cohen AS, Fries JF, et al: The 1982 revised criteria for the classification of systemic lupus erythematosus. Arthritis Rheum 25:1271-1277, 1982.

Upton J, Albin RE, Mulliken JB, Murray JE: The use of scapular and parascapular flaps for cheek reconstruction. Plast Reconstr Surg 90:959-971, 1992.

Maxillofacial and Craniofacial Prosthetics

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Reconstructive plastic surgery has made great strides in restoring defects of the maxillofacial and craniofacial regions with the use of autogenous tissues. Not all defects, however, are amenable to correction by surgical reconstruction. Often, tissues and structures of this region are highly complex or specialized, thus negating the possibility of reconstruction with autogenous tissue. Examples include replacement of an eye, ear, or other facial structure. In many of these situations, a *prosthesis* is the best option. Intraoral defects requiring replacement often need a *maxillofacial prosthesis* to restore the teeth, jaws, or surrounding structures of the stomatognathic system. Replacements for external structures of the head and neck region are usually termed *craniofacial prostheses.*

Etiopathogenesis/Causative Factors

Defects requiring maxillofacial or craniofacial prostheses are almost exclusively either congenital anomalies or defects caused by trauma or ablative surgery. Radiation therapy may also lead to tissue destruction resulting in the loss of normal facial form. Advanced stages of diseases such as tuberculosis, noma, and syphilis can result in destruction of facial structures; although these conditions are infrequent in developed countries, they are still occasionally seen in the third world.

Pathologic Anatomy

In addition to the actual defect that must be reconstructed with a prosthesis, the anatomic structures surrounding a defect are often abnormal in position and contour. Scarring or alterations in the insertion and origin of muscles and other soft tissues may affect how a prosthesis used to reconstruct an ear, orbit, or other craniofacial structure might appear. An abnormal foundation for the prosthesis may necessitate a prosthesis that is also similarly abnormal to blend into the surrounding structures. One example is fabrication of an auricular prosthesis for a patient with craniofacial microsomia and microtia. If the surrounding structures are also restricted in their development, with a lower hairline and small mandibular body/ramus and zygoma, the fabricated auricular prosthesis should not be placed in perfect symmetry with the auricle on the unaffected side; instead, it should blend into the surrounding local craniofacial environment to give a natural-appearing result.

Diagnostic Studies

The most helpful diagnostic as well as pretreatment study in prosthetic reconstruction of a maxillofacial or craniofacial defect is a high-resolution computed tomographic (CT) scan with three-dimensional reformatting. These are particularly important for any facial prosthesis that will be retained by osseointegrated titanium implants. CT scans provide information on the quantity and quality of bone in the region where the implants will be placed. Similarly, appropriate CT scans show vital structures that may affect implant positioning, such as the course of the facial nerve during placement of osseointegrated implants in the parietotemporal region to retain an auricular prosthesis for a patient with unilateral craniofacial microsomia.

Placement of almost any implant-retained intraoral prosthesis similarly requires a CT scan with three-dimensional reformatting (i.e., Dentascan) to assess the quality and quantity of the recipient bone

and vital structures surrounding the defect. In the case of non–implant-retained maxillofacial prostheses (such as a maxillary obturator prosthesis or a speech bulb prosthesis for a patient with a cleft palate), a panoramic radiograph and a full set of dental periapical radiographs should be obtained to evaluate the teeth that will be used to retain the prosthesis, as well as the quality of bone supporting the dentition.

Nasoendoscopy and videofluoroscopy are useful in assessing the effectiveness of a soft palate obturator prosthesis fabricated to assist in velopharyngeal closure.

Reconstructive Goals

The two primary goals of any maxillofacial or craniofacial prosthetic reconstruction are to restore normal form and function. These goals may not be attained completely with the current state of technology in the field of intraoral and extraoral prostheses. Although eyes and periorbital structures may be aesthetically replaced by a prosthesis, the *function* of the human eye cannot be "reconstructed" by a prosthesis. Each prosthesis has a reconstructive goal specific to the particular anatomic structure that it is replacing, but in general, most prostheses, whether intraoral or extraoral, achieve greater aesthetic than functional success.

Treatment

Extraoral Craniofacial Prostheses

AURICULAR PROSTHESIS. In older adult patients, in whom most defects are the result of surgical removal of the ear for the treatment of carcinoma or trauma, the external ear is most often replaced by a prosthesis (Fig. 1). In contrast, a congenitally absent ear in a child is most appropriately replaced by an autogenous reconstruction. Although an autogenous ear reconstruction does not provide the detail in form that a well-constructed prosthesis does, an autogenously reconstructed ear does not require replacement on a regular basis. As children grow, they are more likely to accept the less than ideal form of an autogenously reconstructed ear as part of their normal body image. Autogenous ear reconstruction in an adult does not always provide the anatomic detail that adults have come to consider as a normal part of their body image.

Auricular prostheses are fabricated from custom-colored silicone polymer blended to match the surrounding skin. The prosthesis is retained by medical-grade adhesives or by clips or magnets attached to osseointegrated titanium implants placed in the parietotemporal bone (Fig. 2). Fabrication of an auricular prosthesis is not entirely cosmetic inasmuch as studies have demonstrated that a prosthesis improves sound collection in patients with a missing auricle.

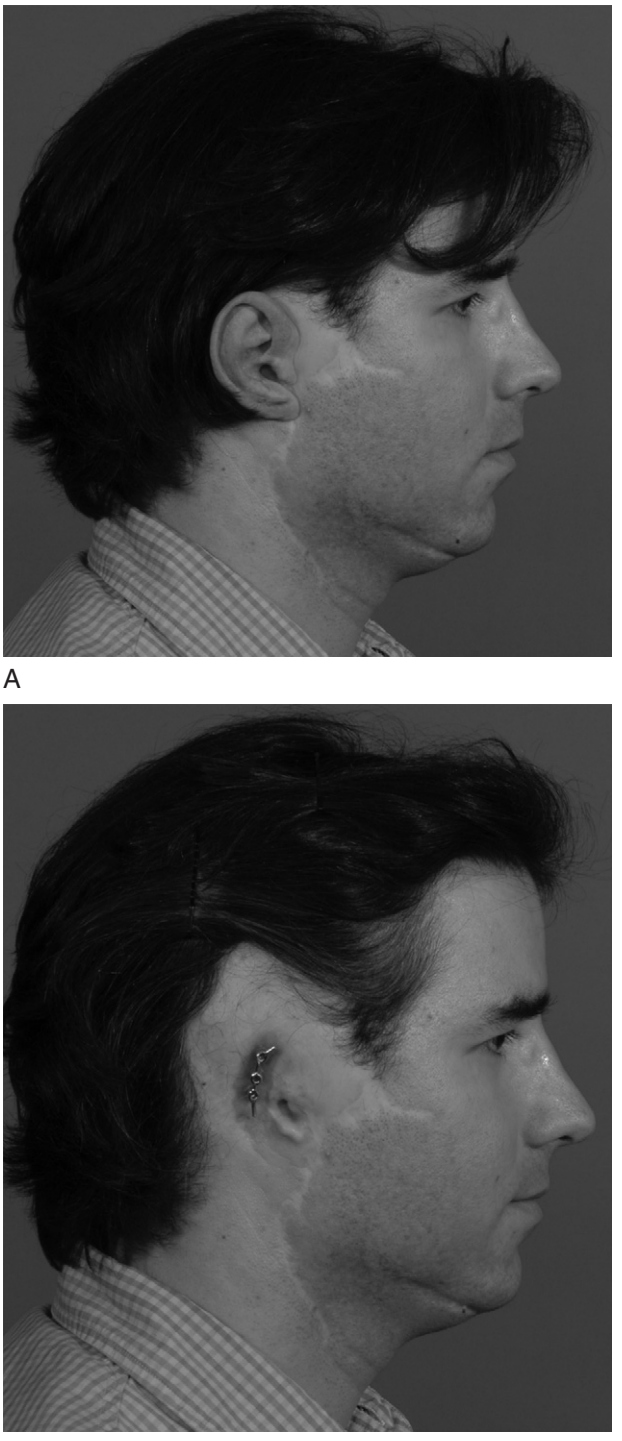


Figure 1 • A, Patient with an implant-retained auricular prosthesis in place after suffering traumatic avulsion of the external ear in a cycling accident. **B,** Prosthesis removed, showing underlying implant.

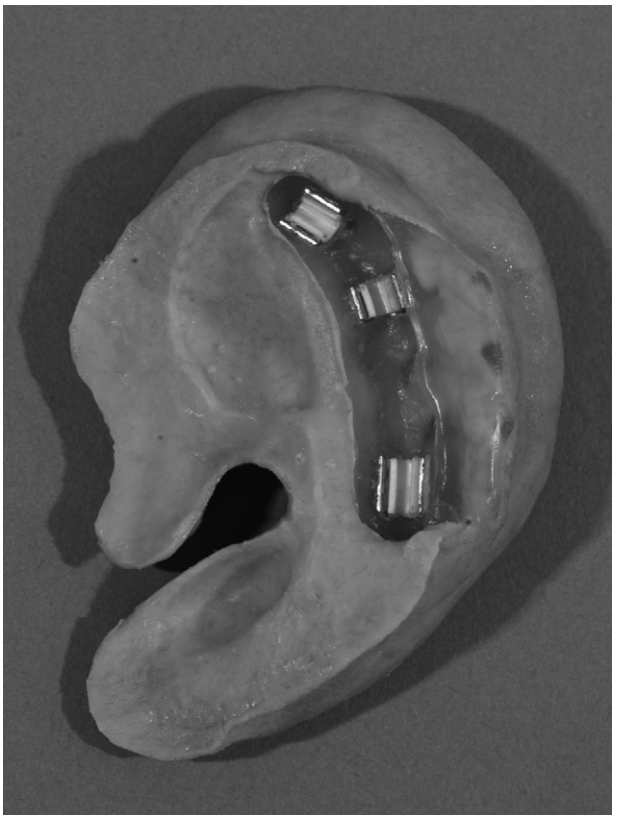


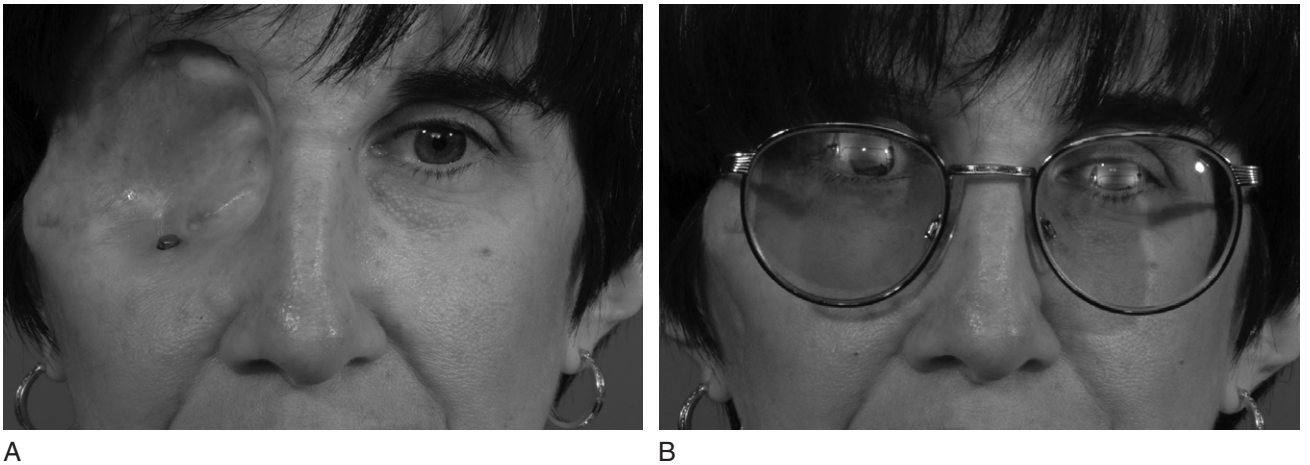
Figure 2 • Tissue surface of an implant-retained silicone elastomer auricular prosthesis. Retentive clips engage a bar attached to implants.

OCULAR PROSTHESIS. When an eye is lost as a result of trauma but the contents of the orbit remain (such as the extraocular muscles), the globe may be replaced with an ocular prosthesis. The prosthesis is custom fabricated from acrylic polymer and can provide a remarkable aesthetic match in most situations.

ORBITAL PROSTHESIS. When the contents of the orbit have been exenterated and the surrounding bone and soft tissues have been resected, a prosthesis can provide a suitable simulation of the lost structures. An orbital prosthesis contains an ocular prosthesis to replace the globe, and silicone elastomeric polymer is used to reproduce the skin of the eyelids and surrounding soft tissue. An orbital prosthesis may be retained by adhesive or by clips or magnets attached to implants placed into the bones surrounding the defect (Fig. 3). The superior orbital rim and the inferior lateral orbital wall provide the most ideal implantation sites.

An excellent aesthetic result can be achieved with the use of adhesive or implants for retention of an orbital prosthesis. Patients with an orbital prosthesis should be encouraged to wear eyeglasses not only to protect the remaining eye but also to cover the skin-prosthesis margin.

NASAL PROSTHESIS. Nasal defects are most often reconstructed surgically with autogenous tissue. Frequently, however, reconstruction is delayed after resection to observe for local recurrence. During this period (often 12 to 18 months), a patient with a partial or total rhinectomy benefits from a nasal prosthesis. When used as a short-term or interim prosthesis, the nasal prosthesis (fabricated from custom-colored silicone elastomeric polymer) is retained by adhesive. If a nasal prosthesis is to serve as a long-term or definitive restoration, consideration should be given to placing osseointegrated titanium implants for retention and avoidance of adhesives, which over time degrade the thin silicone margins of the prosthesis. In addition to the aesthetic role that a nasal prosthesis plays, it also



A B

Figure 3 • **A,** Patient after orbital exenteration with osseointegrated implants in place. **B,** Patient with prosthesis in place. Note patient is wearing glasses both to hide the margins of the prosthesis and to protect the remaining eye.

serves to protect the nasal mucosa from direct contact with cold dry air in the winter months. A nasal prosthesis helps to provide the function of an intact external nose in that it assists in warming, filtering, and moistening inspired air before it enters the lower respiratory tract.

FACIAL PROSTHESIS. Extensive facial defects as a result of trauma or treatment of advanced skin cancer may be restored with a facial prosthesis. Facial prostheses often encompass combined areas such as those resulting from the loss of an eye, nose, and cheek. A facial prosthesis is most often fabricated from custom-colored silicone elastomeric polymer and may be retained by adhesives, by clips and magnets that attach to endosseous implants, or by extensions of the prosthesis into soft tissue undercuts within the defect.

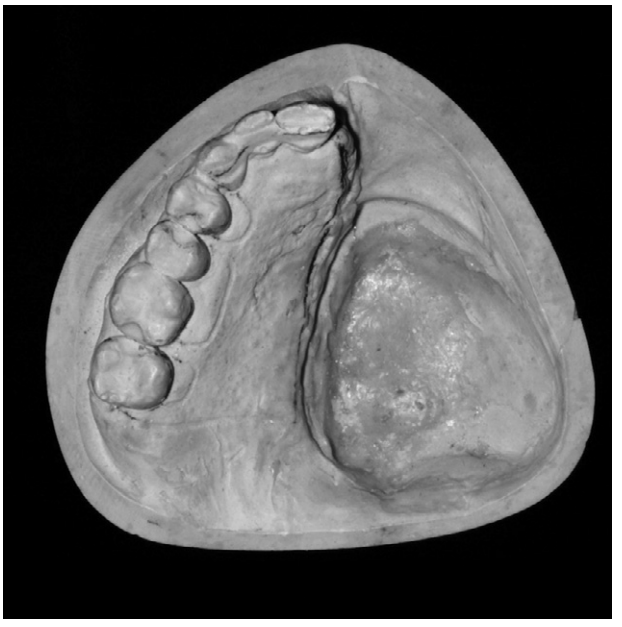
CRANIAL PROSTHESIS. Large cranial defects that cannot be restored with autogenous bone grafts may be candidates for reconstruction with a cranial prosthesis. A cranial prosthesis may be very accurately fabricated from CT data and a stereolithographic model or from traditional techniques of impressioning the defect indirectly. Cranial prostheses may be fashioned from acrylic reinforced with stainless steel mesh and may be wired into place to protect the underlying brain and restore cranial form.

NASAL STENTS. Custom nasal stents may be fabricated to provide support for an internal nasal valve that is deformed or scarred as a result of surgery or trauma. Stents may be made of hard acrylic or flexible silicone and may be modified in conjunction with reconstructive surgery. Serially modified stents may also be used in an attempt to enlarge the circumference of a constricted external naris or nostril.

Intraoral Maxillofacial Prostheses

OBTURATOR PROSTHESIS. Restoration of maxillary defects resulting from tumor resection is most predictably achieved with an obturator prosthesis (Fig. 4). An *immediate* or *surgical obturator* is fabricated before ablative surgery and inserted while in the operating room after maxillectomy to restore palatal form and provide separation of the oral and nasal cavities. An obturator is usually preferable to an autogenous reconstruction and should be considered when maxillary resection is necessary.

Before insertion of an immediate obturator, the surgical defect may be lined with a split-thickness skin graft to provide greater resistance to abrasion by the prosthesis. The keratinized skin graft also contracts at its junction with the oral mucosa to create a band of scar tissue that provides a "purse string" effect around the obturator. The scar tissue greatly improves retention, stability, and the peripheral seal of the obturator. An immediate obturator



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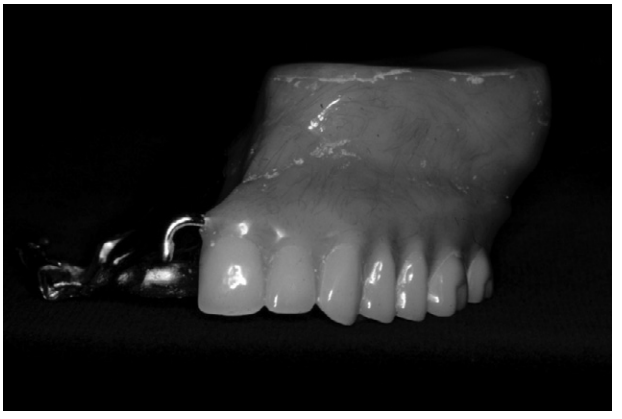

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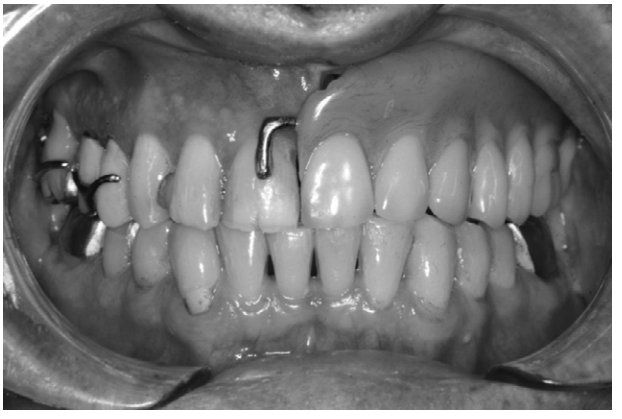

C

Figure 4 • A, Postresection model of maxillary defect. **B,** Maxillary prosthesis. **C,** Prosthesis in place.

is used by the patient during the healing period and may easily be modified as the contours of the defect change over time. The prosthesis is fabricated from processed acrylic and wire clasps, and teeth are used to provide retention. When a patient is edentulous in the maxilla, existing dentures may be modified in the operating room to provide retention by engaging undercuts in the defect. An immediate obturator may also be wired into place during the initial healing period if no other means of retention are available.

After the peripheral tissues surrounding the defect have healed, a *definitive obturator prosthesis* may be fabricated. Additional healing time should be allowed, especially if the surrounding tissues have been irradiated. A definitive obturator is constructed from tissue-colored acrylic and prosthetic teeth and is retained by a metal base and custom cast metal clasps that engage teeth proximal to the defect. A definitive obturator may also be retained by osteointegrated implants or by engaging tissue undercuts in an edentulous patient. Placement of osseointegrated implants in bone that has been irradiated is associated with a higher incidence of failure and remains controversial. Hyperbaric oxygen therapy before and after implant placement is believed to reduce the risk of implant loss in irradiated bone.

A definitive obturator can be used to restore a large defect of the maxilla that encompasses both hard and soft palatal tissue and can assist in velopharyngeal closure. Neurologic deficits caused by trauma, tumors, or cerebrovascular insults that result in impaired or deficient velopharyngeal function may also be improved by a *soft palate obturator*. Such prostheses are commonly referred to as *speech bulb prostheses*. These obturators provide the function of the soft palate and other structures of the velopharyngeal mechanism and are fabricated with the assistance of nasoendoscopy and a speech pathologist to assess the quality of its function.

CLEFT PALATE PROSTHESIS. A patient with a cleft palate may benefit from a variety of intraoral prostheses, depending on the age of the patient. Before surgical repair, the alveolar segments adjacent to a unilateral cleft may be moved closer together and the nasal cartilage repositioned with a *nasoalveolar molding appliance*. An infant with a bilateral cleft lip and palate may also have the alveolar segments and premaxilla repositioned and the columella lengthened nonsurgically with a *bilateral nasoalveolar molding appliance*.

As a child with a cleft palate grows, a *pediatric speech bulb appliance* may be used in some situations to improve velopharyngeal closure. Speech bulb obturators are indicated when pharyngeal flaps or pharyngoplasty procedures have been unsuccessful.

Removable partial-denture prostheses or *overdenture prostheses* are effective in replacing missing or malpositioned teeth in both adolescent and adult cleft palate patients. These prostheses may include an obturator portion if required. In situations in which only a maxillary lateral incisor is congenitally absent as a result of the cleft, an endosseous dental implant may be placed into the cleft site if adequate bone is present, either after early gingivoperiosteoplasty or after alveolar bone grafting. The single dental implant is a conservative method of replacing the missing incisor and preserves the teeth proximal to the cleft that might otherwise have been prepared for a fixed dental prosthesis. Adult cleft patients with a need for extensive prosthetic dental rehabilitation may be reconstructed with fixed dental prostheses ("fixed bridge"), removable prostheses, implant-supported prostheses, or a combination of these.

MANDIBULAR RESECTION PROSTHESIS. A patient who has undergone mandibular resection is best reconstructed with vascularized autogenous free flaps. The usual donor site is the fibula; the iliac crest is also a potential donor site. Prosthetic dental reconstruction may be possible for many autogenously reconstructed mandibular resection patients when combined with placement of titanium endosseous dental implants. The dental implants may then be used to retain either a fixed or a removable dental prosthesis to restore facial form and provide masticatory function.

Patients who elect to forego autogenous reconstruction or whose reconstruction has failed may have a *mandibular resection prosthesis* fabricated to replace missing teeth. In an edentulous post-resection patient without adequate teeth to provide sufficient retention for a prosthesis, a mandibular resection prosthesis often provides improvement in cosmesis only and is minimally functional. Dentate patients undergoing mandibular resection who are not immediately reconstructed autogenously are candidates for a *mandibular guidance appliance*. The unreconstructed/resected mandible is pulled to the resected side because of a lack of post-resection muscle balance. A mandibular guidance appliance assists in maintaining the pre-resection dental occlusion on the nonresected side by guiding the teeth on the nonresected side into a normal occlusal relationship.

Postoperative Care

Autogenous reconstruction provides living tissue that is capable of renewing and regenerating itself, whereas a prosthesis is a nonrenewable device that requires maintenance and, eventually, replacement. The life span of a prosthesis varies. Extraoral silicone prostheses usually last for 2 to 3 years before they must be replaced. Implant-retained prostheses last longer than adhesive-retained pros-

theses because chemical components in the adhesive lead to decreased flexibility in the silicone polymer and make the edges brittle and prone to tearing. Over time, the color of the prosthesis also fades.

The skin surrounding endosseous craniofacial implants requires diligent daily hygiene to maintain optimal tissue health. The adhesive residue must be removed daily before fresh adhesive is applied to an adhesive-retained prosthesis. Both implant and adhesive-retained prosthesis must be removed for some period each day to maintain tissue health.

Immediate obturator patients require regular postsurgical care during the initial weeks of healing. Approximately 7 to 10 days after surgery and placement of the prosthesis, the patient is seen to remove the surgical packing placed between the skin graft and the superior surface of the prosthesis. The prosthesis is then modified on an almost weekly basis. Soft denture lining material is added to the tissue-contacting surface of the prosthesis to maintain stability and improve retention.

Pearls and Pitfalls

- The plastic surgeon and prosthodontist must understand the capabilities and techniques that each has to offer.
- In any treatment plan requiring fabrication of a prosthesis, the result is improved by *preoperative* consultation with a maxillofacial prosthodontist.
- The maxillofacial prosthodontist serves as an integral member of the craniofacial, cleft palate, or head and neck cancer reconstructive team.
- Autogenous tissue reconstruction of maxillary defects, if not properly planned, may prevent prosthetic dental rehabilitation. Poorly positioned soft tissues may not provide a solid foundation for a prosthesis, and an improperly positioned osteomyocutaneous free flap may prevent the placement of a removable or even an implant-retained prosthesis.
- Soft tissue reconstruction after orbital exenteration may preclude placement of an orbital prosthesis.

SUGGESTED READINGS

Beumer J III, Curtis TA, Marunick MT: Maxillofacial Rehabilitation—Prosthodontic and Surgical Considerations. St. Louis, Ishiyaku EuroAmerica, 1996.

Desjardins R: Early rehabilitation management of the maxillectomy patient. J Prosthet Dent 38:31, 1977.

Granstrom G, Jacobsson M, Tjellstrom A: Titanium implants in irradiated tissue: Benefits from hyperbaric oxygen. Int J Oral Maxillofac Surg 7:15, 1992.

Thorne CH, Brecht LE, Bradley JP, et al: Auricular reconstruction: Indications for autogenous and prosthetic techniques. Plast Reconstruct Surg 107:1241, 2001.

Urken ML, Buchbinder D, Constantino PD, et al: Oromandibular reconstruction using microvascular composite flaps: Report of 210 cases. Arch Otolaryngol Head Neck Surg 124:46, 1998.

Acute Management of Head and Neck Trauma

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LARRY HOLLIER

Treatment of head and neck trauma can often present challenging scenarios both in preservation of life and in restoration of form and function. Although the plastic surgeon usually serves on the consultation service after assessment and stabilization have been provided by emergency center staff, the potential for rapid changes in the disposition of the patient or for missed diagnosis exists. In addition, many patients with injuries to the head and neck region have other significant injuries. Such injuries require that the plastic surgeon have a strong understanding of the acute management of head and neck trauma.

Primary Survey

The approach to treatment of trauma in the United States is outlined in the course and materials of *Advanced Trauma Life Support for Doctors* (ATLS). The strategy is to attend to the most life-threatening aspects of an injury via a primary survey before complete assessment of the patient's injuries. On completion of the primary survey, the secondary survey is begun and involves a comprehensive assessment (from "head to toe") of the patient.

The primary survey follows the acronym "ABCDE," which outlines the following sequence of care:

- **A** Airway maintenance with cervical spine (C-spine) protection
- **B** Breathing and ventilation
- **C** Circulation with hemorrhage control
- **D** Disability: neurologic status
- **E** Exposure/Environmental control: the patient is completely undressed while preventing hypothermia

Although listed as a sequence in the order of priority, several assessments can be performed simultaneously. In addition, a change in the patient's status or completion of an intervention on the patient requires reassessment of the patient, again starting with "airway." Moreover, completion of the survey does not exclude the importance of future reassessment of the patient, again starting with "airway."

Airway with Cervical Spine Protection

As the patient arrives in the emergency center, a rapid assessment of airway patency is made. A quick indication of the patient's status can be made by vocalization; if the patient is able to speak, the airway is patent. Furthermore, if the patient is able to answer and respond appropriately to questions, the primary neurologic assessment is considered negative for central nervous system injury. If airway patency is questionable, a visual inspection of the pharynx and larynx is made to detect any obstructing foreign bodies that could easily be removed with a finger sweep. Care is taken to protect the C-spine by not changing the head position in relation to the body.

In the context of head and neck trauma, there are several indications for intubation. First, ATLS training recommends intubation of a patient with a Glasgow Coma Scale (GCS) score of 8 or less. Other indications would include an inability of the patient to protect the airway because of severe mandibular fractures that allow the tongue to fall back into the pharynx, severe maxillary fractures compromising the airway, and severe nasopharyngeal bleeding raising the risk of aspiration. An indication that the patient is having difficulty keeping the airway open in the face of severe maxillofacial fractures is refusal to lay supine with a preference for a seated position while leaning forward.

Subsequent to the decision to intubate the patient is the decision regarding nasotracheal versus orotracheal intubation. If either route is taken, it is important to keep the C-spine immobilized; the favored technique is a two-person in-line intubation in which one individual manually stabilizes the patient's neck while the other intubates. Nasotracheal intubation has previously been recommended for trauma and is often indicated for trauma to the oropharyngeal area or mandible. However, nasotracheal intubation is contraindicated in the presence of apnea or suspected injury to the cribriform plate. Cribriform plate fractures may manifest as oral, nasal, or auricular drainage of cerebrospinal fluid. Nasotracheal intubation is also relatively contraindicated in the presence of a suspected intracranial injury because of a documented increase in intracranial pressure during nasotracheal intubation. In the context of a suspected laryngeal injury, neither orotracheal nor nasotracheal intubation should be performed, and a surgical airway is indicated. Laryngeal injury is suspected if the patient demonstrates hoarseness, subcutaneous emphysema in the neck, or palpable fractures of the thyroid or cricothyroid cartilage. In the emergency center, the safest procedure is a cricothyroidotomy, with tracheostomy performed later in the operating room after the patient has been further stabilized.

In conjunction with the airway, the C-spine is assessed and protected in the primary survey. Relative injury patterns in the different regions include the cervical spine in 55%, the thoracic spine in 30%, and the lumbar spine in 15%. Complete spinal cord injuries (total loss of motor and sensory function distal to the lesion) occur in 43% to 46% of spinal cord injuries. Variables that correlate with C-spine injury include a fall of more than 10 feet, subjective numbness, sensory loss, motor loss, neck spasm, neck tenderness, objective sensory loss, and weakness or loss of anal sphincter tone.

A victim of trauma arriving in the emergency center via ambulance should have the C-spine immobilized via a backboard and a soft/Philadelphia-style collar with the head further fixed to the backboard. The neck should remain immobilized with a backboard and collar in place until C-spine injury has been excluded. The cervical collar alone limits cervical motion only by approximately 50%, which is not sufficient to protect an injured C-spine. It is important to remember that a pressure sore can begin to form after only 2 hours on a backboard.

It is the role of the physician to determine the need for additional studies to evaluate for the presence of C-spine injury. The evaluation may include only a physical examination or radiographs ranging from the standard three-view radiographic series (lateral, anteroposterior, and odontoid), to flexion/extension films, to C-spine computed tomography (CT). In the presence of an injury, a lateral C-spine radiograph is approximately 85% sensitive, and the combination of lateral, anteroposterior, and odontoid films is approximately 92% sensitive; additional views provide no further sensitivity. Any abnormality detected by plain radiograph should be further evaluated with C-spine CT.

Debate exists regarding the extent of studies/examinations required to exclude C-spine injury. ATLS training teaches that any patient sustaining maxillofacial or head trauma should be presumed to have an unstable C-spine injury and that the neck should remain immobilized until adequate physical examination and radiographic studies rule out such injury. The NEXUS low-risk criteria for C-spine injury, which help exclude the need for radiographic studies, include the absence of midline tenderness, a normal level of alertness, no evidence of intoxication, no abnormal neurologic finding, and no painful distracting injuries. In an examination of their own clinical data, Stiell and colleagues (2001) calculated a sensitivity of 99.6% and a specificity of 12.4% for the NEXUS exclusion criteria. Furthermore, from their data they derived the Canadian C-Spine Rule Exclusion Criteria with 100% sensitivity and 42.5% specificity: absence of any high-risk factor (age older than 65 years, dangerous mechanism of injury, or paresthesias in the extremity), the presence of any low-risk factor allowing for safe assessment of range of motion (simple rear-end motor vehicle collision, sitting position in the emergency center, ambulatory at any time since the injury, delayed onset of neck pain, or absence of midline C-spine tenderness), and the ability of the patient to actively rotate the neck 45 degrees to the left and right.

Attempts to correlate specific injuries with C-spine injury have yielded minimal consensus other than that the forces required to fracture the C-spine generally had to be applied toward the upper part of the face and scalp. A retrospective analysis of isolated penetrating gunshot wounds to the head suggested that C-spine immobilization and diagnostic radiography were not necessary and could even delay or complicate emergency airway management.

After a C-spine injury is diagnosed, it is important to document the deficit with regard to the anatomic level, clinical severity, and "sacral sparing" (presence/absence of anal tone). Acute management includes the administration of intravenous steroids (a bolus of prednisone 30 mg/kg over a 15-minute period and 5.4 mg/kg/hr for the next 23 hours), administration of an H_2 blocker/sucralfate/proton pump inhibitor, administration of prophylactic heparin, placement of a Foley catheter, and placement of pneumatic compression devices.

Breathing

Establishment of a patent airway does not guarantee adequate gas exchange. In addition to examination of the lips, mucous membranes, and extremities for evidence of hypoxemia, the adequacy of breathing can be assessed by auscultation, a chest

radiograph, arterial blood gas measurement, or continuous monitoring with pulse oximetry. With pulse oximetry it is important to remember that spurious values can result from hypothermia or the presence of carbon monoxide or methemoglobin.

Inadequacies in gas exchange result from several different mechanisms ranging from physical to metabolic. In a trauma patient, however, the first mechanisms to consider are injuries that can impair gas exchange, including tension pneumothorax, a flail chest with pulmonary contusion, massive hemothorax, or an open pneumothorax.

Circulation

Any finding of hypotension must first be considered to result from hypovolemia and treated as such until proved otherwise. Assessment of the circulation of a trauma patient in the emergency center includes obtaining venous access with at least two large-bore peripheral intravenous catheters. Further assessment includes examination of the level of consciousness, pulse, and skin color, all of which can be indicators of hypotension. It is important to remember that pulse is not a satisfactory indicator of volume status in the young, elderly, or patients taking β-blockade medications. Finally, assessment of the circulation includes examination for the sources of hemorrhage and control of bleeding, if possible, in the emergency center.

For control of hemorrhage, the first maneuver should be direct pressure. In the context of head and neck trauma, hemorrhage from scalp lacerations can be quickly controlled by stapling the free edges or approximating the edges with large sutures (with later revision along with proper wound care). Facial and neck lacerations are best controlled with direct pressure until proper studies or wound exploration and repair (or both) can be performed. Blind clamping with hemostats can result in damage to critical neurovascular structures and ducts in these regions. Profuse bleeding from the nose or mouth may occur, particularly when the maxillary artery is lacerated. Along with intubation to secure the airway, the patient's posterior oropharnyx and nasopharynx should be packed. If this measure fails to control the bleeding, the patient should be brought to the interventional radiology suite for embolization.

Disability

Disability is determined by rapid assessment of neurologic function. In the evaluation of head and neck trauma, an important caveat is that intracranial injuries often accompany facial fractures; complete assessment of the patient includes a CT scan of the head. Assessment of disability first begins with a brief history as taken from observers or the medical transport team, with a focus on the initial status of the patient and whether it is improving or deteriorating. When examining the patient, rapid indicators of an intact central nervous system are the ability of a patient to speak and respond properly to questions and commands. The international standard in quantifying this examination is the Glasgow Coma Scale (GCS) score (Table 1). When recording the score, it is important to document the deficits in each category for future referral.

TABLE 1 Glasgow Coma Scale

ASSESSMENT AREA	SCORE
Eye Opening (E)	
Spontaneous	4
To speech	3
To pain	2
None	1
Best Motor Response (M)	
Obeys commands	6
Localizes pain	5
Withdraws from pain	4
Decorticate flexion	3
Decerebrate extension	2
None	1
Best Verbal (V)	
Oriented	5
Confused	4
Inappropriate	3
Incomprehensible	2
None	1

Acute management in the emergency center includes ventilation, fluid resuscitation, reduction of intracranial pressure and, as indicated, administration of anticonvulsant and antibiotic medications. As mentioned previously, a GCS score of 8 or less is considered an indication for intubation. Indications for intubation of a head-injured patient include the following: avoidance of hypoventilation, protection of the C-spine, minimization of gastric distention, avoidance of maneuvers that might increase intracranial pressure, and the concomitant use of muscle relaxants. In penetrating trauma to the head, the patient should be hyperventilated with supplemental oxygen to a target P_{CO_2} of 30 mm Hg. Fluid resuscitation of a head-injured patient involves the use of isosmotic fluids, including lactated Ringer's solution or normal saline, until a target mean arterial pressure of 90 mm Hg is attained. In addition to proper ventilation, intracranial pressure can be reduced through an intravenous bolus infusion of mannitol (1 g/kg). Administration of anticonvulsants is recommended when an intracranial lesion has been demonstrated by CT scan or is suspected by clinical evaluation. Prophylactic antibiotic administration is recommended if the dura has been violated. Finally, in the acute management of head trauma, steroids have not been shown to improve outcome.

After stabilization has been initiated, intracranial head trauma necessitates the assistance of a neurosurgeon for further management. Of particular importance to any physician caring for a potential head trauma patient are the signs of impending uncal herniation as manifest by Cushing's triad: systemic hypertension, bradycardia, and hypoventilation.

Exposure/Environmental Control

The last step in the primary survey is removal of all the clothing of the patient to detect injuries that could be hidden by garments. Removal of wet clothing also prevents the development of hypothermia. The patient, once exposed, needs to be kept warm by means of a warm examination room and the use of blankets and additional warming devices, including warming blankets and bags of warmed intravenous fluids. Total exposure prepares the patient for a complete secondary survey.

Secondary Survey

The secondary survey is a careful reevaluation of the patient, including a "head to toe" physical examination. The first step is a complete history. In addition to the routine questions regarding past medical history, allergies, medications, ingestion of alcohol or other mind-altering substances, and time of the last meal, questions aimed at the particulars of the trauma are important.

The "physics" involved in the trauma predict the path or extent of damage, or both, and give the physician a tailored approach to the trauma patient. For example, blunt trauma has a wider extent of injury than penetrating trauma. With blunt trauma, specifically motor vehicle collisions, the direction of impact and the speed of impact are important factors in the injuries suffered. With penetrating trauma, specifically gunshot wounds, the extent of energy is dependent not only on the muzzle velocity of the gun but also on the size, profile, tumble, and fragmentation of the missile. A history of the injury can be obtained from relatives, witnesses, the emergency transport team, and any law enforcement personnel who come to the emergency center with the patient.

The history is followed by a complete physical examination and the use of radiographs and other studies designed to evaluate specific injuries as outlined in the following sections. With head and neck trauma, the physical examination should follow an orderly fashion proceeding methodically through the several different "systems" present in the head and neck region. First, the patient, if conscious, should be questioned regarding any abnormalities in dental occlusion, diplopia, or paresthesias. Second, a visual assessment is made by noting any asymmetry in facial expression or extraocular movement, changes in visual acuity, sources of bleeding, or drainage of fluid from the ears, eyes, nose, or mouth. Cerebrospinal fluid leaks from the ears or nose are often copious and initially bloody but become clearer over time. The entire head and neck region should be palpated for areas of tenderness, crepitation, bony abnormalities, and lacerations specifically within hirsute regions. In addition, the mandibular or maxillary dental surfaces can be palpated, again noting step offs, malalignment, and loose, broken, or avulsed teeth. Paresthesias can be elucidated by palpation or "pinprick" examination of the skin areas innervated by the supraorbital, infraorbital, and mental divisions of the trigeminal nerve.

The physical examination and treatment of the head and neck in a trauma patient can be further subdivided into several different systems, each requiring specific approaches to rule out and treat injuries: the eye, face, and neck.

Eye

Treatment of any trauma affecting the upper part of the face requires an evaluation of the eye and orbit. First, the lid should be examined for lacerations or ptosis. With full-thickness lid lacerations, the integrity of the underlying globe requires assessment. It is important to remember that because of Bell's phenomenon (superolateral rotation of the globe with eye closure), even lacerations of the upper lid may be accompanied by injuries to the inferior aspect of the globe. Lacerations of the medial quarter of the upper or lower lid also increase the possibility of damage to the lacrimal drainage system and as such must be carefully evaluated and documented.

After examination for the presence of lacerations, visual acuity should be assessed; however, it is often difficult to evaluate acuity accurately in the emergency setting. A key in determining a subtle problem with the optic nerve is an observed change in the patient's color perception, particularly with the color red. A difference between the two eyes in terms of color desaturation indicates potential compromise of the optic nerve. A rapid method to assess such injury is to place a penlight against the pulp of the finger to make the nail bed red. The next component of the examination should be evaluation of extraocular movements. Diplopia in either the primary field of gaze or the extremes of gaze is abnormal and may be caused by damage or entrapment of the extraocular muscles or damage to cranial nerves III, IV, or VI.

Pupillary size and response to light should be evaluated. Differences in the resting size of the pupils are abnormal and should be documented. A difference in pupillary reflexes is of particular concern. A normal pupil should respond to both direct and consensual light stimulation. A pupil that fails to constrict with either direct or consensual

light indicates motor dysfunction, as with injury to cranial nerve III or after the application of topical dilators. A pupil that fails to constrict with direct stimulation but exhibits normal consensual constriction indicates difficulty in the perception of light in the affected eye, as with optic nerve damage. This abnormality is termed an afferent pupillary defect.

The anterior chamber of the eye should be examined for clarity and with lateral illumination for hyphema. Corneal abrasions frequently manifest as photophobia and pain and may be diagnosed by using fluorescein and a Wood lamp. Treatment involves irrigation and the application of topical ophthalmic antibiotic ointment.

The lens should be transparent. A vitreous hemorrhage gives a black reflex, as opposed to the red reflex usually visualized with ophthalmoscopy. The retina is examined for hemorrhage, tears, or displacement.

When the orbit has been subjected to substantial force, a CT scan is indicated to evaluate for the presence of fractures. The CT scan should be performed at 1.5-mm intervals through the entire orbit and optic canal and should be done in both the axial and coronal planes. Indications for repair of an isolated orbital fracture include persistent diplopia in primary gaze, enophthalmos exceeding 2 mm, and symptomatic entrapment of the extraocular muscles. Most orbital floor fractures can be observed for 7 to 10 days before repair to allow any soft tissue swelling to resolve.

Facial Bones

The patient's face should be palpated for areas of tenderness or irregularity in bony contour. Of particular importance is the presence of malocclusion. Patients can detect even subtle changes in the normal bite relationship that are not apparent to the examining physician; such changes are indications of an underlying fracture of the mandible or possibly the maxilla.

Any significant findings on physical examination require CT scanning. Although some clinicians "screen" patients for fractures with a plain radiographic series, most prefer to proceed immediately to a CT scan because essentially all definitive care is based on CT. The one exception is a mandibular fracture, in which the best combination of radiographs is a Panorex and an anteroposterior view of the mandible.

Both axial and coronal CT images should be obtained. However, coronal images require hyperextension of the neck, which is contraindicated when the C-spine is not yet cleared of injury. In these situations, coronal reformations may be constructed from the axial image data, but the resolution of reformatted data is of poorer quality.

Although many patients with thoroughly evaluated, isolated facial fractures may be safely discharged from the hospital with follow-up scheduled within the next 24 to 48 hours, most mandible fractures require admittance for pain control and antibiotic administration. Surgery should be undertaken fairly rapidly in these patients because of difficulties experienced with eating and the propensity for these fractures to become infected.

Nerves

In the examination of a patient with acute head and neck trauma, facial nerve function must be assessed. Especially in patients with facial lacerations, care should be exercised in evaluating the entire spectrum of facial movements: forehead elevation, forced eye closure, upper lip elevation, and lower lip depression. The most frequent mistake made when performing such evaluation is to assess lower lip depression by having the patient smile, a maneuver that does not produce lower lip depression. Rather, patients should be asked to show the examiner their teeth, a more sensitive method of detecting the absence of lip depression. Any abnormality in function in conjunction with overlying facial lacerations should be presumed to be secondary to nerve transection and it requires exploration in the operating room. Some clinicians advocate that lacerations medial to the lateral canthus need not be explored because there is extensive arborization of the buccal branches at this level that allow for subsequent reinnervation. In general, repair should be attempted as soon as possible and certainly within the first 72 hours after injury. Beyond this time, the distal nerve stumps cannot be stimulated with a hand-held nerve stimulator to demonstrate muscular movement.

Ear

Lacerations of the ear are common in facial trauma. There are several unique concerns in this region. First and foremost is the formation of a hematoma. Because cartilage has poor vascularity, auricular hematomas may cause pressure necrosis and subsequent resorption of ear cartilage—the classic "cauliflower ear." The presence of any hematoma mandates immediate evacuation and placement of a bolster dressing to prevent reaccumulation.

Infection of the ear cartilage is particularly troublesome, again because of the poor vascular supply. Severe pain is the hallmark of auricular chondritis. If the ear cartilage becomes infected, the patient should be admitted to the hospital for intravenous antibiotics, and conservative débridement of the site of infection may be indicated.

When repairing ear lacerations involving the cartilage, it is generally unnecessary to place a separate suture layer in the cartilage for support. Because of the close adherence of skin to the lateral aspect of the ear, skin approximation alone is sufficient.

Nose

Nasal fractures are among the most common injuries of the face. The diagnosis can be based on physical examination alone; radiographs typically offer little additional information. Appropriate physical examination includes intranasal inspection with the use of a speculum to visualize the septum. Particular attention should be paid to the presence of a septal hematoma, which mandates immediate drainage. Allowing the hematoma to consolidate may lead to loss of cartilage within the septum and a subsequent saddle nose deformity.

Nasal fractures should be reduced. Reduction may be done at the time of injury before swelling has become severe. If the patient is seen at a later point, attempts at closed reduction can be delayed for several weeks to allow for the swelling to subside. It is important to counsel patients that even after successful closed reduction, subsequent reconstructive rhinoplasty may be necessary.

Neck

The most important caveat in the management of penetrating neck wounds is to refrain from probing or locally exploring any wound in the emergency center because of the risk of dislodging a blood clot that may be sealing a serious vascular injury. Any injury deep to the platysma should be considered in this category. For the purpose of evaluation, the neck has been divided into three zones: zone I, above the clavicles and below the cricoid cartilage; zone II, between the cricoid cartilage and the angle of the mandible; and zone III, above the angle of the mandible to the base of the skull. Although management of these injuries is the subject of a great deal of controversy, there is a tendency for selective exploration of most penetrating injuries to the neck.

The decision to operate is based on a graduated diagnostic protocol, with surgery performed if any step is positive. The initial step is the physical examination. Findings mandating operative exploration include a hematoma or significant bleeding, neurologic changes, airway difficulties, crepitus, or hematemesis. Angiography may be performed to rule out an injury to the major blood vessels of the neck. Care should be taken when interpreting these results in zone I because the abundance of great vessels in this region may obscure subtle injuries. In addition to angiography, evaluation of penetrating neck wounds, especially zone II injuries, includes a barium swallow to detect clinically occult esophageal injuries. Any finding on the barium study is confirmed by rigid esophagoscopy. If all studies are negative, the patient is admitted for close observation.

Pearls and Pitfalls

- Although the plastic surgeon typically serves in the context of a consulting service, the potential for rapid changes in the patient's condition makes it imperative that the surgeon fully understand acute management of the injuries.
- Control of the airway is critical.
- Cervical spine injury must be suspected and ruled out.
- Care must be taken not only to rule out and address life-threatening injuries but also to reestablish function and achieve an aesthetically acceptable repair.

SUGGESTED READINGS

Ali J, Aprahamian C, Bell RM, et al: Advanced Trauma Life Support for Doctors, Student Manual. Chicago, American College of Surgeons, 1997.

Babcock JL: Cervical spine injuries. Diagnosis and classification. Arch Surg 111:646-651, 1976.

Britt LD, Peyser MB: Penetrating and blunt neck trauma. In Mattox KL, Moore EE, Feliciano DV (eds): Trauma. Norwalk, CT, Appleton & Lange, 2000, pp 437-450.

Broocker G, Mendicino ME, Stone CM: Injury to the eye. In Mattox KL, Moore EE, Feliciano DV (eds): Trauma. Norwalk, CT, Appleton & Lange, 2000, pp 401-414.

Cooper PR: Gunshot wounds of the brain. In Cooper PR (ed): Head Injury. Baltimore, Williams & Wilkins, 1993, pp 355-372.

Klyachkin ML, Rohmiller M, Charash WE, et al: Penetrating injuries of the neck: Selective management evolving. Am Surg 63:189-194, 1997.

Lillard PL: Five years experience with penetrating craniocerebral gunshot wounds. Surg Neurol 9(2):79-83, 1978.

Maisel RH, Hom DB: Penetrating trauma to the neck. Otolaryngol Head Neck Surg 3:1707-1720, 1998.

Marion DW, Przybylski GJ: Injury to the vertebrae and spinal cord. In Mattox KL, Moore EE, Feliciano DV (eds): Trauma. Norwalk, CT, Appleton & Lange, 2000, pp 451-471.

Robinson RJS, Mulder DS: Airway control. In Mattox KL, Moore EE, Feliciano DV (eds): Trauma. Norwalk, CT, Appleton & Lange, 2000, pp 171-194.

Schultz RC: Soft tissue injuries of the face. In Aston SJ, Beasley RW, Thorne CHM (eds): Grabb and Smith's Plastic Surgery. New York, Lippincott-Raven, 1997, pp 371-382.

Shires G, Thal E, Schwartz SI: Trauma. In Schwartz SI, Shires G, Spencer F (eds): Principles of Surgery. New York, McGraw-Hill, 1989, pp 2132-2182.

Stiell IG, Wells GA, Vandemheen KL, et al: The Canadian C-spine rule for radiography in alert and stable trauma patients. JAMA 286:1841-1848, 2001.

Facial Soft Tissue Injuries: Evaluation and Repair

PAUL N. MANSON

As a surgeon evaluates the patient with facial injuries, it is critical to identify and differentiate the various injuries, formulate a treatment plan for correction of the individual problems, and complete that plan.

Etiopathogenesis

Soft tissue injuries of the face most often result from motor vehicle accidents, falls, sporting accidents, or assaults. If a soft tissue laceration exists, approximately 25% of patients in a motor vehicle accident have an underlying facial bone fracture. Soft tissue injuries can be divided into contusions, abrasions, and lacerations. Lacerations that transect arteries can produce uncontrolled hemorrhage, especially partially transected arteries. Deep lacerations may extend to the salivary glands and ducts, the facial mimetic muscles, and cranial nerves V and VII. There is generally a male preponderance. Approximately half the patients in motor vehicle accidents have used drugs or alcohol before the motor vehicle accident.

Facial soft tissue injuries occur in a "T-shaped" pattern over the forehead, brows, lips, and chin (Figs. 1 and 2). They occur most commonly along relaxed skin tension lines, at muscular or vascular interfaces that cleave soft tissue, and over bony prominences. Lacerations are more common over the more rigid facial bones and less common around regions of low fracture tolerance, such as the nose and zygomaticotemporal area. The combined use of air bags and seat belts confers the highest level of protection against facial lacerations, cervical spine injuries, and facial fractures.

Pathologic Anatomy

The soft tissue layers of the face include the skin, subcutaneous tissue, the superficial musculoaponeurotic system (SMAS) with its contained mimetic muscle, additional fine areolar tissue, and mucosa. The sensory nerves originate from the trigeminal nerve (cranial nerve V) or the cervical nerves. The facial nerve (cranial nerve VII) exits the skull at the stylomastoid foramen, enters the parotid gland, travels in the lateral aspect of the face and runs centrally beneath the facial muscles to innervate the mimetic system from below the muscles. Exceptions to inferior innervation of the facial muscles are the buccinator, levator labii superioris, orbicularis oris, and depressor angularis oris, which are innervated by cranial nerve VII from above (Fig. 3).

The soft tissue contains the parotid gland, which is positioned superoinferiorly from the zygomatic arch to the inferior border of the mandible and posteroanteriorly from the ear to the midportion of the cheek. Stenson's duct empties into the mouth opposite the second maxillary bicuspid and is located along a transverse line from the ear canal to the base of the nose. It is in close proximity to the buccal branch of the facial nerve (cranial nerve VII) so that injury to one structure is usually accompanied by injury to the other (Fig. 4).

The mechanism of the injury dictates the amount of crushed or devitalized tissue (or both) present in an injury. Tissues may be contused, crushed, torn, avulsed, or lacerated. Combination injuries are the most common, and hematoma is invariable. Crushed tissue should be excised if marginally viable; however, conservative excision of tissue is recommended in critical areas such as the eyelids, eyebrows, nostril rim, and vermilion border.

The blood supply to the face is profuse and predominantly comes from branches of the external carotid: the facial, internal maxillary, and superficial temporal arteries. Arterial and venous connections occur between the internal and external carotid arteries and the arteries in the face; around the skull base the tributaries of the internal jugular

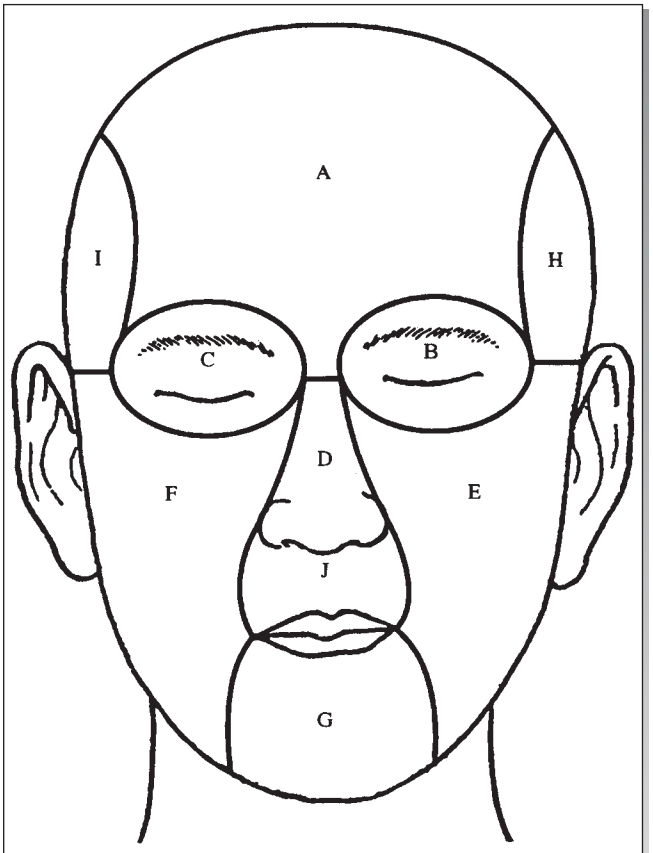

Figure 1 • Designated areas of the face. **A,** Forehead; **B,** left periorbital region; **C,** right periorbital region; **D,** nose; **E,** left cheek; **F,** right cheek; **G,** chin; **H,** left temporal region; **I,** right temporal region. (© Johns Hopkins University. Lee RH, Gamble WB, Mayer M, Manson PN: Pattern of facial lacerations from blunt trauma. Plast Reconstr Surg 99:1544-1554, 1997.)

and intracranial circulation have valveless communication.

The lymphatics of the face drain posteriorly and laterally. Partially avulsed tissue is frequently associated with lymphedema, which is exacerbated long-term by soft tissue scarring and fat necrosis. A "trapdoor" deformity may be produced by fat necrosis and lymphedema. Generous excision of the potentially ischemic fat tissue is desirable; however, in critical anatomic areas, excision may produce tissue discrepancies that are difficult to resolve.

Diagnostic Studies

Precise inspection performed under local or general anesthesia is the most helpful diagnostic tool. Such inspection allows determination of the depth and direction of the wound and permits assessment of the soft tissue structures injured. The age of the wound determines the necessity for débridement by sharp excision of the wound edge and for reinspection of the wound at 48-hour intervals, including inspection of marginally viable tissue.

Radiologic studies can assist in detection of deeper injuries, but they are far less important than careful inspection. Plain radiographs are seldom of value in soft tissue injuries unless a foreign body is identified. Most glass, for instance, is not leaded and therefore not apparent on radiographic study.

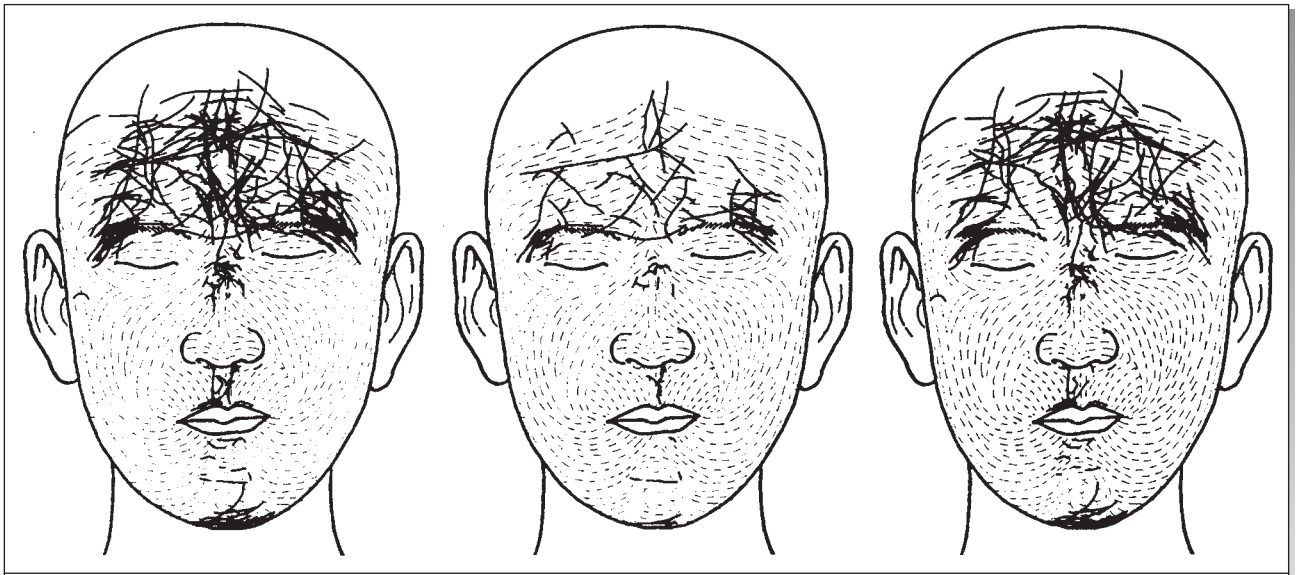

Figure 2 • Composite tracings of lacerations on a face template showing the cleavage lines of Nakano (1983). (© Johns Hopkins University. Lee RH, Gamble WB, Mayer M, Manson PN: Pattern of facial lacerations from blunt trauma. Plast Reconstr Surg 99:1544-1554, 1997.)

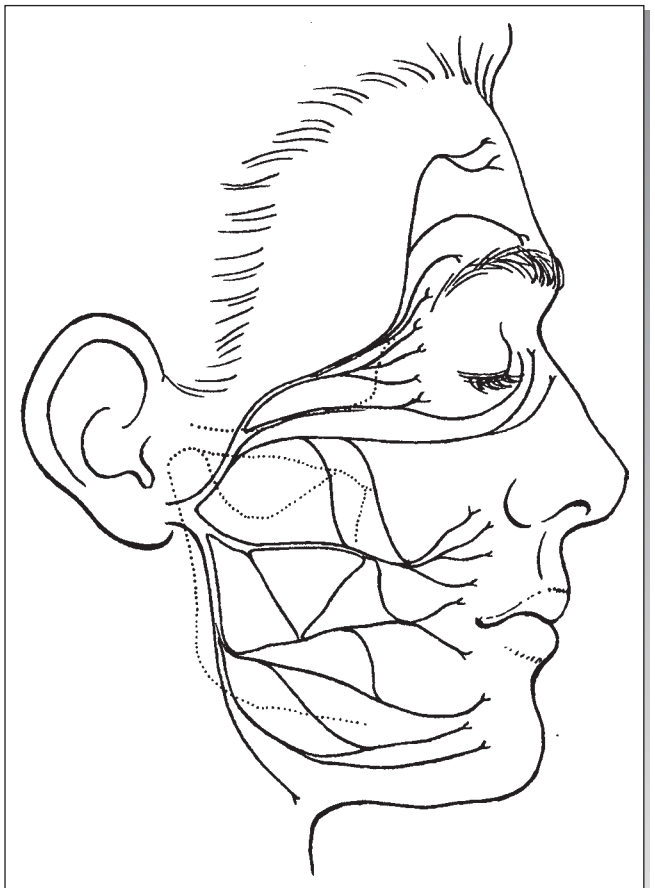

Figure 3 • Facial nerve branches and their ramifications: from above, temporal, orbital, buccal, marginal mandibular, and cervical nerves. (© Johns Hopkins University.)

Photographs

A complete photographic record of the wound should be obtained, as well as a written description for medicolegal documentation. Photographs supplement and enhance the value of written medical reports. They also help the patient appreciate the severity of the injury and the effectiveness of treatment.

Reconstructive Goals

The reconstructive goals of soft tissue repair include (1) removal of contaminated material and devitalized tissue; (2) sculpting of the wound edge to produce a flat even repair; and (3) performance of a layered repair, which splints the wound in an anatomic position and minimizes dead space as well as hematoma and scar tissue formation.

The final scar outcome can be minimized by avoidance of tension, placement of deep sutures, avoidance of forceps or manipulative damage to the skin, and supportive taping after suture removal. In all soft tissue wounds an order of repair is identified, first proceeding with repair of underlying fractures or the application of arch bars. Soft tissue wounds may be used in appropriate circumstances as an access for fracture repair. The soft tissue wound should not be extended to accomplish a repair, however, because such extension increases the extent of facial scar formation. The glandular repair is performed first followed by muscle or nerve repair (or both). Repair of fat and subcutaneous tissue is performed before repair of the dermis, with skin closure as the final step.

Inspecting Soft Tissue Wounds

All soft tissue wounds should be inspected to determine the exact extent of damage and presence of foreign material. The ends of transected structures (such as ducts or muscles) should be identified. Removal of devitalized soft tissue and foreign bodies is necessary to prevent infection, delayed healing, and abnormal scar formation. Foreign material reduces the bacterial inoculum necessary to cause infection from 10^5 organisms per gram of tissue to 10^2 organisms per gram. Bleeding should be controlled with precise ligation of arteries or veins. Cautery of lesser bleeding structures must not damage surrounding fat or muscle. Any hematoma represents a nonvascularized region and is therefore not exposed

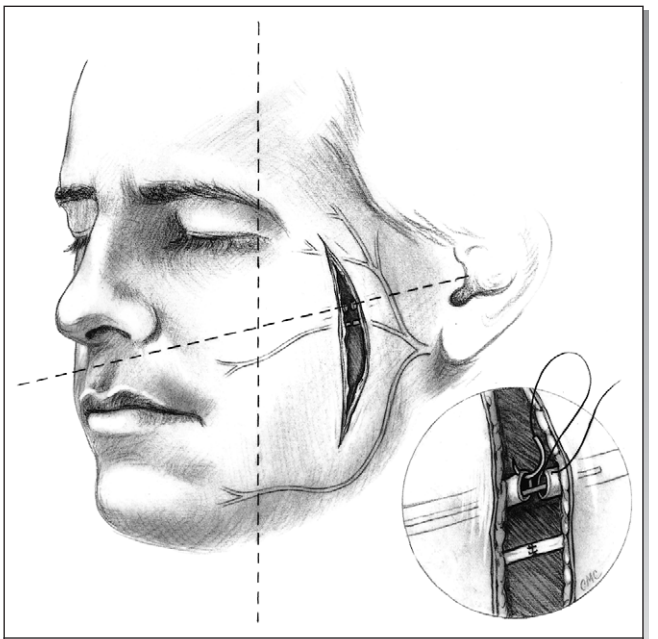

Figure 4 • The buccal branch of cranial nerve VII and Stenson's (parotid) duct travel adjacent to each other on a line from the ear canal to the base of the nose. The *inset* shows repair of the duct over a stent. (© Johns Hopkins University.)

to white blood cells or antibiotics. In areas in which soft tissue has been avulsed, layers of soft tissue are separated or contamination has been significant, or there has been laceration of glands, prevention of fluid accumulation within the wound is accomplished with suction drainage. Progressive necrotizing infections occur in contaminated wounds in which soft tissue viability is compromised and cleansing is inadequate.

Cleaning Soft Tissue Wounds

Contamination is directly proportional to the length of time that has elapsed from the moment of the injury. Organisms inoculated into a wound become encased in a protein-fibrin coagulum after several hours and are thereby protected from antibiotics and dislodgment by normal irrigation fluids. The wounds must therefore be surgically débrided, pressure irrigated, or scrubbed. One should be alert for the presence of material driven beyond the surfaces of the wound because it may be difficult to identify. Many materials are not radiopaque, which makes detection of embedded material in soft tissue challenging, especially if the foreign material is driven beyond the wound surface and into surrounding soft tissue.

The skin and tissue edges may be cleansed with antiseptics, detergent soaps, or physiologic solutions such as saline. Occasionally, a solvent is necessary to remove material that is not soluble in saline. The surface of the skin, if contaminated by a "traumatic tattoo," should be cleansed of all foreign material by scrubbing and prying out embedded particles. Primary surface dermabrasion may also be considered; however, it can increase the depth of the cutaneous injury and result in more prominent, hypertrophic scarring. If the wound edge is ragged and devitalized, sharp débridement of the edge should be completed unless that tissue cannot be sacrificed without creating distortion.

All facial wounds are potentially contaminated, and deep, narrow wounds (such as puncture wounds) are more difficult to cleanse. Immunization with tetanus toxoid should be considered. In contaminated wounds, if the patient has not been immunized, simultaneous intramuscular injection of 250 IU of Hyper-Tet (tetanus immune globulin) and 0.5 mL of tetanus vaccine is recommended. Two additional boosters at monthly intervals complete the immunization.

Anesthetic Techniques

Usually, a general anesthetic is preferred for significant facial injuries. If general anesthesia is to be induced and the patient needs to undergo intermaxillary fixation, nasal, retroocclusal, or tracheal intubation is preferred. The intermaxillary fixation should be completed before any soft tissue repair work is performed. Placement of a tracheostomy may provide better exposure for the facial injuries by moving the tracheal access site away from the face. Local anesthesia by nerve block is always possible for a limited soft tissue injury repair.

Abrasions

An abrasion must be cleansed of any foreign material. Infection must be prevented to avoid conversion of a partial-thickness to a full-thickness injury. Any dirt, grease, carbon particles, or other pigments must be scrubbed out of the wound and a light lubricating dressing applied and changed frequently. The wound should not be permitted to dry out because desiccation may contribute to tissue loss. In partial-thickness or full-thickness dermal injuries, an allograft may be applied to prevent desiccation of the wound surface.

Contused and Avulsed Wounds

Contusions usually result in extensive edema, ecchymosis, and hematoma within the tissue. This can produce tissue scarring and rigidity and permanent tissue thickness, pigmentation, or swelling. The only treatment that can be rendered is gentle handling to prevent additional tissue damage. The tissue of a partially avulsed flap should not be folded, twisted, or placed under tension by tape or sutures. Marginally viable tissue often survives when managed gently.

Hematomas

Hematomas can be either localized or diffuse. Most hematomas are diffuse and gradually become resorbed. Moist, warm heat is used after the third day to promote resorption. On occasion, a facial hematoma is localized and can be evacuated. The patient should be taken to the operating room, a small incision placed in a camouflaged area, and a suction tip advanced into the hematoma cavity. The area can be irrigated free of all clot. A suction drain may be used postoperatively.

Patients with extensive ecchymosis or hematoma must avoid sun exposure to prevent hyperpigmentation. Hydroquinone ointments may decrease the tendency for hyperpigmentation. Thick, woody, chronically edematous soft tissue (the result of fibrosis after contusion) partially responds to the application of pressure or compression silicone sheets and to massage with a bland lubricating ointment.

Lacerated Wounds

The edges of a laceration must be excised. Removal of the crushed tissue margin during repair of lacerations results in a much better appearance of the scar and converts an avulsed, contused, and irregularly torn surface to a straight incision (Fig. 5).

The wound should be repaired in layers, commencing with repair of any ducts or nerves and proceeding to approximation of muscle, fat, dermis, and epidermis in layers. Absorbable sutures of the finest grade should be used, and their size should be just large enough that they have sufficient strength to hold the repaired structures. The skin sutures should be fine, and the dermal sutures should provide support and alignment for the cutaneous sutures, which are removed on days 3 to 5 (see Fig. 5B and C).

Animal and Human Bites

Animal and human bites are common injuries, and creation of a surgically clean wound is essential. Irrigation of the wound with copious amounts of saline and sharp débridement of devitalized tissue permit excision of the contaminated wound edges. Human and canine saliva contain necrotizing enzymes and potent microorganisms that can induce progressive soft tissue infection and necrosis. Prophylactic antibiotics should be administered because surgical excision of the wound surface is seldom complete. *Pasteurella multocida* infection from cat bites should be treated with penicillin. Human bites must be treated with gram-positive, gram-negative, and aerobically/anaerobically effective antibiotics.

Injury of the Facial Nerve

Branches of the facial nerve that are transected should be repaired under microscopic magnification. In theory, the distal branching and communication achieved by the plexus of facial nerve fibers make regeneration common in the midportion of the face despite the absence of direct facial nerve suturing anterior to a line drawn vertically, parallel to the lateral canthus of the eyelid. In practice, however, any nerve deficit should prompt a thorough search of the wound for nerve ends, and any nerve branches (electrical stimulation of nerve ends to aid in detection may be performed in the first 48 hours) should be approximated with microsurgical techniques. Precise repair of the facial muscles permits some element of nerve regeneration by neurotization of muscle. Muscle repair should be undertaken for this reason, as well as the fact that activity and balance are better restored to the face when the muscle ends are approximated.

Injury of the Trigeminal Nerve

The sensory branches of the trigeminal nerve arborize extensively, and repair is generally impractical unless the transection has occurred adjacent to one of the major foramina, such as the infraorbital or mental foramen. Transection of the supraorbital nerve is surprisingly frequent with lacerations around the eyebrow, and repair should be considered. Repair of the sensory nerves is perhaps the best way to avoid painful neuroma formation.

Lacerations of the Parotid Gland

When duct injury is suspected, retrograde irrigation of the distal orifice of the parotid duct or the lacrimal canaliculus may allow detection of a subtle duct injury by evidence of escape of clear fluid into the wound. Such detection can be accomplished by dilating the duct orifice, inserting a small plastic catheter, and irrigating with saline.

Lacerations of the parotid gland and duct should be repaired at the time of initial wound closure. A temporary parotid fistula to the skin surface is common, and draining the subcutaneous aspects of the wound minimizes the fistula. The parotid duct is located parallel to a line drawn from the tragus of the ear to the midportion of the upper lip; it traverses the middle third of this line (see Fig. 4). The proximity of the buccal branch of the facial nerve to the parotid duct produces simultaneous injuries in many cases. The orifice of Stensen's duct can be dilated with a small lacrimal dilator and irrigated to detect the presence of fluid within the wound. A silicone tube should be placed within the duct as a stent, brought out through Stensen's orifice, and securely anchored within the mouth. Repair is accomplished with fine sutures over the stent. Duct ligation produces considerable swelling of the gland and eventual atrophy; however, chronic infection requiring parotidectomy has been described after the use of this technique.

Lacerations of the submandibular gland or duct can be treated by duct ligation or removal of the gland.

Eyelid Lacerations

Eyelid lacerations should be repaired with fine absorbable suture material internally, with the conjunctiva, tarsus, and orbicularis repaired separately.

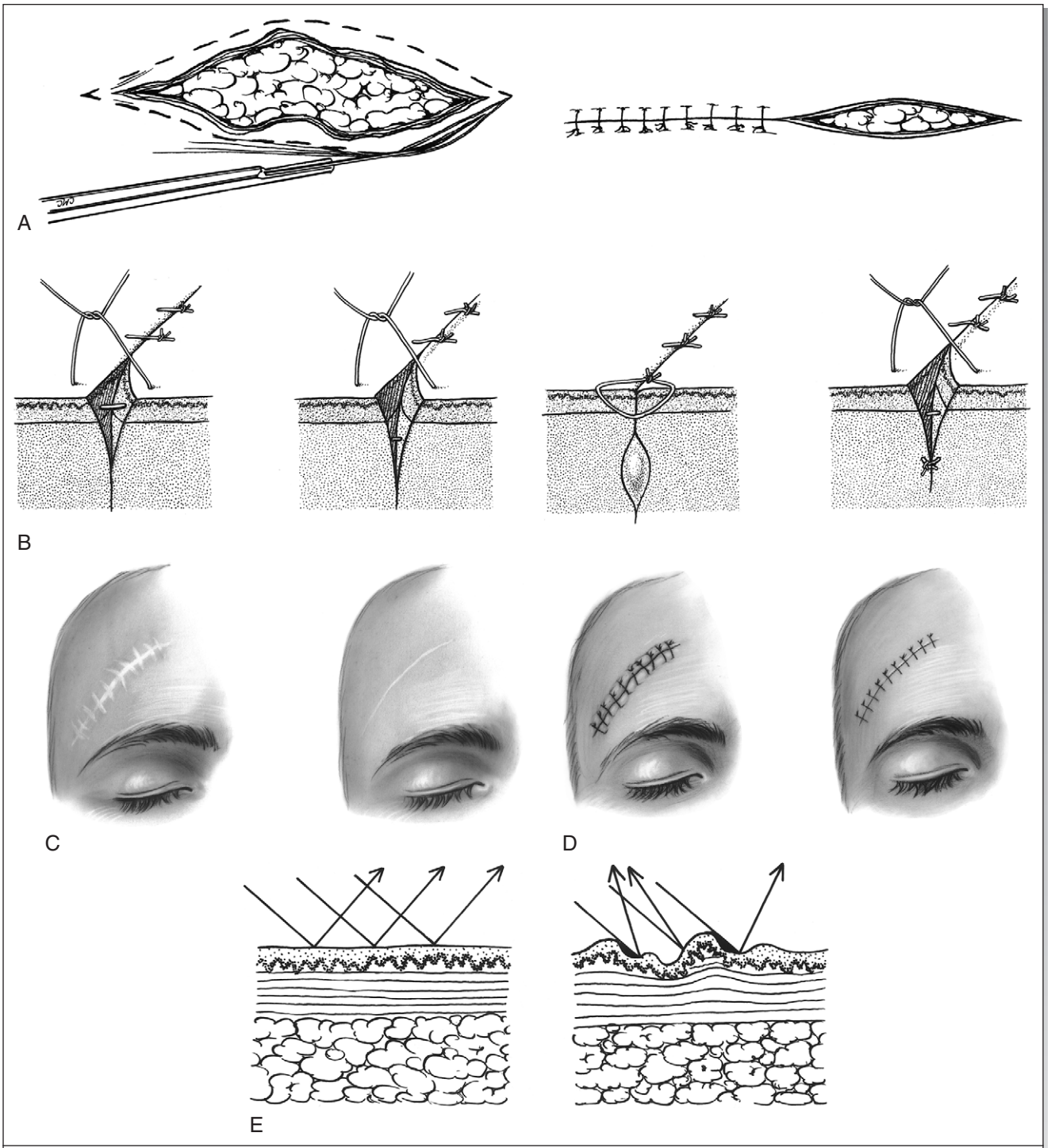

Figure 5 • A, Resection of 1 or 2 mm of skin is often all that is required to convert a posttraumatic wound into a surgically excised wound. **B,** Proper placement of sutures predictably creates a flat scar. More tissue must be incorporated by the suture on the deeper aspect of the bite. Layered closure is important. **C,** Early removal of sutures prevents suture marks from adding to the scarring. A scar is pictured with and without suture marks. **D,** Excess tension creates an edematous wound edge that is ischemic and has more scarring. Irregularly placed sutures (left) produce an uneven closure. Carefully spaced, precise repair with fine sutures creates an even repair. **E,** Irregular surfaces create irregular reflection of light, which exaggerates scarring. (© Johns Hopkins University.)

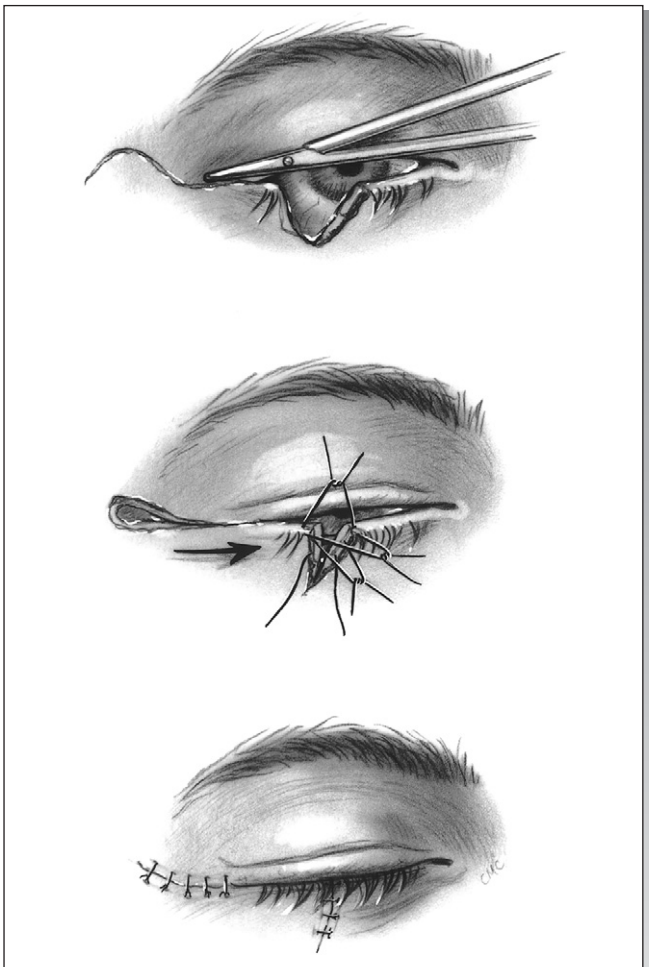

Figure 6 • Eyelid repair should be performed in layers with precise alignment of the cilia, "gray line," and internal lid margin. A lower eyelid defect has been repaired by canthotomy and advancement of tissue lateral to the canthal ligament into the lower lid. (© Johns Hopkins University.)

The knots should be tied in the wound away from the globe or conjunctival surface. In the case of defect, the lid is mobilized by a lateral and superiorly directed incision and canthotomy of the inferior limb of the lateral canthus, permitting medial mobilization of the remaining lower lid (Fig. 6). The skin sutures consist of three sutures of fine silk. They are located superiorly at the gray line, internal lid margin, and cilia and are tied with the posterior suture limbs captured by the more anterior sutures to avoid irritation of the corneal surface.

Lacrimal System Laceration

Lacerations near the medial canthus may sever the lacrimal canaliculi or damage portions of the lacrimal sac. If a lacrimal canaliculus or sac is severed, the ends are identified under microscopic magnification and reunited over Silastic tubing. The upper and lower lacrimal system should be intubated with soft Silastic tubing and the ends of the tubing tied in the nose. One cannot depend on an intact upper canaliculus to provide adequate drainage for the entire lacrimal system.

Lacerations of the Nose

Lacerations of the distal portion of the nose are common and may involve the skin, cartilage, and mucous membrane. To avoid distortion of contour, all structures should be repaired and débridement should be minimal. Soft, nonadherent gauze or Silastic splints may be helpful in positioning or supporting the soft tissue structures. The large glands in the skin of the distal half of the nose are prominent and can contribute to irregular scar formation.

Lacerations of the Lips

Lacerations of the lips may involve skin, the orbicularis oris, or the facial muscle system and mucosa. All structures must be precisely aligned and repaired, especially the pars orbicularis (thick band of orbicularis muscle) underneath the vermilion of the lip. The vermilion of the lip has two edges, and both should be aligned precisely by sutures placed at the anterior and posterior margins (Fig. 7). The white roll should be identified and carefully aligned before injection of vasconstricting solutions. Brilliant green skin marking solution on a needle can be used to "tattoo" the vermilion border before injection of local anesthesia. Accurate approximation

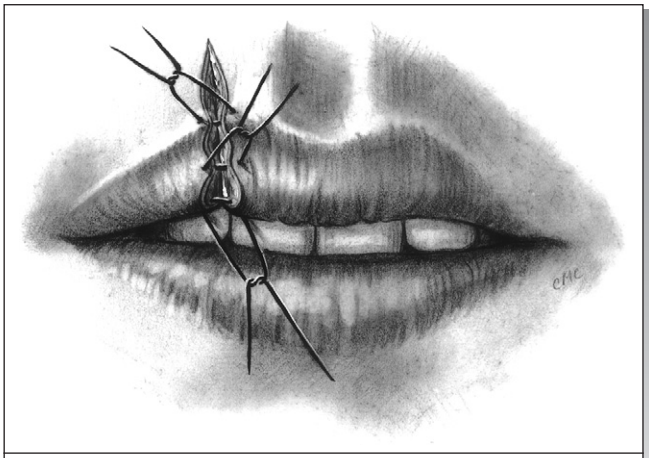

Figure 7 • Lip repair must first align the various bundles of the orbicularis and then proceed with approximation of the lip vermilion edges (anterior and posterior) and white roll, each with individual sutures. (© Johns Hopkins University.)

of the orbicularis muscle sets up alignment of the vermilion. If portions of the lip vermilion are débrided, a "step" deformity is produced because of the varying thickness of the vermilion across the surfaces of the lip. Débridement should be deferred in the area of Cupid's bow because it can produce an uncorrectable deformity.

Injuries of the Eyebrow

Injuries of the eyebrow should be treated without débridement because the eyebrow is of varying thickness along its length. Resection creates a "step" deformity that is solvable only by hair unit transplantation. Secondary (delayed) scar treatment can be performed in areas of hair loss by implanting hair unit grafts transferred from the posterior scalp. Associated supraorbital nerve transection should be identified and repaired.

Injuries of the Auricle

The ear is frequently involved by lacerations and contusions and is prone to hematoma formation. Lacerations of the auricle are usually associated with full-thickness "through and through" lacerations of the cartilage. Even subtotal ear avulsions generally survive when attached by a small pedicle. The cartilage should be minimally débrided, sutured in place, and the skin closed with carefully placed sutures. If lacerated, the ear canal should be repaired and stented. Avulsed tissue may be replaced as a graft after freshening the wound edges by excision of approximately 1 mm of wound margin. The part should be protected by placing it in sterile saline-soaked gauze in a container placed in another container with ice. Replantation of severed parts has also been accomplished through the use of leeches. Definitive reconstruction can be considered if the initial result is unsatisfactory.

Replantation of Avulsed Facial Parts

Replantation of ears, lips, parts of the nose, scalp, and face have been described and should be considered. Successful treatment requires considerable microsurgical expertise but can make a complex multistage reconstruction unnecessary, the results of which would probably be inferior to replantation. In some cases, it may be difficult to establish venous anastomoses, and one could consider leech treatment.

Wounds That Involve Missing Tissues

Generally, facial wounds that cannot be closed because of avulsion or tissue loss should have a skin-to-mucosa or temporary allograft cover placed under proper tension. The existing tissue should be advanced into proper position to prevent contraction. Full-thickness loss of the lips or nose may be repaired secondarily by flaps or grafts. In general, the primary use of composite grafts or flaps may result in loss of the grafted or transferred tissue because of the associated vascular injury in the area.

Pearls and Pitfalls

- Accurate primary treatment can prevent the need for complex secondary reconstruction.
- Meticulous layered repair of the injury with identification of severed glands, ducts, nerves, and muscles provides the basis for better opportunities for secondary reconstruction.
- Scar revision of malaligned structures is efficacious; however, scar revision after accurate primary repair often leads to minimal improvement.
- Dermabrasion may smooth superficial skin irregularities.

SUGGESTED READINGS

Clark N, Bierly B, Manson P: High energy ballistic and avulsive facial injuries: Classification, patterns and an algorithm for primary construction. Plast Reconstr Surg 98:583-601, 1996.

Hussain K, Wijejunge D, Grubnic S, Jackson I: A comprehensive analysis of craniofacial trauma. J Trauma 36:34, 1994.

Lee RH, Gamble WB, Mayer M, Manson PN: Pattern of facial lacerations from blunt trauma. Plast Reconstr Surg 99:1544-1554, 1997.

Lee R, Gamble WB, Robertson B, Manson P: The McFontzl classification system for soft tissue injuries of the face. Plast Reconstr Surg 103:1150, 1999.

Manson P: The importance of serial débridement and "second look" procedures in high energy ballistic and avulsive facial injuries. Op Tech Plast Reconstr Surg 5:236-246, 1998.

Manson P: Reconstruction of traumatic cutaneous facial defects. Op Tech Plast Reconstr Surg 5:368-378, 1998.

Zide BM, Swift R: How to block and tackle the face. Plast Reconstr Surg 101:840-851, 1998.

Fractures of the Frontal Bone

JAMES P. BRADLEY

Seventy percent of frontal sinus injuries result from motor vehicle accidents, 20% from assaults, and 10% are caused by falls or industrial and sporting accidents. Frontal sinus fractures are less common than other facial fractures, representing only 5% to 15% of all maxillofacial fractures. Two to three times more force is required to cause a frontal sinus injury. Reasons for operative intervention include correction of forehead contour and prevention of infectious complications. Untreated frontal sinus fractures may result in meningitis, brain abscess, and even death. Complications may appear years after injury. Obstruction of sinus drainage or trapped mucosa in a fracture line may gradually lead to mucocele formation. Subsequent bacterial colonization (mucopyocele) may be followed by spread of infection to the brain or orbit. Alternatively, chronic infection may result from incomplete frontal sinus obstruction. A potentially lethal course after frontal sinus injury is avoided by proper management in a timely fashion.

Embryology and Anatomy

Like other sinuses, the frontal sinus begins as an outgrowth of the nasal chamber. Growth of the frontal sinus is unique because it occurs mostly after birth. As an evagination of the middle meatus, it begins to invade the frontal bone between 1 and 3 years of age but becomes radiographically recognizable only by 7 years of age. Growth of the frontal sinus may continue into early adulthood (until 20 years of age). The small or nonexistent frontal sinuses in children make the frontal bone less likely to be involved in a fracture. Accessory sinuses may exist if more than one ethmoid sinus invades the frontal bone.

The anatomic boundaries of the frontal sinus vary, particularly in the extent of the superior and lateral walls. The thick anterior table provides contour for the glabella, brow, and lower part of the forehead. The thin posterior table separates the sinus air space from the frontal lobes of the anterior cranial fossa. The floor of the frontal sinus overlies the ethmoid air cells medially and the orbits laterally. The nasofrontal duct originates from the posteromedial sinus floor.

The physiologic function of the paranasal sinuses is not completely known. However, the frontal sinuses provide protection to the brain from injury because the air-filled compressible cavities may absorb impact. A functioning frontal sinus contains respiratory epithelium that secretes mucus and drains into the nasofrontal duct. No functional deficit has been observed with either surgical obliteration or developmental agenesis of the frontal sinus.

Diagnosis

The initial evaluation should follow standard trauma management and the advanced trauma life support (ATLS) protocol as described in the chapter "Acute Management of Head and Neck Trauma." A history of the events surrounding the accident should be elicited, as well as any past history of nasal or sinus disease. Awake patients should be assessed for visual difficulty, supraorbital anesthesia, pain, and change in sense of smell. In unconscious patients, signs of injury to be noted include soft tissue forehead injuries (lacerations, contusions, hematoma, crepitus), palpable bony depression of the brow, and cerebrospinal fluid (CSF) rhinorrhea. However, visible brow depression may not be appreciated in the first 48 hours because of overlying soft tissue swelling.

Diagnostic Studies

Plain radiographs are used more for a screening tool and follow-up than for definitive diagnosis. Caldwell's view (posteroanterior) may be the optimal projection. It demonstrates the septa of the

frontal sinus and margins of the dura. Other helpful views include the submental vertex and lateral projections, which allow for visualization of the anterior and posterior tables. Waters' view (30 degrees occipitomental) allows visualization of the periphery of the frontal sinus. With computed tomography (CT) scans, 1-mm sections through the forehead and orbits should be obtained. Even with the improved imaging of modern CT scans, however, the status of the nasofrontal duct is only indirectly determined. CT evidence suggesting an injury to nasofrontal duct drainage includes the following: fracture of the frontal sinus floor, fractures of the nasoethmoid complex, and inferiorly located, depressed fractures of the posterior wall. Isolated fractures of the anterior table and transverse linear fractures through the posterior and anterior walls (above the floor) are not associated with fractures of the nasofrontal duct.

Associated Injuries

Traumatic injury to the frontal sinus may be associated with significant intracranial, dural, or orbital injuries. Therefore, ruling out these injuries in patients with frontal sinus fractures is critical, particularly before attempting operative repair. In addition, appropriate consultation with a neurosurgeon or ophthalmologist should be considered.

CSF leakage in conjunction with frontal sinus fracture denotes posterior table displacement and dural disruption. Colorless fluid escaping through an open frontal bone fracture or rhinorrhea should alert the clinician to a CSF fistula. However, there may not be any outward sign of CSF leakage in the case of nasofrontal duct obstruction and in the absence of a forehead laceration. CSF may be differentiated from normal secretions by its low protein level (<0.5 g/L), low potassium level (2.5 to 3.5 nmol/L), and a glucose concentration greater than 30 mg/dL. By contrast, normal nasal secretions and serum have higher protein and potassium levels, and the nasal secretions have a glucose level less than 14 mg/dL. CSF rhinorrhea may also be caused by fractures of the cribriform plate, fovea ethmoidalis, sphenoid sinus, and temporal bone (via the eustachian tube). If it is necessary to localize the site of CSF leakage, fluorescein- or indium-labeled radioactive tracers may be injected via lumbar puncture. Labeled pledgets in the middle meatus, cribriform plate, and posterior inferior meatus help to identify the region of CSF drainage.

Goals of Reconstruction

The status of the nasofrontal duct and the anterior and posterior walls directs management (Fig. 1). The preoperative plan may have to be modified during surgery, depending on the intraoperative findings. Unless a neurosurgical or ophthalmologic emergency exists, patients with an intracranial injury may be observed for at least 48 hours before correction of frontal sinus fractures.

Injury to the nasofrontal recess requires obliteration of the frontal sinus. As mentioned earlier, radiographic diagnosis may be difficult. Intraoperative assessment of nasofrontal duct status may be accomplished by testing for drainage into the nose after the instillation of methylene blue into the frontal sinus. Repair of the nasofrontal duct with placement of Silastic tubes yields poor results.

Anterior wall fractures that are nondisplaced (or displaced less than 2 mm) on CT scan may be treated by observation alone, whereas anterior wall fractures that are more displaced require operative intervention. With mild comminution of the anterior wall, open reduction plus plate fixation with preservation of the frontal sinus is possible. More severe comminution of the anterior wall requires obliteration of the frontal sinus.

Posterior wall fractures that are minimally displaced (less than a table's width of displacement) without demonstrable CSF leakage may be monitored. However, some surgeons propose operative exploration of all posterior wall fractures. Posterior wall fractures with minimal displacement but a CSF leak may be observed for 7 days. If resolution occurs, patients are managed only by close observation and antibiotic therapy. If a CSF leak persists, obliteration or cranialization of the frontal sinus should be performed. A patient with a posterior wall fracture displaced greater than a table's width and a CSF leak should also undergo obliteration or cranialization of the frontal sinus. Likewise, if the posterior wall is displaced and a CSF leak exists without evidence of bony comminution, obliteration or cranialization should be performed. However, a noncomminuted, displaced posterior wall fracture without evidence of CSF leakage may be observed. In cases in which observation is allowable, endoscopic exploration of the frontal sinus may serve as an adjunct to minimize the incidence of missed injuries.

Surgical Treatment

A coronal flap provides the best exposure to the frontonasorbital/ethmoid region (Fig. 2). In patients with male pattern baldness in whom the scar cannot be hidden in the hairline, a brow incision usually heals with an acceptable scar. If present, traumatic lacerations in the forehead may be sufficiently large for exposure ("open sky" approach). An osteoplastic flap may be used for frontal sinus exploration. A template is made from a plain x-ray film and a sinusotomy is performed through the anterior wall. For endoscopic exploration, a small incision may be made in the lower medial aspect of the brow. A bur

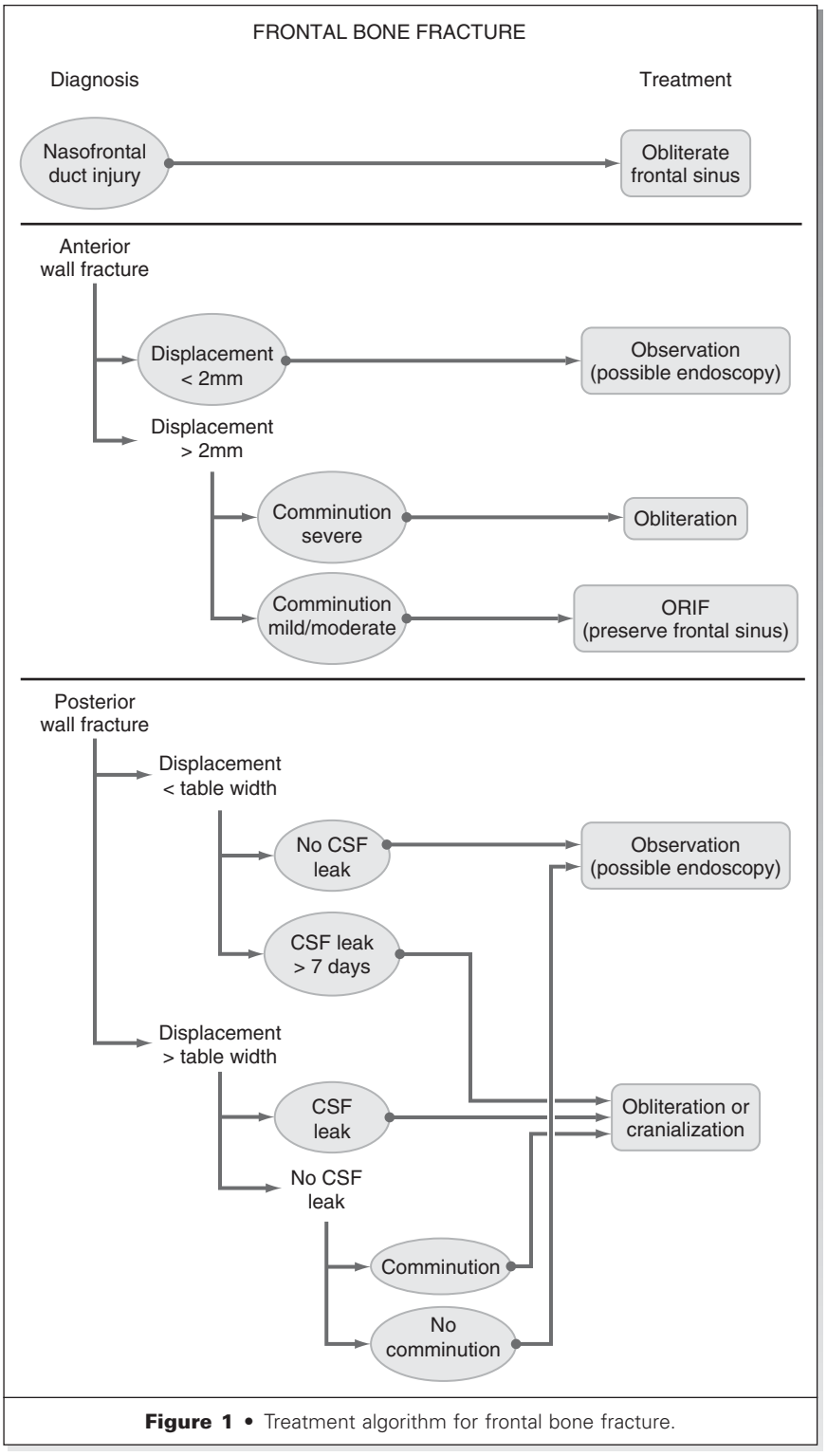

Figure 1 • Treatment algorithm for frontal bone fracture.

is used to enter the anterior floor of the frontal sinus.

Frontal sinus obliteration involves ablation of the frontal sinus mucosa, obliteration of the nasofrontal duct, and filling of the resultant cavity. Meticulous burring of the irregular bony cavities is necessary to remove all mucosal elements. Plugging of the nasofrontal duct, accessory ducts, and the residual frontal sinus cavity may be performed with cranial bone grafts. Other options include iliac bone, autogenous fat grafts, a pericranial flap, or prosthetic material, including bone substitutes (e.g., Norion,

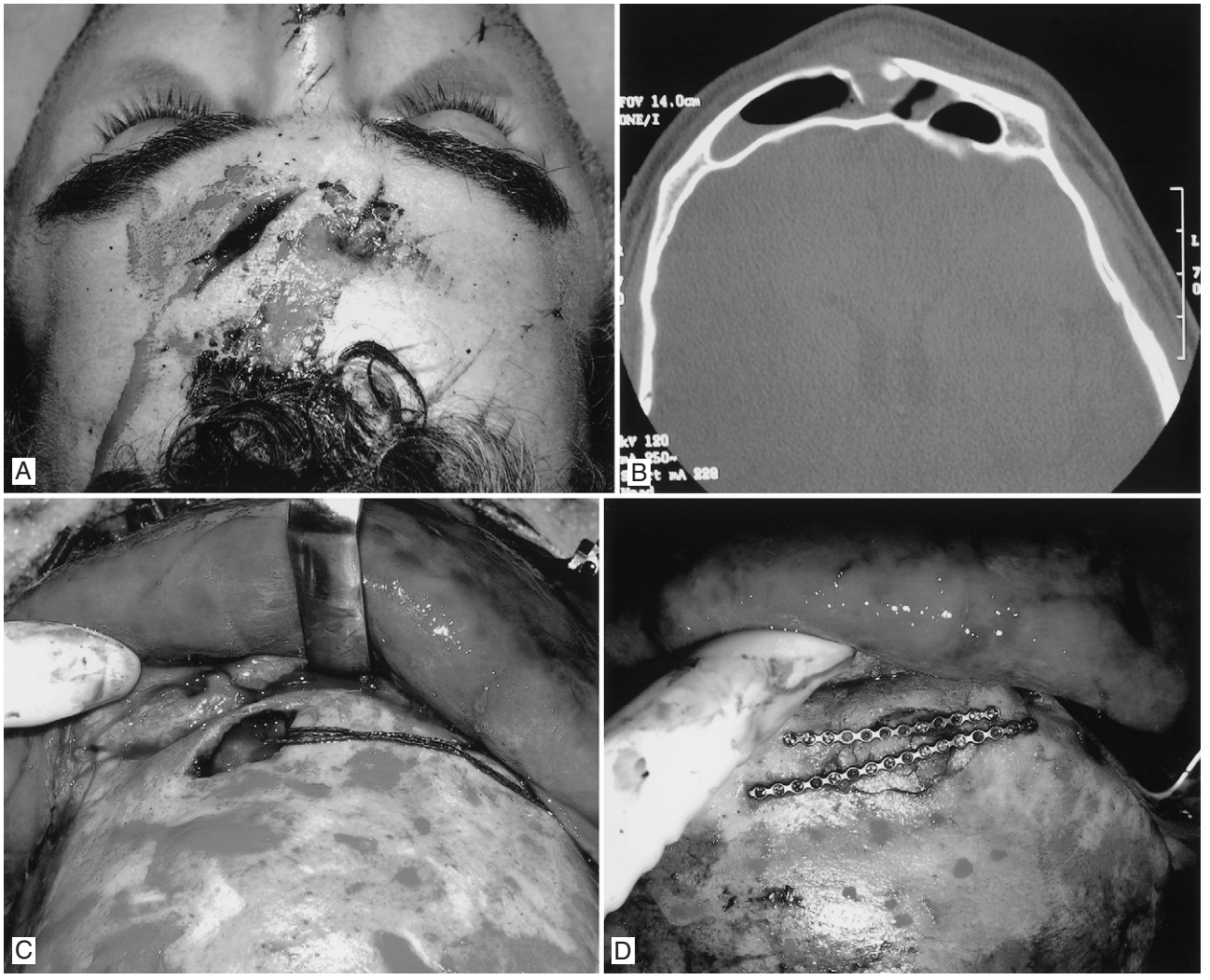

Figure 2 • Patient with an open frontal bone fracture after a motor vehicle accident. **A,** Bird's-eye view showing a forehead laceration and soft tissue contusion. **B,** Axial computed tomography scan demonstrating a displaced anterior wall fracture. **C,** Coronal incision and removal of minimally comminuted fracture fragments expose the frontal sinus. Instillation of methylene blue dye demonstrated that the nasofrontal duct was functional with drainage into the nose. **D,** Open reduction plus internal fixation of the frontal bone fracture was performed with titanium plates, and the frontal sinus was preserved. (Courtesy of J. Losee.)

bone source, hydroxyapatite). Cranialization of the frontal sinus also involves ablation of the mucosa and plugging of the nasofrontal duct. However, with cranialization, the comminuted posterior wall is removed so that the brain may expand into the frontal sinus defect.

Postoperative Care

Broad-spectrum antibiotics and head elevation for dependent drainage are advocated. Patients treated nonoperatively require follow-up evaluations to make certain that the sinuses remain clear. Caldwell's view of the frontal sinus obtained immediately after injury demonstrates a sinus opacified with blood. If a repeat Caldwell's view taken a week after the injury shows persistent radiographic opacification, a problem with drainage is likely and exploration should be performed.

Pearls and Pitfalls

- High-resolution CT scanning directs the need for surgical intervention.
- Surgeons must have high suspicion for nasofrontal duct injury.
- In the event of CSF leak, neurosurgical consultation is indicated.

- A mucocele is a serious complication and may occur years after the injury.

SUGGESTED READINGS

Olson EM, Wright DL, Hoffman HT, et al: Frontal sinus fractures: Evaluations of CT scans in 132 patients. AJNR Am J Neuroradiol 13:897, 1992.

Schultz RC: Frontal sinus and supraorbital fractures from vehicle injuries. Clin Plast Surg 2:93, 1975.

Stanley RB Jr: Management of severe frontobasilar skull fractures. Otolaryngol Clin North Am 24:139, 1991.

Xie C, Mehendale N, Barrett D, et al: Thirty year retrospective review of frontal sinus fractures: The Charity Hospital experience. J Craniomaxillofac Trauma 6:7, 2000.

Midfacial Fractures

PAUL N. MANSON

The midface, by virtue of its exposed position, is frequently injured by a wide variety of accidents. The component fractures range from the most frequently fractured projecting portion, the nose, to the zygoma and internal orbit and finally to the less frequently injured components, the maxilla and palate. This chapter reviews midfacial fractures and their treatment, with each fracture pattern discussed individually. It is important to note that many of the fractures are found in combination and that treatment of fracture combinations should be anticipated.

Etiopathogenesis

Auto accidents and altercations are responsible for the majority of midfacial fractures. Although air bags and seat belts have decreased the incidence of facial fractures in severe automobile accidents, many patients who otherwise would have not survived are now being seen with facial trauma requiring reconstruction. In addition, fractures can result from a fall associated with syncopal episodes. Underlying medical conditions should be investigated, including seizure disorders, arrhythmias, stroke, and drug use.

Patient Evaluation/Diagnostic Studies

Evaluation of maxillofacial injuries consists of a physical examination and computed tomography (CT) scans. Plain radiographs are seldom of value. The physical examination should begin with a history and progress to a directed and general facial examination. Most injuries should be detected by physical examination, even in a noncooperative patient. The examination begins by assessing facial symmetry and noting any skin or soft tissue injuries such as contusions or lacerations.

Orderly palpation of bony contours begins in the upper part of the face and progresses to the orbital rims, the nose, the nasoethmoidal area, and the zygomas, with note taken of their anteroposterior and vertical position, crepitus, or movement. Intraoral palpation plus visualization for evidence of hematoma is performed, followed by assessment of occlusal relationships. Inspection of the internal aspect of the nose is performed to detect septal swelling, fracture, or hematoma. Sensory nerve evaluation may show specific injury to portions of the terminal branches of the trigeminal nerve (cranial nerve V) that is often the result of fractures. Neuromotor evaluation consists of an orderly examination of the cranial nerves. A search for visual symptoms should emphasize visual acuity (cranial nerve II) (at the least, pupillary reactivity and light perception), extraocular motion (cranial nerves III, IV, and VI), and sensory evaluation (cranial nerve V). A search for occult lacerations should include the eyelids in addition to the mucosal surfaces, the ear canal, the intraoral and intranasal areas, the pharynx, the floor of the mouth, and the hard and soft palate. Occlusion should be evaluated and excursion of the lower jaw noted. Bleeding from the teeth or gums, absent or loose teeth, instability of the dental arches, and palatal or gingival lacerations should raise suspicion of fractures involving the jaws.

Eye Examination

Evaluation for double vision in all fields, vision in the peripheral fields, intraocular pressure, and fundus examination should be performed in all patients with blunt injuries around the orbit. Lid lacerations should suggest possible globe puncture. Hyphema and corneal abrasion suggest more extensive eye injury. Hyphema, retinal detachment, and globe rupture dictate deferral of facial bone surgery.

Radiographic Evaluation

Clinical findings identify areas of suspected injury and direct appropriate CT scans. Axial and coronal CT scans of the area with suspected injury and the surrounding bones should be obtained. Old photographs can be procured from the patient or family and are useful in demonstrating preinjury facial structure and asymmetry. A history of previous visual acuity problems implies a preinjury ocular deficit.

Dental and orthodontic records can contribute to knowledge of the preinjury occlusion.

A Panorex radiograph is useful for diagnosis of dental injuries; however, a cooperative patient is required.

Pneumocephalus and Cerebrospinal Fluid Rhinorrhea

Upper midfacial and nasoethmoidal orbital fractures are sometimes accompanied by pneumocephalus and cerebrospinal fluid (CSF) rhinorrhea. In the absence of displaced fractures, CSF fistulas are managed "expectantly" with a short period of prophylactic antibiotics. Most CSF fistulas in the absence of displaced fractures close spontaneously. CSF fistulas with displaced fractures often close after reduction of the fractures without a separate dural repair. The use of antibiotics for prolonged treatment of CSF fistulas merely selects organisms resistant to the antibiotic and thus should be avoided.

Treatment

Definitive treatment of facial injuries should be accomplished as early as possible. The operations and reduction maneuvers are easier at this stage, and therefore the amount of soft tissue stripping and injury is reduced. Patients who have multisystem injuries commonly suffer a sequence of complications that begins within days after the injury and includes systemic sepsis, pulmonary problems, and deterioration in liver and renal function. Early management of midfacial injuries allows definitive reduction to be completed *before* such issues prevent return to the operating room. Sequential partial management with the operation separated into logical steps (e.g., upper midfacial and then lower midfacial repair) can be an option to avoid prolonged surgery in the face of hemodynamic instability. Some patients benefit from periodic return to the operating room for irrigation and débridement, cleansing of the teeth and intraoral and nasal areas, and confirmation of the accuracy and rigidity of fixation and arch bars.

Nasal Fractures

The nose is the most commonly fractured bone in the adult midfacial skeleton. The bony nasal pyramid represents the upper portion of the nose. The cartilaginous part of the nose consists of the septum with its attached upper lateral cartilage and its separate lower lateral cartilage. The forces that produce nasal fractures are directed anteroposteriorly, laterally, or in both directions (Fig. 1). Each type of displacement must be analyzed for injury to the nasal bone, cartilage, and septum. The lateral and anteroposterior components of displacement of the nasal fracture may be separately typed by degree and can therefore be used to guide graded treatment approaches.

Patients with posterior displacement usually have widening of the nasal bridge and flattening of the nasal dorsum. Lacerations, edema, crepitus, pain, and ecchymosis are often present in the perinasal and orbital areas. Bleeding from the nose is universal. Both external (skin) and internal (mucosal) lacerations are frequent and should be repaired when possible. External lacerations may provide a site through which inspection or more extensive open reduction can be performed. Septal hematomas should be drained to prevent necrosis of the cartilage of the septum as a result of pressure as the hematoma resolves. Nasal obstruction may be the result of swelling or displacement of structures.

Plane I and Plane II Laterally Displaced Nasal Fractures

Treatment of nasal fractures is based on the type and degree of displacement. Three degrees of displacement are categorized for both laterally and posteriorly displaced nasal fractures. In laterally displaced nasal fractures, plane I fractures are unilateral and plane II fractures are bilateral, worse on one side. Plane I and II injuries are managed by elevation and outfracture of the unilateral nasal bone, along with completion of any contralateral fracture to achieve stability. The septum must also be mobilized to enhance centralization of the nasal pyramid. Without completion of the septal fracture, deviation of the nasal pyramid recurs. General anesthesia is used because it allows more effective completion of the fracture without patient discomfort. It also provides protection of the airway from aspiration if bleeding is produced by fracture reduction. Oxymetazoline (Afrin) on pledgets is used for intranasal vasoconstriction along with 0.5% lidocaine (Xylocaine) with 1:200,000 adrenaline as a field block and vasoconstrictor.

A septal laceration that requires repair or a septum that needs incision and drainage to evacuate a hematoma may require specific anesthesia. The nasal bones are outfractured with a reversed

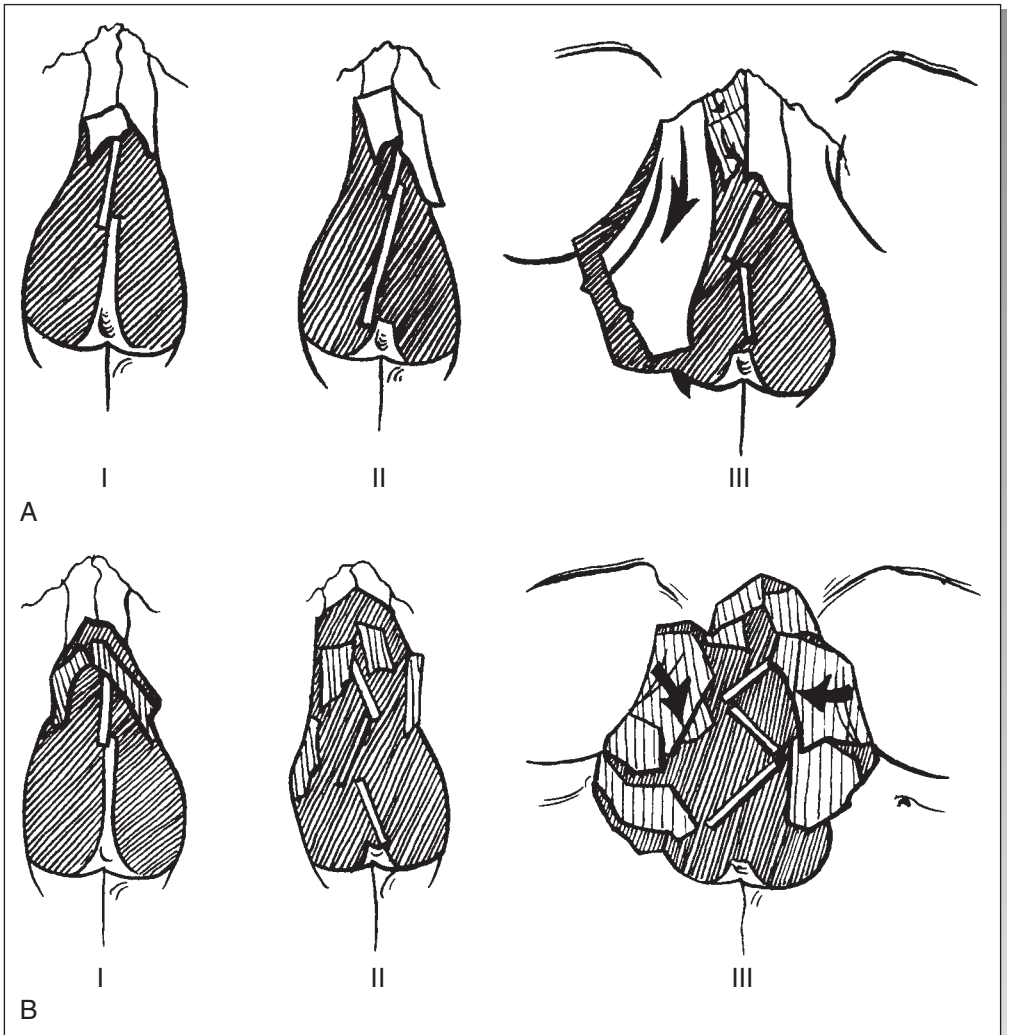

Figure 1 • Classification of fractures of the nose. **A,** Lateral displacement patterns. **B,** Anteroposterior displacement. Fractures with moderate anteroposterior displacement may require bone grafting and open reduction. Plane I and II fractures may occur in combination. Plane III fractures are nasoethmoidal orbital fractures.

No. 3 scalpel handle. Palpation is used to mold the nasal pyramid into its proper position. An osteotomy is rarely required to complete a "greenstick" (incomplete) fracture. Asch forceps are used to complete the fracture of the septum and reposition the septum into the midline. Hematomas can be drained through a vertical or L-shaped incision, depending on the location. A small latex drain may be placed in the hematoma cavity to allow egress of blood or fluid. Alternatively, "quilting" sutures of 4–0 plain gut can be laced through the septum to prevent the septal hematoma from reaccumulating. Petroleum-saturated packing may be used under the nasal fossa (the distal portion of the nasal bones) to support slumping distal nasal fracture fragments. A Doyle splint (Xomed Co, Jacksonville, Fla) is placed on each side of the septum and fixed by a 2–0 nylon suture penetrating the septum.

Plane III Laterally Displaced Nasal Fractures

Plane III nasal injuries (see Fig. 1, right) represent a dislocation of at least half the nose that extends into the piriform aperture and medial part of the orbit, along with dislocation of the heminasoethmoidal area. The fracture is generally "greenstick" and incomplete at the suture between the internal angular process of the frontal bone and the frontal process of the maxilla. The nondisplaced portion of the fracture need not be entered surgically, whereas the remainder of the fracture must be approached through the mouth (labiobuccal sulcus) and the medial aspect of the lower eyelid. A short medial incision just below the inferior portion of the tarsal plate usually leaves an inconspicuous scar. The skin incision is completed and dissection is performed to

separate the skin from the orbicularis for several millimeters inferiorly. The orbicularis is transected to reach the infraorbital rim and medial part of the maxilla. The inferomedial aspect of the orbit may be reduced and plated through this approach. Despite being the first stage of a nasoethmoidal orbital fracture, telecanthus is not observed because the fracture is displaced medially and inferiorly. The medial displacement is usually moderate and nearly closes the nasal airway on the injured side. This fracture is frequently mistaken for a nasal fracture and mistreated by closed reduction of the nasal pyramid alone. After open reduction of the medial part of the orbit and piriform aperture, the remainder of the nasal fracture (which is often unilateral) may be managed by closed nasal reduction.

Frontal Impact Nasal Fractures

Nasal fractures caused by frontal impact are characterized by three degrees of displacement (see Fig. 1, bottom panel). Plane I injuries involve posterior displacement of just the inferior ends of the nasal bones and the septum. Septal height is lost, and patients often have a residual dorsal hump. Reduction can elevate the ends of the nasal bones by placing packing in the nasal fossa. Reduction of the nasal septum merely reduces septal cartilage overlap. The bony-cartilaginous discrepancy is corrected in a second stage by either augmentation of the dorsum or reduction of the height disparity between the bone and cartilage.

Plane II frontal impact fractures involve the distal half of all of the nasal bones, depending on the severity of the force. The septum collapses in proportion. These injuries require treatment ranging from closed reduction and percutaneous splinting with bolsters to immediate open reduction with bone or cartilage grafting.

Plane III injuries are either unilateral complete or bilateral complete nasoethmoidal orbital fractures. They are discussed in the next section.

Extensive Combined Frontal and Lateral Fractures of the Nose

These fractures represent combinations of frontal and laterally displaced injuries and are managed by combining the recommendations discussed in the sections on frontal and lateral fractures.

Pearls and Pitfalls—Nasal Fractures

- Septal Doyle splints and "quilting" sutures are used to position the septum and prevent recurrence of hematoma.
- The most frequent mistake in the treatment of a nasal fracture by closed reduction is failure to complete the fracture thoroughly. The nasal bones should be able to be freely moved to either direction if mobilization has been accurate, and they should be easily "palpated" into position to remain where placed.
- Occasionally, an osteotomy is necessary to complete a greenstick nasal fracture that is slightly displaced but not unstable.
- All patients with nasal fractures should be warned that a secondary procedure is necessary for airway obstruction or correction of appearance in at least half the cases.

Nasoethmoidal Orbital Fractures

Nasoethmoidal orbital fractures are severe fractures of the central third of the upper midfacial skeleton (Fig. 2). The nasal bones, medial orbital rims, and the piriform apertures are involved. Various degrees of injury occur, ranging from inferior displacement with a greenstick fracture at the nasofrontal junction (the junction of the frontal process of the maxilla and the internal angular process of the frontal bone) to a comminuted, severely displaced fracture. Nasoethmoidal fractures are isolated in a third and extended in two thirds of cases to involve either the frontal bone or the zygoma or maxilla (or

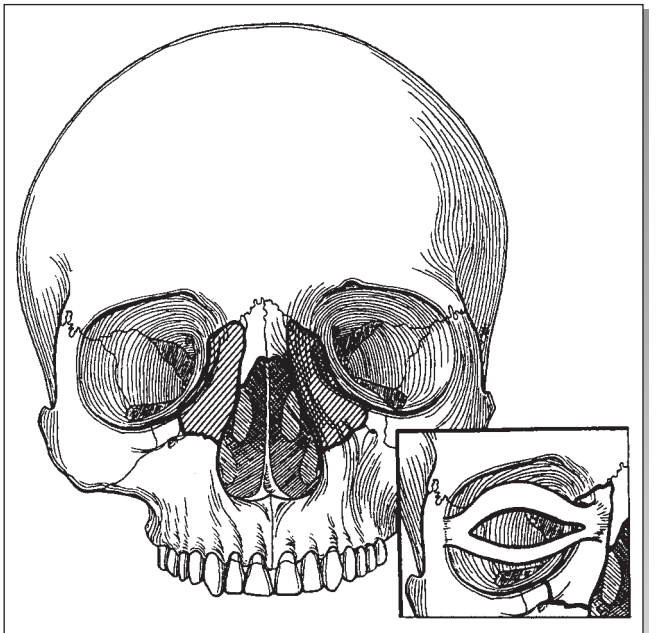

Figure 2 • The central fragment of the nasoethmoidal complex is *shaded*. The area of attachment of the anterior medial canthal ligament is demonstrated on the central fragment in the *inset*. (From Markowitz B, Manson P, Sargent L, et al: Management of the medial canthal tendon in nasoethmoidal fractures: The importance of the central fragment in treatment and classification. Plast Reconstr Surg 87:843-845 1991.)

both). A third are unilateral and two thirds are bilateral injuries. The central feature characterizing nasoethmoidal orbital fractures is displacement of the bony attachment of the medial canthal tendon. Mobility of this fragment may be assessed by direct digital palpation over the canthal tendon. "Bimanual examination" consisting of a clamp inside the nose with its tip underneath the canthal ligament and an index finger tip palpating externally over the canthal tendon insertion may be performed for confirmation. Digital manipulation allows the central fragment to move between the palpating index finger and the intranasal clamp (Fig. 3).

Clinical Examination

Lacerations of the forehead and nasal area are present in approximately a fourth of patients. A spectacle hematoma (a periorbital hematoma confined to the distribution of the orbital septum) and a displaced nasal fracture are often present. In more severe injuries, the nose is foreshortened, upturned, widened, and flattened. Nasopharyngeal hemorrhage can occur and may occasionally be severe. Palpation of the nose may reveal a lack of skeletal and cartilaginous support either proximally, distally, or both, depending on the degree and extension of the injury. In at least 20% of cases, a CSF leak is present as a result of a bone injury in the cribriform area of the anterior cranial fossa.

When the degree of comminution of the fracture is sufficient, the medial canthal tendon and its attached frontal process of the maxilla move laterally and produce telecanthus, or an increased distance between the medial commissures of the palpebral fissures. Telecanthus may be initially obscured by swelling. As time passes, the palpebral fissure shortens, the internal commissure of the eyelids becomes rounded, and enophthalmos occurs as a result of an enlarged orbital volume, with the globe sinking downward, medially, and posteriorly.

Radiographic study shows four fractures: the medial orbital wall, orbital floor, infraorbital rim, and nose and nasofrontal region. Opacification of the ethmoidal and maxillary sinuses is common. Some nasoethmoidal orbital fractures are difficult to document on CT scan, thus making "bimanual" examination essential for confirmation of a significant injury (see Fig. 3).

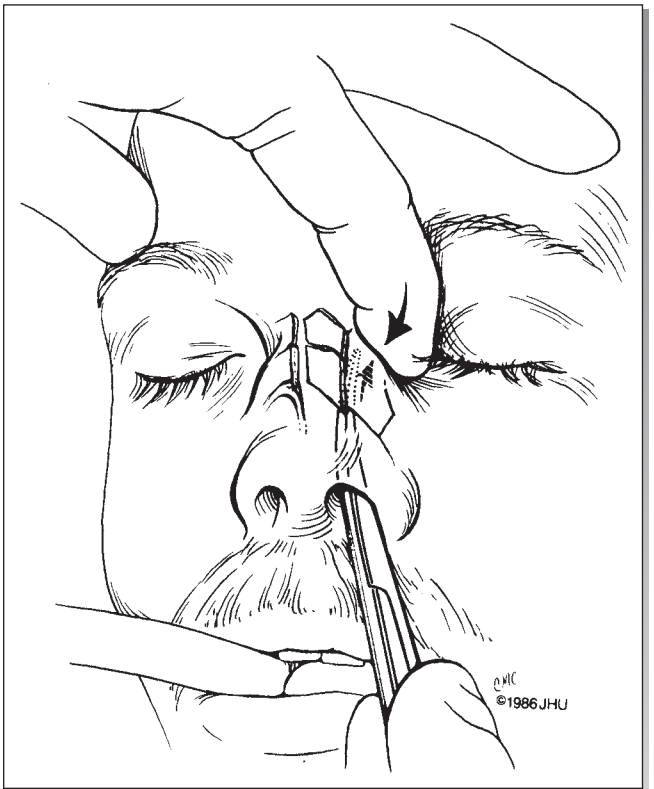

Figure 3 • Bimanual examination is the most effective way to confirm the presence of a mobile nasoethmoidal fracture. In significant fractures, the "central fragment" is mobile between an external palpating index finger over the canthal ligament and the tip of a clamp placed intranasally under the canthal insertion. Placement of the clamp underneath a nasal fracture fragment yields a false-positive test result. (From Paskert JP, Manson PN: The bimanual examination for assessing instability in nasoethmoidal orbital fractures. Plast Reconstr Surg 83:165, 1989.)

Patterns of Nasoethmoidal Orbital Injuries

Nasoethmoidal orbital injuries consist of four types: a localized central midface injury, a unilateral injury extended superiorly into the frontal bone or inferiorly into the orbital rim and zygoma, a high LeFort II or III fracture with a nasoethmoidal orbital component, or a panfacial fracture that may or may not involve the frontal bone.

Surgical Technique

Superior and inferior exposure is necessary for visualization of the peripheral buttresses of a nasoethmoidal orbital fracture. Occasionally, a laceration may substitute for one of the exposures. Lacerations should not be extended because the scar is generally worse than after elective incisions. Inferior approaches include the lower lid incision and labiobuccal sulcus incision. A lower lid incision, either a midtarsal, conjunctival, or subciliary incision, provides access to the orbital rim and the upper portion of the piriform aperture, whereas a gingival buccal sulcus incision provides access to the lower aspect of the piriform aperture and the LeFort I level. The superior approach is generally a coronal incision. Two local incisions may be occasionally chosen in localized fractures: a vertical midline incision over the root of the nose (not extended into the distal two thirds of the nose) or the horizontal limb

alone in the Converse open sky approach. Either of these local incisions may be used for exposure of the upper portion of a nasoethmoidal fracture. They are inadequate for exposure of the frontal sinus and the frontal bone.

The author divides nasoethmoidal fractures into four types by treatment options. Type I is an incomplete fracture, mostly unilateral but occasionally bilateral, that is displaced only inferiorly at the infraorbital rim and piriform margin. Inferior surgical approaches alone are necessary for reduction and fixation (Fig. 4*A* and *B*).

Type II nasoethmoidal orbital fractures section the entire nasoethmoidal area as a unit (Fig. 4*C* and *D*). These injuries are not true nasoethmoidal orbital fractures because telecanthus cannot occur. The central fragment is usually rotated and posteriorly displaced, and considerable canthal distortion occurs. They are treated by combined superior and inferior approaches, but neither type I nor type II requires canthal stripping because the canthus is not unstable and remains attached to the large bone fragment.

Type III nasoethmoidal orbital fractures (Fig. 5) are comminuted nasoethmoidal fractures; however, the fractures do not involve the bony insertion of the medial canthal tendon. The central fragment may be sizable and can be reduced to the canthal ligament–bearing fragment of the other side with a transnasal wire (Fig. 5). The remainder of the pieces

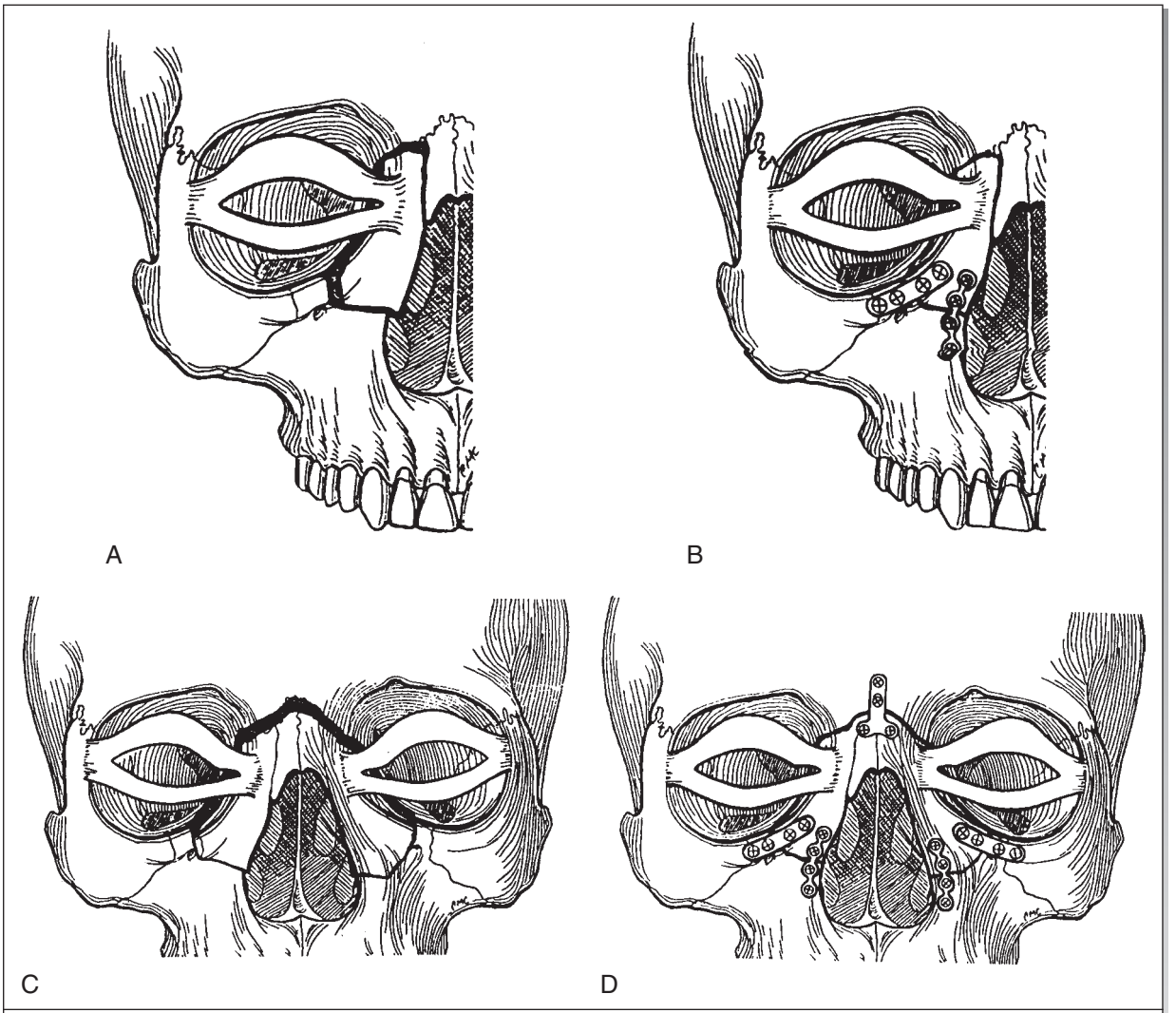

Figure 4 • Type I and II injuries. **A,** Type I injury. **B,** Treatment of type I injuries. Unilateral single-segment fractures undisplaced superiorly may be treated with inferior alone approaches and stabilization (piriform aperture and inferior orbital rim). **C,** Bilateral single-segment injuries (Type II) displaced at the articulation with the frontal bone. **D,** Displacement superiorly at the internal angular process of the frontal bone requires superior approaches in addition to inferior approaches for reduction and stabilization. Bilateral single-segment injuries require superior and inferior approaches for rigid fixation. (From Markowitz B, Manson P, Sargent L, et al: The management of the medial canthal tendon in nasoethmoid-orbital fractures: Repair of the central fragment in diagnosis, treatment and classification. Plast Reconstr Surg 87:843-853, 1991.)

of the nasoethmoidal orbital skeleton are assembled with interfragment wiring or microplates and then united by junctional plate and screw fixation to the frontal bone, the infraorbital rim, and the LeFort I level of the maxilla.

Type IV nasoethmoidal orbital fractures (Fig. 6) are characterized by either avulsion of the canthal tendon (uncommon) or extension of the fractures underneath the canthal tendon insertion. Because the fracture fragments are small, the canthus tendon must be detached for reduction. Therefore, canthal tendon reattachment is required. Both the left and right medial canthal tendons should be reattached with a separate set of transnasal wires for each canthus (Fig. 7). In general, bony reduction of the intercanthal distance should be 5 to 7 mm less per side than the soft tissue distance. Bone grafts are frequently required for restoration of the nasal dorsum. A cartilage graft may be needed for the columella to stabilize this area and prevent nasal shortening. The nasal septum is frequently perforated and should be repaired. If dislocated, the septum may be reattached to the anterior nasal spine with a wire.

After fracture repair, the soft tissue is held to the nasal bones with soft Xeroform-wrapped felt bolsters transfixed by two transnasal wires. Careful daily observation of the skin pressure produced by the bolsters is required to prevent skin necrosis. Meticulous daily cleansing underneath the bolsters is also necessary.

The lacrimal system is always injured in nasoethmoidal fractures, but seldom by direct transsection.

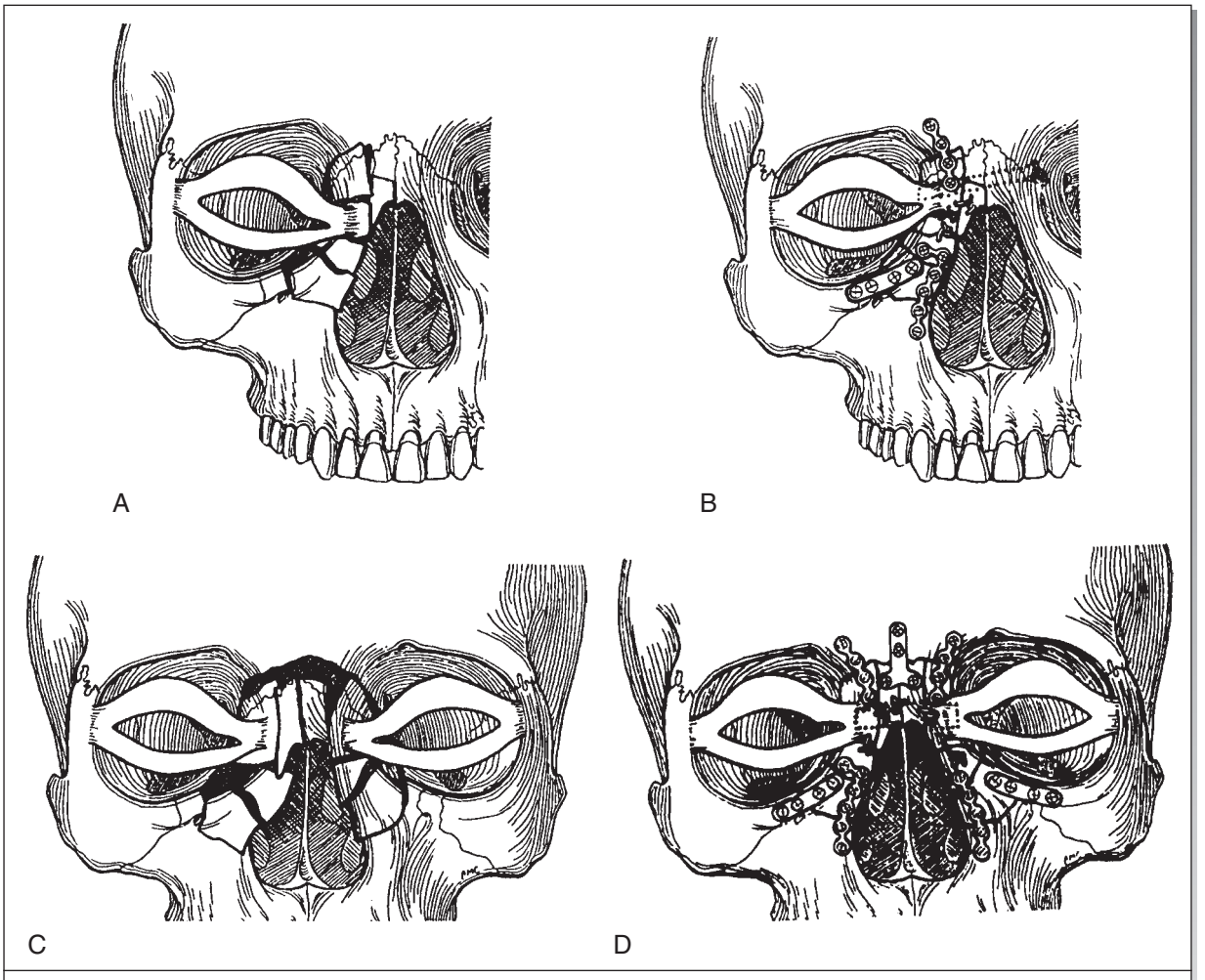

Figure 5 • Type III injury. **A** (unilateral) and **C** (bilateral), The comminuted fracture does not extend into the area of insertion of the canthus. **B** (unilateral) and **D** (bilateral), Treatment of type III injuries. Fractures that are comminuted external to the canthal insertion require (1) transnasal reduction of the medial canthal tendon–bearing bone fragments, (2) interfragment wiring to link all fragments, and (3) junctional rigid fixation. *Left*, Completed reduction, unilateral fracture. The transnasal reduction wires are *dotted* behind the canthal ligament. *Right*, Completed reduction, bilateral fracture. Bilateral repair requires transnasal passage of the transnasal reduction wire posterior and superior to each canthal ligament. (From Markowitz B, Manson P, Sargent L, et al: The management of the medial canthal tendon in nasoethmoid-orbital fractures: Repair of the central fragment in diagnosis, treatment, and classification. Plast Reconstr Surg 87:843-853, 1991.)

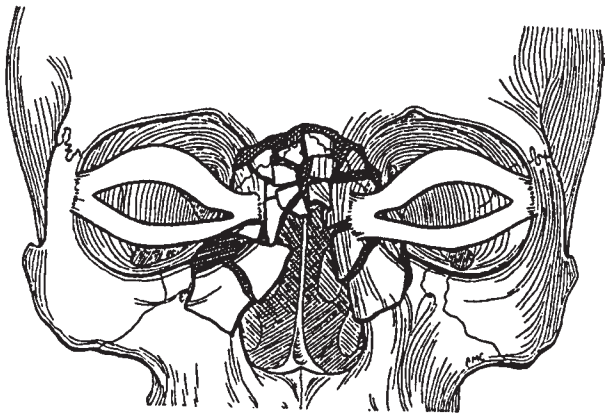

Figure 6 • Type IV injuries. Fractures extend into the canthal-bearing segment. Canthal avulsion may be present. (From Markowitz B, Manson P, Sargent L, et al: The management of the medial canthal tendon in nasoethmoid-orbital fractures: Repair of the central fragment in diagnosis, treatment and classification. Plas Reconstr Surg 87:843-853, 1991.)

When a direct laceration occurs, the lacrimal system must be repaired.

The internal orbit must be reconstructed by placing grafts between intact bone at the back of the orbit and the infraorbital rim. In the medial orbital region, the posterior ethmoidal foramen forms the posterior boundary of the fracture in more extensive injuries. Medial orbital grafts may be stacked against the displaced ethmoidal bones until normal contour is achieved.

Fractures of the Zygoma

Zygomatic fractures are the second most common midfacial fracture and may occur in isolation or as a component of a more extensive fracture such as a LeFort III fracture. The physical findings of a zygomatic fracture consist of a deformity of the eye and eyelids with recession of cheek prominence.

In some high-energy injuries, the zygoma can be laterally displaced with increased midface width on the affected side. In lower-energy injuries, the zygoma is displaced medially with narrowing of the midface. Zygoma fractures are almost always accompanied by periorbital and subconjunctival hematoma. Hypoesthesia is present in the peripheral branches of the infraorbital nerve, including those to the teeth, ipsilateral cheek, sidewall of the nose, and upper lip. The upper teeth may be hypoesthetic because the anterosuperior dental alveolar nerve is a branch of the infraorbital nerve.

Zygomatic complex fractures involve fractures of the orbital floor and lateral aspect of the orbit. If the bony displacement is sufficient, the globe drops posteriorly and enophthalmos is produced. The globe also sinks inferiorly such that the eye is inferiorly depressed. Because the lateral canthal ligament is attached to Whitnall's tubercle, a rounded eminence on the internal aspect of the frontal process of the zygoma 10 mm below the zygomaticofrontal suture, the lower lid will sink inferiorly if the zygoma is inferiorly displaced. Unilateral epistaxis is almost universal because the fractures communicate with the maxillary sinus. Because of the orbital floor fracture, double vision or diplopia may occur, usually as a result of contusion of the extraocular muscles. The increased size of the orbit produces enophthalmos and orbital dystopia. Hematomas may be observed around the cheek and the eye and in the upper buccal sulcus. If the posterior depression of the zygomatic body is sufficient or if the zygomatic arch is depressed medially, there may be impingement on the coronoid process of the mandible and restriction of mandibular motion. These abnormalities produce pain and difficulty in chewing and may prevent normal occlusion.

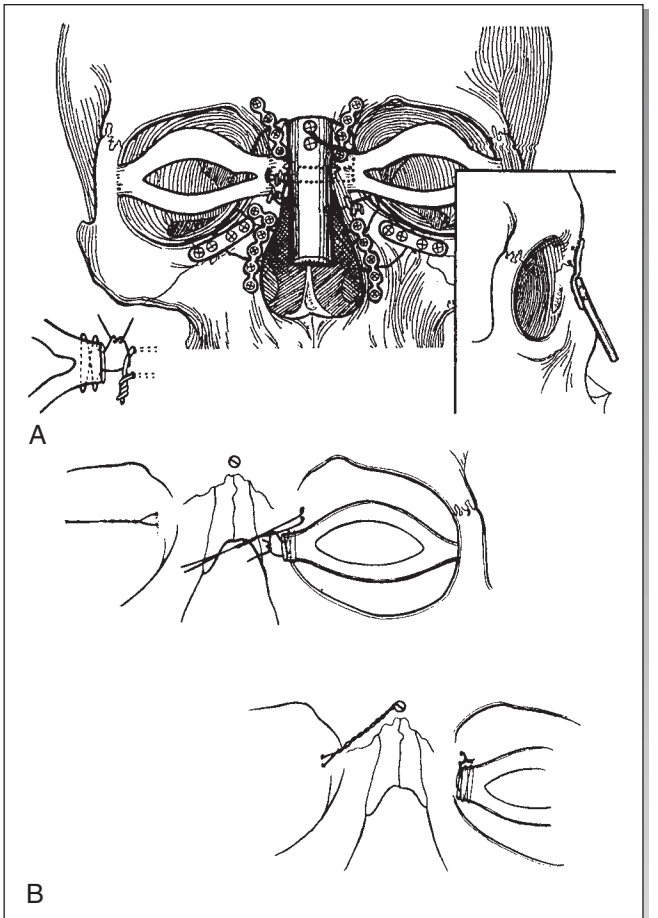

Figure 7 • Fracture management requires canthal detachment and reattachment by transnasal wiring to the contralateral tendon mechanism **(A)** or separately fixed screws or wires **(B).** (From Markowitz B, Manson P, Sargent L, et al: The management of the medial canthal tendon in nasoethmoid orbital fractures: Repair of the central fragment in diagnosis, treatment and classification. Plas Reconstr Surg 87:843-853, 1991.)

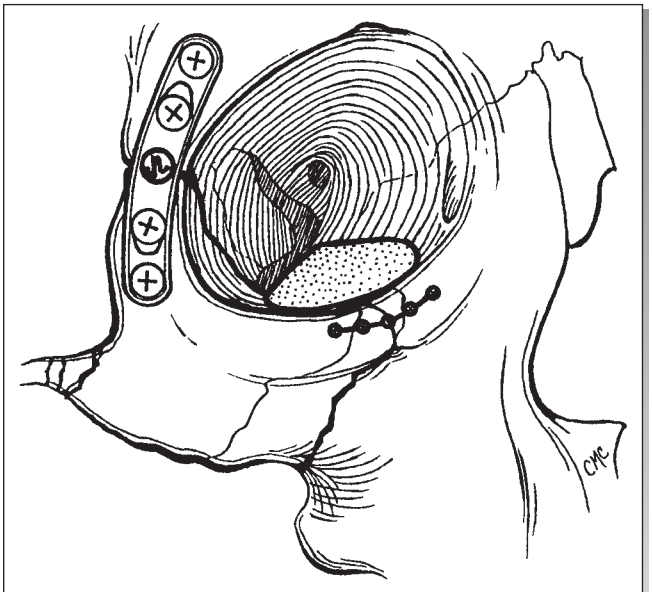

Figure 8 • Anterior approaches to the zygoma may involve a single plate at the zygomaticomaxillary buttress or exposure and plating of the inferior orbital rims, zygomaticomaxillary buttress, and zygomaticofrontal frontal suture. (© Johns Hopkins University.)

Incomplete Fractures

Greenstick fractures of the zygoma are incomplete at the zygomaticofrontal suture. They are displaced inferiorly and as such can be managed with inferior approaches alone. If the orbital floor is not comminuted, reduction of the zygoma reduces the floor.

Isolated Zygomatic Arch Fractures

Isolated fractures of the zygomatic arch can be caused by a lateral blow and most frequently result in a "W-shaped" deformity with depression of the lateral midcheek region just anterior to the ear and the glenoid fossa. Isolated fractures of the zygomatic arch are classically reduced in "closed" fashion through the Gillies temporal approach, but they may alternatively be approached either through the mouth or through a lateral brow incision. The reduction maneuver in the Gillies approach requires an incision in the temporal hair-bearing scalp that is deepened to expose the fibers of the temporalis muscle (the incision must extend deep to the deep temporal fascia). An elevator is passed directly underneath the depressed portion of the arch, and the surgeon lifts the depressed portion of the arch in a gentle, but steady fashion. The fractures usually snap back into place and do not require support. This maneuver may be used for isolated arch fractures or for the arch alone in complete zygomatic fractures that demonstrate medial displacement of the zygomatic arch.

Zygomatic Complex Fractures

For fractures that are displaced at the zygomaticofrontal suture with medial displacement of the arch, an "anterior approach" is preferred (Fig. 8). Exposure for reduction and fixation can be achieved at three places: an incision over the zygomaticofrontal suture (which is generally through the lateral limb of an upper lid blepharoplasty or a laceration or preexisting scar); a lower lid incision (which could be either a subciliary, midtarsal, or conjunctival incision); and a labiobuccal sulcus incision, which exposes the attachments of the zygoma to the maxilla (Fig. 8). An elevator may be placed within the maxillary sinus and used to lever the zygoma into position. Because one cannot view the three separate approaches simultaneously, positioning wires are usually placed (e.g., in the zygomaticofrontal suture and the infraorbital rim) and the reduction commenced with plate and screw fixation at the zygomaticomaxillary articulation. In fractures with medial displacement, which generally includes medial displacement of the arch, the arch can additionally be maneuvered through a Gillies approach in the manner of a closed reduction. More than 90% of zygomatic fractures may be managed with anterior incisions alone.

A comminuted zygoma fracture with lateral displacement of the arch and the zygoma or extreme posterior displacement of the zygomatic body requires complete visualization, including both anterior incisions and a coronal (scalp) incision, to expose the zygomaticofrontal suture and the zygomatic arch (obviating the need for blepharoplasty or brow incision; Fig. 9). Dissection is carried out via the coronal incision beneath the deep temporal fascia to expose the bone fragments of the zygomatic arch. After placement of the positioning wires, open reduction of the zygomatic arch and zygomaticomaxillary buttress is achieved. Positioning (temporary interfragment) wires are useful because three separate fracture sites cannot be simultaneously visualized. Wires allow some degree of rotation while the other areas are being reduced. The proper medial position of the zygomatic arch is determined by alignment in the lateral orbit of the orbital process of the zygoma with the greater wing of the sphenoid. Generally, 1.3-mm plates are used at the zygomaticofrontal suture and orbital rim, and 1.5- or 2.0-mm plates are used in the zygomaticomaxillary buttress and arch to resist muscular forces.

If the lateral canthus is detached in the reduction, it needs to be reattached with a separate wire to the inner aspect of the lateral orbital rim, just below the zygomaticofrontal suture, so that the proper three-dimensional draping of the canthus is reestablished. It is also important to resuspend the midfacial soft tissues to support the lower lid and prevent facial asymmetry. The periosteum can usually be suspended from the zygomaticofrontal plate and the infraorbital rim plate.

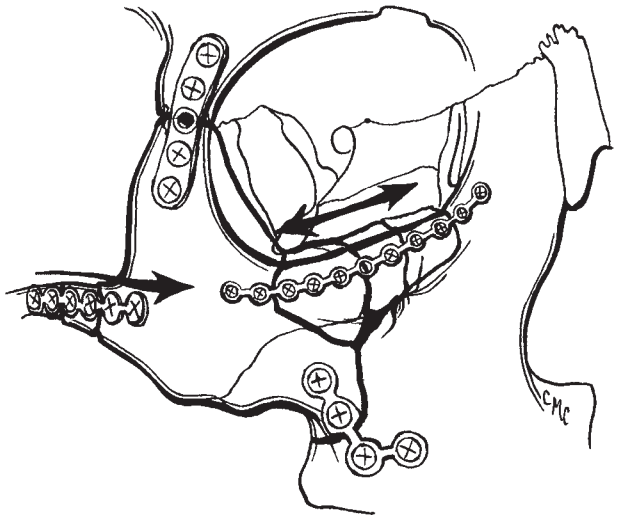

Figure 9 • The posterior approach to the zygoma involves the approaches illustrated in Figure 8, the anterior approaches, plus a plate along the zygomatic arch. The arch is properly positioned by observation of the alignment in the lateral wall of the orbit obtained at the time of arch reduction. (From Manson P, Clark N, Robertson B, et al: Subunit principles in midface fractures: The importance of sagittal buttresses, soft tissue reductions, and sequencing treatment of segmental fractures. Plast Reconstr Surg 103:1287, 1999.)

In upper LeFort fractures, one sees retrusion of the midfacial structures. As the swelling subsides, the midface usually elongates and is flattened. The face in comminuted upper-level LeFort fractures is rounded, wide, and short. CSF fistulas occur in 25% of upper LeFort II and III fractures.

Incomplete LeFort Fractures

It is important that the maxilla be fully mobilized and positioned in normal occlusion with the mandible before fixation. In incompletely mobilized LeFort fractures, the mandible may be distracted out of the glenoid fossa to meet an immobile, displaced maxillary segment. Proper condylar seating in the glenoid fossa is paramount. If it cannot be obtained, the incomplete LeFort segment should be disimpacted by mobilization, osteotomy, or traction elastics before establishing maxillomandibular fixation (MMF). Eric arch bars are applied to the teeth, and the jaws are placed in MMF. MMF reduces the LeFort I segment to the mandible and positions the LeFort I segment with regard to the cranial base if the mandible is intact. If the mandible is not intact, it should be anatomically reconstructed before initiating MMF. Similarly, if a sagittal fracture of the palate or an alveolar fracture is present, it should be reduced and fixated before establishment of MMF.

LeFort Maxillary Fractures

Maxillary fractures were classified by LeFort on the basis of "weak areas" of the maxilla. He defined three "great lines of weakness" (Fig. 10). A LeFort I fracture is a horizontal fracture between the maxillary alveolus and the upper craniofacial skeleton. In a LeFort II fracture, the central nasomaxillary segment is separated from the zygomatic and nasofrontal aspects of the facial skeleton. A LeFort III fracture is a "craniofacial" disjunction in which all the facial bones are separated from the cranium. One generally sees combinations of fractures in the same injury, such as the presence of a LeFort I and II fracture within a LeFort III fracture. LeFort fractures are often more severe on one side; thus, a higher LeFort level fracture will be seen on the most injured side (see Fig. 10).

LeFort fractures traverse many of the aforementioned fractures lines. As a result, they produce symptoms that are found with other facial fractures. Mobility of the maxillary alveolus and teeth along with malocclusion is the "sine qua non" of most LeFort fractures. The fractures may be incomplete, in which case movement may be absent. Malocclusion is, however, still present and confirms the presence of a fracture that is dislocated but not mobile. Malocclusion, therefore, is the hallmark of diagnosis and is present in all displaced LeFort fractures. Mobility of the maxilla occurs in fractures that are not incomplete or impacted.

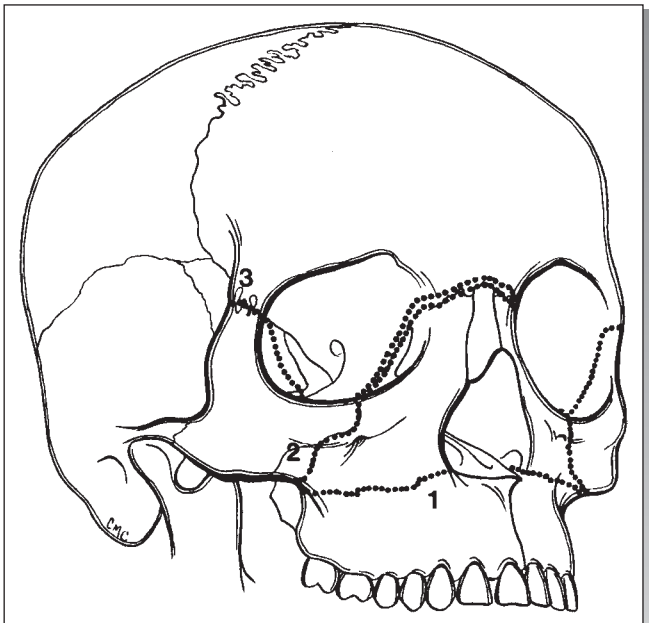

Figure 10 • A LeFort I fracture (1) is a transverse fracture separating the segment carrying the maxillary occlusion from the upper part of the maxilla. In a pyramidal or LeFort II fracture (2), a central fragment containing the occlusion and the nose is separated from the zygomatic aspects of the midface. A LeFort III fracture (3) separates the zygomas and nose from the skull base. Combinations of these fractures are frequent, and the fractures are usually worse on one side than the other. (© Johns Hopkins University.)

Exposure of the LeFort fracture line is achieved through bilateral labiobuccal sulcus incisions. Both the nasomaxillary and the zygomaticomaxillary buttresses are likewise exposed and fixated.

LeFort I Fractures

LeFort I fractures may be treated by plate and screw fixation over the nasomaxillary and zygomaticomaxillary buttresses bilaterally as described earlier (Fig. 11). Postoperatively, the patient may be released from MMF and observed. The arch bars are left in place so that wires or elastics can be reestablished if there is any change in occlusion. In general, 6 to 8 weeks is required for healing. Patients are observed for several weeks after apparent healing, with the arch bars left in place so that stability of fracture position and solid bony union are ensured.

More complicated maxillary fractures require reconstruction of the horizontal and vertical buttress system of the facial skeleton (Fig. 12). Conceptually, the upper midfacial region is related to the frontal bone, and the lower midfacial region below the LeFort I level is related to the mandible.

LeFort II Fractures

In LeFort II fractures, plate and screw fixation is used at the zygomaticomaxillary buttress as in LeFort I fractures. In addition, the infraorbital rim is exposed via a lower orbital incision, and the fracture here is also fixated. If the fracture traverses the lower nasal bones and cartilage, closed reduction of the nose is usually sufficient.

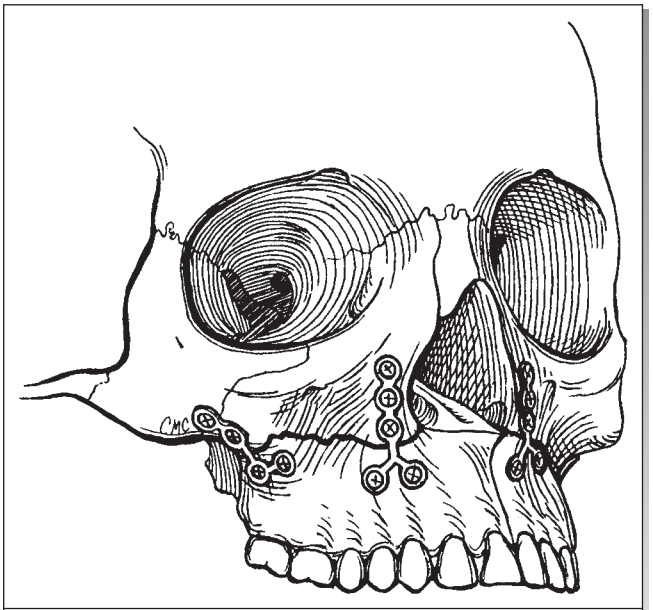

Figure 11 • Open reduction and fixation of a LeFort I fracture with plates across the four buttresses of the maxilla. (© Johns Hopkins University.)

In the case of a fracture that disconnects the nasofrontal junction from the frontal bone, a coronal or local incision over this area is required for reduction and fixation (Fig. 13).

LeFort III Fractures

For LeFort III fractures, a coronal incision exposes the nose at the internal angular process of the frontal bone, the zygoma at the zygomaticofrontal suture, the lateral and medial aspects of the orbit, and the zygomatic arch. Reduction at the lateral aspect of the orbits and reestablishment of facial projection by reconstruction of the zygomatic arches are important principles. The infraorbital rims and medial orbital wall are also often involved and can be examined through orbital incisions. Fractures are reduced and fixated (Fig. 14).

Edentulous LeFort Fractures

In edentulous LeFort fractures, one may need to extend the fixation to the alveolus to find sufficient bone for stable fixation. Bone grafting may be added to the anterior aspect of the maxilla over the maxillary sinus and at the piriform aperture for augmentation. Intermaxillary fixation should be accomplished temporarily with dentures or splints as an initial positioning technique for the lower midface region and alveolus, similar to a dentulous patient. The dentures may be secured to the alveolus with screws or wires. The denture fixation may be discontinued postoperatively. The upper and lower midface areas are stabilized in units and connected at the LeFort I level as the final step in the reduction of a significant fracture.

Sagittal Fractures of the Maxilla

Sagittal fractures of the maxilla may manifest as alveolar fractures or as maxillary arch instability (Fig. 15). Fractures that divide the palate in an anteroposterior plane should be treated by direct open reduction in the roof of the mouth and at the piriform aperture. This maneuver converts the fractured alveolus into a “one-piece” LeFort I segment, which can be managed in the fashion of a standard LeFort I fracture. Because the alveolar reductions are not as stable, the patient should remain in MMF for a 4- to 6-week period until stability and normal occlusion are ensured. The use of an acrylic palatal splint is helpful in cases in which the dental alveolus is subject to rotation (see Fig. 15).

Orbital Fractures

Fractures may occur in any of the four portions of the internal orbit. They may extend to involve small rim fractures as well (Fig. 16).

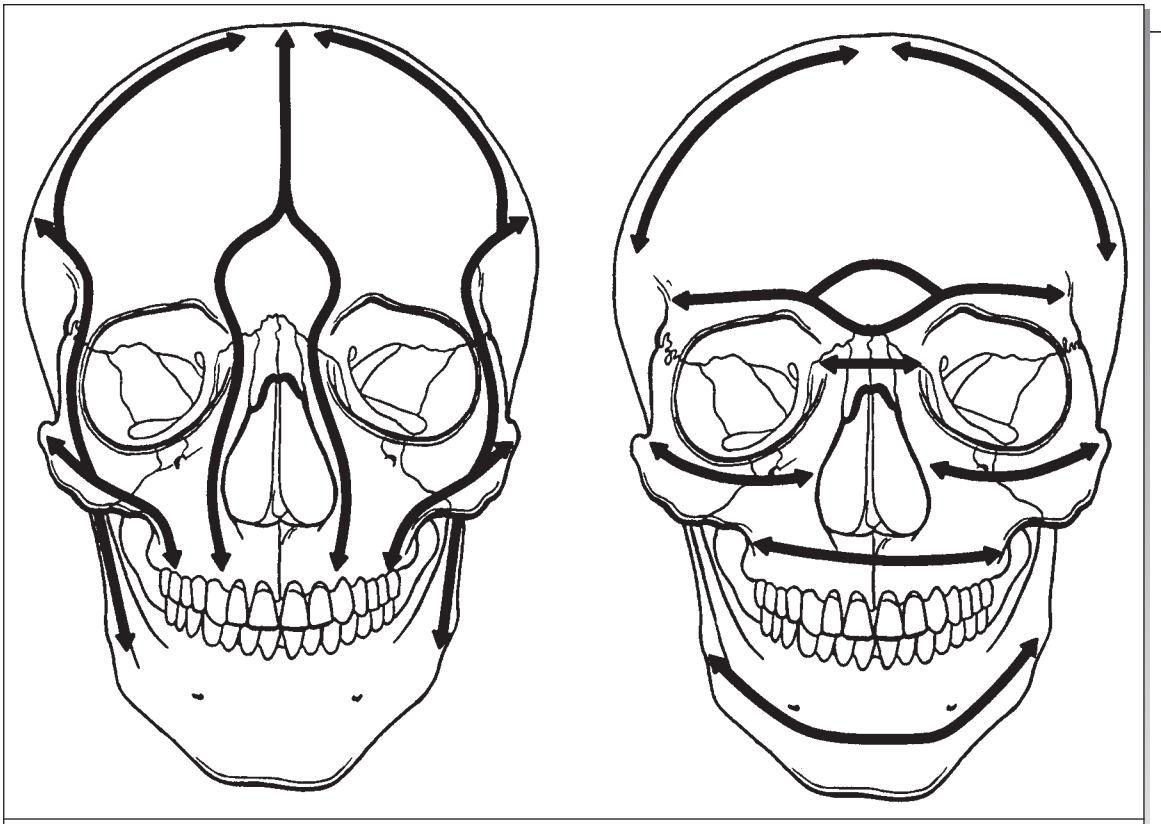

Figure 12 • The vertical and horizontal buttress system of the face. (From Manson PN, Clark N, Robertson B, et al: Subunit principles in midface fractures: The importance of sagittal buttresses, soft tissue reductions, and sequencing treatment of segmental fractures. Plast Reconstr Surg 103:1287-1306, 1999.)

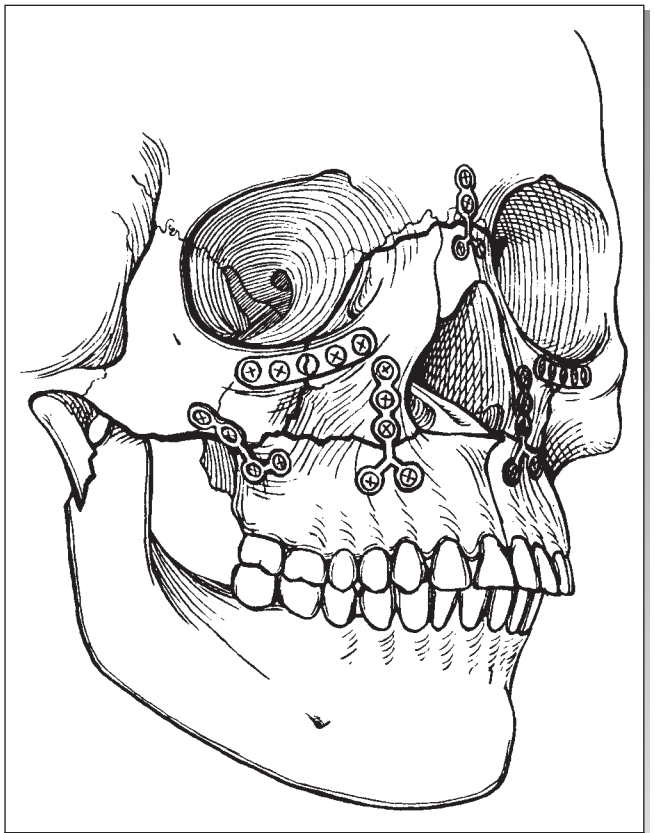

Figure 13 • Open reduction and internal fixation of a combination LeFort I and LeFort II fracture by fixation at the nasofrontal junction, inferior orbital rims, and LeFort I level. (© Johns Hopkins University.)

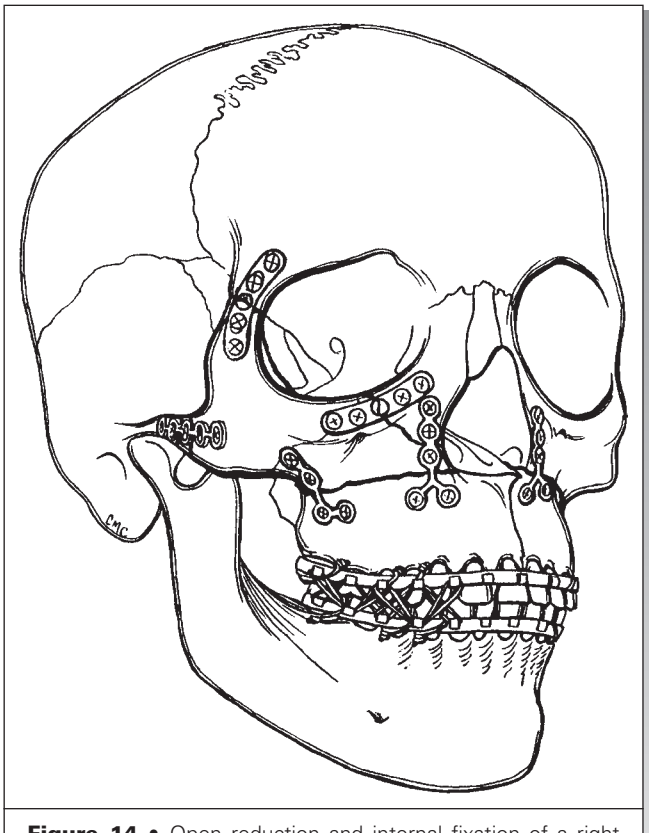

Figure 14 • Open reduction and internal fixation of a right LeFort III, bilateral LeFort I fracture by plates placed at the zygomaticofrontal suture, inferior orbital rim, zygomatic arch, and the four buttresses at the LeFort I level. (© Johns Hopkins University.)

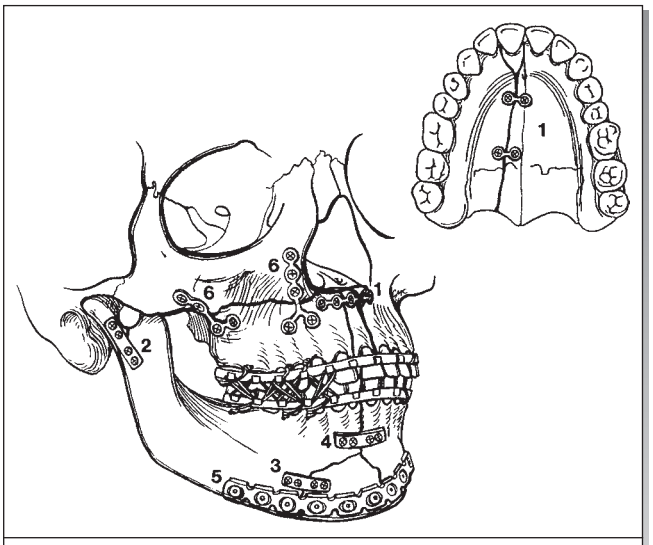

Figure 15 • Segments of sagittal fractures of the maxilla, if large, can be united by fixation in the roof of the mouth, which limits transverse width and displacement of the fracture; such fixation may be combined with reduction of the fracture at the piriform aperture. This technique creates a one-piece occlusal segment, which can then be treated like a LeFort I fracture segment. (From Hendrickson M, Clarke N, Manson P: Sagittal fractures of the maxilla: Classification and treatment. Plast Reconstr Surg 101:319-332, 1998.)

The most common orbital fractures are fractures of the orbital floor. Epistaxis is often present as a result of blood in the maxillary sinus, and many patients have hypoesthesia or anesthesia in the infraorbital nerve distribution. Depending on the extent of contusion of the ocular soft tissues, the patient may have diplopia on superior or inferior gaze. If a significant portion of the floor is dislocated, the eye may displace inferiorly and medially. Most of the fractures are confined to the thin internal section of the orbits; however, some involve small segments of the orbital rim.

In orbital floor fractures, the inferior rectus muscle or its fascia may be incarcerated. If the muscle is incarcerated, it represents an emergency condition in which release should be accomplished as soon as possible to avoid vascular compromise of the entrapped muscle because of the fine ligament system that extends throughout all the orbital soft tissue. Tethering of fat in the fracture site may limit excursion of an extraocular muscle even though the muscle itself is not tethered by the fracture. Freeing of the fat releases the extraocular muscle ligament system and allows normal eye motion.

Fractures may also involve the medial orbital wall, either with or without an orbital floor component. The medial rectus muscle is often contused in these fractures, but it is rarely incarcerated.

All patients with orbital fractures require ophthalmologic evaluation, and visual acuity must be documented. Pupil reactivity, the presence of double vision, eye mobility, visual fields, intraocular pressure, and funduscopic examination are essential components of the evaluation.

Diagnosis

Orbital fractures are evaluated by axial and coronal CT and by physical examination, including forced duction tests. Topical anesthesia is instilled into the conjunctival sac, and the insertion of the extraocular muscles onto the globe is grasped with forceps. The eye is rotated, and resistance implies mechanical restriction. In addition, CT may also demonstrate soft tissue incarceration by showing the relationship of the perimuscular soft tissue to the fracture.

Surgical Reduction

The need for surgery in orbital fractures depends on the resultant orbital volume and the potential for enophthalmos. The presence of functionally limiting double vision as a result of entrapment of either an extraocular muscle or perimuscular fat by virtue of the fine ligament system must be excluded. When an extraocular muscle is sufficiently entrapped to

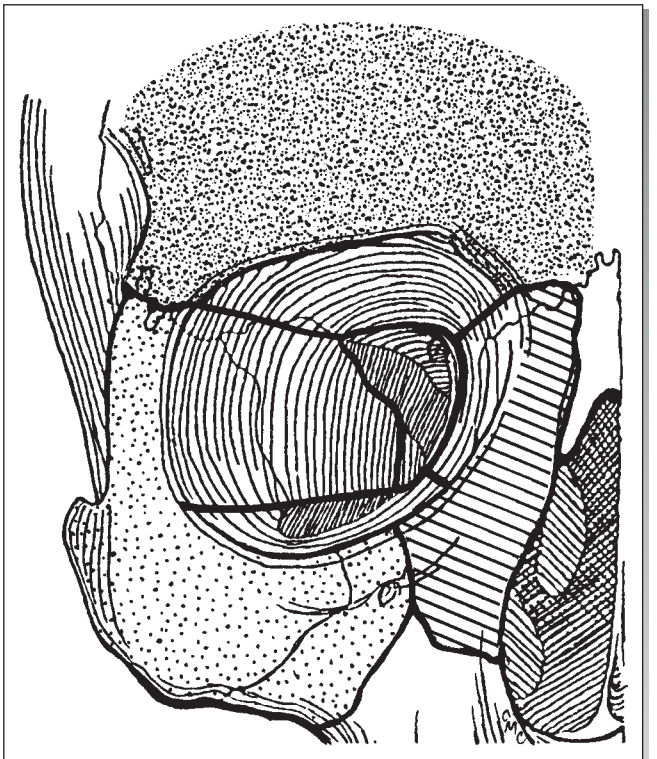

Figure 16 • Orbital fractures divide the internal orbit into four areas and the rim into three areas. "Buttresses," or thickened areas of bone, separate the four internal areas through the inferior orbital fissure, the inferior medial orbit, the upper portion of the medial orbit, and the lateral aspect of the orbital roof. When the inferoorbital buttress is disrupted, stable fixation of the reconstructed material is required for support. (© Johns Hopkins University.)

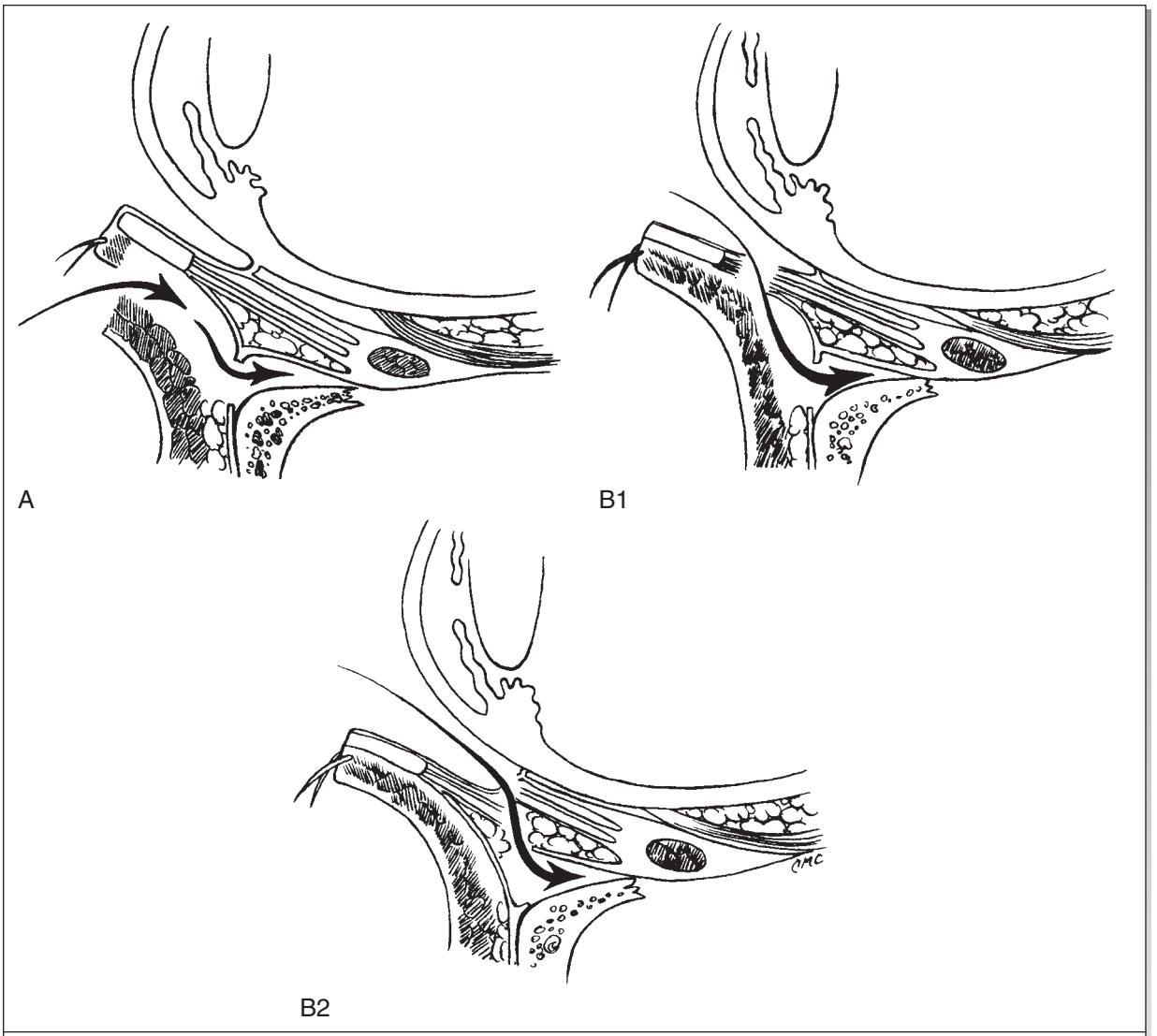

Figure 17 • A, Cutaneous lower lid incisions include the subciliary, midtarsal, and orbital rim incisions. **B,** Conjunctival incisions include the fornix (B1), infratarsal (B2), and medial conjunctival (not shown) incisions. The medial aspect of the orbit can be approached through the medial extension of the conjunctival incision behind the lacrimal area. (© Johns Hopkins University.)

cause double vision, a positive forced duction examination is present, and CT evidence of incarceration of orbital soft tissue is seen. Such fractures benefit from release of the orbital soft tissue to improve ocular range of motion.

Enophthalmos or a globe positional change occurs when the orbital volume increases by approximately 3 cc. The eye is displaced medially, posteriorly, and inferiorly. In medially impacted zygoma fractures, hypoesthesia may indicate compression of the infraorbital nerve by the fracture and could be an indication for surgical decompression by fracture reduction. However, the presence of an isolated infraorbital nerve sensory deficit is not in itself an indication to proceed with reduction of an orbital fracture. Reduction of orbital floor fractures is performed through a lower lid incision, which can include either a subciliary, midtarsal, or conjunctival incision (Fig. 17). The arcus marginalis is identified and incised and the subperiosteal dissection plane entered. As the dissection approaches the fracture, the periorbita is most often herniated into the maxillary sinus and is difficult to dissect. Dissection is first performed on all sides of the fracture in which the periosteal plane is undisturbed. It is often necessary to extend the dissection to the lateral and medial walls to identify the uninvolved regions of the orbit. After the periphery of the fracture is defined, the periorbita is teased out of the maxillary sinus and the posterior ledge of the fracture is identified. It is important to locate the posterior ledge because it allows accurate reconstruction of orbital volume. After all borders of the fracture are identified and the periorbita

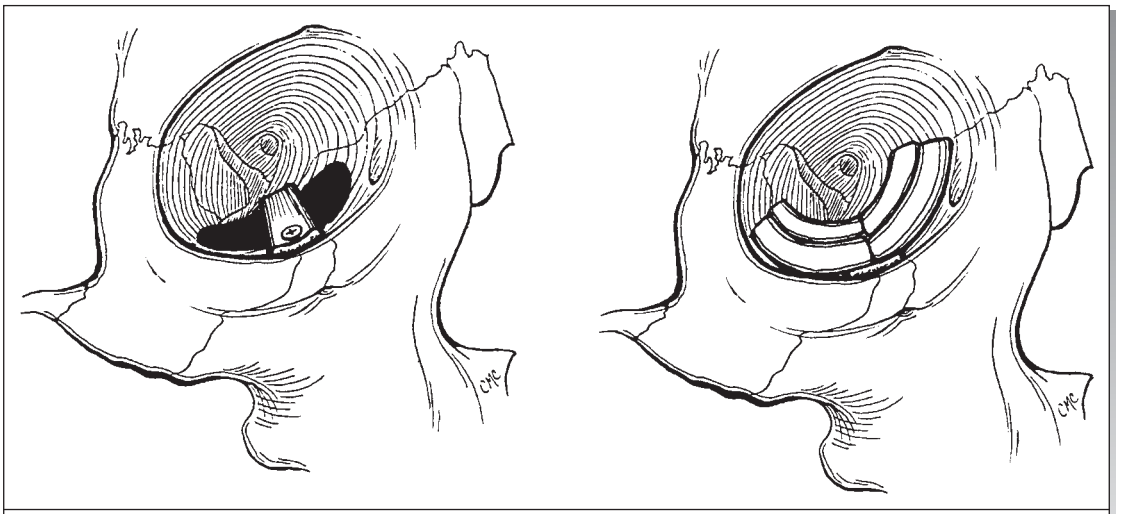

Figure 18 • Bone grafts to reconstruct the lower portion of the orbit must be positioned and angled appropriately to recreate the shape of the internal orbital wall that was fractured. They must be supported when necessary. (© Johns Hopkins University.)

reduced, the floor can be reconstructed with a thin bone graft, Medpor (Porex Medical, Fairburn, GA), or titanium mesh.

Forced duction examination should be performed before the dissection, after the dissection, and after the insertion of reconstructive material to ensure that there is no limitation in the movement of the extraocular muscle system. The exact volume of the orbit should be reconstructed by the bone graft (Fig. 18). In cases in which the operation was performed because of entrapment, release of the perimuscular or muscular soft tissues should improve the forced duction examination, and this fact should be confirmed at the time of surgery.

Orbital Roof Fractures

Supraorbital fractures usually involve the orbital roof. They are frequently minimally displaced, and treatment is observation. Some fractures can be inferiorly displaced and require open reduction via an intracranial procedure.

Fractures of the Medial Orbit

Medial orbital fractures are common and often found in conjunction with a floor fracture. Separate exposure of the medial orbital wall may be required and involves a conjunctival incision extended behind the lacrimal system to expose the medial orbital region. These fractures can be treated by placement of either alloplastic material or bone grafts to correct the volume enlargement of the medial orbit. Dissection should extend to the posterior border of the fracture, which in extensive injuries may approach the posterior ethmoidal foramen. The anterior ethmoidal foramen is approximately 15 mm and the posterior ethmoidal foramen 25 mm posterior to the medial orbital rim. In the dissection, the anterior ethmoidal vessels must be cauterized and divided to expose the medial orbital wall. More extensive fractures require a coronal incision. Medial orbit reconstruction is accomplished by placement of bone grafts with the smooth surface toward the extraocular muscles or with alloplastic material such as Barrier Medpor.

SUGGESTED READINGS

Crawley W, Azman P, Clark N, et al: The edentulous LeFort fracture. J Craniofac Surg 8:298-308, 1997.

Glassman N, Iliff N, Vander Kolk C, et al: Rigid fixation of orbital fractures. Plast Reconstr Surg 86:1103-1109, 1990.

Gruss J: Complex craniofacial trauma. Evolving concepts in management: A trauma unit's experience. J Trauma 30:377, 1990.

Gruss JS, Van-Wyck L, Phillips JH, et al: The importance of the zygomatic arch in complex midfacial fracture repair and correction of post traumatic orbito-zygomatic deformities. Plast Reconstr Surg 85:878, 1990.

Hendrickson M, Clark N, Manson P: Sagittal fractures of the maxilla: Classification and treatment. Plast Reconstr Surg 101:319-332, 1998.

Manson P, Clark N, Robertson B, et al: Subunit principles in midface fractures: The importance of sagittal buttresses, soft tissue reductions and sequencing treatment of segmental fractures. Plast Reconstr Surg 103:1287-1306, 1999.

Manson PN, Clifford CM, Su CT, et al: Mechanisms of global support and posttraumatic enophthalmos: Part I. The anatomy of the ligament sling and its relation to intramuscular cone orbital fat. Plast Reconstr Surg 77:193, 1986.

Markowitz B, Manson P, Sargent L, et al: The management of the medial canthal tendon in nasoethmoid orbital fractures: The importance of the central fragment in diagnosis, treatment and classification. Plast Reconstr Surg 87:843-853, 1991.

Phillips J, Forrest C: LeFort fractures. In Prein (ed): A-O Manual of Internal Fixation in the Craniofacial Skeleton. New York, Springer-Verlag, 1998.

Fractures of the Mandible

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H. PETER LORENZ

Optimal mandibular fracture treatment restores form and function to the mandible. Dental occlusion, bony anatomy, and facial aesthetics are returned to their preinjury state with fracture reduction and fragment stabilization. This chapter provides an overview of closed and open surgical treatment of mandibular fractures and their postoperative care.

Etiopathogenesis

After the nasal bones, the mandible is the most commonly fractured facial bone. Common causes are motor vehicle accidents and physical assault. Mandible factures are usually multiple so that when a single fracture is identified, one should seek to identify additional fracture sites. They are also associated with other facial fractures, and thus other fractures should be excluded as well.

Pathologic Anatomy

The mandible is a bilateral bone fused in the midline. Each hemimandible has a vertical and horizontal component. The condyle, coronoid, and ramus are part of the vertical segment, whereas the body and parasymphysis form the horizontal segment. Fractures occur in areas of weakness, such as unicortical areas (e.g., the condylar neck and angle), foramina (e.g., the mental foramen), or sites of long dental roots. In dentulous adults and children, fractures are most common in the weak subcondylar region, whereas in edentulous patients, the body and the angle are the most frequent sites of fracture. In the stage of mixed dentition, the body and symphysis are also susceptible to fractures because they are weakened by the developing tooth follicles.

The muscle groups are categorized by their direction of pull. The mandibular *elevators* are the masseter, medial pterygoid, and the anterior part of the temporalis. The depressor-retractors are the geniohyoid and digastric. The main *protrusor* (pushing the mandible anterior) is the lateral pterygoid. The *retractors* (pulling the mandible posterior) are the posterior component of the temporalis and the deep part of the masseter. *Lingual rotation* is caused by the mylohyoid. The pull of these muscles is normally balanced, but in the presence of fractures, the muscles act as deforming forces and can cause displacement of the fragments.

Diagnostic Studies

Complaints of malocclusion and dental or lip numbness often indicate a mandible fracture. Other findings include intraoral lacerations between teeth, fractured or loose teeth, and uneven areas along the dental arch. An ear canal laceration may signify a condylar head fracture or dislocation. An anterior open bite commonly occurs with bilateral condylar/subcondylar fractures. Loss of facial/dental sensation, temporomandibular joint (TMJ) crepitance, trismus, and mandibular deviation with mouth opening should also be noted.

Plain film mandibular radiographic series have largely been supplanted by computed tomography (CT) and are not routinely requested. CT evaluates not only the mandible but also the facial skeleton and brain. Each portion of the mandible, including the location of the condylar head with regard to the glenoid fossa, is clearly seen, and three-dimensional reconstructive images can also be obtained. Three-dimensional images are additionally helpful because they distinctly show the obliquity of a fracture and the position of the fragments. Panoramic roentgenograms provide additional information by demonstrating tooth involvement and root injury.

Goals of Reconstruction

Ideal mandible fracture management returns the patient to the preinjury occlusion and function in the shortest amount of time. For most fractures, internal rigid fixation in conjunction with maxillomandibular fixation (MMF) is used to anatomically align the fracture fragments and restore occlusion.

Treatment of mandible fractures is ideally performed as soon as feasible but can be delayed for 2 to 3 weeks for treatment of concomitant life-threatening injuries. Occasionally, urgent MMF can be performed in the acute setting to control bleeding at the fracture site in an unstable patient. Even 4 to 6 weeks after injury, the fracture callus can be mobilized without an osteotomy. After 6 to 8 weeks, however, the fracture is most likely healed, and an osteotomy will be required for fragment realignment.

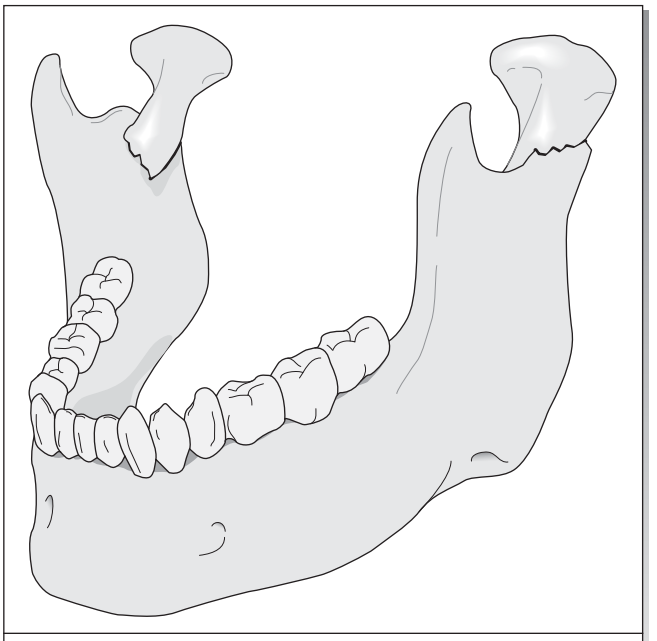

Figure 1 • Mandible with bilateral displaced subcondylar fractures that are amenable to maxillomandibular fixation.

Treatment: Closed versus Open Surgical Fixation

Preoperative counseling is given regarding the risk for local nerve injury, infection of hardware/bone, possible need for hardware removal, hematoma formation, dental injury, and an inability to achieve preinjury occlusion. The locations of intraoral and possible extraoral incisions and the need for MMF are discussed. Instructions for postoperative diet and oral hygiene are given.

Closed Surgical Treatment by Maxillomandibular Fixation

Greenstick fractures, condylar and subcondylar fractures, and fractures with minimal displacement are amenable to closed reduction and MMF (Fig. 1). Adequate dentition and periodontal health are required. For successful management, the segments must remain stable and reduced after MMF is established. For example, subcondylar fractures treated with MMF alone should remain stable with proper vertical ramus height reestablished. The technique of MMF involves securing arch bars to all teeth. Normal occlusion is established by aligning the wear facets of the interdigitating maxillary and mandibular teeth. MMF is held in position with either wire loops or tight elastics.

Two weeks in MMF followed by 2 weeks of a soft, no-chew diet are adequate for younger, healthy patients with condylar/subcondylar fractures. During mobilization, patients should be closely monitored, and asymmetries or deviations should be corrected with elastics to bring the mandible to the midline. Longer periods, up to 6 weeks, are necessary if delayed healing is expected (i.e., patients with periodontal disease, multiple injuries, immunosuppression, or diabetes). Six to 8 weeks are required for parasymphyseal and body fractures treated solely by MMF.

Open Surgical Treatment by Internal Rigid Fixation

Open reduction with internal rigid fixation (ORIF) is the treatment of choice for most mandibular fractures, including symphyseal/parasymphyseal fractures, multiple mandibular fractures, displaced fractures in edentulous patients, and displaced fragments subject to rotation (unstable fractures). Other indications for ORIF include angle fractures, an inability to obtain preinjury occlusion by closed reduction, the presence of a foreign body, severe condylar angulation, a condyle displaced outside the glenoid fossa, a noncompliant patient, an unstable midface, concomitant head trauma, and patient choice.

For fractures of the angle, body, or parasymphysis, a plate (generally 2.0 to 2.4 mm) is placed with a minimum of two bicortical screws on each side of the fracture. The screws must be inserted below the inferior alveolar canal. In addition, a tension band is necessary to prevent separation of the fracture edge at the superior border (Fig. 2). The tension band and plate should be small (generally a 1.3- to 1.5-mm plate) with unicortical (4 mm in length) screws to avoid injury to the tooth roots. When dentition is present on either side of the fracture, an arch bar can function as a tension band and a second plate is not necessary (Fig. 3).

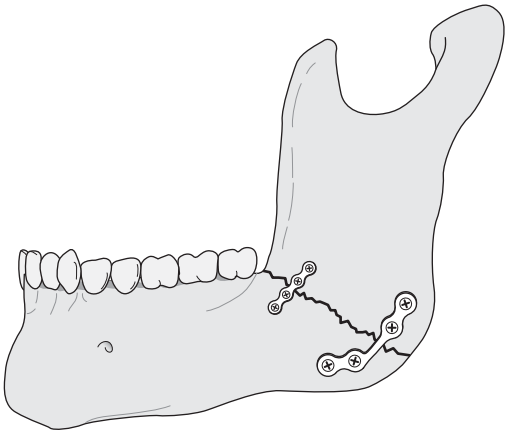

Figure 2 • Mandibular angle fracture repaired with a tension band and plate. As no dentition spans the fracture, a tension band is required.

The intraoral approach is recommended for exposure of the fracture sites. It can be combined with stab incisions in the cheek skin for transcutaneous placement of screws (for angle and body fractures) or with preauricular incisions (for condylar head or neck fractures).

After MMF is achieved, the mucosa and muscle are incised approximately 0.5 cm from the dentogingival line. The fracture is approached subperiosteally and all callus and debris removed. The mental nerve can be identified exiting the mental foramen below the first premolar and should be preserved. After fixation, the incision can be closed in watertight fashion with interrupted sutures.

For repair of fractures of the body and angle, the intraoral approach requires transcutaneous placement of drill bits and screws. Specialized cheek retractors and percutaneous drill/screw equipment are available in most mandibular fixation sets. The extraoral (Risdon) approach is made through an incision 2 cm below and parallel to the inferior border of the mandible. The Risdon incision carries the disadvantages of a visible scar and the risk of injury to the marginal mandibular nerve.

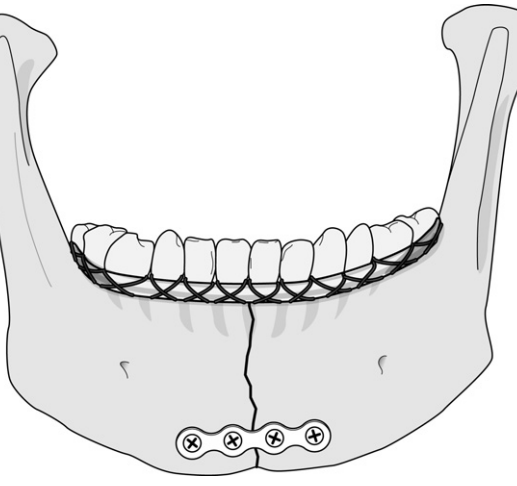

Figure 3 • Mandibular parasymphyseal fracture repaired with an arch bar and plate.

SURGICAL TECHNIQUE. Prophylactic intravenous antibiotics are administered preoperatively. Steroids to decrease facial edema can also be used. Nasal endotracheal intubation is performed. In circumstances in which nasal intubation is not possible, an armored oral tube can be passed in the retromolar region while MMF is being established.

The surgical sequence is as follows:

1. Arch bars are placed. Twenty-four-gauge wires are used to secure the arch bars to the molars and premolars, and 26-gauge wires are used on the canines and incisors.
2. The fractures are exposed and the fragments mobilized.
3. MMF is achieved with looped wires. Preinjury occlusion is established with simultaneous bony reduction. Interosseous wires or bone-approximating clamps are often needed to hold the reduction before plating.
4. Rigid plate fixation is performed. The plates are slightly overbent to prevent splay of the lingual cortex of the fracture when the plate is secured against the buccal cortex. A tension band, either a separate plate or an arch bar spanning the fracture, is necessary to prevent separation of the superior fracture edge.
5. The MMF is removed and the occlusion checked to ensure proper positioning of the condylar heads in the glenoid fossae. If a condylar head is subluxing or dislocating with jaw opening and closing, the occlusion is incorrect and the plating must be redone.
6. The incisions are closed. If postoperative MMF is necessary, it is established with wire or dental elastics. MMF is commonly used to minimize postoperative pain. If smaller plates (<2.0 mm) are used along the inferior border or more than one fracture is present, postoperative MMF is recommended for 2 weeks to maintain fracture immobilization.

Splints and Models. The use of preoperative models and splint construction can be particularly helpful to restore preinjury occlusion and proper dental relationships. Splints are most helpful for palatal and alveolar fractures. They provide additional strength for compound maxillary-mandibular fractures and for fractures in near-edentulous patients. Splints generally provide more stability, assist with

precise occlusal relationships, and save time in the operating room.

Teeth in the Fracture Line. Indications for removing affected teeth include fracture of the tooth or roots, severe periodontal disease, displacement of the tooth from the alveolar socket, a tooth interfering with reduction, and a tooth without bite function. With antibiotic coverage and oral hygiene, most teeth can be salvaged. Loose teeth should be wired solidly to adjacent teeth. The risk of infection, late dental loss, possible root canal treatment, and potential prosthodontic work should be discussed preoperatively with the patient.

Maxillomandibular Screws. In a near-edentulous patient, arch bars either cannot be placed or would not provide stable MMF. Self-tapping screws can be inserted directly through the gingiva into the anterior aspect of the maxilla and mandible, just medial (mesial) to the canine roots. The screws allow for easy application of wire or elastics for MMF. The screws are removed when MMF is no longer required.

Condylar Fracture/Temporomandibular Joint Dislocation

Condylar fractures are the most common mandibular fracture in adults. Patients are often initially found to have pain in the preauricular region, an ear canal laceration, malocclusion, limited jaw opening, and deviation with jaw opening. The vertical height of the fractured ramus/condyle is reduced, which results in premature contact of the posterior teeth and leads to an anterior open bite deformity. Condylar fractures can be further divided into *intracapsular* (*condylar head*) and *extracapsular* (*condylar neck* or *subcondylar*). Condylar head (intracapsular) fractures are best treated conservatively by closed treatment consisting of 2 weeks of MMF followed by guiding elastics and a liquid diet for an additional 2 weeks. Nondisplaced condylar neck fractures are unstable and should also be treated by closed reduction and MMF. In adults, indications for open reduction and internal fixation (ORIF) of condylar fractures include fragments that interfere with jaw excursion, the presence of a foreign body, displacement into the middle cranial fossa, severe angulation of the condylar head (>30 degrees), bilateral fractures with a persistent open bite, and combined bilateral subcondylar and LeFort fractures (ORIF for subcondylar fractures is necessary to establish proper mandibular vertical height).

Open reduction is usually performed through a preauricular incision. The procedure can be endoscopically assisted or combined with an intraoral or a modified Risdon incision. Care is taken to minimally disrupt the periosteum and vasculature of the condylar head because of the associated risk for either premature TMJ ankylosis (disk loss) or fibrous nonunion at the fracture site, or both. Plate and screw fixation is preferred, but the distal fracture segments are often too small and wire fixation must suffice.

Another option for smaller fragments is placement of lag screws in an inferior-to-superior direction through an incision below the angle of the mandible. The postoperative regimen involves MMF for 2 weeks followed by guiding elastics and jaw physical therapy. Patients should be advised of the risk for TMJ ankylosis and limited jaw opening.

Extracapsular fractures involving the neck and subcondylar region can best be treated by intermaxillary fixation alone for 2 weeks followed by guiding elastics and physical therapy.

Mandible Fractures during Childhood

Early diagnosis and treatment are imperative because fracture healing is faster in the pediatric population. Greenstick fractures are more common because of the elasticity of young bones. If malocclusion is present, MMF is necessary for 2 weeks, followed by a soft diet for 2 additional weeks. In the mixed-dentition phase, occlusal relationships can be difficult to establish. Arch bar placement on deciduous and partially erupted permanent teeth can be less secure. Other wire fixation techniques that can be used are acrylic splints and circummandibular, circumzygomatic, infraorbital, and piriform sinus wires.

ORIF for displaced fractures in a child is similar to that in an adult. Smaller plate sizes (1.3 to 1.5 mm) can be used, depending on jaw size. Monocortical screws 3 to 5 mm in length are used to avoid injury to the tooth buds. The miniplates and screws should be removed after healing is complete in 3 to 4 months. Dense fibrous tissue can envelop the hardware and may interfere with later bone growth. Resorbable systems are advantageous in this respect and may be a better alternative in the future.

The condyle is a mandibular growth center in children. Injury to the mandible can lead to TMJ ankylosis or mandibular growth disturbances, or to both. When growth is disturbed, the resultant deformity is inversely related to the age of the child. Children with condylar and subcondylar injuries should be monitored until they reach skeletal maturity. These fractures are treated by closed reduction and MMF for 2 weeks, followed by early mobilization. Open reduction is not recommended because of the risk of condylar devascularization. Physical therapy for young children involves activities designed to improve mandibular range of motion.

Edentulous Mandible

An edentulous mandible is structurally weaker than a dentulous one. With tooth loss, there is resorption

of bone along the alveolar ridge together with loss of strength and vertical height. Splints can be fabricated to establish the proper vertical height, and MMF can be achieved with circummandibular wires or MMF screws. If the patient's dentures are available, they can be used to establish the jaw relationships and MMF can be applied. The degree of displacement of the fractures guides the treatment modality. Minimally displaced fractures are treated with a soft diet and close observation. Displaced or highly mobile fragments must undergo ORIF. During dissection, periosteal stripping is minimized because the subperiosteal plexus is a major source of blood supply to the edentulous mandible. A single 2.4- to 2.7-mm compression plate with bicortical screws is placed along the inferior border. Primary bone grafting should be performed if a bony defect is present at the fracture site.

Postoperative Care

Factors that affect the patient's postoperative regimen include the anatomy of the fracture, the quality of rigid fixation, and the compliance of the patient. Longer periods of MMF may be required if bicortical screw fixation cannot be achieved or smaller plates are used. Details of postoperative care are listed in Table 1.

Complications

Complications specific to repair of mandibular fractures include infection, malocclusion, facial nerve injury, nonunion (fibrous union), and TMJ ankylosis and trismus. Abscesses need to be drained, cultured, and treated with antibiotics. Facial cellulitis that progresses or does not improve with antibiotic therapy is an indication to débride the fracture site and remove the hardware. Whenever possible, the hardware should be retained for 6 to 8 weeks to produce bony union. If infection develops after bony union has occurred, the hardware should be removed at the time of drainage and débridement.

Postoperative malocclusion can occasionally be improved with secondary orthodontic therapy. If facial asymmetry is present, however, secondary mandibular reconstruction is probably necessary. Hypoesthesia in the distribution of the mental nerve usually resolves over a period of several weeks but may take several months. Physical therapy for the TMJ is important in attaining adequate jaw opening.

TABLE 1 Postoperative Care

Diet (while in MMF): Full liquid, high-protein diet with high-calorie supplements
Oral hygiene: Half-strength chlorhexidine gluconate mouthwash three times a day and after meals
Antibiotics: A 7-day course (perioperative: ampicillin/sulbactam IV; postoperative: ampicillin/clavulanate elixir)
MMF: Assess weekly and tighten as necessary
After MMF removal: Soft diet with or without guiding elastics for 2 weeks. Advance to regular diet
Follow-up: Weekly to assess patient comfort, occlusion, nerve sensation, oral hygiene, and any signs of jaw locking and/or popping.

MMF, maxillomandibular fixation.

Pearls and Pitfalls

- When establishing preinjury occlusion, examine the displaced fragments for lingual/buccal canting and for small posterior open bites.
- Slight overbending of the plates is recommended to prevent splay at the lingual cortex of the fracture when the plate is seated against the buccal cortex by the screws.
- Release MMF before closure of the incision. Check jaw opening/closing to ensure that the condyles are seated properly in their fossae. Seating can be a problem with comminuted ramus, angle, and body fractures.
- Make certain that an arch bar or second plate spans the upper aspect of the fracture and functions as a tension band.
- Dental models and splints assist in establishing the occlusion and maintaining stability in complex fractures.
- Use "training" elastics—a single dental elastic on each side—to guide the jaw during excursion postoperatively. Elastics counteract the pull of the pterygoid and masseter muscles and can treat and possibly prevent later development of mandibular deviation with jaw excursion.
- When treating fractures by internal or external fixation, use elastics for postoperative MMF. Elastics are safer than wires if an airway emergency develops. Wires are useful for intraoperative MMF and for fractures treated by MMF alone.

SUGGESTED READINGS

Crawley WA, Sandel AJ: Fractures of the mandible. In Ferraro JW (ed): Fundamentals of Maxillofacial Surgery. New York, Springer-Verlag, 1997, pp 192-202.

Eckelt U: Problem areas—condylar neck. In Booth PW, Schendel AS, Hausamen JE (eds): Maxillofacial Surgery. London, Churchill Livingstone, 1999, pp 207-220.

Luhr HG: Fractures of the atrophic mandible. In Booth PW, Schendel AS, Hausamen JE (eds): Maxillofacial Surgery. London, Churchill Livingstone, 1999, pp 195-206.
McCarthy JG, Kawamoto H, Grayson BH, et al: Surgery of the jaws. In McCarthy JG (ed): Plastic Surgery, vol 2. Philadelphia, WB Saunders, 1990, pp 1188-1465.
Posnick JC: Management of facial fractures in children and adolescents. Ann Plast Surg 33:442-457, 1994.

Facial Fractures in Children

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JOSEPH GRUSS

Although pediatric facial injuries represent only a small percentage of all facial trauma, they are unique and challenging problems. Pediatric facial injuries differ from those in adults with respect to fracture patterns, influence of the injury and treatment on future facial growth, evolving dental relationships, and bony remodeling. Anatomic growth and ossification proceed from the skull to the midface and finally to the mandible. The paranasal sinuses aerate and the permanent dentition erupts, thereby further altering the size, proportion, and strength of the facial skeleton. Such anatomic changes influence the patterns of injury and the goals of treatment.

Etiopathogenesis

It is rare for a child younger than 5 years to sustain a facial fracture, and as puberty approaches, the incidence and patterns of facial fracture resemble those in the adult population. Children younger than 5 years reside with adults in a protected environment and have little independence and only gentle interaction with their peers. Injury in this age group is most often the result of motor vehicle accidents, falls, or abuse. In the 5- to 10-year-old age group, children enter school and participate in sports. Their activities, such as skate boarding and bicycling, are less supervised. With behavioral differences in play and social interactions, the incidence of fractures increases and tends to peak near the age of 10 years in the male population. Automobile accidents (passenger or pedestrian/cyclist) are the most frequent mechanism. Penetrating injury also begins to appear in this age group. The advent of the air bag has certainly reduced mortality for adult patients, but its effects on children can be catastrophic. However, appropriately restrained, back seat pediatric passengers sustain significantly less facial trauma during automobile accidents. As the child progresses into adolescence and becomes the driver of the car or the wielder of the weapon, an adult mechanism of trauma predominates.

Pathologic Anatomy

Facial skeletal injury is usually discussed in terms of three large regions—skull/frontal sinus, midface, and mandible—and the vertical/horizontal buttress systems that support them. This anatomic structure in the adult face results from differential growth during childhood. The skull or cranial vault dominates the pediatric face. At birth, the skull-to-face ratio is 8:1. The skull grows rapidly during the first 2 years of life. However, as the face grows, the skull-to-face ratio approaches 2:1 in adulthood. Facial growth is rapid from 4 to 7 years and includes aeration of sinuses and mineralization of bones. The infant's nasal cavity is small, as are the paranasal and frontal sinuses. In addition, the small maxillary sinus is strengthened by the presence of tooth buds. The maxillary sinus grows with the midface and reaches its adult size only when the permanent dentition erupts. Thus, the short, elastic face of an infant elongates to become the pillared, hollow structure of adolescence and adulthood.

Mandibular growth begins in the first branchial arch but proceeds with constant remodeling. Alveolar bone is added as the teeth develop. Endochondral ossification at the condyle is responsible for downward and forward growth of the mandible. Facial growth is a complex, dynamic process with many functional and anatomic interactions that are each dependent on the other. A mechanism to control this process, however, is not clear. Consequently, predicting alterations in growth resulting from trauma or the techniques used to repair injury remains difficult. Malunited fractures, however, continue their growth in abnormal positions, and the facial disturbances worsen with time.

Diagnostic Studies

Diagnosis of pediatric facial fractures can be difficult if one does not maintain a high index of suspicion for such injuries. The historical details of the injury are often unavailable, and in addition, clinical examination of an injured child can be difficult. As with any patient, the physical examination proceeds in an orderly fashion with observation and palpation of all involved areas. The presence of injured teeth is an indication of underlying bony injury. Neurosurgical and ophthalmologic consultation should be liberally applied because of the high incidence of associated injuries.

With the possible exception of the Panorex radiograph, plain film radiographs of the facial skeleton are not helpful in pediatric trauma patients. Due to the absence of aerated sinuses and a larger percentage of cancellous bone in children, plain radiographs have a high false-negative rate. The newer generation of computed tomographic (CT) scanners is fast and offers exceptional resolution. A non–contrast-enhanced CT study that includes the maxillofacial skeleton also affords the ability to image the brain and the upper cervical vertebrae.

Treatment Methods—Regional Approaches

Treatment methods for managing adult facial fractures include early intervention, wide exposure of all fracture elements, anatomic reduction with rigid fixation, and the use of early bone grafts. These principles must be carefully applied to pediatric patients. There is certainly a role for conservative management of carefully selected pediatric injuries. Fractures can often be "greenstick"-type injuries because of the cartilaginous nature of the immature skeleton. After appropriate reduction, they may require only one-point miniplate fixation rather than the traditional rigid, multipoint fixation as required in adults.

Fixation itself can be problematic in pediatric patients because of concerns of plate migration and growth limitation. When internal fixation has been used, interval removal has been advocated; however, the judicious use of rigid internal fixation has not been significantly problematic. Bioabsorbable plating materials have been introduced and accepted in craniofacial surgery, and experimental investigations with absorbable fixation systems are continuing in trauma patients. Polymers of polylactic acid, polyglycolic acid, or a combination are most prevalent. In pediatric patients with significant growth potential, absorbable plating systems may be advantageous.

The fracture patterns unique to pediatric patients are addressed according to their regional distribution.

Frontal-Orbital-Zygomatic Fractures

Fractures of the orbitozygomatic region are rare in children, and the type of fracture pattern is age related. The maxillary sinus has yet to develop, and the adult dentition waiting to erupt adds extra strength to the bone. In addition, the relatively large size of the skull in comparison to the face of a child also increases the number of skull fractures.

In younger children, trauma to the orbital soft tissues is more likely to produce an orbital roof fracture, whereas in the older pediatric population, as in adults, the same mechanism generates a floor blowout fracture into the aerated maxillary sinus. Before the age of 7 years, pressure near the orbital rim or forehead is transmitted to the orbital roof, whereas after the age of 7, when the sinuses have aerated, force is transmitted to the orbital floor.

When the orbital roof is fractured, the pulsatile pressure of the central nervous system is transmitted into the globe. On physical examination, the patient often demonstrates vertical dystopia and exophthalmos. Hypoesthesia of the supraorbital nerve is common. CT scans in both the axial and coronal views assist in the diagnosis and are also essential to define any underlying brain injury.

Orbital roof fractures in children are unique injuries and often occur without concomitant facial fractures. Orbital roof fractures can be grouped into those that are *displaced* and those that are not. Fractures that are displaced move either into the cranial vault or into the orbit. It has been reported that between 50% and 90% of orbital roof fractures are not displaced. Conservative management is appropriate. However, accurate reduction of a *displaced* orbital roof fracture is essential to prevent malunion and future orbital asymmetry. In a young child, displaced orbital fractures remodel and flatten. Downward pressure on the globe is transferred to the growing skeleton, thereby creating true orbital dystopia (Fig. 1). Untreated fractures also have a tendency for the development of an associated encephalocele. When this occurs elsewhere on the skull and is associated with a dural injury, it has been termed a "growing" skull fracture.

Exposure for reduction is accomplished through a coronal incision, and reduction of the fracture segments frequently requires an intracranial approach. Rigid fixation is applied, and if comminuted segments are present, the roof is bone-grafted to stable segments. Care is taken to reestablish the curved nature of the roof with molded split calvarial bone grafts. This maneuver is essential to maintain orbital volume and to isolate the anterior cranial fossa, as well as its deformational forces, from the growing orbit.

In patients older than 7 years, the pathophysiology of the orbital blowout fracture mirrors that seen in adults. Treatment plans are dictated by accurate diagnosis. Injuries necessitating surgical repair are divided into those in need of emergent, urgent, or

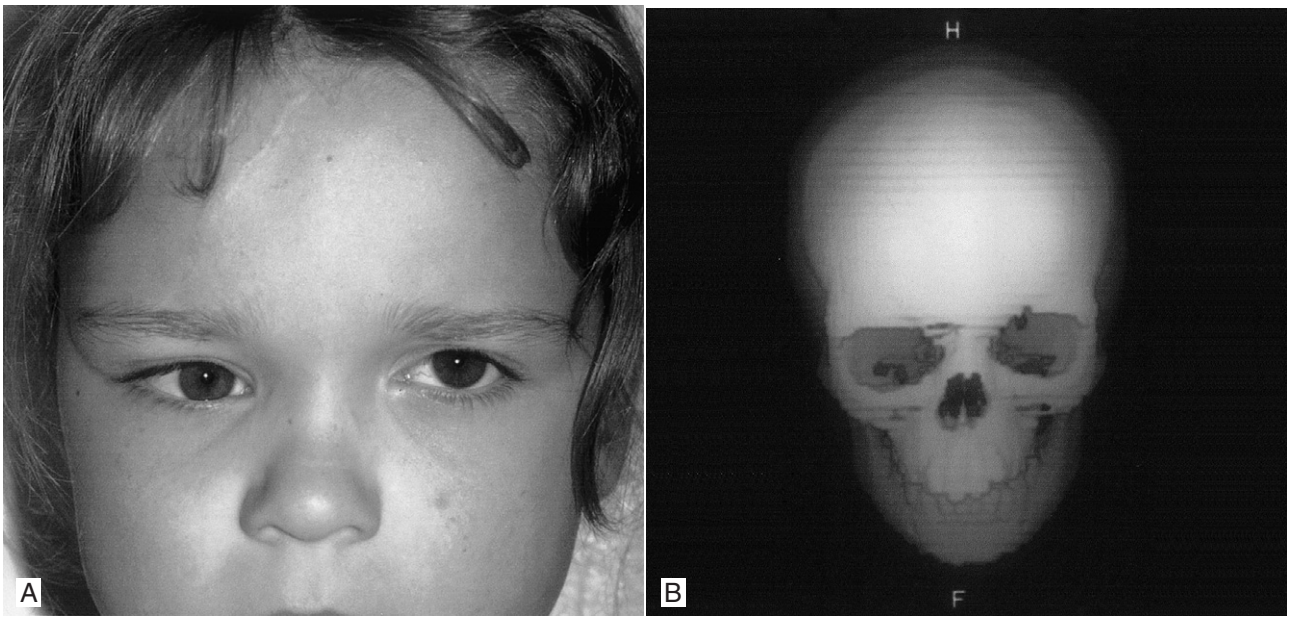

Figure 1 • A, Delayed manifestation of an untreated orbital roof fracture. Notice the true orbital dystopia. **B,** Three-dimensional computed tomography of the same patient demonstrating the flattened, remodeled orbital roof and bony orbital dystopia.

delayed treatment. The findings of diplopia in central gaze, early enophthalmos, or muscle entrapment mandate earlier repair. Ophthalmologic examination should document a forced duction test when muscle entrapment is suspected.

Unique to the pediatric population is the orbital floor "trapdoor" fracture. This injury is often the result of low-velocity injury and has also been called the "white eye" blowout fracture. It is accepted that young bones respond to traumatic force with more elasticity than do those of adults. Consequently, transmission of deformational forces to the floor of the orbit in a child produces transient displacement of the bony floor (Fig. 2), and the orbital contents can herniate through the defect. As the elastic bone recoils from its trauma, the orbital floor springs back into its natural position and traps the herniated orbital contents. Such entrapped structures sustain ischemic injury and fibrosis. Emergency surgical release is essential to regain complete ocular motility.

CT scanning can assist with this diagnosis because the entrapped ocular muscle can be seen within the maxillary sinus, below an apparently intact orbital floor (Fig. 3). In addition, sagittal reconstructed views provide information regarding the location of the blowout fracture with respect to the axis of the globe. Floor fractures posterior to the globe have a higher incidence of delayed enophthalmos, and one should maintain a low threshold for surgical intervention.

The surgical approach to the orbit is similar to that in adults. Every attempt is made to use a transconjunctival approach. The herniated contents are reduced into the orbit, and if possible, the floor is reconstructed with autogenous bone graft. The graft is stabilized with a wire or a lag screw; alloplastic material is avoided. If a "trapdoor" injury is suspected, the herniated contents are gently approached with a small rongeur or bone punch. In effect, the floor defect is enlarged to free the soft tissues so that they are not further damaged by the blunt trauma of pulling them through the closed "door." The created floor defect is repaired with autogenous bone graft. For small defects, a pericranial shave graft is used.

Isolated zygomatic fractures are rare in children. The large force required to injure this bone often results in fracture-dislocation of the zygoma. Treat-

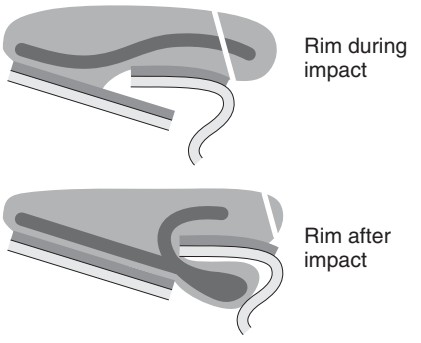

Figure 2 • Schematic representation of a "trapdoor" fracture. As the elastic orbital floor recoils, the herniated rectus muscle and fat are trapped.

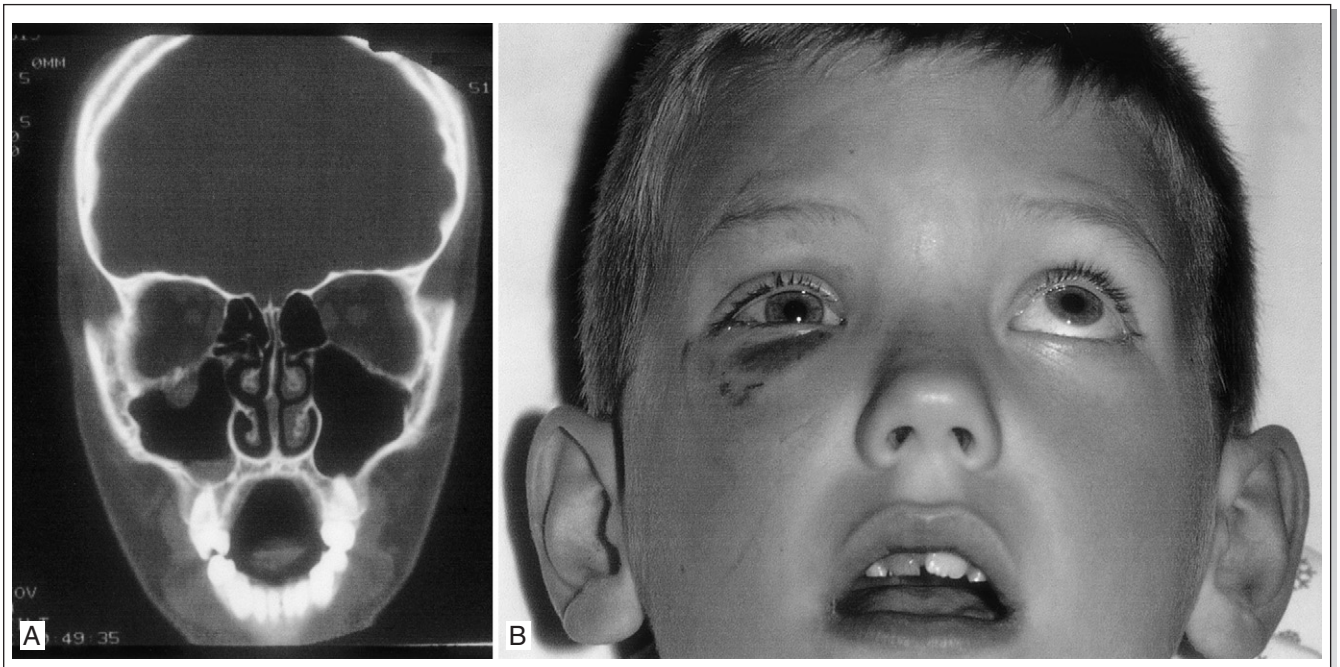

Figure 3 • A, Computed tomographic examination demonstrates the herniated fat and muscle trapped beneath an apparently intact orbital floor. **B,** The eye often shows little signs of injury. However, the inferior rectus muscle is clearly impaired.

ment of this injury is similar to that in adults. However, given the rapidity of bone healing in children, early intervention is essential. Unlike the situation in adults, attempts to mobilize a partially healed zygoma fracture in a child can result in new fracture lines.

Midface Fractures

DOG BITES. Dog bites are a significant health care problem. Although facial dog bites often result in various soft tissue injuries, a few cases are associated with facial fractures. Facial fractures are commonly not even suspected by the medical personnel initially caring for dog bite injuries. Fractures associated with dog bites have occurred predominantly in the orbital, nasal, and maxillary regions of the midfacial skeleton. These areas of facial fracture correspond to the most frequent sites of soft tissue dog bite injury. The nose, lips, and cheeks have been designated the "central target area" and are the most common structures damaged by facial dog bites.

Management focuses on prevention of infection and repair of skeletal and soft tissue deformities. Although these fractures are caused by a contaminated mechanism, a dog bite, the actual technique of repair should not differ. The stability afforded by rigid fixation has been shown to improve rates of bony union and decrease rates of infection.

Although facial fractures as a result of dog bites are not common, they should be actively excluded in any young child with dog bite injuries to the head or midface. As for other suspected bony injuries, a CT scan is the diagnostic tool of choice. The index of suspicion for a fracture should be especially raised when large breeds of dogs capable of crush-type injuries are involved.

LEFORT FRACTURE PATTERNS. Midface fractures in children are rare and account for only 0.5% of all facial fractures in this population (Fig. 4). In fact, LeFort I level fractures are rarely seen until after the age of 10 years when the maxillary sinus has aerated and the permanent teeth have erupted.

Traumatic forces dissipate across the pediatric skeleton by following the path of least resistance, which often results in a unilateral LeFort injury with a palatal split because the palatal suture is late in ossifying. Fractures tend to be oblique and pass around unerupted teeth and through foramina or open sutures.

Exposure techniques are the same for children as for adults. However, the timing of dental development and eruption complicates the application of rigid fixation. Occlusion is first established with the assistance of acrylic splints secured with circummandibular and piriform aperture wires. Rigid fixation can proceed with miniplates applied along the buttresses. Recently, resorbable plating systems have also been used.

Pediatric fractures heal rapidly, and maxillomandibular fixation may be removed sooner than in adult patients. However, it also accentuates the

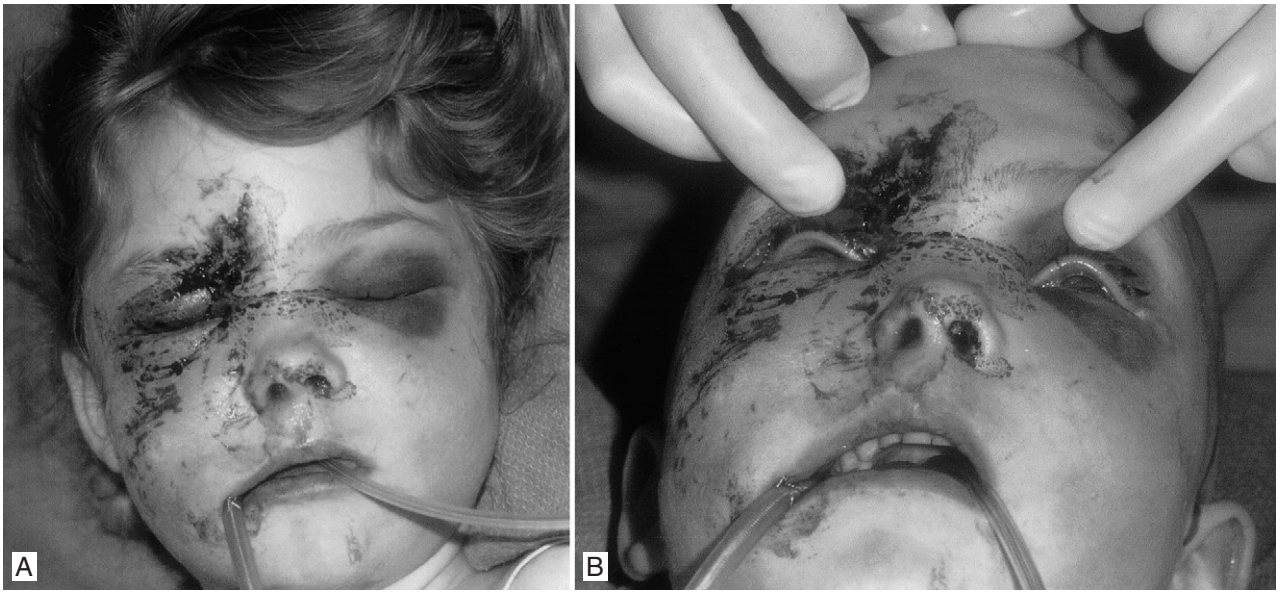

Figure 4 • A, A young girl was examined after being kicked by a horse. The periorbital ecchymosis and malocclusion indicated the severity of her injury. **B,** Enophthalmos is apparent.

need for prompt CT diagnosis and early surgical intervention to prevent malunion. As discussed with zygomatic fractures, it is difficult to remobilize a partially healed fracture in the pediatric population, and attempts to do so can produce new and different fracture patterns.

NASAL-ORBITAL-ETHMOIDAL FRACTURES. As in adult patients, nasal fractures occur frequently in children. The nose of a child is proportionally more cartilaginous than that of an adult; consequently, radiographic examination is unreliable, and nasal fractures can go undiagnosed. It may be that unrecognized nasal fractures are responsible for later nasal deviation or contour irregularity that appears to have no other cause.

Treatment of nasal fractures in children is similar to that in adults. The septum must be carefully evaluated for the presence of hematoma. Drainage is accomplished with an L-shaped incision in a dependent position. Transseptal quilting stitches are placed to prevent the reaccumulation of blood. Under general anesthesia, the bony pyramid is reduced by using an elevator placed within the nasal vault, and the outfractured bones are remodeled by palpation. The nose is packed with Vaseline gauze and a splint is applied. The packing is removed within 72 hours.

When the traumatic force is severe, as in motor vehicle accidents or large animal kicks, the bony structures of the nose may be impacted into the frontal sinus, if developed, and along the medial orbital walls into the interorbital space. Other oblique fracture extensions are often present. The nasal pyramid appears flat, the intercanthal distance is widened, and the nasal tip is elevated. Periorbital hematomas are often present bilaterally, and cerebrospinal fluid rhinorrhea can be observed. Detailed physical examination and high-level clinical suspicion lead to early CT confirmation of the nasal-orbital-ethmoidal fracture and prompt surgical reduction.

Management varies little from that in adult patients. The majority of nasal-orbital-ethmoidal fractures in children are large-segment injuries with the medial canthal tendon firmly attached to its central bone. This finding facilitates rigid fixation with microplates or resorbable plates. Fixation frequently extends from the frontal bone to the inferior orbital rim and piriform aperture buttress. The buttress may need to be exposed through an upper buccal sulcus incision. In severe injuries, a split calvarial bone graft or primary rib graft is cantilevered from the glabella to provide support for the nasal dorsum and soft tissues (Fig. 5).

One of the most confounding issues involving pediatric facial fractures is that of growth. The midface growth pattern requires a complex interaction of bone deposition and resorption in the presence of masticatory forces and sinus aeration. Even with appropriate treatment, traumatic disruption of these interactions can lead to facial deformity. Similarly, the lack of precise anatomic reduction can result in malunion, and the future growth of malunited bones can also produce facial deformity. The unpredictable nature of facial growth after trauma should be emphasized to the family members preoperatively (Fig. 6).

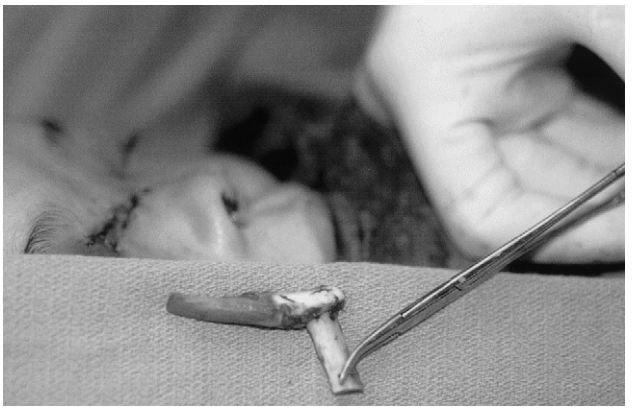

Figure 5 • Nasal support was achieved with a costochondral rib graft and columellar strut.

Dentoalveolar and Mandibular Fractures

Mandible fractures are the most common pediatric facial fractures, and it has been reported that they account for as many as 41% of all pediatric fractures. Treatment modalities and concerns regarding growth disturbances can be discussed with reference to three specific anatomic injury sites: alveolus, angle/body/ramus, and condyle.

ALVEOLAR FRACTURES. Fractures of the teeth and alveolar bone are frequent in children. Maxillary injury occurs early, as the child is learning to walk and is prone to frequent falls, whereas mandibular injury occurs later in development. Treatment options vary depending on whether the injury occurred to permanent or deciduous dentition. Children younger than 3 years have incomplete development of the deciduous tooth roots. Consequently, fractured or even avulsed teeth can often survive if supported with bracketed orthodontics. Dental impressions can be taken under anesthesia and a splint fabricated for adequate support. Because the time from injury to stabilization can affect tooth survival, these injuries require prompt attention.

Teeth impacted into the maxilla by trauma are managed expectantly. Although some injury to the underlying tooth buds may have occurred, impacted deciduous teeth can reerupt, and dental development may proceed. As the child ages, injury to the deciduous teeth or permanent teeth is more likely to produce tooth loss. Similar prompt stabilization of the segments with bracketed orthodontic appliances or acrylic splints is appropriate. Fractured tooth crowns should be treated with dental protection and restoration.

ANGLE/BODY/RAMUS FRACTURES. The anatomic distribution of mandibular fractures changes with age. Children younger than 6 years experience almost only dentoalveolar fractures. Condylar fractures first predominate and are discussed in the next section. Condylar fractures become surpassed

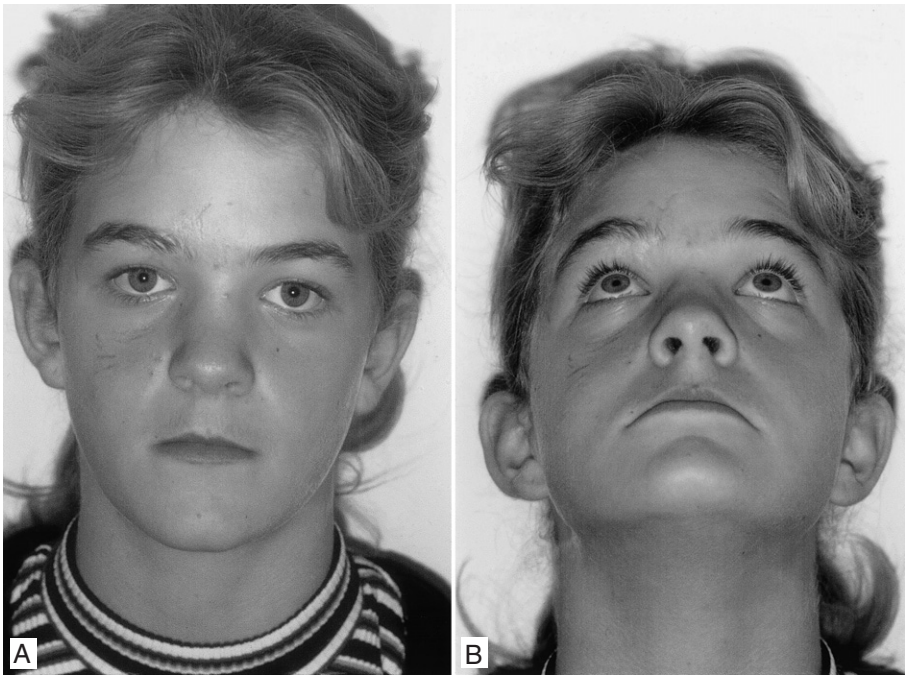

Figure 6 • **A,** Ten years after a severe midface injury and open reduction/fixation. **B,** Inferior view demonstrating adequate zygomatic projection and no significant enophthalmos.

by body and angle fractures as the child becomes older. The characteristics of fracture patterns also change with age. Before the eruption of permanent dentition, irregular greenstick patterns are common. The elastic, cartilaginous bone is occupied by developing tooth follicles, and this type of anatomic geography influences fracture lines and the application of internal rigid fixation.

Treatment goals are the same for children as for adults. However, the varying stages of dental development complicate treatment. If the fracture patterns are incomplete and the child maintains the premorbid occlusion, conservative therapy is adequate. A soft diet is maintained for approximately 2 weeks. The rapidity of bony healing in a child may allow for a shorter therapeutic course. However, when the fracture segment is comminuted or displaced or the premorbid occlusion is not maintained, rigid stabilization is required.

Maxillomandibular fixation and internal rigid fixation of the mandible vary greatly with the age of the patient. For an adolescent with permanent dentition, arch bars and internal fixation may be applied as in an adult. However, the teeth cannot be used adequately for fixation during the stage of mixed dentition or before eruption of the permanent dentition. During this developmental stage, skeletal wires are required to support maxillomandibular fixation. Circummandibular wire loops (26 gauge) are placed and connected to wire loops through the nasal spine or lateral piriform aperture. An occlusal splint may also be used. Circumzygomatic or orbital rim wires are avoided in young patients because they can cut through the cartilaginous structure.

Internal reduction with fixation of the fracture proceeds through an intraoral approach. Miniplates or microplates must be placed at the farthest inferior border of the mandible to avoid injury to unerupted tooth buds. Subtle degrees of malocclusion are tolerated in children because of the adaptive remodeling associated with the eruption of permanent dentition and masticatory forces, especially in children younger than 4 years.

CONDYLE FRACTURES. Management of condylar fractures continues to be debated. Controversies center around the impact of trauma and surgical repair on growth and the lack of long-term studies to document outcomes. For classification, condylar fractures are grouped as either intracapsular or extracapsular.

Intracapsular fractures or crush injuries occur in younger children. During the first 3 years of life, the condyle is short and conical. This type of anatomy protects it from fracture but increases the frequency of intracapsular crush injury. It is this population of patients sustaining crush injuries who are the most prone to growth abnormalities. Although the cartilage of the condylar head may not be a primary growth center for the mandible, it does grow in concert with other facial bones. Any alteration in normal function and development can change the interrelated growth of the face and produce internal joint derangement or mandibular deformity. Consequently, maintaining normal function is of primary importance. After a brief period of rest and soft diet, active physiotherapy is instituted with a liberal prescription of chewing gum!

Extracapsular condylar fractures often involve the condylar neck. It is not uncommon in older children to observe a unilateral condylar neck fracture associated with a contralateral parasymphyseal fracture. Similarly, bilateral condylar fractures occur after a fall or other impact on the center of the chin. The pull of the lateral pterygoid frequently angles the condylar segment medially. When the condylar head remains in the fossa and the premorbid occlusion is maintained, conservative management is preferred. A brief period (7 days) of maxillomandibular fixation is instituted, followed by active range-of-motion exercises.

When the condylar head is dislocated from the fossa or normal occlusal relationships cannot be established, open surgical reduction is required because dislocated fractures will not "remodel" sufficiently to provide normal function. Access to the condylar neck is achieved via a preauricular approach. Dissection is carried out along the cartilage of the external auditory canal so that the parotid and facial nerve branches are mobilized anteriorly. The joint capsule is exposed, and the fractured, displaced condylar neck is identified. Reduction involves both lateral traction and external rotation. Fixation is achieved with a miniplate whenever possible, and physiotherapy is instituted.

The condyle should also be opened and surgically repaired when penetrating injuries are associated with bone loss and retained foreign bodies. Techniques originally used in adults are applied. There are many options for the treatment of penetrating condylar injuries. The temporomandibular joint may be replaced with an autogenous costochondral rib graft. If possible, preserving the articular cartilage during the initial débridement permits the patient to maintain a functional upper joint space within which the rib graft can rotate and translate.

Postoperative Care

For children, the postoperative management of facial fractures is primarily supportive. Anticipated edema can leave children unable to open their eyes for 48 to 72 hours. Intraoral hygiene is maintained by frequent rinsing with a solution combining equal parts water, hydrogen peroxide, and mint mouthwash. Eyes are rinsed with a balanced salt solution, and supportive Frost sutures, if placed, are removed at 72 hours.

Pearls and Pitfalls

- Pediatric facial fractures are relatively uncommon.
- Legislative advances requiring infant or car seat restraint have significantly lowered the impact of automobile accidents.
- When injuries occur, there are many issues that separate the care of facial fractures in children from those in adults. The unique anatomy of the pediatric craniofacial skeleton produces different fracture patterns than in their adult counterparts. The orbital roof and the orbital trapdoor patterns are clear examples. The presence of developing teeth alters the pattern of midface fractures such that they stray from the expected LeFort classifications.
- Any injury to the craniofacial skeleton may alter growth. Displaced fractures that are not anatomically reduced may produce deformities that are progressive. These deformities are often accentuated with growth, for example, an unrepaired orbital roof fracture. In contrast, displaced fractures that are reduced and stabilized by standard exposure and rigid fixation usually maintain normal growth potential.

SUGGESTED READINGS

Andreasen J: Etiology and pathogenesis of traumatic dental injuries: A clinical study of 1,298 cases. Scand J Dent Res 78:329-342, 1970.

Arbogast K, Durbin DR, Kallan MJ, et al: The role of restraint and seat position in pediatric facial fractures. J Trauma 52:693-698, 2002.

Bartlett SP, DeLozier JB 3rd: Controversies in the management of pediatric facial fractures. Clin Plast Surg 19:245-258, 1992.

Grant JH III, Patrinely JR, Weiss AH, et al: Trapdoor fracture of the orbit in a pediatric population. Plast Reconstr Surg 109:482-484, 2002.

Gruss JS: Naso-ethmoid-orbital fractures: Classification and role of primary bone grafting. Plast Reconstr Surg 75:303-317, 1985.

Haug RH, Van Sickels JE, Jenkins WS: Demographics and treatment options for orbital roof fractures. Oral Surg Oral Med Oral Pathol 90:238-246, 2002.

Kelly K, Manson P, Vander Kolk C, Markowitz B: Sequencing LeFort fracture treatment. J Craniofac Surg 1:168-178, 1990.

Manson PN, Van der Kolk C, Defresne CR: Facial trauma. In Oldham KT, Colombani PM, Foglia RP (eds): Surgery of Infants and Children. New York, Lippincott-Raven, 1997, pp 429-445.

Messinger A, Radkowski MA, Greenwald MJ, Pensler JM: Orbital roof fractures in the pediatric population. Plast Reconstr Surg 84:213-216, discussion 217-218, 1989.

Palmer J, Rees M: Dog bites of the face: A 15 year review. Br J Plast Surg 36:315, 1983.

Reedy BK, Bartlett S: Pediatric facial fractures. In Bentz M (ed): Pediatric Plastic Surgery. Stamford, CT, Appleton & Lange, 1998, pp 463-486.

Posttraumatic Orbital Deformities

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OMER R. OZERDEM

This chapter provides an overview of the surgical treatment of patients with posttraumatic orbital deformities. This subject includes a wide variety of pathology, often in patients with a history of one or more previous operations.

Etiopathogenesis

Posttraumatic orbital deformities occur in patients without a history of primary treatment of orbital fractures or in patients with a history of primary treatment but without restoration of normal anatomy. In the ideal situation, a patient receives primary treatment from a surgeon who is experienced with a number of surgical approaches to reconstruction of the craniofacial skeleton, including the use of autogenous bone grafts.

Principles of Primary Care to Avoid Posttraumatic Deformities

TIMING. Patients with major facial injuries often have associated cranial or cervical spine injuries. However, unless administration of a general anesthetic would put the patient at significant clinical risk, the best results are obtained with prompt treatment. Delay invites the development of fibrosis and contracture and precludes optimal anatomic restoration. Nonanatomic treatment adds scar and makes definitive treatment even more difficult.

SURGICAL EXPOSURE. Major panfacial fractures often require a *coronal* incision for exposure of the upper part of the orbits and zygomatic arches (Fig. 1); however, a coronal incision may not be required for orbitozygomatic fractures if the zygomatic arch is not comminuted. An isolated orbitozygomatic fracture can be adequately approached through the lateral extent of an upper eyelid (blepharoplasty) incision (to access a frontozygomatic fracture), an upper buccal sulcus incision (to access a zygomaticomaxillary fracture; Fig. 1), and a lower eyelid incision (to access an infraorbital rim fracture). A subciliary (*transcutaneous*) incision is associated with a high incidence of lower lid retraction and should be avoided. A *subtarsal* incision made 5 mm lower in an eyelid crease (below the tarsus) gives a satisfactory aesthetic result with a lower incidence of lid retraction. A *transconjunctival* incision alone provides adequate exposure of the orbital floor and can be used in patients with blowout fractures. If the infraorbital rim is comminuted and plating with multiple screws is required, exposure through a conjunctival approach often requires the inclusion of a lateral canthotomy.

PROPER FRACTURE REDUCTION. Reduction of the fracture segments is dependent on adequate surgical exposure. A coronal incision, for example, exposes the lateral orbital wall, which has to be aligned perfectly for proper fracture reduction. If a fracture cannot be reduced, all measures, including osteotomies, must be performed before fixation.

CHOICE OF MATERIAL FOR ORBITAL FLOOR RECONSTRUCTION. Primary autogenous bone grafts are the material of choice for orbital floor reconstruction. Primary bone grafts consolidate with the patient's native bone and do not undergo late infection or extrusion. Less scar forms over a

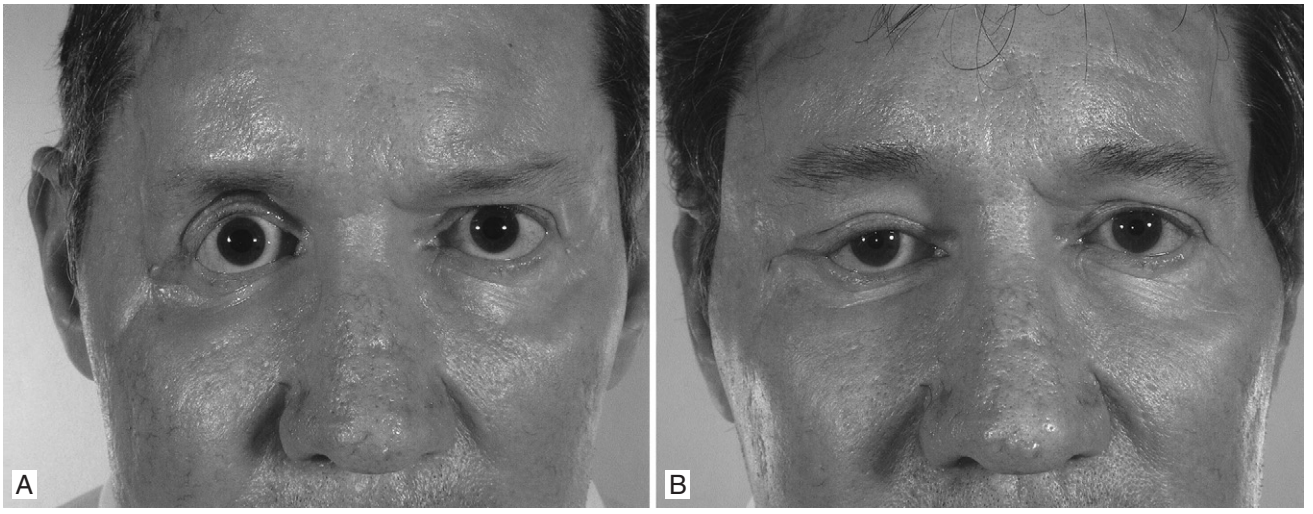

Figure 1 • A, Malunited right zygomatic complex fracture. Inferior displacement of the cheek and enophthalmos are present in addition to scar retraction of the lower lid. **B,** The patient underwent corrective osteotomy of the zygomatic complex with accurate reduction through a combined coronal, intraoral, and transconjunctival/lateral canthotomy approach. The zygomatic complex was reduced and the lower eyelid repositioned.

bone graft than over an alloplastic implant and allows for better eye movement. If reexploration of the orbit is required at a later date, it is easy to elevate the thin neoperiosteum, in contrast to the difficulty encountered when dissecting an orbital floor in which an implant is present, particularly one that is perforated.

Pathologic Anatomy

Skeletal deformities may include defects or displacement of any of the four walls of the orbit. The external framework of the orbit may have been well aligned and fixated, but persistent defects in portions of the internal orbit can cause enophthalmos or other abnormalities in globe position (hyperglobus and hypoglobus). The nose may have been fractured with a depressed bridge, deviation, or foreshortening.

Soft tissue abnormalities may include telecanthus (increased distance between the medial canthi); canthal malposition; eyelid malposition, including lower lid retraction or ectropion; upper eyelid ptosis; or injuries or missing portions of any of these structures. The globe itself may have been damaged, with resultant loss of light perception or pupillary constriction, and corneal scarring, posttraumatic cataracts, or glaucoma may be present. If damage or scarring involving the extraocular muscles has occurred, there may be diplopia.

Interruption leading to malfunction of the nasolacrimal apparatus is common.

Preoperative Evaluation and Diagnostic Studies

Physical examination is important and includes palpation and observation of eyelid/canthal/eye position and eye movement. A funduscopic, slit lamp examination and the Schirmer test are best performed by an ophthalmologist. Patency of the lacrimal system can be demonstrated by passage of dye from the conjunctival cul-de-sac into the nasal cavity.

A computed tomographic (CT) scan is the most valuable imaging study; plain radiographs are not indicated because CT scans provide superior visualization of the bony orbit and its contents. Axial and coronal views are routine, and sagittal views should be obtained to assess the contour of the orbital floor. Three-dimensional scans provide a view of displaced larger structures, such as the body of the zygoma, but one can usually obtain more precise information from two-dimensional images. Magnetic resonance imaging provides further information about the brain and intraorbital soft tissue structures, but it does not visualize bone well.

Reconstructive Goals

Reconstruction should result in a normal periorbital appearance with optimal function of the affected eye. Fractures should be reduced and missing structures replaced with similar tissue material.

Treatment

Skeletal Deformities

Preoperative CT scans detail the anatomic areas that require correction. Defects of the orbital floor, with or without an implant and without displacement of the outer orbital framework, can be approached through a lower eyelid incision alone.

If there is a significant defect in the medial orbital wall or a displaced zygoma, a coronal and, often, an intraoral incision is preferred. Dissection of the orbit must be circumferential and extended into the depths of the orbit as far as normal, nondisplaced bone (the "shelf"). In most posttraumatic orbits, dissection farther than 38 mm from the infraorbital rim along the orbital floor is not required (the optic foramen is usually found at approximately 42 mm). The optic nerve is at greatest risk during dissection of the medial orbital wall; one can coagulate the anterior ethmoidal vessels, which are only several millimeters from the optic canal. After the posterior, nondisplaced bone is identified, an autogenous bone graft should be placed so that it rests on top of the ledge or shelf of bone posteriorly. It should be fixed on its anterior aspect to the orbital rim with either plates or wires.

If skeletal structures are displaced, even minimally, it is better to liberate and move them into anatomic position (Kawamoto) than to correct the deformities with onlay camouflaging grafts. Resulting defects of the internal orbit or anterior aspect of the maxilla should be bone grafted; calvarial bone grafts are the first choice, but iliac or rib grafts are also satisfactory donor bones.

Enophthalmos

As one places bone grafts across internal orbital defects to diminish orbital volume and correct posttraumatic enophthalmos by projecting the globe, one should be aware of any sudden changes in intraocular pressure (determined by simple digital palpation or a Schiøtz tonometer). If the globe suddenly becomes much firmer on palpation, some of the bone graft should be removed because a prolonged increase in intraocular pressure may result in visual loss.

Medial Canthal Deformities/Telecanthus

Medial canthal repositioning or canthopexy usually requires identification of the medial canthal mechanism through an external incision, complete mobilization, and transnasal wiring by making a hole with a piercing awl superior and posterior to the lacrimal fossa. It is not generally possible to replace a segment of bone with the tendon attached as one can in a primary operation. An onlay nasal bone graft also improves the contour of the medial canthal area. If one uses a coronal approach, the lateral canthal tendon should be repositioned to the temporal aponeurosis. After extensive orbital dissection and bone grafting, the use of a symblepharon (ocular) conformer may prevent chemosis of the conjunctiva (Fig. 2).

Eyelid Ptosis and Extraocular Muscle Imbalance

Eyelid ptosis and extraocular muscle imbalance should not be addressed until at least 6 months after skeletal orbital surgery because there may be considerable improvement attendant to the improvement in orbital shape, volume, and globe position. One should *never* attempt to correct upper eyelid ptosis before the enophthalmos has been corrected by reduction of orbital volume.

Postoperative Care

The most important examination after extensive orbital surgery is that involving the function of the eye. Dressings should not be placed over an operated eye, and patients with sighted eyes should be checked for visual acuity in the recovery room. In the event of loss of light perception, the patient should be returned immediately to the operating room for removal of some of the bone grafts. In patients with absence of light perception preoperatively, one does not need to check for visual acuity.

The use of cranial bone grafts for orbital surgery has reduced the length of hospitalization because there is less donor site morbidity. Passive drains are placed in the temporal fossae after a coronal dissection, and they are removed the following day. A small drain can be placed in the orbital cavity, particularly in previously bone-grafted patients, to facilitate egress of blood. Drains are removed the following day. Antibiotics are given prophylactically; steroids are not routinely used.

After extensive dissection through the lower eyelid, it is wise to suspend the lower eyelid with a lateral Frost suture by passing a 5–0 nylon suture through the skin of the upper eyelid, exiting at the upper gray line, passing through the lower gray line and tarsus, and returning through the upper eyelid. The two ends of the suture are suspended to the brow with a short segment of Steri-Strip and reflected downward; another Steri-Strip is applied and the sutures are reflected upward for a final Steri-Strip. The suspensory suture is removed after 4 to 6 days.

Patients with coronal incisions may shower and gently wash their hair on the second postoperative day.

When orbital floor bone grafts have been placed, nose blowing should be avoided for the first month.

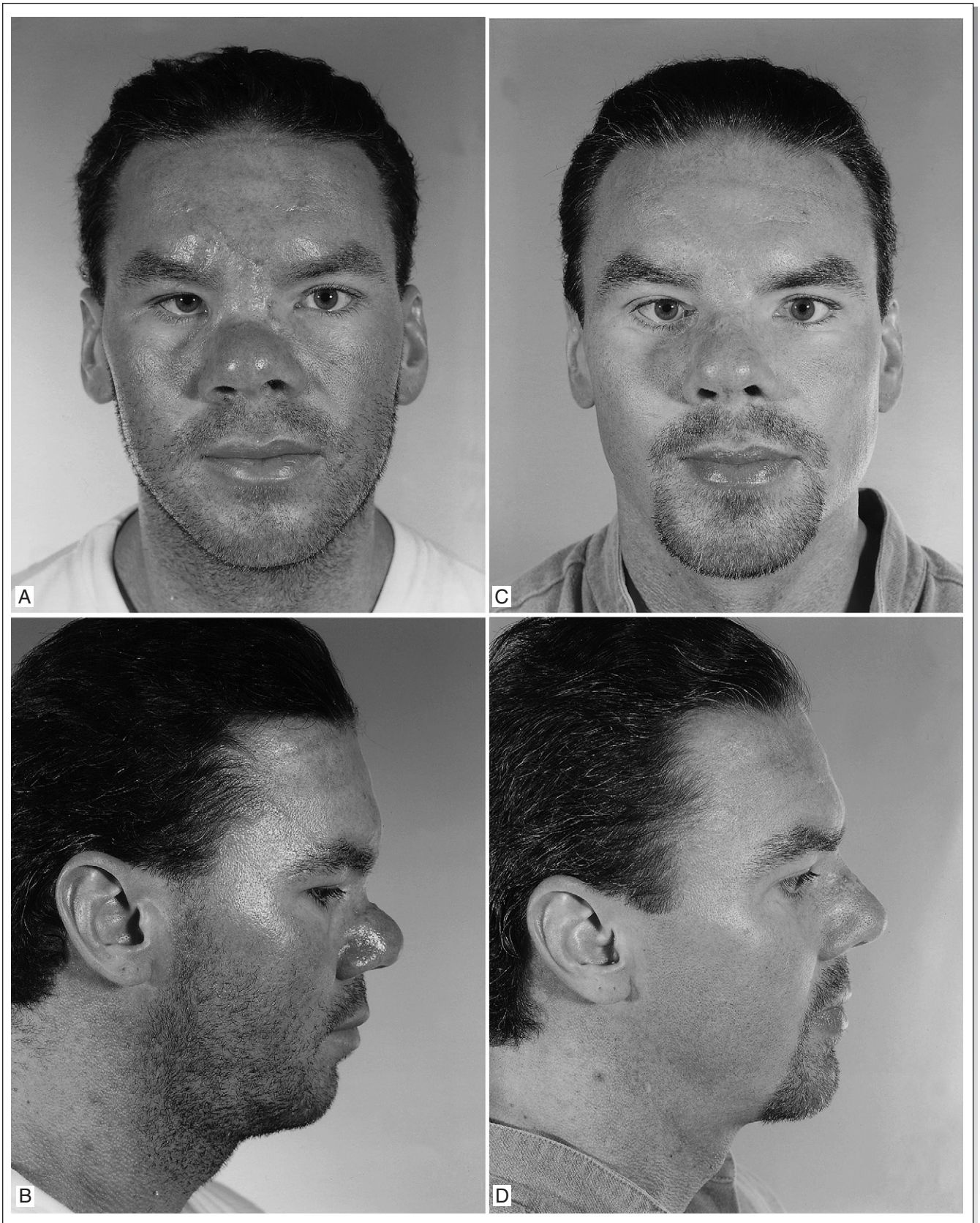

Figure 2 • **A** and **B,** Telecanthus and nasal collapse after a naso-orbito-ethmoid fracture. **C** and **D,** The patient underwent a calvarial bone graft to the nasal dorsum with transnasal wiring of the medial canthi.

Complications

The most significant complication is loss of visual acuity. It is presumed that venous pressure in the orbit increases to the point that it affects the retinal circulation. As mentioned earlier, the patient must be returned to the operating room for exploration of the orbit and removal of bone grafts.

Corneal damage may also occur; either corneal protectors or a temporary tarsorrhaphy suture can decrease the incidence of corneal injury.

Hematomas are unusual if the temporal fossa has been properly drained postoperatively. Small TLS drains placed in the orbital cavity may be helpful in preventing intraorbital fluid collections, especially if the orbit has been closed by previous bone grafting.

Infections are almost nonexistent in bone-grafted orbits if an eye is present. Nasal bone grafts are more vulnerable, particularly if the overlying tissue cover or the underlying lining is of questionable quality. If an infection does occur, and frequently it may be several weeks before one is recognized, intravenous antibiotics should be instituted and adequate drainage provided. If exploration of the wound is required, one can often wait 4 to 6 weeks. At that time, all hardware and sequestered bone should be removed.

Pearls and Pitfalls

- Orbital reconstruction should be undertaken only by properly trained, experienced surgeons.
- Precisely conceptualize the anatomic deformity (provided by preoperative imaging and adequate dissection).
- Adequate surgical exposure along with satisfactory lighting is mandatory.
- Retrieve herniated orbital contents from the paranasal sinuses.
- Restore bony orbital volume and correct contour deformities with bone grafts.
- Relocate a displaced zygoma by osteotomies.
- Achieve rigid fixation of the displaced segments.
- Use only autogenous bone grafts.
- Reattach medial and lateral canthal tendons.
- Provide adequate soft tissue cover.

SUGGESTED READINGS

Jackson IT: Classification and treatment of orbitozygomatic and orbitoethmoid fractures. The place of bone grafting and plate fixation. Clin Plast Surg 16:77, 1989.

Kawamoto HK: Late posttraumatic enophthalmos: A correctible deformity? Plast Reconstr Surg 69:423, 1982.

Wolfe SA: Posttraumatic orbital deformities. In Wolfe SA, Berkowitz S (eds): Plastic Surgery of the Facial Skeleton. Boston, Little, Brown, 1989, p 575.

Face Lift and Brow Lift

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DOUGLAS S. STEINBRECH

Facial aging is one of the most common reasons for plastic surgical consultation. The increased use of nonsurgical antiaging modalities has not resulted in reliable improvement in many of the characteristic aging changes, and therefore surgical lifting of the face and brow remains the best solution for ptotic facial tissues.

Etiopathogenesis

The process of facial aging represents a combination of gravitational effects, atrophy, and aging of the skin itself. Gravitational changes and atrophy yield the characteristic changes shown in Figure 1.

Aging of the skin is manifested as fine wrinkles and irregular pigmentation. At the histologic level, cutaneous aging is characterized by flattening of the dermal-epidermal junction, decreased number of melanocytes and Langerhans cells, reduction in the amount of glycosaminoglycan ground substance, progressive dropout of elastic fibers, and diminution in the total amount of collagen as well as the fraction of type III collagen. Such histologic deterioration correlates with the following clinical findings: thinning of the skin, decreased resistance to shearing forces, reduced elasticity, immunologic changes, and increased susceptibility to ultraviolet light and cutaneous malignancies. *Face and brow lifting, the subject of this chapter, addresses only the effects of gravity and atrophy, such as jowls and submental excess. It has no effect on fine wrinkles or the quality and texture of the skin.* Cutaneous aging is treated by chemical or resurfacing techniques, subjects addressed in the earlier chapter "Cutaneous Resurfacing and Fillers."

Pathologic Anatomy

All patients have individual aging patterns, but certain characteristics are seen to varying degrees in every patient (see Fig. 1), including the following:

1. Brow ptosis
2. Glabellar creases
3. Excess eyelid skin
4. Lower lid laxity
5. Tear trough deepening
6. Malar descent
7. Deepening nasolabial folds
8. Drooping nasal tip
9. Perioral wrinkles
10. Jowls
11. Platysmal bands
12. Loss of the cervicomental angle
13. Skin laxity

Diagnostic Studies

Patients seeking a face and brow lift are usually in the fifth to seventh decade of life. Although most patients are in good health, the importance of preoperative risk assessment for elective surgery in this age group cannot be overstated. All patients should have standard preoperative screening with a complete history, physical examination, blood analysis, and electrocardiogram (ECG). Patients with any abnormalities, including a history of systemic

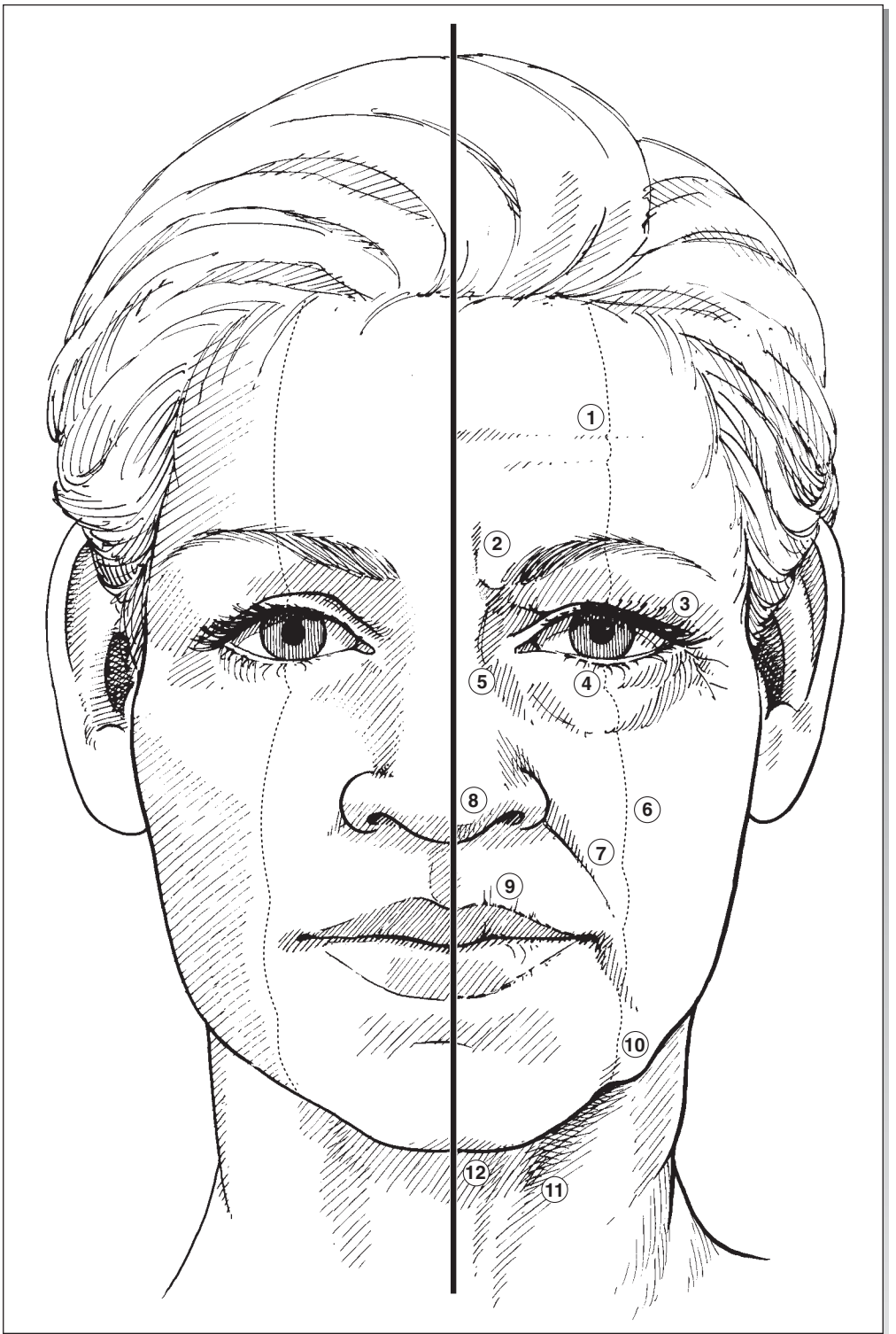

Figure 1 • Characteristic facial aging. 1, Brow ptosis; 2, glabellar creases; 3, excess eyelid skin; 4, lower lid laxity; 5, tear trough deepening; 6, malar descent; 7, deepening nasolabial folds; 8, drooping nasal tip; 9, perioral wrinkles; 10, jowls; 11, platysmal bands; 12, loss of the cervicomental angle and skin laxity.

medical problems (hypertension, diabetes, asthma, heart disease, etc.), borderline hypertension found at physical examination, or any abnormality on ECG should obtain complete medical clearance before surgery. It is also helpful to review all patients with the anesthesiologist before surgery. Stringent clearance not only helps to avoid unexpected complications but also promotes trust between the plastic surgeon and patient.

All patients should undergo standardized preoperative photographs (frontal, right and left lateral, three quarters, and clenched face views). The clenched face view should highlight the platysma bands and show a full smile with tightly closed eyes. Photographic analysis aids in diagnosis of the pathologic and age-related features most concerning to the patient and is an important tool for communicating the goals of surgery. Photographs also provide documentation of the preoperative condition, including the degree of deformity, asymmetry, and facial nerve status. Most patients have facial asymmetries, and some have preoperative paralysis of certain facial muscles. Finally, photographs are also used for comparison of the preoperative and postoperative appearance.

Reconstructive Goals

The goal of any facial rejuvenation procedure is restoration of a more youthful face and the characteristic curves that are lost with age. Face and brow lifts reverse only certain aspects of facial aging, and thus the goals of a face and brow lift are specific. The current trend is toward an emphasis on redistribution of facial volume rather than tension, which tends to flatten the desirable contours. The specific goals of a face lift include the following:

1. Improvement in neck contour
2. Redistribution and excision of excess neck skin
3. Softening of the platysma bands
4. Defining the mandibular angle
5. Reduction of jowls
6. Elevation of the malar fat pad
7. Redistribution and excision of excess facial skin with improvement in some of the deeper lines

The goals of a brow lift are to establish optimal brow position. The ideal female brow anatomic criteria are as follows:

1. Medial pole of the brow on a perpendicular line even with the alar base
2. Lateral pole of the brow on an oblique line tangent to the lateral canthus and alar base
3. Medial and lateral poles of the brow on the same horizontal level
4. Apex of the brow curvature even with the lateral limbus
5. Apex of the brow 2 to 3 mm above the supraorbital rim

Ideal brow positioning in men is more difficult. A man with the aforementioned criteria would appear feminine. The male brow should lie at approximately the level of the rim and should be only slightly arched.

Brow lifts are often sought for correction of transverse forehead rhytides. The deep creases are frequently the result of chronic frontalis contraction in an effort to elevate a ptotic brow or upper eyelid. It is important to identify and correct this condition to have a long-term effect on these rhytides. A ptotic brow is treated with a brow lift, but a ptotic upper lid is not. The latter should be identified and addressed via blepharoplasty or ptosis procedures (or both).

Normal Anatomy

The anatomy that is relevant to the face and brow is best conceptualized as a series of layers. The five layers of critical anatomy are the skin, subcutaneous fat, superficial musculoaponeurotic system (SMAS)/muscle layer, a thin layer of fascia, and the facial nerve. These five layers are present in all areas of the face and forehead, but they vary in quality and thickness, depending on the anatomic area. The third layer (SMAS) is the most heterogeneous. It is fibrous, muscular, or fatty, depending on the location in the face. The muscles of facial expression are part of the SMAS layer (e.g., frontalis, orbicularis oculi, zygomaticus major and minor, and platysma). In the temporal region, this layer is represented by the superficial temporal (or temporoparietal) fascia.

FACIAL NERVE. Dissection in the subcutaneous plane, superficial to the SMAS/muscle layer, is safe in all areas of the face and forehead because the nerve branches innervate the respective facial muscles on their deep surface. Dissection deep to the SMAS, superficial to the facial nerve branches, requires care.

There are three exceptions to the rule just mentioned: the buccinator, mentalis, and levator anguli oris muscles are innervated via their superficial surface and lie deep to the facial nerve. These muscles, however, are not in danger during face lifts. It is essential to note that although the facial nerve branches are deep to the SMAS/muscle layer, at some point the nerve fibers turn superficially to innervate the facial muscles.

The frontal branch of the facial nerve consistently travels deep to the superficial temporal fascia. Any dissection in the sub-SMAS plane, whether as part

of a composite rhytidectomy or standard dissection of the SMAS as a separate layer, necessitates a change in surgical planes at the zygomaticus major muscle to avoid muscular denervation. Dissection begins in the sub-SMAS plane, but rather than continuing in that plane deep to the zygomaticus major, the dissection changes to a more superficial plane and passes over the zygomaticus muscle. In this manner the innervation to that muscle is preserved.

PAROTID-MASSETERIC FASCIA. The parotid-masseteric fascia is a filmy, areolar layer between the third layer of the face (the SMAS) and the fifth layer (the facial nerve branches). This thin, transparent layer lies immediately on the surface of the facial nerve branches anterior to the parotid gland. In the neck, this layer is similar in quality and is known as the superficial cervical fascia. If one elevates the platysma muscle, the marginal mandibular nerve can be seen through this thin, transparent layer. An analogous layer exists in the temporal region and scalp, where it is known as the innominate fascia or the subgaleal fascia. Just as it is convenient to think of the galea–frontalis–temporoparietal fascia–SMAS–orbicularis oculi–platysma layer as a single layer, it is convenient to think of the subgaleal fascia–innominate fascia–parotid–masseteric fascia–superficial cervical fascia as a single layer.

RETAINING LIGAMENTS. In at least two areas the layers are condensed and less mobile with respect to each other. These two "ligaments" are the zygomatic and mandibular ligaments. They are important for two reasons: first, they restrain the facial skin against gravitational changes at these two points and delineate structures such as the anterior border of the jowl. Second, the ligaments must be surgically released during a face lift to adequately redrape the tissue distal to these points.

PLATYSMA. The platysma muscle has a medial border that tends to become redundant with age and contribute to the appearance of bands in the submental region. The medial borders of the two platysma muscles decussate to a variable degree in the midline of the neck.

MALAR FAT PAD. The malar fat pad is superficial to the SMAS layer in the cheek. Other important cheek structures include the zygomaticus major and minor muscles. The malar fat pad appears to descend with age, and such descent leads to hollowing out of the infraorbital region and deepening of the nasolabial creases. Techniques have been described to mobilize the malar fat pad to restore volume to the upper part of the face. In the extended SMAS technique, the fat pad can be mobilized in continuity with the SMAS layer. The malar fat pad has also been elevated independently of both the SMAS layer and the skin to facilitate greater flexibility in redraping. In addition, the malar fat pad can be bluntly elevated with finger dissection (finger-assisted malar elevation [FAME]) deep to the orbicularis oculi such that it remains attached to the skin flap. With this technique, the fat pad can be repositioned during skin redraping.

GREAT AURICULAR NERVE. With the head turned 45 degrees to the contralateral side, the great auricular nerve consistently crosses the superficial surface of the sternocleidomastoid muscle approximately 6.5 cm below the caudal edge of the bony external auditory canal at a position just posterior to the external jugular vein. The vein and nerve are deep to the SMAS/platysma layer, except where the terminal branches of the great auricular nerve pass superficially to provide sensibility to the skin of the earlobe. Injury to the main trunk of the great auricular nerve as it passes over the sternocleidomastoid muscle may result in the development of a neuroma or permanent hypoesthesia on the lower portion of the ear.

BUCCAL FAT PAD. The buccal fat pad is deep to the buccal branches of the facial nerve, anterior to the masseter muscle, and superficial to the buccinator muscle. Access to the buccal fat pad can be achieved by performing sub-SMAS dissection and spreading between the buccal branches of the facial nerve in the cheek or, through the mouth, by a stab wound in the buccinator muscle. Despite occasional indications to remove the fat pad, removal of cheek fat tends to ultimately make the patient look older. As a general rule, rejuvenation of the face involves redistribution, not removal, of fat.

FOREHEAD. The frontalis muscles are vertically oriented extensions of the galea that begin at the level of the anterior hairline, extend inferiorly, and insert into the dermis of the forehead skin. Transverse forehead lines result from chronic frontalis contraction. Innervation is provided by the frontal branches of the facial nerve entering the muscle on its deep surface, and loss of frontalis innervation always results in brow ptosis on the affected side.

There is never a single frontal branch (temporal branch) of the facial nerve, but rather multiple branches (between two and five) that pass over the zygomatic arch.

The corrugator muscles arise from the periosteum along the superomedial orbital rim and insert into the dermis of the medial aspect of the eyebrow. Contraction pulls the brow medially and downward, thus producing a scowling appearance, and results in vertical glabellar creases. The procerus muscles originate from the surface of the upper lateral cartilage and nasal bones and insert into the skin in the glabellar region. Contraction pulls the forehead down and the root of the nasal skin up, thereby causing transverse wrinkles at the root of the nose.

Sensory innervation to the forehead is via the supraorbital nerve, the supratrochlear nerve, and the infratrochlear nerve, all branches arising from the first division of the trigeminal nerve (V1). If the corrugator muscle is approached through an upper eyelid incision, the supratrochlear branches will be encountered first when approaching the muscle from its superficial surface. However, when performing a coronal lift and approaching the corrugator muscle from its deep surface, the supraorbital nerve is encountered first and the supratrochlear branches are visualized only after the corrugator muscle is resected.

The frontal branches of the facial nerve emerge from beneath the parotid gland on a line extending from 0.5 cm below the tragus of the ear to a point 1.5 cm above the lateral aspect of the brow and pass deep to the SMAS over the zygomatic arch.

Treatment

Face Lift

The incisions used for a classic face lift are illustrated in Figure 2. Approximately 10 minutes before the first incision, half the face (and submental area if a submental approach is planned) should be injected for both hemostasis and hydrodissection. Injection of 40 to 60 mL of 0.3% lidocaine with 1:300,000 epinephrine allows sufficient volume while maintaining a dose within the limit of safety.

Depending on the position of the patient's hairline, sideburn, and history of previous surgery, the incision is initiated either a short distance above the ear in the temporal aspect of the scalp or along the anterior temporal hairline. In patients with previous face lifts, the sideburn may already be significantly raised and posteriorly displaced, and the final skin excision associated with the temporal scalp incision will move the sideburn posteriorly and superiorly. In such situations, an anterior hairline incision should be considered. These patients (i.e., those undergoing secondary lifts) are usually in the seventh decade of life, and their thinner skin generally heals without noticeable scars, thus making an anterior hairline incision acceptable. In both cases, the horizontal incision at the new sideburn level will set its height.

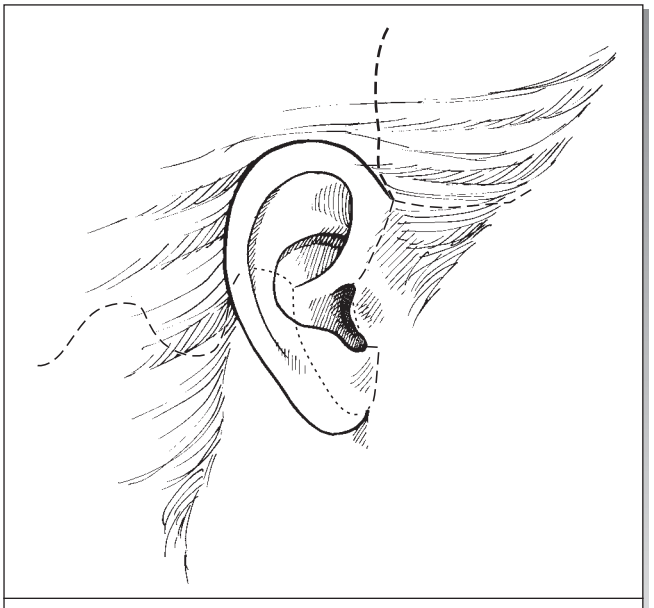

Figure 2 • Incisions for a face lift. The incision allows preservation of the sideburn and temporal hairline. The downward direction at the posterior hairline helps to avoid a "step off" deformity.

The incision proceeds caudally along the junction of the ascending helix of the ear and the cheek. It should be kept on the nonvelous skin of the ear because this scar is prone to anterior migration. The authors place the incision identically in men and women, but some surgeons prefer to make it slightly farther from the ear in men because it will be hidden in the beard line. The incision is made either at the posterior margin of the tragus or in the pretragal region, usually in a natural skin crease. A posttragal incision is more difficult and requires redraping the tragal skin with significant excess to prevent distortion of the tragus.

The incision passes beneath the earlobe and extends into the retroauricular sulcus. The retroauricular incision is placed slightly up on the ear because it is also prone to migration and is best hidden if the final scar rests in the depth of the sulcus. The incision traverses the hairless skin in the retroauricular region at a point sufficiently high to be invisible if the patient were to have short hair or be wearing hair in a ponytail. The incision extends along the hairline for a short distance and passes back into the occipital scalp in the form of either an "S" or an inverted "V" (see Fig. 2).

Regardless of the technique chosen for face lifting, the incisions and the final draping are critical. They are designed so that on completion of the procedure, the incisions will be virtually invisible and will not distort the hairline, ear, cheek, retroauricular sulcus, or posterior hairline. If the incision is performed properly and the patient experiences uncomplicated wound healing, it should be difficult even for the surgeon or the hairdresser to find the incisions.

SUBMENTAL DISSECTION AND UNDERMINING. In patients with abundant excess skin in the submental region, a submental incision is made and subcutaneous undermining performed. In younger patients with early aging changes, the submental incision is omitted and undermining is performed via the lateral incisions alone. A decision is made regarding the submental crease. In patients in whom the crease is not abnormally deep, an incision is made just caudal to the crease. Dissection is

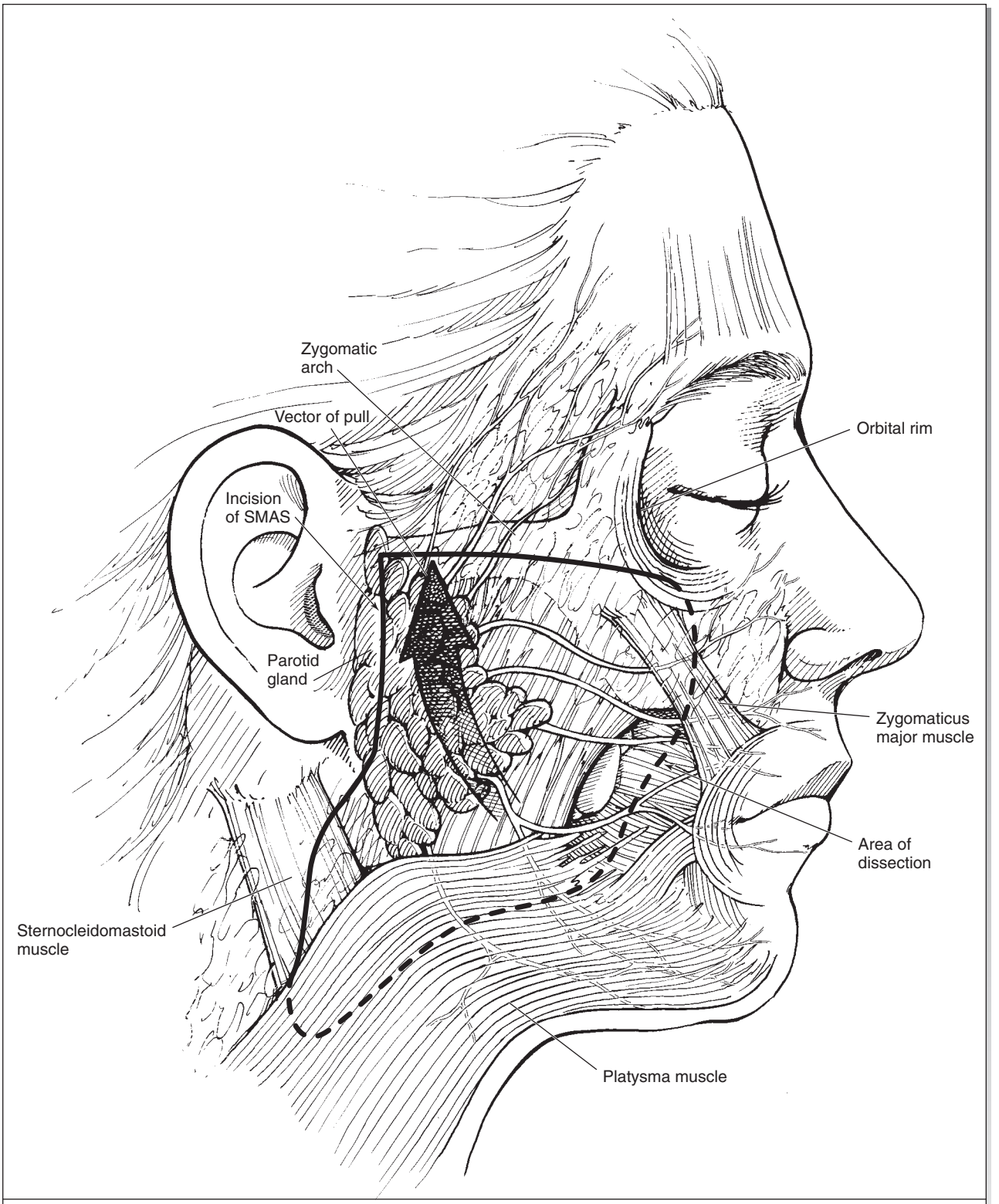

Figure 3 • Dissection of the superficial musculoaponeurotic system (SMAS). *Solid lines* are incisions, and *dashed lines* are the limit of the SMAS dissection. The *arrow* is the direction of pull on the SMAS.

performed retrograde to undermine the crease. In patients in whom the crease is deep, the crease is completely excised.

Extensive subcutaneous undermining is performed in the submental region. The platysma is defatted under direct vision and the anatomy of the medial borders of the platysma identified. Subplatysmal fat is trimmed if necessary. Midline plication of the platysma muscles is performed with numerous buried absorbable sutures. A wedge of platysma is excised bilaterally caudal to the plication to break the continuity of the platysmal bands.

UNDERMINING THE FACE. The cheek flap is developed, followed by the temporal and retroauricular flaps. Subcutaneous dissection is continued with the fiberoptic retractor superficial to the orbicularis oculi muscle. In the lower part of the face, the mandibular ligament is released and dissection is performed in continuity with the previous submental dissection. Two-finger countertension, provided by the assistant, allows excellent subcutaneous visualization of the dissection.

HIGH, EXTENDED SMAS/PLATYSMA FLAP. A transverse incision is made in the SMAS at approximately the level of the infraorbital rim, above the zygomatic arch (see Fig. 3). An intersecting vertical incision is made in the SMAS in the preauricular region and extended along the anterior border of the sternocleidomastoid muscle well below the angle of the mandible. The SMAS and platysma muscle are elevated in continuity. The SMAS is always elevated beyond the anterior border of the parotid gland. If less dissection is performed, insufficient release of the SMAS occurs. Dissection is continued to expose the zygomaticus major muscle if maximal mobilization is desired. The SMAS is rotated in a mostly cephalad direction with a slight posterior vector and secured to the temporal fascia. A second set of sutures is placed low in the neck from the platysma muscle to the mastoid periosteum. The posterior border of the cheek SMAS is incised to allow a tongue of excess SMAS to be transferred behind the ear to reinforce tightening of the platysma below the angle of the mandible. This tongue of SMAS is sutured to the periosteum over the mastoid process. Additional sutures are placed along the entire suture line to reinforce this maneuver. If the SMAS has been fully elevated and rotation is in the correct orientation, an aesthetically pleasing malar augmentation results without any need for alloplastic material. This improved appearance is due to the "dog-ear" of SMAS at the orbital rim; the more rotation, the more "dog-ear."

CERVICAL DEFATTING. After the SMAS/platysma has been rotated, the platysma is defatted beneath the inferior border of the mandible. Such defatting should be performed judiciously because excessive skeletonization of the neck results in an unpleasant "Popsicle on a stick" appearance.

HEMOSTASIS. Hemostasis is obtained by using a fiberoptic retractor and electrocautery with insulated forceps to avoid damage to the skin edge.

REDRAPING. The skin is redraped in a vector that is slightly less vertical than the SMAS elevation. In the face the vector is oblique, and in the neck, it is closer to transverse. It is important to avoid redraping the transverse neck creases up onto the face. Tension of the entire procedure is borne by two sutures, one at the top of the ear anteriorly and one at the apex of the postauricular incision. The remaining skin is trimmed so that there is no gap between the skin edges and therefore no tension on the closure. Beneath the earlobe, the skin is trimmed so that the incision is tacked up beneath the earlobe to exaggerate the separation of the earlobe from the cheek. It is important to avoid excising too much skin around the earlobe, which invariably leads to a "pixie ear deformity." In the postauricular region, an incision is made along the posterior hairline, and the hairline is approximated precisely so that there is no step off.

In patients in whom an anterior hairline incision is made, excess skin is removed along this incision, again making sure that there is no tension on the closure. In patients in whom a short temporal incision has been made within the hairline, a transverse wedge is always removed below the sideburn so that this structure is not elevated above the apex of the ear.

TRAGUS. In patients in whom an incision is made in the pretragal region, the facial skin is trimmed so that there is no gap between the skin edges. In patients in whom an incision has been made at the posterior border of the tragus, a tragal flap of skin is created that is larger in all dimensions than necessary to cover the tragus. This technique allows the inevitable contraction of the skin to occur without deformation of the tragus. The area directly in front of the tragus and the skin flap directly over the cartilage should be defatted.

DRAINS. Closed-suction drains are placed in the neck bilaterally and brought out through separate stab wounds in the hair-bearing scalp.

CLOSURE. Closure is performed with buried monofilament absorbable sutures in several key locations: the aforementioned root of the helix, the sideburn area, and the anterior hairline. A suture is also placed at the base of the earlobe such that it purchases the skin of the flap, the earlobe, and the perichondrium of the ear. This maneuver decreases the risk of a pixie deformity and provides a natural-appearing earlobe. Another suture is placed in the same manner at the postauricular skin closure to

ensure that the resulting scar does not migrate past the retroauricular sulcus. The anterior skin closure is generally performed with fine nylon, the postauricular incision is closed with plain gut, and incisions in the hair are closed with staples.

Short-Scar Technique

The short-scar technique has become a popular alternative. It is useful in younger patients with minimal excess neck skin. The incision is not extended beyond the earlobe, thereby completely avoiding a posterior incision into the hairline. This technique relies on more vertical redraping of the skin. The anterior hairline incision may need to be extended to redrape the skin. The vertical vector is transmitted across the mandibular border and eliminates a certain amount of submental excess. Skin and SMAS undermining and elevation are identical. Bunching of skin behind the earlobe often occurs but improves with time, although revision is occasionally necessary at this location.

Forehead/Brow Lift

The classic coronal forehead/brow lift is no longer the gold standard for brow elevation. It has the disadvantage of a longer scar, frequent itching postoperatively, and areas of alopecia if skin excision is used as the method of brow fixation. Exposure to the brow musculature is not better than that achieved endoscopically. Newer techniques allow the same degree of elevation without a long scalp incision.

ENDOSCOPIC BROW LIFT. The endoscopic approach to forehead/brow lifting has become popular in recent years. The main advantages of the endoscopic approach are a shorter operative procedure, a decrease in itching, and shorter incisions to camouflage. Release of the ligaments necessary to allow brow elevation can be performed with this access, and fixation is as effective as any other form of brow lifting.

LIMITED INCISION BROW LIFT. A limited incision, lateral brow lift can be performed without an endoscope. The incision is made lateral to the temporal line of fusion to avoid injury to the deep branch of the supraorbital nerve. Fixation relies on suturing the galea. This technique results in a roll of excess skin in the temporal region that gradually dissipates over a period of a few weeks.

INTERNAL BROWPEXY. An alternative to the aforementioned procedures is direct browpexy performed through the upper blepharoplasty incision. Dissection is performed under the orbicularis, and the brow tissue is sutured directly to the periosteum over the supraorbital ridge at the appropriate level. This approach is minimally invasive and may be ideal for men and women with thinning hair. Its long-term results are not known.

FIXATION. Fixation methods have undergone an evolution. The two most successful means of fixation at this time appear to be a cortical tunnel technique and an absorbable fixation device. In the cortical tunnel technique, a hole is drilled through the outer table of the skull through vertical access incisions at four points behind the hairline. Sutures are placed to fix the advanced forehead/brow.

Various absorbable devices have been described that allow fixation of the brow in a more superior position from behind the hairline. Most surgeons perform the fixation at only two points if these devices are used. The dissolvable clips offer several advantages. First, they are easy and quick to place. Second, the absorbable clip devices offer the surgeon the flexibility to adjust the position of the lift intraoperatively, immediately postoperatively, or within 48 hours postoperatively in the office.

TECHNIQUE. Having performed all the brow lifting techniques described in this chapter, the authors currently use a modified limited incision, lateral approach technique. A zigzag incision is made 2 cm posterior to the hairline in the temporal region. The incision does not extend medial to the temporal fusion line so that it will not damage the deep branch of the supraorbital nerve. The dissection is performed with endoscopic assistance or under direct vision with a fiberoptic retractor. The lateral orbital ligaments are released, and the periosteum is incised and spread under the lateral aspect of the eyebrow. The forehead is advanced along the desired vector, and fixation is accomplished with an absorbable fixation device.

Postoperative Care

Ice compresses can be used in the initial postoperative period. Patients are advised to remain still and quiet for 3 days. Drains and dressings are usually removed on postoperative day 1. Oral antibiotics and pain medication are prescribed. The preauricular nylon sutures are removed on postoperative day 3 to 5, and the postauricular sutures and staples are removed on postoperative day 7 or 8.

Face Lift Complications

Hematomas

Hematomas are the most frequent complication after face lifts (0.9% to 8.0% incidence) and vary from large collections of blood that threaten skin flap survival to small collections that are obvious

only when the facial edema has subsided. The incidence of hematomas in men (8.7%) is more than twice that in women, but this difference seems to be decreasing with strict blood pressure control. Most major hematomas occur during the first 10 to 12 hours postoperatively. The cause of hematomas is multifactorial, but they correlate most closely with perioperative blood pressure control. Antiplatelet medication also increases the risk for postoperative hematomas, and the use of these medications is therefore discontinued 2 weeks before the surgical procedure.

The most common manifestation of a hematoma is pain localized to one side of the face or neck in an apprehensive, restless patient. Because pain is unusual after an uncomplicated face lift, it must be regarded as a sign of a hematoma until proved otherwise. Rather than provide analgesics for pain relief, the dressing is removed immediately to permit examination.

Treatment of expanding hematomas is always surgical. Historically, the flaps were elevated and the bleeding point was sought. However, rarely was a distinct bleeding vessel found. Alternatively, the patient is given blood pressure control and anxiolytic medications to decrease systolic pressure below 110. Limited sutures or staples are removed from the sideburn, retroauricular, or posterior hair incision (whichever is closest to the largest aspect of the collection). Catheters are inserted and the hematoma evacuated. The area is irrigated first with saline until clear and then with a 0.25% lidocaine, 1:400,000 epinephrine solution. Gentle pressure is placed on the flap for 20 minutes. This regimen controls most hematomas without any need for surgical intervention. If bleeding persists, the flaps should be elevated and pinpoint hemostasis achieved in the operating room.

Small hematomas that are not apparent until the edema begins to subside may be treated with a No. 11 blade placed tangentially through the skin to allow drainage and satisfactory healing. Hematomas that are not detected and evacuated during the period of liquefaction result in skin firmness, irregularity, and hemosiderin discoloration. Warm compresses, gentle daily massage, and intralesional injection of dilute steroids are occasionally helpful.

Skin Slough

Full-thickness skin slough always results in some degree of permanent scarring. The postauricular and mastoid areas are most frequently involved because of thin skin in an area farthest from adequate circulation, but the resulting scars may be concealed by the ear and hair. Skin slough (incidence of 1% to 3%) is caused mostly by (1) undiagnosed hematomas, (2) a skin flap that is too thin or damaged during flap dissection, (3) excessive tension on wound closure, and (4) cigarette smoking (12-fold increase in relative risk). Patients are required to abstain from smoking for 3 weeks preoperatively and 2 weeks postoperatively.

The areas of skin slough epithelialize and contract dramatically. The resulting scar is always better than would be anticipated from the initial wound appearance. Depending on the size of the necrotic area, it may be possible to excise the scar and readvance the facial skin, but it generally takes several years before sufficient skin laxity allows for a secondary lift.

Nerve Injury

Nerve injury in subcutaneous facialplasty is rare (0.9%), but occurs more frequently with extended SMAS and composite procedures. Most nerve injuries recover within a few weeks and probably represent cautery insults. A nerve that has been cut will not recover, although injuries in the buccal area most often improve as a result of cross innervation. Intraoperative transection of the facial nerve necessitates immediate microsurgical repair. It is more likely, however, that nerve injury is not recognized during surgery, and the surgeon and patient are placed in the difficult position of waiting for return of function.

Transient numbness of the lower two thirds of the ear, the preauricular area, and the cheeks for the first 2 to 6 weeks postoperatively occurs as a result of interruption of small sensory nerves during surgery and is unavoidable. The most common avoidable nerve injury during facialplasty is that to the great auricular nerve. This nerve is injured when dissection is too deep and the fascia over the sternocleidomastoid muscle is pierced. When injury to the great auricular nerve is recognized during surgery, immediate, meticulous repair is performed.

Scarring

Scarring after facialplasty is most commonly caused by vascular compromise of the skin flaps and excess closure tension. Two points of maximum skin tension are established: in the temporal aspect of the scalp just above the ear and at the apex of the postauricular incision. Conservative trimming is especially required in the preauricular area and around the earlobe, where the slightest tension widens the scar and distorts the earlobe.

Hypertrophic Scarring

Such scarring is rare and most commonly occurs at the postauricular incision. Small-volume injections of dilute insoluble steroid often help to flatten the scars.

Pearls and Pitfalls

- It is important to develop a relationship with the patient preoperatively. Patients who are

unrealistic or unstable should be refused treatment.

- Understand the anatomy, with particular attention to the facial nerve, prior to attempting extended SMAS procedures.

SUGGESTED READINGS

Baker D, Conley J: Avoiding facial nerve injuries in rhytidectomy: Anatomic variations and pitfalls. Plast Reconstr Surg 64:781, 1979.

Baker DC: Complications of cervicofacial rhytidectomy. Clin Plast Surg 10:543, 1983.

Baker DL, Aston SJ, Guy CL, Rees TD: The male rhytidectomy. Plast Reconstr Surg 4:514, 1977.

Ellenbogen R: Transcoronal eyebrow lift with concomitant upper blepharoplasty. Plast Reconstr Surg 71:490, 1983.

Furnas D: The retaining ligaments of the cheek. Plast Reconstr Surg 83:11, 1989.

Gosain A, Yousif NJ, Madiedo G, et al: Surgical anatomy of the SMAS: A reinvestigation. Plast Reconstr Surg 92:1254, discussion 1264, 1993.

Mitz V, Peyronie M: The superficial musculoaponeurotic system (SMAS) in the parotid and cheek area. Plast Reconstr Surg 58:80, 1976.

Rees TD, Aston SJ: Complications of rhytidectomy. Clin Plast Surg 5:1, 1978.

Stuzin J, Wagstrom L, Kawamoto HK, Wolfe SA: Anatomy of the frontal branch of the facial nerve: The significance of the temporal fat pad. Plast Reconstr Surg 83:265, 1989.

Blepharoplasty

PIERRE B. SAADEH
JOSEPH G. MCCARTHY

The concept that one is revealed though one's eyes is an assumption so innate that it not usually consciously recognized. The globe, however, is expressionless, and it is the function of the surrounding soft tissue or periorbital complex to convey emotion and well-being. The aging and pathologic processes that result in changes in soft tissue may indicate the need for restorative surgery, specifically, blepharoplasty (Greek: *blepharo*, or "eyelid," and *plasty*, to "form" or "mold").

Etiopathogenesis

Excessive wrinkling of the eyelid skin (*dermatochalasis*) is due to aging changes and sun damage. In contrast, *blepharochalasis* is a familial condition that leads to skin excess as a result of recurrent episodes of swelling of the periorbital tissues. Evidence of a weakened orbital septum manifests as fat herniation. Severe eyelid hooding or levator ptosis, or both, may prompt a patient to complain of visual field defects. Attenuation of tarsal support may accompany other age-related changes and leads to senile ectropion. Graves' disease, diabetes mellitus, scleroderma, hyperlipidemia, myasthenia gravis, Horner's syndrome, and dermatomyositis are conditions that can also be associated with periorbital soft tissue pathology.

Pathologic Anatomy

With aging, a variety of soft tissue changes combine to affect the aesthetics of the periorbital soft tissue envelope: progressive loss of elasticity of the skin, tarsus, orbicularis muscle, and orbital septum. The periorbital fat becomes redundant and may bulge through an attenuated septum.

The upper eyelid crease tends to become less distinct and more elevated. The upper eyelid and periorbita lose a sense of volume. The eyebrows descend more laterally than medially.

Lower eyelid age-related changes include descent of the infratarsal concavity, orbital septal attenuation, fat herniation, and descent of the cheek (zygomatic fat pad) mass. Diminution of lower lid support and the development of scleral show and ectropion are usually signs of advanced aging changes (Fig. 1).

Diagnostic Studies

Preoperative evaluation should include a detailed medical and ocular history and documentation of ocular and periocular anatomy.

The history should focus on the following:

- Subjective assessment of vision
- Presence of dry eye symptoms
- Use of corrective lenses/contacts
- Presence of cataracts, glaucoma, hypertension, endocrinopathy, or facial nerve disorders
- Bleeding history
- Use of anticoagulant medication (warfarin [Coumadin], nonsteroidal antiinflammatory drugs, aspirin)

The presence of a significant ophthalmic history or ocular pathology is an indication for ophthalmologic consultation.

Physical examination includes a general assessment of skin quality (sun damage, elastosis), fat excess, extraocular muscle function, visual acuity, Bell's phenomenon and identification of a "negative vector" in which the globe projects anterior to the inferior orbital rim and malar eminence.

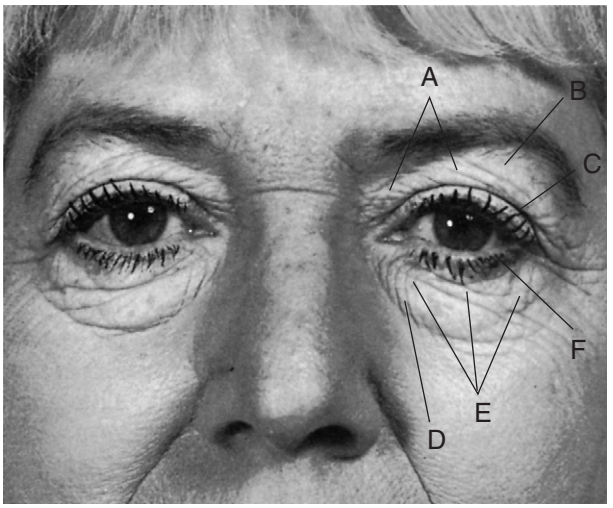

Figure 1 • Periocular age-related changes in the upper lid. **A,** fat herniation and septal attenuation; **B,** hooding with brow ptosis; **C,** asymmetry (elevation of the tarsal crease may also occur). Lower lid: **D,** tear trough; **E,** fat herniation and septal attenuation; **F,** lower lid laxity. (From McCarthy JG: Plastic Surgery. Philadelphia, WB Saunders, 1990.)

Upper lid evaluation identifies skin and fat excess, asymmetry, ptosis, pseudoptosis (brow ptosis), levator dysfunction, lid retraction, and corrugator/procerus muscle hyperactivity. Evaluation specific to the lower lid should be directed at identification of scleral show, ectropion and entropion, and lid position, tone, and support. The snap back test may be a helpful adjunct in the evaluation of lower lid support.

Adjunctive evaluation includes Schirmer testing and tear breakup time. Preoperative evaluation should also assess the patient's general emotional and psychological state. Realistic postoperative expectations are fundamental to a successful outcome.

Professional photographs, including upward and downward gaze, eyes open, eyes closed, squinting, and up-close lateral views, are useful both in the preoperative surgical dialogue with the patient and in the operating room as reference source.

Reconstructive Goals

The primary surgical goals are to enhance periorbital aesthetics and preserve or restore periorbital symmetry and function. Goals of upper lid blepharoplasty include restoration of symmetry, pretarsal show, and well-defined tarsal folds. Lower lid blepharoplasty involves the restoration of a smooth, soft lid, a slightly elevated canthal tilt, and sharp canthal angles while maintaining or supplementing lid support.

Treatment

Anesthesia

The authors' practice is to perform the procedure under continuous sedation with routine monitoring of vital signs. A benzodiazepine sedative is adminis-

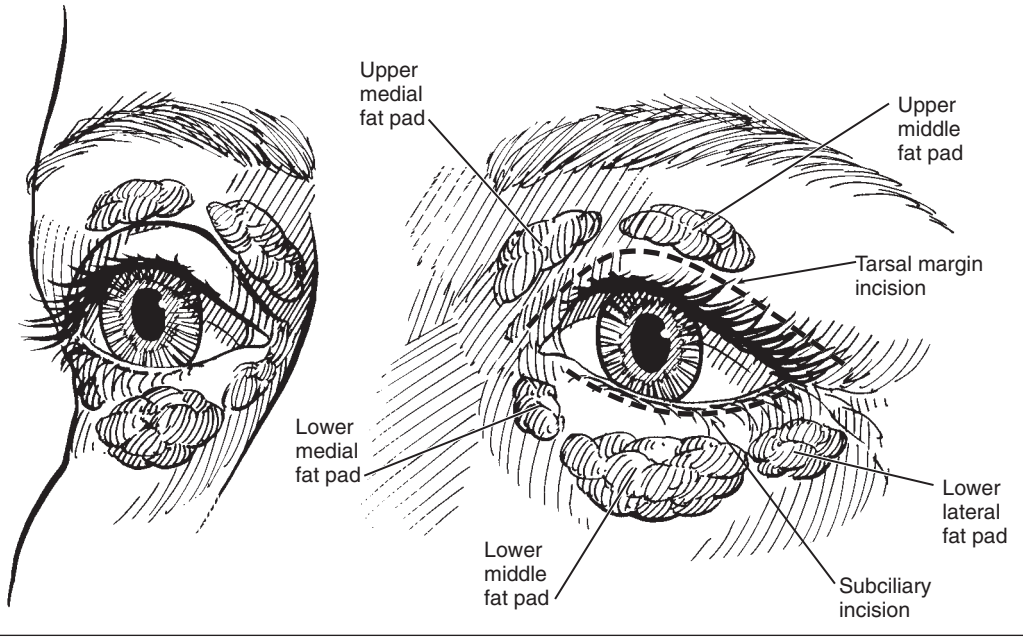

Figure 2 • Location of fat pads of the upper and lower lid incisions.

tered intravenously, and 0.5% lidocaine with 1:200,000 epinephrine is injected tangential to the lid (and globe) to facilitate dissection.

Upper Lid Blepharoplasty

MARKINGS. With the patient in the reverse Trendelenburg position, the supratarsal crease is identified and marked (see Fig. 2). The crease, which results from and is delimited inferiorly by the levator extension into the skin, is located 9 to 12 mm above the ciliary margin and lies near the upper edge of the tarsus (accentuated by retracting and moving the lid in an inferior direction). The lower line curves toward the lid margin and should not extend into the medial canthus. The mark should be limited laterally to a line perpendicular to the lateral orbital rim in younger patients, whereas in older patients, it is usually extended in one of the crow's feet lines.

The superior line of the planned excisional ellipse is determined by the amount of skin overhang present when forceps, applied at several points, gathers skin from the lower line until the lashes begin to rotate upward. Skin excision is maximized laterally but minimized medially.

TECHNIQUE. The skin is removed and a strip of orbicularis is also excised with iris scissors. The orbital septum is opened over the central fat pad with a stab incision made with the sharp scissors (Figs. 3 and 4; see also Fig. 2). A high incision helps to avoid the septum-levator fusion plane. Gentle globe counterpressure is applied and fat teased out with Adson forceps or a curved hemostat. While avoiding overresection, the fat is clamped around its base and amputated with electrocautery.

Another scissors stab incision is then made over the medial fat compartment in an identical fashion, and the fat pad is identified by its whiter appearance. Underresection medially is a common mistake. There is no lateral fat pad in the upper lid; this space is occupied by the lacrimal gland.

Lateral to the canthus, the skin is closed with 6–0 nylon interrupted sutures. The skin margins are approximated medially with a running 5–0 nylon intradermal suture reinforced with skin tapes.

ADJUNCTIVE PROCEDURES—UPPER LID

Excessive Lateral Brow Fullness. Many blepharoplasty candidates have excessive fullness of the lateral aspect of the brow. Causes include lateral brow ptosis, retroorbicular fat excess, an overhanging/prominent supraorbital rim, a prominent lacrimal gland, or a combination of these findings.

Brow ptosis, especially along the lateral aspect, is a normal, expected age-related change. Optimal brow position is determined preoperatively with the patient in the upright position. The extent of lateral upper lid skin resection is adjusted depending on the degree of planned lateral brow elevation. Through the upper lid incision, dissection is carried superolaterally over the orbital septum and toward the orbital rim lateral to the supraorbital notch. A suborbicularis plane is developed superficial to the periosteum to the desired brow position. With the lateral aspect of the brow in an appropriately elevated position, 4–0 Vicryl sutures secure the periosteum to the orbicularis and overlying soft tissue while avoiding the superficial dermis. Care is taken to maintain symmetry and avoid both undercorrection and overcorrection.

Retroorbicularis orbital fat located superficial to the septum has been identified as a potential cause of excess lateral brow fullness and can be trimmed at the time of upper lid blepharoplasty. Conservative resection is emphasized to prevent inadequate soft tissue coverage of the underlying bony brow.

A *prominent or projecting supraorbital rim* (bony excess) can cause lateral brow fullness. Through the upper lid incision, correction is achieved by scoring and elevating the periosteum over the superolateral orbital rim. Tangential ostectomy with a 5-mm osteotome permits careful removal of a thin piece of bone, with care taken to avoid a bony step off. The periosteum should be reapproximated.

The *lacrimal gland,* which takes the place of a lateral fat compartment in the upper lid, accounts for approximately 10% to 15% of all cases of excessive lateral brow fullness. Appropriate management is resuspension of the gland to the orbital rim with absorbable suture material. Excision of the gland should be discouraged because of the potential for keratoconjunctivitis sicca.

Glabellar Furrows. The upper lid blepharoplasty approach affords ample access to the corrugator supercilii and the associated musculature responsible for glabellar furrows, all of which can be approached via a suborbicularis plane provided by the upper lid incision. The vertical head of the orbicularis, the depressor supercilii, and the corrugator are exposed and individually resected. The procerus can also be excised, if indicated. The effectiveness of surgical treatment of glabellar furrowing is limited if the muscles are not completely excised. Injection of botulinum toxin (Botox) in this area, albeit temporary in effect, provides an alternative treatment to corrugator resection.

Inadequate Supratarsal Fold. In the absence of a supratarsal fold, as in Asian eyelids, the fold is recreated by suture fixation of the pretarsal orbicularis to the dermal edge of the incised skin. It is vital to ensure side-to-side symmetry when creating the supratarsal fold.

Lower Lid Blepharoplasty

Lower lid blepharoplasty can be broadly divided into transcutaneous and transconjunctival procedures.

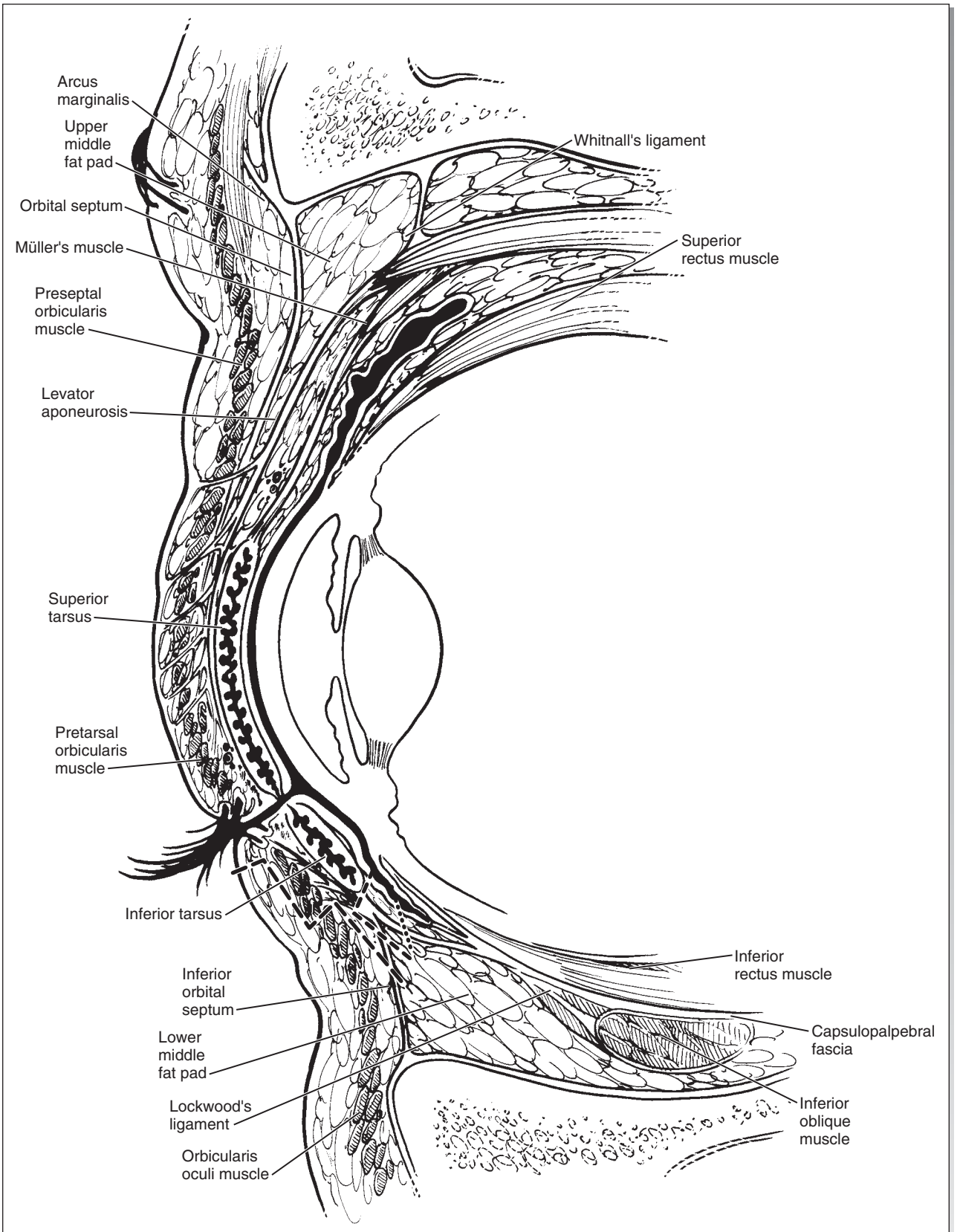

Figure 3 • Cross-sectional anatomy of the midorbit. (See Fig. 4 for details of the lower eyelid incisions.)

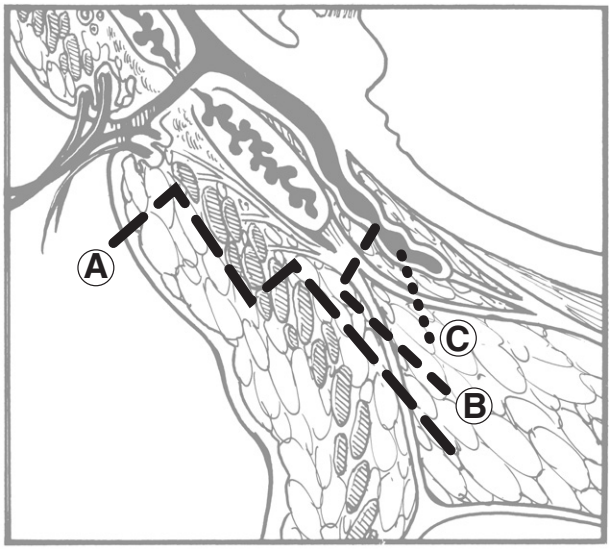

Figure 4 • Surgical approaches to the lower lid fat pads. **A,** Subciliary skin-muscle flap (a step incision leaving the pretarsal orbicularis oculi intact is shown). **B,** Transconjunctival, preseptal. **C,** Transconjunctival, retroseptal.

Although both give adequate access to the lower lid fat pads, transcutaneous approaches also provide definitive surgical solutions for skin excess and orbicularis hypertrophy. However, transcutaneous procedures have a greater, albeit small, potential for ectropion. When the pathology is mostly limited to fat herniation or the patient is more likely to experience lid retraction and its attendant complications, a transconjunctival approach is preferred. It should be noted that numerous skin resurfacing techniques can be used to rejuvenate mildly wrinkled skin in appropriately selected individuals.

TRANSCUTANEOUS BLEPHAROPLASTY

Markings. The lower lid tarsal crease, which is analogous to the upper lid crease, is identified. A gently curving subciliary line is marked 2 mm below the lid margin and is limited medially by the medial canthus. The line is limited, particularly in younger, less wrinkled patients, to the lateral orbital rim. In simultaneous upper and lower blepharoplasty, the planned incisions should be separated laterally by an area of at least 9 mm of skin in the lateral canthal area to avoid vertical scar contracture (see Figs. 2 and 4).

Technique. A full-thickness incision is made along the marked line, and care is taken to avoid damaging the pretarsal orbicularis because this muscle, in conjunction with the underlying tarsus, is important for lower lid support. It is noteworthy to mention that in severe cases of extensive and discrepant skin excess (relative to the orbicularis muscle), a skin-only flap may be raised, redraped, and carefully trimmed as needed to allow for tension-free closure. However, this technique may be associated with a higher rate of lid retraction. Scissors dissection is carried through the infratarsal orbicularis, and a preseptal skin-muscle flap is developed as far as the inferior orbital rim.

Gentle globe pressure aids in identification of the lower lid fat pads. Stab incisions through the septum and removal of the individual fat pads are performed in a fashion identical to that described for the upper lid, with the main difference being the need for resection of the lateral fat pad. Care is taken to avoid injury to the inferior oblique muscle, which separates the medial and central fat pads. Conservative resection will avoid a postoperative "hollowed out" appearance.

The skin-muscle flap is redraped in a superior direction, and excess tissue is excised while allowing for tension-free skin closure. Interrupted 6–0 nylon sutures are used to reapproximate the skin margins.

TRANSCONJUNCTIVAL BLEPHAROPLASTY

Technique. The lower lid is retracted with a Desmarres retractor. The level of the conjunctival incision defines the approach to the fat pads (see Fig. 4). A high incision (3 mm below the lid margin) divides the capsulopalpebral fascia/septal fusion plane and allows for a preseptal approach to the fat pads. Alternatively, a lower incision (2 to 3 mm above the fornix) is chosen to allow direct access to the fat pads in an entirely retroseptal approach. Fat is excised as described previously while avoiding injury to the inferior oblique muscle. Adjustments in skin tension are not possible with this technique.

ADJUNCTIVE PROCEDURES—LOWER LID

Skin Resurfacing. As indicated earlier, skin resurfacing is an alternative to skin excision after transconjunctival blepharoplasty. Resurfacing may be accomplished with lasers (CO_2, yttrium-aluminum-garnet [YAG], or erbium) or chemical peels (trichloroacetic acid [TCA] or phenol/croton oil). Patients are pretreated with hydroquinone and acyclovir to prevent hyperpigmentation and herpes zoster, respectively. These procedures are restricted to fair-skinned individuals (Fitzpatrick grade I to III). Although less invasive by nature, these procedures do not eliminate the possible development of lid retraction, have the potential for prolonged erythema of the lids, and are contraindicated in patients with advanced aging changes.

Canthopexy/Canthoplasty. Unaddressed lid laxity predisposes a patient to lid retraction, exacerbation of dry eye symptoms, scleral show, and corneal abrasion. Numerous procedures have been recommended for correction of lid laxity. Lateral canthopexy can help to restore tarsal height and lower lid support. However, lid laxity is frequently accompanied by horizontal excess and a weak lateral canthal mechanism, findings that require more complicated pro-

cedures, including lid shortening and even tarsal spacer grafts.

Festoons. Festoons, which represent orbicularis laxity in the lower eyelids, may be improved by a combination of partial orbicularis excision and resuspension of unresected orbicularis to the lateral orbital rim. The techniques are fraught with complications.

Tear Trough. A multitude of specialized procedures, including sliding retroseptal fat over the arcus marginalis, suborbicular fat repositioning, and fat grafting, have been described to correct the "tear trough" deformity, which consists of a deep nasojugal groove and/or lower eyelid hollowing (see Fig. 1). Alternatively, midface descent, which accentuates the tear trough, may be addressed by a variety of midface lift procedures that elevate the malar fat and cheek soft tissues and also augment the infraorbital area. The latter procedures are more effective in correcting a postoperative tear trough deformity.

Postoperative Care/Complications

Postoperative care includes head elevation, application of cold compresses, mild analgesics, blood pressure control, and early suture removal.

Although serious complications of blepharoplasty are rare, the following problems have been reported:

Visual Loss

Visual loss is reported to occur after 0.04% of blepharoplasty procedures. It is usually heralded by the rapid onset of orbital pain, proptosis, eyelid ecchymosis, and reduced visual acuity (normal to no light perception). The pathophysiology commences with intraorbital hemorrhage, followed by elevated intraorbital and intraocular pressure and, secondarily, compromise of the ocular circulation.

Hemorrhage is a surgical emergency requiring prompt recognition and management. Initial treatment includes opening the incisions and medically reducing intraocular pressure with intravenous mannitol and acetazolamide (Diamox). Surgical exploration to identify the site of bleeding is performed, although a source is not usually found. Release of the orbital septum and detachment of the lateral canthus can provide additional orbital decompression. Emergency ophthalmologic consultation should be obtained. Anterior chamber paracentesis and orbital decompression are controversial surgical adjuncts.

Corneal Injury

The liberal intraoperative use of drops and lubricants can help to prevent corneal injury. Suspected injury can be assessed with fluorescein staining. Large or deep wounds should be referred to an ophthalmologist, whereas superficial injuries are managed with topical antibiotics and eyelid patching.

Hematoma

Blepharoplasty requires meticulous hemostasis. Causes of hematoma include bleeding fat pad vessels that retract into the orbit and bleeding from the cut margins of the orbicularis. It is imperative to differentiate localized hematoma from a sight-threatening retrobulbar hematoma associated with a firm globe on palpation. The latter dictates therapeutic measures as discussed earlier under "Visual Loss." Superficial eyelid hematomas are located anterior to the septum (without ocular symptoms) and require only observation; needle aspiration is performed, if necessary, after liquefaction.

Diplopia

Diplopia may occur postoperatively as a result of edema, hematoma, or injury to the extraocular musculature. The inferior oblique is the muscle injured most frequently (especially after transconjunctival incisions), followed by the superior oblique muscle. Ophthalmologic consultation is indicated. Permanent strabismus from structural damage to the extraocular muscles or nerves requires referral to a strabismologist.

Ectropion

Overresection of skin or normal scar contracture of the surgical site, particularly in predisposed patients (older patients with lid laxity, negative vector), may lead to ectropion. Patient selection and adjunctive procedures performed at the time of blepharoplasty (i.e., lateral canthoplasty and horizontal lid shortening) can reduce the incidence of ectropion. These procedures can also be performed, after an appropriate time, if ectropion develops after surgery. In most cases, postoperative ectropion can be treated with expectant management. Most mild cases will improve with time; more severe cases may require surgical correction. Lubricants should be employed to avoid corneal exposure as the ectropion resolves.

Dry Eye Syndrome

Patients suffering from dry eye symptoms should be identified both by history and by Schirmer testing. Surgical candidates include patients with mild symptoms who accept the risk that surgery may exacerbate their symptoms. With the approval of an ophthalmologist, resection should be conservative, and postoperative management involves vigorous application of artificial tears or other wetting solutions.

Asymmetry

The key to avoidance of this common complication is accurate preoperative assessment and marking of the patient. Repeat blepharoplasty can usually adequately address significant asymmetry.

Ptosis

Ptosis must be distinguished from dermatochalasis or pseudoptosis preoperatively to properly formulate a surgical plan. The ptosis should be surgically corrected as part of the aesthetic surgical procedure. Postoperative ptosis results from an unrecognized injury to the lid elevator mechanism and should be repaired promptly.

Pearls and Pitfalls

- Dry eye should be recognized preoperatively.
- Avoid overresection of skin and fat.
- The presence of a negative vector should alert the surgeon to the possibility of postoperative lower lid retraction.
- Lateral canthoplasty/canthopexy should be part of lower lid blepharoplasty in patients suspected of having postoperative lower lid laxity.
- Ptosis should be corrected as part of the blepharoplasty.
- Pseudoptosis will limit the aesthetic result of upper blepharoplasty; a concomitant brow lift may also be indicated.
- The presence of Graves' disease precludes blepharoplasty until the ophthalmopathy is corrected.

SUGGESTED READINGS

Fagien S: Advanced rejuvenative upper blepharoplasty: Enhancing aesthetics of the upper periorbita. Plast Reconstr Surg 110:278-291, 2002.

Glat PM, Jelks GW, Jelks EB, et al: Evolution of the lateral canthoplasty: Techniques and indications. Plast Reconstr Surg 100:1396-1405, 1997.

Hamra ST: The role of the septal reset in creating a youthful eyelid-cheek complex in facial rejuvenation. Plast Reconstr Surg 113:2124-2141, 2004.

Jelks GW, Jelks EB: Preoperative evaluation of the blepharoplasty patient. Bypassing the pitfalls. Clin Plast Surg 20:213-223, 1993.

Knize DM: Transpalpebral approach to the corrugator supercilii and procerus muscles. Plast Reconstr Surg 95:52-60, 1995.

Niechajev I: Transpalpebral browpexy. Plast Reconstr Surg 113:2172-2180, 2004.

Primary Rhinoplasty

FRED L. HACKNEY

Primary rhinoplasty is one of the most common and challenging aesthetic surgery procedures. To obtain optimal results, the surgeon must have a thorough understanding of nasal anatomy and physiology, nasofacial aesthetic standards, and surgical technique. The surgeon must also understand the variations from normal anatomy that produce nasal deformities.

Etiopathogenesis

Most primary rhinoplasties are performed for deviations from aesthetic norms that occur spontaneously or are the result of nasal trauma. Either can be accompanied by airway obstruction or external cosmetic deformities, or both. The most common deformity resulting from nasal trauma is displacement of the nasal bones and septum in association with an asymmetric or twisted appearance of the dorsum. Nasal trauma can also result in septal hematoma, which can lead to septal perforation and subsequent loss of nasal support. Other acquired nasal deformities can be secondary to cocaine use, which can lead to nasal perforation with loss of dorsal support.

Pathologic Anatomy

The pathologic anatomy of an aesthetically problematic nose can be divided into three categories: tissue excess, tissue deficiency, or abnormal position or shape of tissues (or any combination of the three).

Examples of excess of tissue include a dorsal hump, overprojecting radix, and overprojecting tip. A dorsal hump is due to an overprojection of the nasal bones and dorsal septum. The proportion of cartilage and bone in a dorsal hump varies depending on the relative length of the nasal bones and septum. Patients may have an overprojecting radix with lack of a nasofrontal angle. The latter usually represents bony overprojection in the area of the nasofrontal suture, but it may also be secondary to excessive thickness of the overlying procerus muscle. An overprojecting tip may be due to an overprojecting or "high" anterior septal angle or increased length and strength of the medial and lateral crura of the lower lateral cartilage.

A long nose can be due to either excess or deficiency of tissue. With an excess of tissue such as a high radix, the nose will appear longer. Likewise, with overgrowth of the cephalic-caudal septum or the upper lateral cartilage, the nose will appear longer. Deficiency of the medial crura can result in drooping of the tip and produce the appearance of a long nose.

A short nose is most commonly due to tissue deficiency. The radix can be deficient with a deep nasofrontal angle, and in severe cases such as Binder's syndrome, the maxilla and nasal bones can be underdeveloped. Overrotation of the tip can result from underdevelopment of the septum or the lateral crura. In addition, the appearance of decreased nasal length can be produced by an obtuse columellar-labial angle secondary to prominence of the caudal septum at the anterior nasal spine.

Other deficiencies in anatomy include a saddle nose deformity and underprojection of the tip. The former can be due to a deficiency of support from the septum, and the latter can occur as a result of lack of support from the anterior septal angle or from deficiencies in the length and strength of the medial and lateral crura or their soft tissue attachments (or both).

The last category of anatomic pathology is abnormal shape or position of the anatomic structures, as seen in deformities of the tip. A *boxy tip* may be due to an increased distance between the tip-defining points, an increased angle of divergence of the intermediate crura, or increased width of each dome (or any combination of these causes). Fullness in the supratip area can be caused by increased convexity of the lateral crura in the scroll area or increased

height of the cartilaginous septal angle. Outward bowing of the alae can be due to increased convexity of the lateral crura in an anteroposterior direction.

Deformity or asymmetry of the dorsum can result from septal deviation and abnormal shape or positioning of the nasal bones. Asymmetry of the tip can also result from deviation of the dorsal and caudal portions of the septum. Increased nasal base width can be attributed to increased width of the base of the nasal bones. Increased width of the dorsum of the nose can reflect increased width of the dorsal aspect of the nasal bones. Likewise, deficiencies in nasal base width can result from the nasal bones being displaced medially at the base, and decreased dorsal width can be secondary to a decrease in nasal bone width at the dorsum or to collapse of the upper lateral cartilage.

Diagnostic Studies

Any evaluation for nasal surgery should include questions regarding nasal trauma, allergies, airway problems, need for medications, and history of sinusitis, asthma, bronchitis, and cocaine use.

The goal of the history should be to evaluate the patient's overall appropriateness for surgery, including physical health and emotional status. A thorough evaluation may prevent postoperative complications and allow the surgeon to make the patient more aware of potential complications.

Diagnostic studies before primary rhinoplasty consist of nasofacial analysis and evaluation of facial proportions. There should be a balance between the vertical dimensions of the upper, mid, and lower portions of the face. The presence of significant abnormalities in the vertical relationships of the face is an indication for a more thorough evaluation for possible orthognathic surgery.

The lip-chin complex should be evaluated because it provides balance with nasal projection and rotation. The lower lip should be positioned 2 mm posterior to the normally positioned upper lip. Ideal chin position varies between men and women; in women the chin is positioned posterior to the lower lip, whereas in men, it can be slightly more anterior. One should also be aware of a tension lip deformity, which is often present in patients with a class II skeletal facial deformity. This deformity is characterized by fullness in the columellar-labial angle and lack of projection in the upper lip. Often associated with an overprojecting nose, it can also occur in patients with deficient tip projection.

When considering lip position, the position of the dentoalveolar segments of the maxilla should also be considered. Patients may have proclined maxillary incisors that support the lip in a more anterior position, a fact that needs to be taken into consideration when evaluating the position of the chin and the overall relationship of the chin-lip complex to nasal projection.

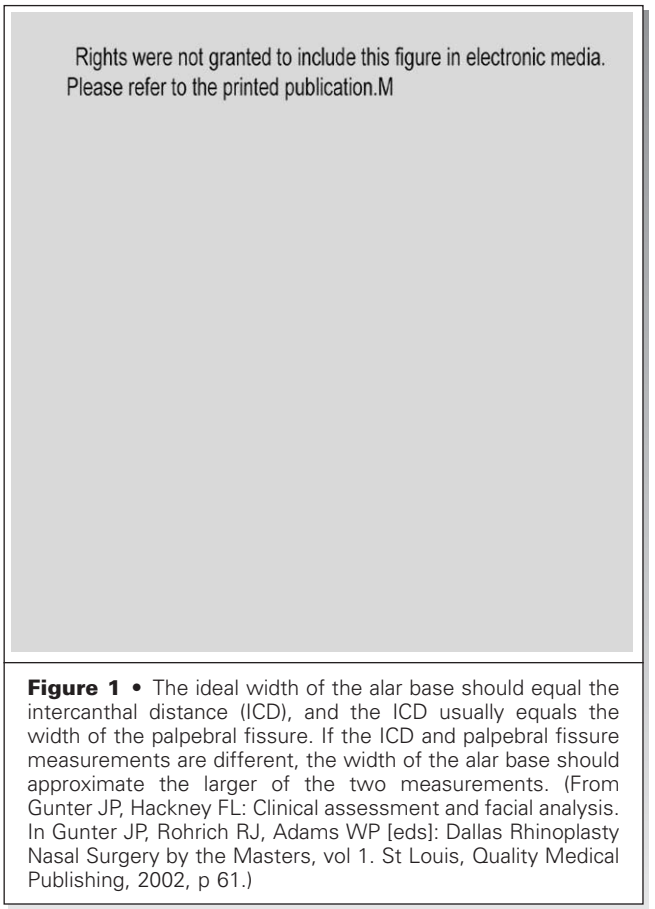

Figure 1 • The ideal width of the alar base should equal the intercanthal distance (ICD), and the ICD usually equals the width of the palpebral fissure. If the ICD and palpebral fissure measurements are different, the width of the alar base should approximate the larger of the two measurements. (From Gunter JP, Hackney FL: Clinical assessment and facial analysis. In Gunter JP, Rohrich RJ, Adams WP [eds]: Dallas Rhinoplasty Nasal Surgery by the Masters, vol 1. St Louis, Quality Medical Publishing, 2002, p 61.)

The nasal examination starts with evaluation of the thickness of the skin. Thick-skinned patients will have prolonged postoperative edema, and the anatomic details of tip refinement will be less noticeable. Conversely, patients with thin skin have more of a tendency to show minor irregularities in the underlining cartilage.

Nasal symmetry is evaluated in the frontal view. The width of the alar base in the frontal view should equal the intercanthal distance (Fig. 1). The width of the bony base should equal 80% of the normal width of the alar base. The patient is also examined for the presence of *dorsal aesthetic lines*, which should extend from the *tip-defining points* to the medial ends of the eyebrows. The tip is evaluated for overall proportions by noting whether the vertical distance between the supratip area and the tip-defining points is equal to the distance between the tip-defining points and the columellar-lobular angle. The contours of the alae are evaluated by noting the outline of the alar rims and columella, which should resemble the wings of a seagull in flight.

On profile view, the patient should be examined in a neutral head position. The position of the radix is evaluated by inspection of the nasofrontal angle.

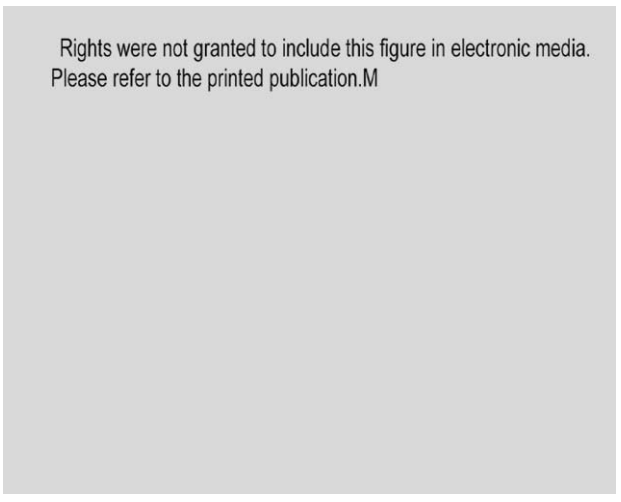

Figure 2 • Ideal tip projection should have 50% to 60% of the tip projection anterior to a line drawn tangential to a normally projecting upper lip. (From Gunter JP, Hackney FL: Clinical assessment and facial analysis. In Gunter JP, Rohrich RJ, Adams WP [eds]: Dallas Rhinoplasty Nasal Surgery by the Masters, vol 1. St Louis, Quality Medical Publishing, 2002, p 65.)

The deepest point of the nasofrontal angle should be positioned between the supratarsal fold and the eyelashes. The depth of the nasofrontal angle should provide an aesthetic break between the forehead and the dorsum of the nose and usually projects 9 to 14 mm from the ocular coronal plane. Tip projection is evaluated by drawing a line tangential to the normally positioned upper lip. This line should divide the distance between the alar base and the tip-defining points such that the amount of tip projection anterior to the line constitutes 50% to 60% of the projection of the tip (Fig. 2). The *tip-defining points* should be the most anteriorly projecting part of the tip. The *nasolabial angle*, which is the angle formed by a line drawn through the anterior and posterior ends of the nostrils and a line perpendicular to the natural horizontal facial plane, is a determinant of tip rotation and should also be evaluated. This angle is generally considered to be normal if it is between 95 and 100 degrees in women and 90 and 95 degrees in men. It is different from the *columellar-labial angle*, which is the angle formed at the junction of the columella and the upper lip. The amount of dorsal projection is determined by drawing a line between the normal position of the nasofrontal angle and the tip-defining points of the normally projecting tip. In women, the dorsum should be approximately 2 mm posterior to this line, and in men, the dorsum should be slightly closer to this line. There are no standards for measurements of the degree of supratip break, but an aesthetic break should be present between the dorsum and the tip and should be more noticeable in women than in men.

In the lateral view, any discrepancies between the alar and columellar relationships should be noted. The alar rim and the roll of the columella should be 1 to 2 mm from a line through the most anterior and posterior positions of the nostril. Deviations from this line identify a retracted or hanging ala or a retracted or hanging columella.

Nasal length and nasal tip projection can also be evaluated in relation to each other. In a patient with ideal nasal length, tip projection should equal 0.67 times the ideal nasal length (Fig. 3). The ideal nasal length should also equal the distance between the stomion and menton in a face with normal facial proportions.

In the basal view, the nose should form an equilateral triangle, with the base of the nose formed by the alar bases and the apex forming the tip. The proper proportion between nostril and lobular length should be two thirds and one third, respectively. Any asymmetry or deformity of the nostrils or columella or protrusion or deviation of the caudal septum should also be noted.

Intranasal examination requires an adequate light source and a nasal speculum. Vasoconstriction should be induced if the mucosa appears to be thickened or edematous. The airways are examined for patency and deviation of the nasal septum, as well as hypertrophy of the turbinates. The nasal septum should be inspected for perforation. The internal nasal valve is the angle formed by the junction of the septum and the caudal edge of the upper lateral cartilage. If less than 10 to 15 degrees and the patient complains of airway obstruction, it is an indication of a collapsed nasal valve.

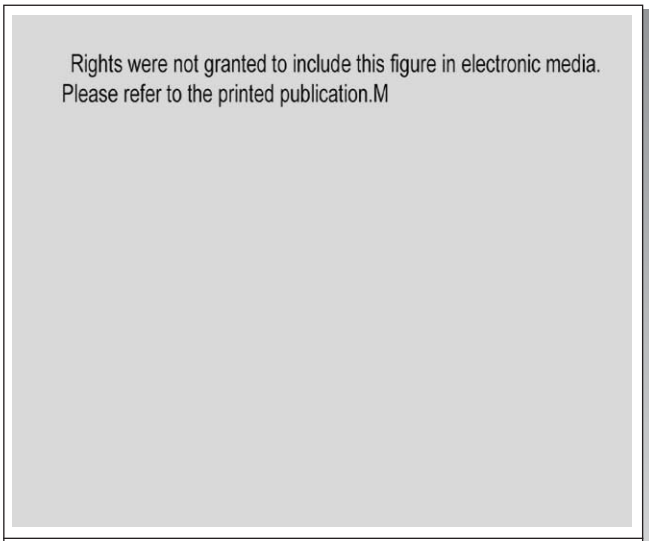

Figure 3 • The ideal nasal length and the ideal tip projection should have a ratio of 1:0.67. (From Gunter JP, Hackney FL: Clinical assessment and facial analysis. In Gunter JP, Rohrich RJ, Adams WP [eds]: Dallas Rhinoplasty Nasal Surgery by the Masters, vol 1. St Louis, Quality Medical Publishing, 2002, p 66.)

Surgical Goals

The goals in a primary rhinoplasty patient are to improve the aesthetic appearance of the nose while keeping it in balance and harmony with the face. If the airway is patent, it should be maintained; if it is obstructed, it should be corrected at the same setting.

Treatment

Anesthesia

The choice between intravenous sedation and general endotracheal anesthesia is generally based on the surgeon's preference and experience.

After induction, local anesthesia with 10 mL or more of 1% lidocaine with 1:200,000 epinephrine is injected along the planned incision lines, the dorsum, the alar base, and the septum. The nose is packed with 1-inch-wide ribbon gauze soaked in oxymetazoline.

Surgical Approach

Proponents of open rhinoplasty and endonasal rhinoplasty can offer pro and con arguments for their preferred procedure. The open approach has the advantage of adequate exposure to visualize the anatomy and perform an anatomic correction. The main potential disadvantage of this approach is the transcolumellar scar; however, if the incision is closed meticulously and the skin edges are everted and properly aligned, the scar is usually imperceptible. Other disadvantages of the open technique are the increased operating time needed to reconstruct the osseous-cartilaginous framework and prolonged postoperative edema. However, the advantages of the open approach, in terms of better visualization and anatomic correction, outweigh these disadvantages.

Open rhinoplasty is begun by designing the transcolumellar incision. It is best designed as a broken line incision, such as an inverted V shape or a stair-step design. The transcolumellar incision is made just anterior to the feet of the medial crura (Fig. 4). A double skin hook is used to retract the alar rim and the index finger is used to evert the lateral crus. A marginal incision is made along the caudal border of the lateral crus and extended laterally from the apex. The incision is extended behind the soft tissue triangle in the dome area and along the lateral sides of the columella to the planned transcolumellar incision. Before the marginal incision is made on each side, a small crosshatch is placed across the caudal border of the lateral crus to provide orientation for closure. The transcolumellar incision is made next with a No. 15 blade, and a No. 11 blade is used to make the smaller stair step or the inverted V portion of the incision.

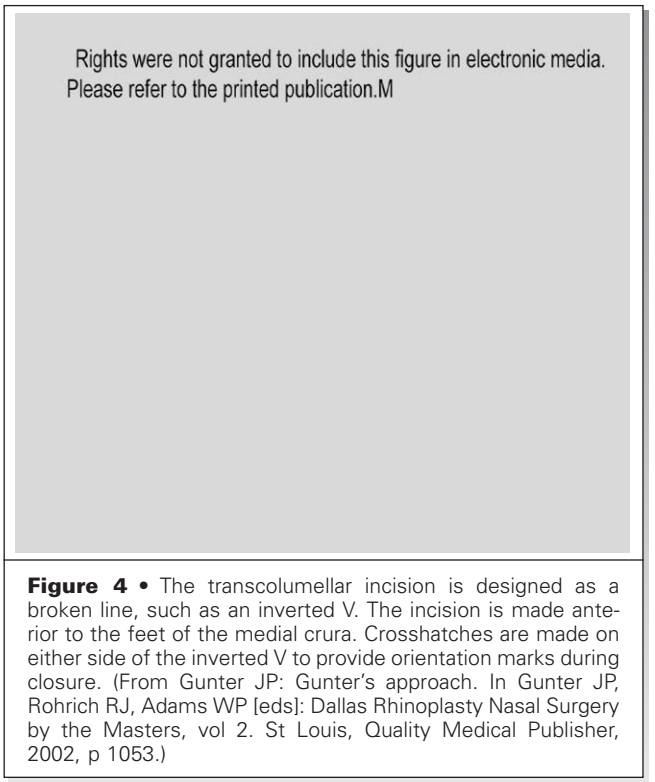

Figure 4 • The transcolumellar incision is designed as a broken line, such as an inverted V. The incision is made anterior to the feet of the medial crura. Crosshatches are made on either side of the inverted V to provide orientation marks during closure. (From Gunter JP: Gunter's approach. In Gunter JP, Rohrich RJ, Adams WP [eds]: Dallas Rhinoplasty Nasal Surgery by the Masters, vol 2. St Louis, Quality Medical Publisher, 2002, p 1053.)

Exposure

Undermining is started at the marginal incision of the lateral crus. A double skin hook is placed inside the alar rim and retracted to spread the incision. Scissors are used to elevate the soft tissue from the superficial surface of the lower crus, and the tissues are dissected off the lateral crus and the dome with a spreading motion. The skin is dissected from the caudal border of the medial crura by inserting the scissors through the incision along the medial aspect of the columella and undermining the skin so that the scissors protrude from the contralateral medial columellar incision. The transcolumellar incision is completed and the skin is reflected over the dorsum. The dissection continues over the upper lateral cartilage and extends 2 mm cephalic to the osseocartilaginous junction, at which point scissors are used to penetrate the periosteum. A periosteal elevator is used to elevate the periosteum over the dorsum. It is important to limit the extent of the lateral subperiosteal dissection over the nasal bones so that the periosteum is not elevated over the planned lateral osteotomy sites.

The soft tissues are elevated from the medial surfaces of the medial crura to expose the anterior septal angle, as well as facilitate dissection of a pocket for a columellar strut. When making the soft

tissue pocket for the columellar strut, one should be careful to leave an intervening layer of soft tissue between the base of the pocket and the anterior nasal spine so that the columellar strut will not be displaced to either side of the anterior nasal spine.

Dorsal Reduction

Surgeons differ in whether the tip projection should be set first or the dorsal projection should be modified first. It is the author's preference to determine the anticipated tip projection but modify the dorsum first.

If dorsal reduction is indicated, the dorsum is initially reduced with a rasp. During rasping, the bony rasp may reduce the dorsum while *displacing* the cartilaginous dorsum inferiorly and actually *not reducing* it. When the rasp is removed, the cartilaginous dorsum may return back to its normal height, which will be higher than the reduced bony dorsum. The surgeon should be aware of this problem and avoid continued rasping and subsequent overreduction of the bony dorsum.

After the bony dorsum has been reduced near its anticipated position, the cartilaginous dorsum is reduced. Submucosal tunnels are developed on either side of the septum, starting at the anterior septal angle. The submucosal tunnels are extended from the septum to the undersurface of the upper lateral cartilage and cephalically beneath the nasal bones. A scalpel or scissors is used to separate the upper lateral cartilage from the septum. Long angled scissors or a scalpel can be used to incrementally lower the dorsal cartilaginous septum to achieve the planned dorsal projection. As this is being done, additional bony reduction with a rasp may be required. It is often necessary to trim the medial edges of the upper lateral cartilage; however, one must be careful not to trim the cartilage excessively because the skin will displace the upper lateral cartilage inferiorly and thereby result in loss of dorsal projection. The bony and cartilaginous dorsum is reduced close to the correct height while keeping in mind that osteotomies may result in some loss of projection if the osteotomies are unstable and move posteriorly when infractured.

Septal Surgery

After trimming of the dorsal or caudal septum has been completed, septoplasty is performed if necessary. Septoplasty is indicated if cartilage grafts are needed to complete the operative plan or if septal deviation with airway obstruction is present. From the anterior septal angle, the mucoperichondrium is elevated from one side of the cartilaginous septum, the anterior two thirds of the perpendicular plate of the ethmoid, and inferiorly to the maxillary crest and vomer. The mucoperichondrium is elevated from the maxillary crest and vomer starting posteriorly and working anteriorly because dissection in an anterior-to-posterior direction facilitates separation of the mucoperichondrium from the osseocartilaginous junction of the maxillary crest and the septum. After exposure has been achieved, a No. 15 blade is used to make the cartilaginous incisions while leaving 10-mm dorsal and caudal cartilaginous struts (Fig. 5). The mucoperichondrium over the area to be dissected is elevated from the contralateral side through the cartilaginous incisions.

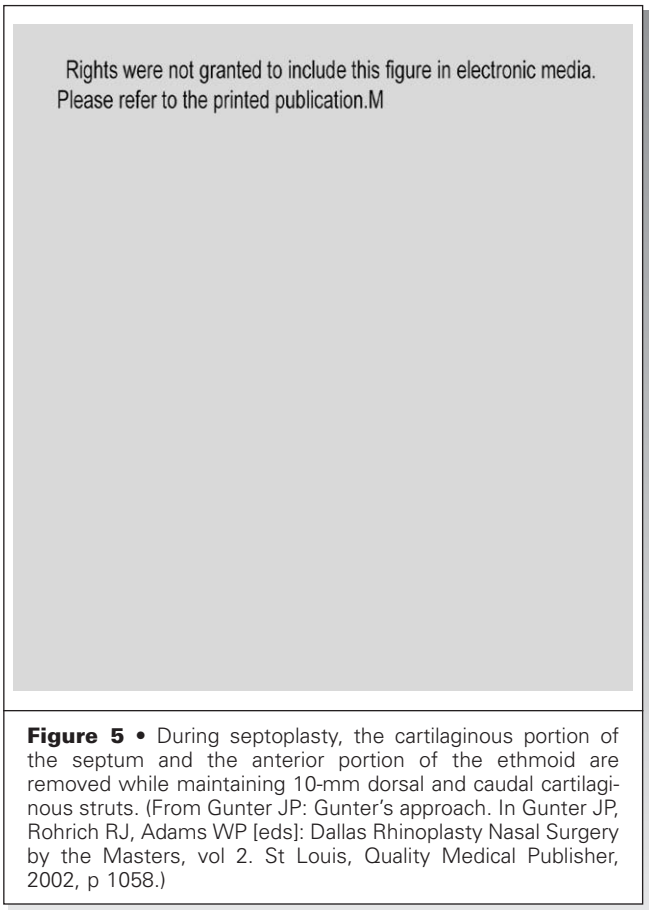

Figure 5 • During septoplasty, the cartilaginous portion of the septum and the anterior portion of the ethmoid are removed while maintaining 10-mm dorsal and caudal cartilaginous struts. (From Gunter JP: Gunter's approach. In Gunter JP, Rohrich RJ, Adams WP [eds]: Dallas Rhinoplasty Nasal Surgery by the Masters, vol 2. St Louis, Quality Medical Publisher, 2002, p 1058.)

After obtaining exposure, the septoplasty is completed by separating the septum off the maxillary crest, starting anteriorly and working posteriorly. The dorsal cartilaginous incision is extended with heavy scissors through the perpendicular plate of the ethmoid. This leaves the cartilage attached by the thin bone of the ethmoid, which is infractured with a curved Freer elevator. The septum should be free except for any residual soft tissue attachments that need to be dissected.

After the septum has been removed, any posterior deviation of the ethmoid that needs to be eliminated to improve the airway can be removed with rongeurs, and spurs along the maxillary crest can be removed with an osteotome. The mucosa is inspected for tears. Only opposing tears must be repaired to prevent septal perforation.

Any residual straight pieces of cartilage remaining after cartilage grafts are carved can be placed

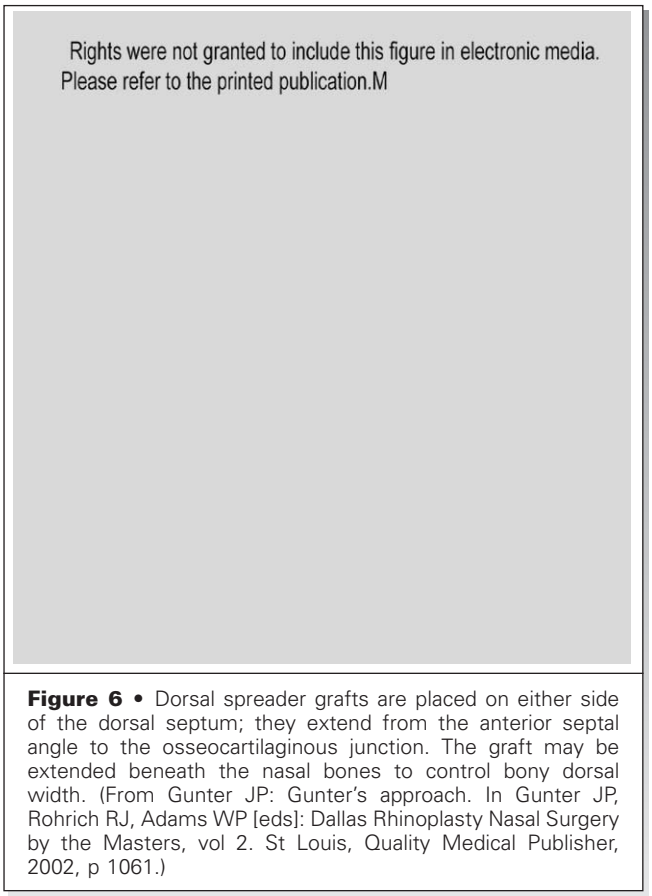

Figure 6 • Dorsal spreader grafts are placed on either side of the dorsal septum; they extend from the anterior septal angle to the osseocartilaginous junction. The graft may be extended beneath the nasal bones to control bony dorsal width. (From Gunter JP: Gunter's approach. In Gunter JP, Rohrich RJ, Adams WP [eds]: Dallas Rhinoplasty Nasal Surgery by the Masters, vol 2. St Louis, Quality Medical Publisher, 2002, p 1061.)

back into the septoplasty site before closure of the mucosa. The cartilage may be used later for revision surgery if necessary.

Dorsal spreader grafts are placed to prevent collapse of the upper lateral cartilage in most patients who require significant dorsal reduction or have short nasal bones (or both). Dorsal spreader grafts are also useful in straightening a deviated dorsal septum. The grafts are carved from septal cartilage and usually measure 20 to 25 mm by 3 to 4 mm. They are placed from the anterior septal angle to the osseocartilaginous junction and may be extended underneath the surface of the nasal bones to control dorsal bony width (Fig. 6). If cartilage grafts are being used to correct septal deviation, the septum can be partially scored on its concave surface to weaken it before the dorsal spreader grafts are placed. The dorsal spreader grafts are sutured to the septum with horizontal mattress sutures. In some patients, the dorsal spreader grafts can be extended beyond the anterior septal angle and the medial crura sutured to the ends of the grafts to increase nasal length. The medial crura can also be advanced anteriorly on the grafts to increase tip projection.

Nasal Tip Surgery

Modification of the tip in open rhinoplasty usually includes the use of a columellar strut. A columellar strut can be placed to increase tip projection and, fundamentally, to provide a more stable infrastructure for modification and shaping of the tip. A columellar strut also prevents narrowing of the columella, which can result if the medial crura are sutured directly together.

When attempting to increase tip projection, the columellar strut is placed by retracting the domes away from the nasal spine and seating the strut posteriorly in the pocket under a moderate amount of tension. A 25-gauge needle is passed through the medial crus on one side, the columellar strut, and the contralateral medial crus (Fig. 7). The medial crura are sutured to the columellar strut with horizontal mattress sutures. One should keep in mind that the caudal edges of the medial crura are normally separated, and the sutures should be placed so columellar width will not be decreased. Care must also be taken to position the sutures so that the caudal edges of the medial crura are even with each other in a caudal-cephalic direction.

The columellar strut can also be used to control the columellar-lobular angle. If the columellar-lobular angle is too obtuse (generally considered to be greater than 45 degrees), the cephalic margin of the medial crura at the angle can be partially transected. The medial crura are sutured to the

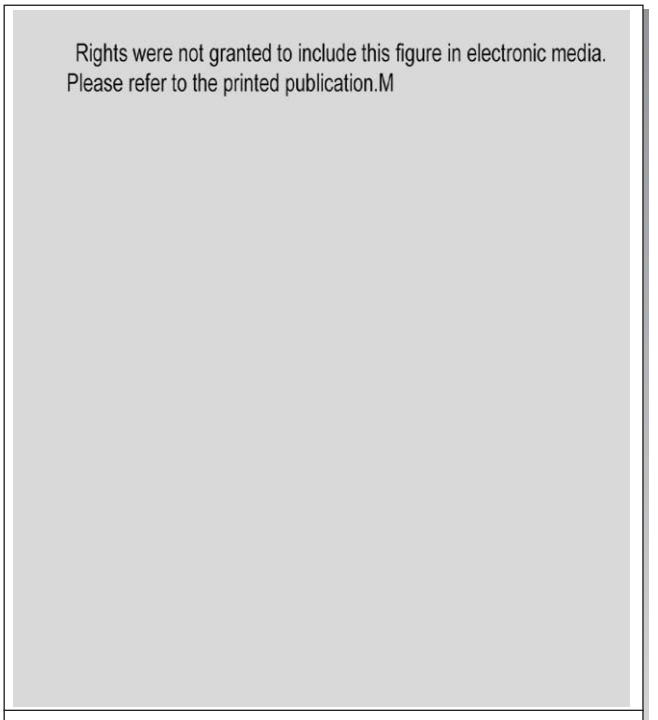

Figure 7 • A columellar strut is placed in the soft tissue pocket while retracting the domes away from the anterior nasal spine. It is stabilized to the medial crura with the use of a 25-gauge needle and horizontal mattress sutures to increase tip projection. (From Gunter JP: Gunter's approach. In Gunter JP, Rohrich RJ, Adams WP [eds]: Dallas Rhinoplasty Nasal Surgery by the Masters, vol 2. St Louis, Quality Medical Publisher, 2002, p 1064.)

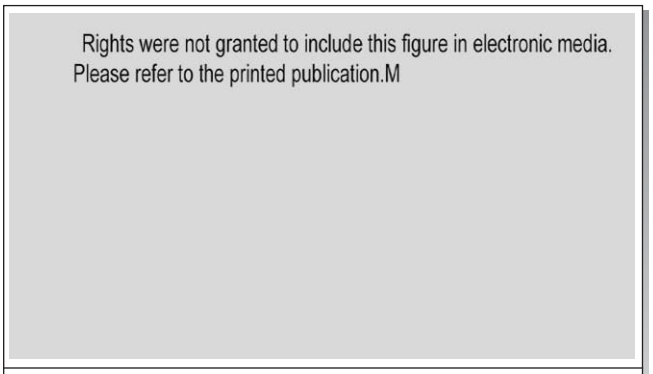

Figure 8 • The columellar-lobular angle can be decreased by scoring the medial crura on the cephalic margin and suturing them to the strut. (From Gunter JP: Gunter's approach. In Gunter JP, Rohrich RJ, Adams WP [eds]: Dallas Rhinoplasty Nasal Surgery by the Masters, vol 2. St Louis, Quality Medical Publisher, 2002, p 1065.)

columellar strut to decrease the columellar-lobular angle (Fig. 8). Conversely, if the columellar-lobular angle is too acute, the caudal margin of the medial crura can be partially transected at the angle and sutured to the columellar strut to increase the columellar-lobular angle (Fig. 9).

The next step in modifying the tip is to evaluate the lateral crura. The most common procedure on the lateral crura is resection of the cephalic margin to decrease tip fullness (Fig. 10). The amount of cartilage removed should be minimal so that the aesthetic result is not compromised. The tip-defining points of the domes should always be maintained. If it is desirable to decrease the distance between the tip-defining points, resection of the cephalic margin can be extended into the intermediate crura and the tip-defining points allowed to move toward the midline.

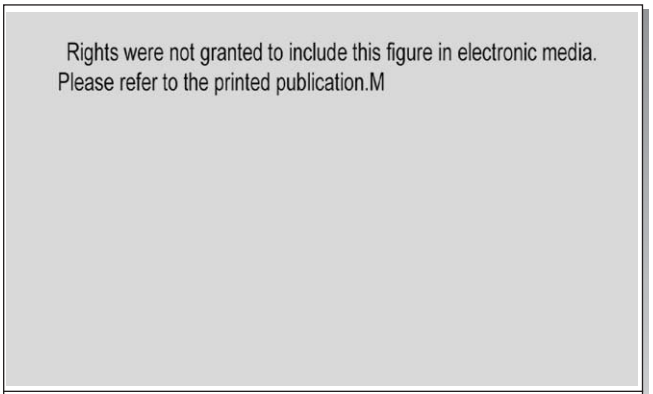

Figure 9 • The columellar-lobular angle can be increased by scoring the medial crura on the caudal margin and suturing them to the strut. (From Gunter JP: Gunter's approach. In Gunter JP, Rohrich RJ, Adams WP [eds]: Dallas Rhinoplasty Nasal Surgery by the Masters, vol 2. St Louis, Quality Medical Publisher, 2002, p 1065.)

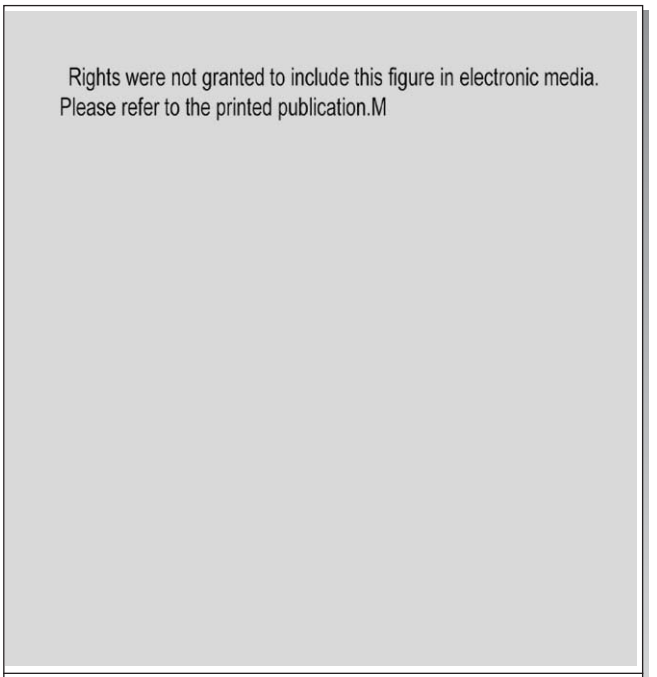

Figure 10 • Trimming of the cephalic margin should be performed only to the extent necessary to accomplish the aesthetic goals while maintaining as much residual lateral crura as possible. (From Gunter JP: Gunter's approach. In Gunter JP, Rohrich RJ, Adams WP [eds]: Dallas Rhinoplasty Nasal Surgery by the Masters, vol 2. St Louis, Quality Medical Publisher, 2002, p 1065.)

Lateral crura strut grafts can be used to correct the position, improve the shape, or reconstruct the lateral crura. Lateral crura strut grafts are carved to a length of 20 to 25 mm and a width of 3 to 4 mm. A pocket is developed by dissecting the vestibular skin starting at the cephalic margin of the lateral crus. The vestibular skin is reflected from the undersurface of the lateral crus while leaving it attached to the caudal border. If the graft is placed to reposition or reconstruct the lateral crura, it is often necessary to extend it superficial to the piriform aperture to provide support. The strut is placed into the pocket and sutured in position with horizontal mattress sutures.

A structurally weakened lateral crus may be seen as alar rim retraction or collapse. In addition to the lateral crural strut graft, another option to correct this deformity is a nonanatomic alar strut. The strut is placed in a pocket between the vestibular and external skin, just above and parallel to the alar rim. The pocket is dissected from the lateral aspect of the marginal incision to the alar-cheek groove. A strut is carved to the appropriate size and sutured in place with absorbable suture. The strut can extend anteriorly as far as necessary to support the alar rim.

If additional tip projection is desired, it can be obtained with suture techniques. Tip projection can be increased by suturing the cephalic borders of the

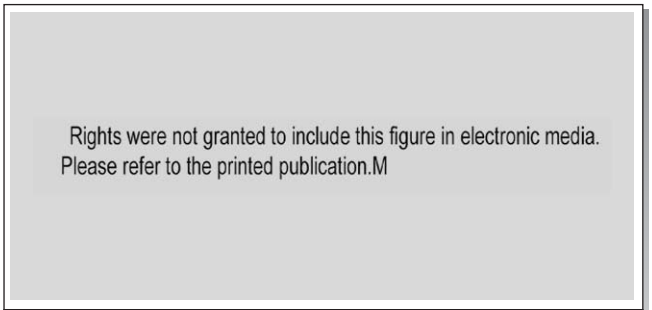

Figure 11 • Transdomal sutures can be placed to narrow the tip and provide angulation. The distance between the tip-defining points can be narrowed by suturing the transdomal sutures together. (From Gunter JP: Gunter's approach. In Gunter JP, Rohrich RJ, Adams WP [eds]: Dallas Rhinoplasty Nasal Surgery by the Masters, vol 2. St Louis, Quality Medical Publisher, 2002, p 1066.)

intermediate crura together. This maneuver is most effective in increasing tip projection if the angle of divergence between the intermediate crura is large. If additional tip projection is indicated, a horizontal mattress suture can be placed through the dome so that the lateral arm of the suture extends lateral to the tip-defining points more than the medial arm extends medial to the tip-defining points. When tied, the suture will recruit part of the lateral crura into the domes and increase tip projection. Each dome is sutured in the same manner. If additional tip projection is still required, one should consider tip grafts.

If necessary, transdomal sutures can be used to control the angulation of the domes and the distance between the tip-defining points to narrow the tip and provide angulation (Fig. 11).

Osteotomies

Medial osteotomies should be considered if the bony dorsum is excessively narrow or wide, if the nasal bones are deviated, or if there are thick nasal bones that would make it difficult to create a greenstick fracture. The medial osteotomy should be performed before the tip is completely stabilized. A 4-mm osteotome is placed at the septal-bony junction and angled toward the medial canthus; the medial osteotomies should end no higher than the medial canthus.

Lateral osteotomies can be performed through an endonasal or percutaneous approach. Proponents of the percutaneous approach believe that the periosteum remains partially intact, which helps to stabilize the nasal bones. Proponents of the endonasal technique believe that this approach is technically easier and gives the surgeon more control. The lateral osteotomy extends no farther superiorly than the medial canthus. The osteotome is angled toward the maxilla until it reaches the nasal-cheek junction, at which point it continues superiorly and curves toward the nasal root (Fig. 12). This angulation preserves a triangle of bone at the base of the inferior turbinate, thereby preventing medial displacement of the turbinate when the osteotomy is infractured. After the osteotomy is completed, gentle digital fracture will complete the greenstick fracture to the level of the dorsum or the medial osteotomy site.

Final adjustments are made to tip projection, the bony dorsum, and the upper lateral cartilage to correct the final relationship between dorsal height and tip projection. The upper lateral cartilage is sutured to the septum and the dorsal spreader grafts.

Closure

Soft plastic septal splints with an incorporated airway are placed in the nose before the closure is started. Closure is begun by aligning the crosshatches and suturing the transcolumellar incision. The marginal incision is closed with interrupted sutures. The surgeon should recognize any distortion of the alar rim that occurs during placement of the sutures when closing the marginal incision. After the closure is completed, the septal splints are brought forward and sutured to the membranous septum with a single suture.

After closure, the patient is evaluated to determine whether resection of the alar base is needed. If alar base resections are indicated, they are performed at this time.

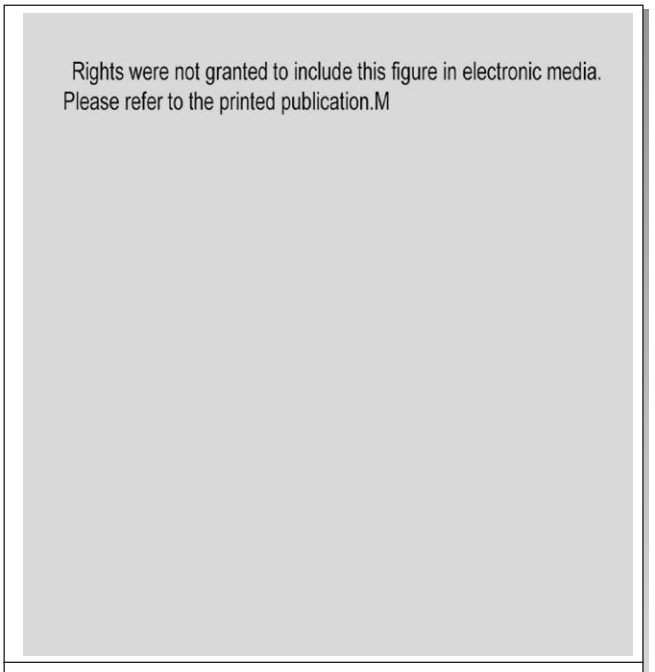

Figure 12 • When performing a lateral osteotomy, Webster's triangle is preserved to prevent medial displacement of the inferior turbinate with infracturing of the bones. (From Gunter JP: Gunter's approach. In Gunter JP, Rohrich RJ, Adams WP [eds]: Dallas Rhinoplasty Nasal Surgery by the Masters, vol 2. St Louis, Quality Medical Publisher, 2002, p 1067.)

An aluminum splint that can easily be molded to the desired shape is used to position the nasal bones.

Pearls and Pitfalls

To avoid the need for revision surgery, the following principles should be adhered to:

- Avoid overresection of tissues. This is most important in reduction of the dorsum and can be achieved by incremental reduction of the bony dorsum, followed by incremental reduction of the cartilaginous dorsum. Trimming of the cephalic margin should be done only to the extent necessary to accomplish the surgical goals. For example, if the goal is to decrease tip fullness, removal of the lateral crural scroll area is usually all that is necessary. This will prevent overresection and weakening of the lateral crura, which can result in postoperative deformity from alar collapse.
- Avoid inadvertent fracture of the dorsal cartilaginous strut at the osseocartilaginous junction. Such fracture can be avoided with proper septoplasty technique by maintaining 10-mm dorsal and caudal struts.
- Avoid onlay grafts, if possible. If onlay grafts are used, they should be stabilized by suture to prevent displacement.
- Stabilize the osseocartilaginous framework. The open rhinoplasty approach involves extensive dissection, which is accompanied by more postoperative scarring and contracture.
- Suturing techniques are useful to reshape and reposition the tip.

SUGGESTED READINGS

Andochick S, Burt JD, Byrd HS, et al: Septal extension grafts: A method of controlling tip projection, rotation and shape. Plast Reconstr Surg 100:999, 1997.

Byrd HS, Hobar PC: Rhinoplasty: A practical guide for surgical planning. Plast Reconstr Surg 91:642, 1992.

Gruber R: Aesthetic and technical aspects of nasal osteotomies. Paper presented at the Dallas Rhinoplasty Symposium, 1996, p 245.

Gunter JP: Nasal tip surgery. Paper presented at the Dallas Rhinoplasty Symposium, 1994, pp 11-53.

Gunter JP, Friedman RM: Lateral crural strut graft: Technique and clinical applications in rhinoplasty. Plast Reconstr Surg 99:943, 1997.

Gunter JP, Hackney FL: Clinical assessment and facial analysis. In Gunter JP, Rohrich RJ, Adams WP (eds): Dallas Rhinoplasty Nasal Surgery by the Masters, vol 1. St Louis, Quality Medical Publishing, 2002, pp 53-71.

Gunter JP, Hackney FL: Decreasing tip projection: An algorithm. In Gunter JP, Rohrich RJ, Adams WP (eds): Dallas Rhinoplasty Nasal Surgery by the Masters, vol 1. St Louis, Quality Medical Publishing, 2002, pp 359-365.

Gunter JP, Rohrich RJ: Augmentation rhinoplasty: Dorsal onlay grafting using shaped autogenous septal cartilage. Plast Reconstr Surg 86:39, 1990.

Gunter JP, Rohrich RJ, Friedman RM, Hackney FL: Importance of the alar-columellar relationship. In Gunter JP, Rohrich RJ, Adams WP (eds): Dallas Rhinoplasty Nasal Surgery by the Masters, vol 1. St Louis, Quality Medical Publishing, 2002, p 105.

Hubbard TJ: Surgical correction of the long nose. In Gunter JP, Rohrich RJ, Adams WP (eds): Dallas Rhinoplasty Nasal Surgery by the Masters, vol 1. St Louis, Quality Medical Publishing, 2002, p 528.

Sheen JH, Sheen, AP: Aesthetic Rhinoplasty, 2nd ed. St Louis, Quality Medical Publishing, 1998 (reprint of 1987 ed.).

Sullivan PK, Varma M, Rozelle A: Optimizing bone-graft nasal reconstruction: A study of nasal bone shape and thickness. Plast Reconstr Surg 97:327, 1996.

Tebbetts J: Shaping and positioning the nasal tip without structural disruption: "A systematic approach." Plast Reconstr Surg 94:61, 1994.

Secondary Rhinoplasty

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Success in secondary rhinoplasty relies not only on technique but also on communication and understanding between the patient and surgeon. Before undertaking secondary rhinoplasty, the surgeon is best advised to allow the normal scarring and healing to take its course, usually at least 1 year after the previous intervention. This period also allows for emotions and anxiety over the first procedure to dissipate.

Etiopathogenesis

The reasons for a suboptimal outcome in primary rhinoplasty are multifaceted. It is likely that the most important factor influencing the outcome of the initial operation is the surgeon's experience, including an appreciation of the potential problems and pitfalls associated with rhinoplasty. In addition, biologic factors such as a history of wound-healing problems, skin conditions (e.g., seborrhea), or bleeding disorders can affect a patient's results. The role that the airway may play in functional outcomes after rhinoplasty is discussed in the next chapter, "The Nasal Airway."

A perfect outcome in rhinoplasty is difficult to achieve. Irregularities measuring only a quarter of a millimeter may result in a visible flaw. Such subtleties may, on occasion, lead to unrealistic demands on the part of the patient. If imperfections after the initial surgery are negligible, it is difficult to justify surgical correction. When a secondary corrective operation is to be undertaken, a detailed examination of the nose, discussion of the findings, and a detailed review of the goals of the operation are necessary to ensure a successful outcome.

Pathologic Anatomy

Imbalances in facial features may contribute to a problem perceived by the patient as purely nasal. If not addressed, these imbalances may result in even more patient dissatisfaction after secondary procedures. Examples include prominence or retrusion of the forehead; malar, maxillary, or mandibular anomalies; and various chin or submental disharmonies.

Systematic examination of the nose, including the internal aspect of the nose, is essential. The texture of the nasal *skin* should be evaluated. Thick, sebaceous skin often prevents optimal nasal definition. Patients with excessively sebaceous skin may benefit from a short to intermediate treatment course with a retinoid. If so treated, the operation should be postponed to avoid hypertrophic scarring or excessive bleeding.

Secondary rhinoplasty patients are more likely to have *telangiectasia* of the nasal skin, which may increase with repeat procedures. In addition, the thickness of the *dorsal skin* should be noted because markedly thin skin may result in visibility of small imperfections on the dorsum of the nose. Skin thinness may be intrinsic to the patient or may have resulted from dissection during the initial operation in a subcutaneous, rather than subperiosteal plane. Correction involves soft tissue augmentation with either a dermal or fascial graft.

Scars from the previous operation should be evaluated because their appearance may be indicative of the patient's healing potential after the secondary rhinoplasty.

A poorly defined *radix* is common after dorsal reduction and may result in an unnatural slope of the nose in the profile view (Fig. 1).

A residual *dorsal hump* may be present, specifically caudally. Alternatively, the dorsum may have been overresected.

Malpositioned *nasal* bones or upper lateral cartilage may interrupt the lines from the medial aspect of the brow to the tip. A step deformity may exist if the osteotomies were performed too anteriorly on the nasal bones.

The *upper lateral cartilages*, which control the shape of the middle nasal vault, may be asymmetric

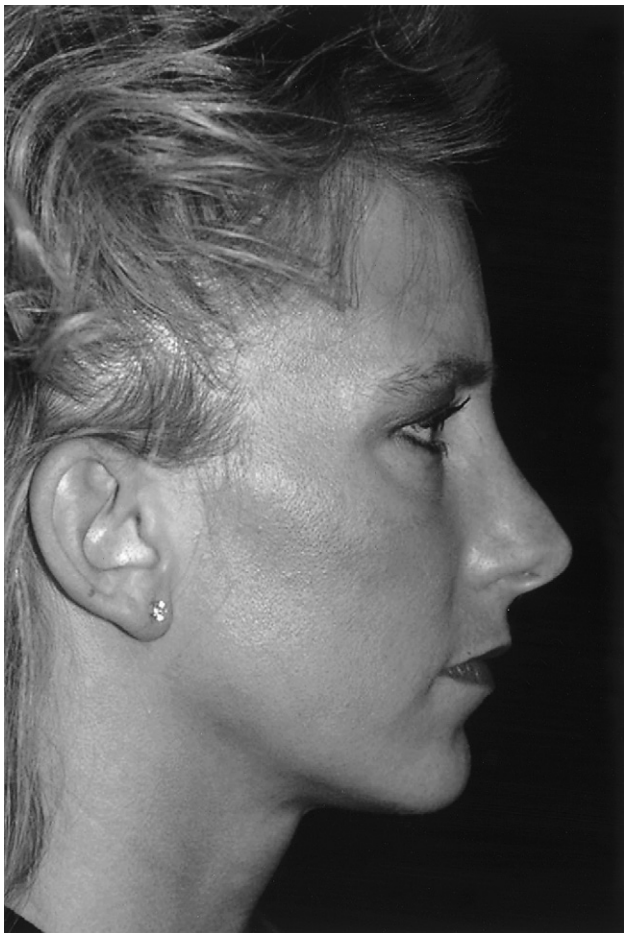

Figure 1 • A profile view of a patient after primary rhinoplasty illustrates underresection of the radix.

or overresected. This asymmetry is often due to an anterior septal deviation, although it may (less frequently) be caused by unilateral overresection of the upper lateral cartilage with subsequent displacement and malalignment. Furthermore, overresection of the upper lateral cartilages may result in collapse of the middle nasal vault and an inverted V deformity (Fig. 2).

The *nasal cavities* should be fastidiously examined to evaluate functional disturbances of the airway (see the "The Nasal Airway" chapter).

Supratip fullness may be present and is often due to inadequate resection of the caudal dorsum. Overresection of the caudal dorsum can also result in dead space in the supratip area, which fills with blood and can lead to excessive scarring and fullness in the area, especially in patients with thicker nasal skin. In addition, inadequate tip projection can contribute to a supratip deformity.

Abnormalities of the nasal tip can range from minor imperfections to significant tip dysmorphology. The most common secondary tip deformity is an underprojected tip.

Weakness of the *lower lateral cartilage* may affect the alar rims. The alar rims may be retracted or even notched because of inadequate cartilage support by the lateral crura of the lower lateral cartilage.

Abnormalities of the *columella* can be inherent to the columella or secondary to the position of the alar rim. Optimal columellar show in the lateral view is between 3 and 4 mm. Patients with a wide nasolabial angle and suboptimal tip rotation are prone to excessive columellar show or a hanging columella (Fig. 3). Conversely, the columella may be retracted, creating the unattractive appearance of hooding by the alar rims in the profile view. Less frequently, this finding is secondary to alar rims that extend too far caudally and obscure the columella.

Asymmetry of the *nostrils* and an abnormal nostril shape can result from widely spaced medial crura footplates, a deviated nasal spine, or weakness of the lower lateral cartilage. The domes should be examined for asymmetry and malposition.

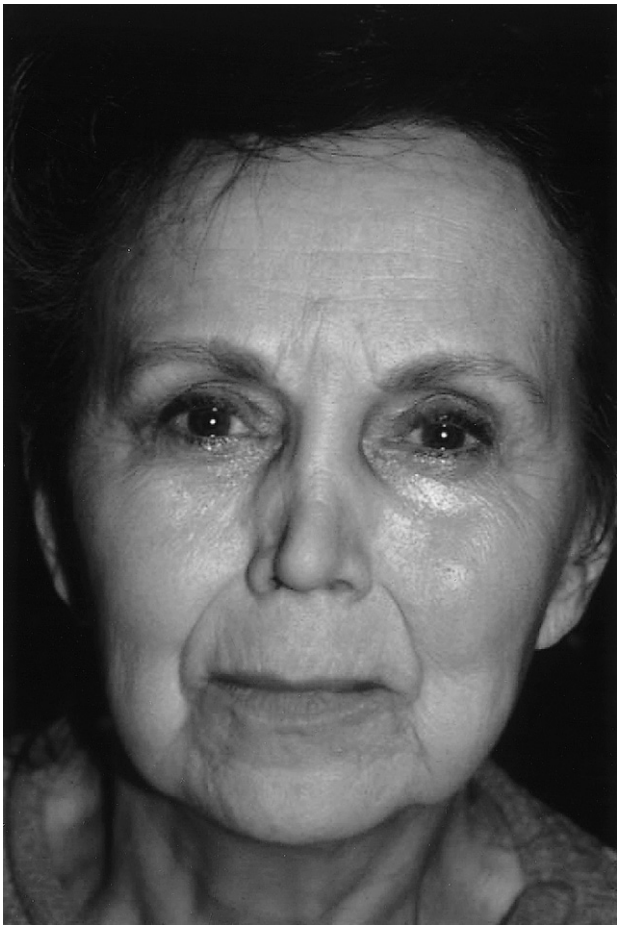

Figure 2 • Collapse of the middle nasal vault and an inverted V deformity after primary rhinoplasty, as well as overresection of the upper lateral cartilage. (From Guyuron B, Michelow BJ, Englebardt C: Upper lateral splay graft. Plast Reconstr Surg 102:2169-2188, 1998.)

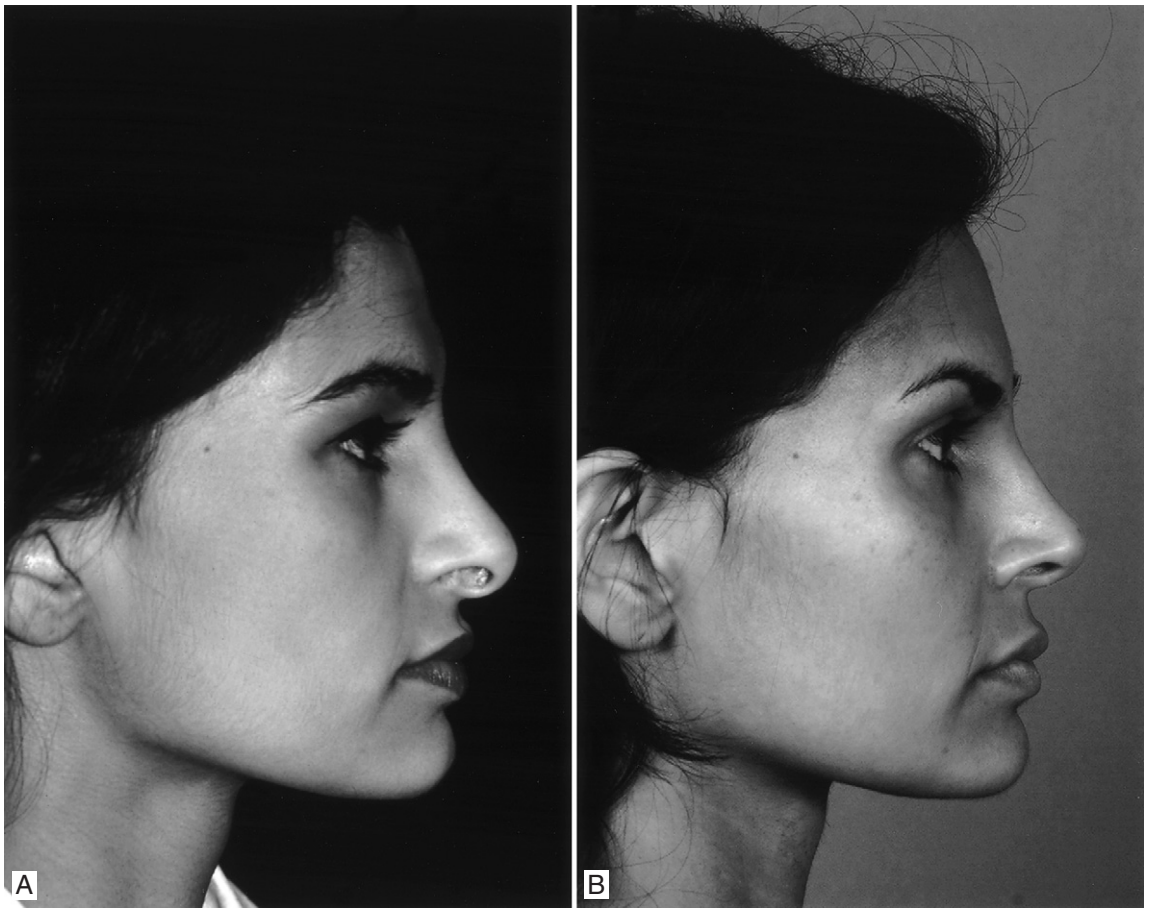

Figure 3 • A, Appearance after primary rhinoplasty with inadequate tip rotation and a hanging columella. **B,** Correction of the columella deformity after secondary rhinoplasty.

The *alar base* may be wide, flared, or too thick and thereby result in a disproportionate appearance of the caudal aspect of the nose.

Diagnostic Studies

Life-sized photography is the most useful preoperative planning tool. Analysis of life-sized photographs defines subtle as well as obvious problems of the nose and allows for accurate surgical planning. Occasionally, symptoms justify study of the paranasal sinuses by computed tomography.

Reconstructive Goals

Reconstructive goals in secondary rhinoplasty are patient specific. The following abnormalities should always be addressed in surgical planning:

1. An underprojected or overprojected radix
2. A residual dorsal hump or overresected dorsum
3. Asymmetric or depressed nasal bones
4. Middle nasal vault collapse or nasal airway obstruction (or both) as a result of septal deviation, turbinate hypertrophy, internal valve collapse, or synechiae
5. A supratip deformity
6. Tip asymmetry, underprojection or overprojection of the tip, excess tip width, or a "pinched" tip deformity
7. Collapse of the lower lateral cartilage
8. Columellar retraction or excess, a wide columella, or caudal protrusion of the columella
9. Nasal spine or medial crura footplate abnormalities
10. Excessive width and asymmetry of the alar bases
11. A retracted alar rim

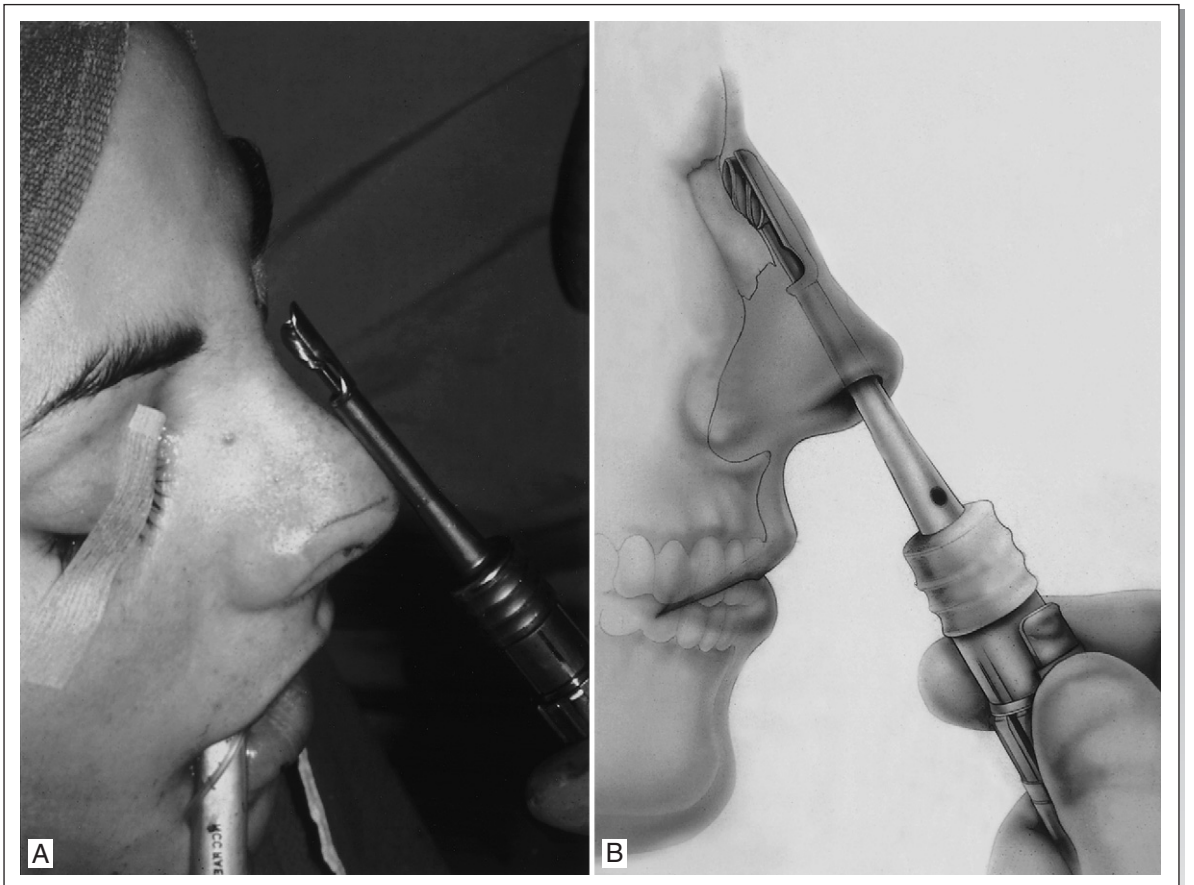

Figure 4 • A, A guarded bur and the vector of its introduction into the nose. **B,** Position and vector of the guarded bur intranasally.

Treatment

The surgical exposure required in large measure determines whether an open or closed approach should be used in secondary rhinoplasty. Minor revisions can be performed through an endonasal (closed) approach, but major revisions are best performed via an open technique.

Problems and Their Treatment

OVERPROJECTED RADIX. The ability to attain only limited exposure with a rasp or an osteotome makes it difficult to achieve optimal radix contour and position at the nasofrontal junction. This difficulty often results in an overprojected radix postoperatively. A guarded bur can reduce the excess bony protrusion of the nasion safely and effectively (Fig. 4).

UNDERPROJECTED RADIX. If an underprojected radix is continuous with an underprojected dorsum, the radix can be addressed with the same dorsal cartilage graft made longer and also shaped to address the radix deficiency. If the dorsum is at the optimal level, however, a smaller cartilage graft may be shaped, gently crushed, and placed in a soft tissue pocket created at the radix in the subperiosteal plane.

RESIDUAL DORSAL HUMP. A residual dorsal hump is best reduced by using a rasp with incremental reduction. Use of an osteotome may result in overresection of the dorsum.

OVERRESECTED DORSUM. An overresected dorsum is best treated with a septal cartilage graft because of its flat panel shape, thickness, and long-term reliability. The graft should be cut into a fusiform shape so that the caudal portion of the graft is narrower than the central segment. The septal graft is gently crushed and the borders beveled. If greater thickness is needed for the reconstruction, two layers of the graft may be used. If, according to operative reports, the majority of the septal cartilage has been removed previously, one should still consider exploration of the septum for the availability of residual cartilage. There may be just enough for a dorsal graft.

In the event that insufficient septal cartilage is available, conchal cartilage is the second choice for minor dorsal deformities. However, this residual cartilage may prove inadequate in both size and shape for major dorsal deformities. In this situation, a costochondral rib graft remains a viable option. Costochondral cartilage is prone to warp when uneven surface shavings are applied. Carving out a core segment of the costochondral cartilage helps to avoid long-term warping. Alternatively, a Kirschner wire can be buried in the core of the cartilage graft to maintain its shape. The graft should be narrower and thinner cephalically and caudally, and all of the margins should be beveled. Alloplastic material should not be used.

ASYMMETRIC NASAL BONES. An osteotomy is often required for correction of asymmetric nasal bones. Lateral osteotomies can be performed either percutaneously or through a stab wound in the nasal vestibule. If the first osteotomy was performed too anteriorly and resulted in a step-off on the nasal sidewall, a new posterior osteotomy can correct the deformity.

Regardless of the type of osteotomy, the soft tissues and periosteum surrounding the nasal bones are less mobile because of the formation of scar tissue after the primary operation. In patients with multiple trauma or previous osteotomies, it may be necessary to perform medial, anteroposterior, and lateral osteotomies, all of which can result in more extensive calcification and scarring.

NASAL BONE DEPRESSION. Nasal bone depression can be addressed without an osteotomy if the depressed bone and attached upper lateral cartilage do not compromise the airway. The depression can be camouflaged with a cartilage graft. If the airway is narrowed, the nasal bone can be outfractured.

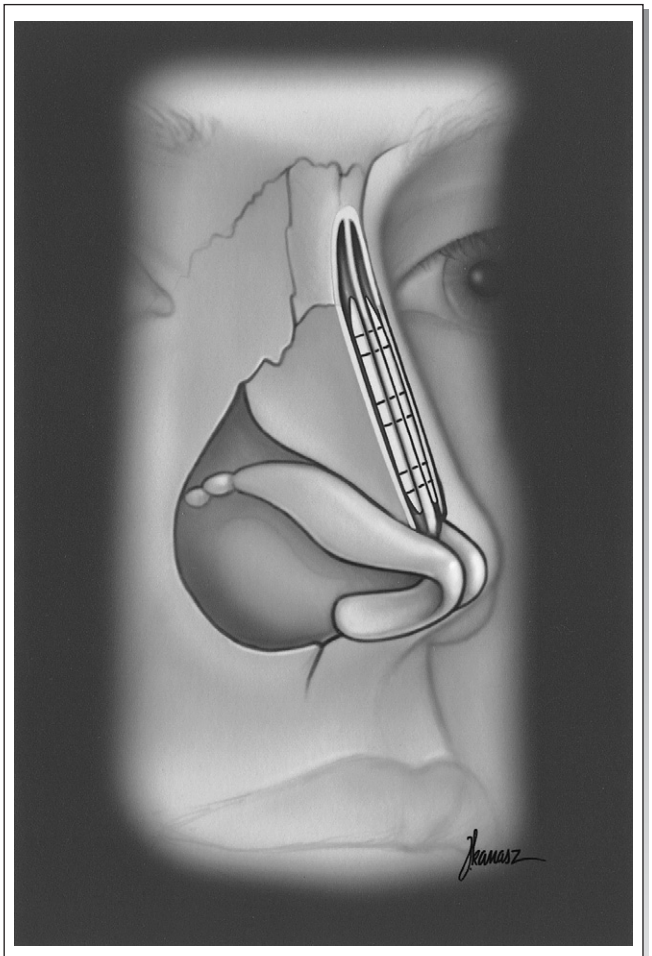

Figure 5 • Illustration depicting the length and position of spreader grafts. Spreader graft length is equivalent to the distance from the junction of the nasal bones and upper lateral cartilage to the caudal end of the upper lateral cartilage.

UPPER LATERAL CARTILAGE COLLAPSE. Collapse of the upper lateral cartilages can cause airway obstruction by narrowing the internal nasal valves and can result in a noticeable V-shaped deformity of the middle nasal vault (see Fig. 2). In milder cases, the collapse can be addressed with spreader grafts. Through an open approach, the spreader graft is placed between the upper lateral cartilage and the septum in a submucoperichondrial position. A piece of cartilage with a length equivalent to the distance from the junction of the nasal bones and upper lateral cartilage to the septal angle is used as a spreader graft (Fig. 5). If necessary, two pieces of cartilage can be overlapped to create a greater distance between the septum and the upper lateral cartilage. The spreader grafts, whether unilateral or bilateral, should be sutured in place with horizontal mattress sutures to secure the graft to the septum.

For major collapse of the upper lateral cartilage, a splay graft produces an effect similar to that of a spreader graft, but in a more predictable and potent manner. A splay graft is used when the upper lateral cartilages are too short and attenuated, thus reducing the effectiveness of the spreader graft. A splay graft spans the dorsum of the nose by arching from the medial aspect of one upper lateral cartilage to the medial aspect of the contralateral upper lateral cartilage.

The mucoperichondrium on the medial aspect of each upper lateral cartilage is elevated up to its posterior aspect to create pockets for the splay graft (Fig. 6). More flexible cartilage, such as conchal cartilage, is the ideal donor. Septal cartilage can also be used if it is sufficiently supple. The septal cartilage may need to be crushed gently to render it more flexible and avoid excessive widening of the lower portion of the nose. The leading anterior edges of the upper lateral cartilages are advanced anteromedially and sutured to the graft to create sufficient strength of the internal valve and to ensure the desired contour.

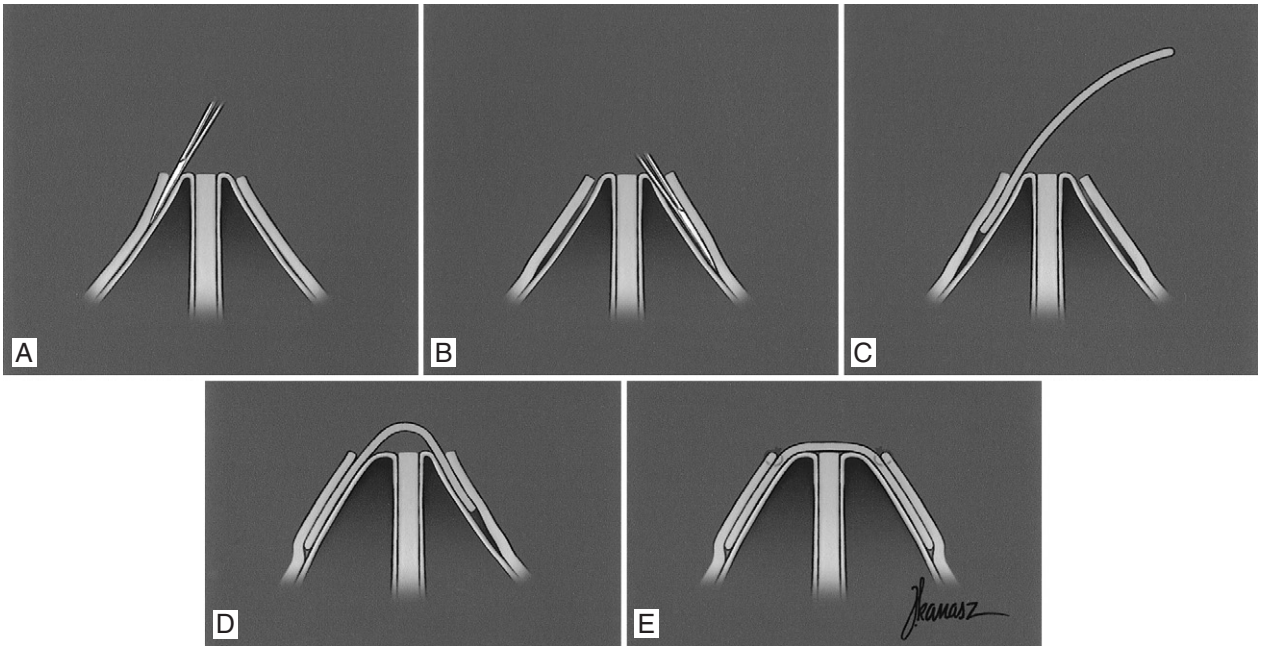

Figure 6 • Splay graft. **A** and **B,** The mucoperichondrium on the medial aspect of each upper lateral cartilage is elevated up to its posterior aspect to create pockets for the splay graft. **C** and **D,** Once the cartilage has been shaped, it is inserted into the pockets that have been created. **E,** The cartilage is sutured to the septum with 5–0 PDS sutures.

SUPRATIP DEFORMITY. A supratip deformity results from overresection or underresection of the caudal dorsum or inadequate tip projection. When the caudal dorsum is overresected, a dead space is created that may fill with blood and protein-rich fluid. This fluid results in scar tissue formation and excess supratip fullness. In a patient with thick nasal skin, the same sequence of events can occur in the absence of caudal dorsum overresection because the skin may be too rigid to conform to the newly shaped underlying caudal dorsum. A supratip suture may be used to close the dead space in both these scenarios. After the nasal sleeve is redraped, the supratip break is marked with blue dye by passing a 25-gauge needle through the skin over the underlying caudal dorsum. The suture approximating the supratip skin to the caudal dorsal angle is tied lightly to avoid necrosis of the skin.

When a supratip deformity is due to underresection of the caudal dorsum during the primary operation, a supratip suture can be used to eliminate any resultant dead space after removal of the excess dorsal septum. If insufficient tip projection is causing the supratip deformity, a tip augmentation technique may be selected.

NASAL TIP DEFORMITIES. The most common deformities in secondary rhinoplasty are nasal tip deformities. Direct visualization (open approach) of the tip structures is necessary for a final diagnosis and optimal correction.

The focus of nasal tip correction centers on the use of sutures for stabilizing and reshaping the cartilage, minor cartilage excision, and the addition of cartilage grafts. Eight common tip suture techniques can be used alone or in conjunction with other approaches for correction of the nasal tip:

1. A medial crural suture approximates the medial crura and strengthens support of the nasal tip. There is also a varying degree of narrowing of the columella.
2. An interdomal suture approximates the domes and can equalize asymmetric domes.
3. Transdomal sutures narrow the domal arch while pulling the lateral crus medially.
4. A lateral crural suture enhances the concavity of the lateral crus and can reduce the interdomal distance.
5. A medial crural anchor suture increases tip projection, rotates the tip cephalically, and retracts the columella.
6. A tip rotation suture shifts the tip cephalad and retracts the columella.
7. A medial crural footplate suture approximates the footplates, narrows the columella base, and improves the undesirable shape of the nostrils.
8. A lateral crural convexity control suture affects the shape of the lateral crus.

Common problems in the tip area include inadequate tip projection, excessive tip projection, and

excess or suboptimal tip width. Inadequate tip projection can be addressed with the previously described suture techniques, with tip cartilage grafting alone, or with a combination of the two measures. The size of the lobule and columella is a key factor in deciding which technique should be used. For a patient with a small infratip lobule and an underprojected tip, a tip graft or a suture technique commonly narrows the tip and provides more projection. If the columella is short, a columellar strut or anchor suture would be a better choice. A combination of these techniques usually provides the best results.

Excessive tip projection may be addressed by resection of the medial crura footplates or the end portion of the lateral crura, or a combination of both. One must take into account the tripod effect. For a short and overprojected tip, removal of the footplates reduces tip projection and causes caudal rotation. When the nose is long, resection of the posterior portion of the lateral crura reduces tip projection and shortens the nose. On rare occasions pathologically deformed domes may be resected to alleviate this problem. Finally, excessive tip width can be altered with a combination of the previously described suture techniques if it is believed that scarring and decreased cartilage flexibility will require some trial-and-error attempts before the optimal result is achieved.

LOWER LATERAL CARTILAGE COLLAPSE. In patients with collapse of the lower lateral cartilage, a lower lateral cartilage strut can be used to remedy the deformity. An appropriately sized piece of cartilage is placed on the undersurface of the lower lateral cartilage such that it spans from the piriform aperture to the ipsilateral dome. The strut is sutured in place with 6–0 PDS suture.

COLUMELLAR RETRACTION. If the columella appears to be retracted, several similar options exist to enhance it. An interposition cartilage graft may be added to the caudal septum, in effect protruding the overlying medial crura and therefore the columella. However, this measure may move the tip caudally as well. Alternatively, a columellar cartilage strut may be used. The strut is placed between the medial crura and extends from the anterior nasal spine to the anterior septum. The columella advances caudally, but tip projection also increases. If this is not the desired effect, only a cartilage graft should be used because it does not affect the domes or the alar rim but only increases the caudal projection of the columella.

EXCESSIVE CAUDAL PROTRUSION OF THE COLUMELLA. In patients with excessive caudal protrusion of the columella (see Fig. 3), a rectangular-shaped piece of cartilage can be resected from the cartilaginous caudal border of the septum. A proportionate amount of lining is also removed from the membranous septum parallel to the columella. Cephalic rotation of the nasal tip would result if the resected segment were triangular and based anteriorly. In an underprojected nose with excessive columellar show, the evagination technique may be used. This technique involves overlaying the footplates and medial crura and fixing them over the caudal septum in a more anterior position.

NASAL SPINE AND MEDIAL CRURA FOOTPLATE ABNORMALITIES. These abnormalities affect the base of the columella and can be addressed through a hemitransfixion incision. Deviation of the base of the columella may be due in part to an asymmetric anterior nasal spine that affects not only the base of the columella but also the caudal aspect of the nose. Greenstick fracture of the nasal spine into a more symmetric midline position can alleviate this source of caudal nose deviation. With a wide columellar base and underprojected tip, the footplates of the medial crura can be approximated by a suture after resecting the intervening soft tissues. These steps narrow the columellar base and strengthen nasal tip support. In an overprojected nasal tip with a wide columellar base, correction would involve resection of the footplates and approximation of the medial crura.

ABNORMALITIES OF THE ALAR BASE. Alar base abnormalities are common. Wide alar bases are encountered most frequently, but varying degrees of asymmetry exist. Alar base resection can be undertaken by removing tissue from either the nostril sill or the lateral aspect of the alar base. A combination of the two can be performed simultaneously, thus reducing nostril size and alar base width. With medial movement of the alar base, there is also caudal relocation of the alar rim. A *retracted alar rim* is addressed by using a lower lateral crura strut, which advances the rim caudally. In more notable retraction, V-Y advancement of the vestibular lining and rim can be undertaken. A composite chondrocutaneous graft may be used to advance the alar rim caudally.

Figures 7 and 8 illustrate representative case studies.

Postoperative Care and Complications

Postoperatively the nose is secured with tape. This tape should help the skin envelope adapt to the underlying osteocartilaginous framework. It should help eliminate dead space, especially in the supratip area. A light splint is used to protect the nose and maintain the position of the nasal bones.

Routine complications such as infection, bleeding, and numbness should be discussed, as well as possible problems specific to the patient's care. The

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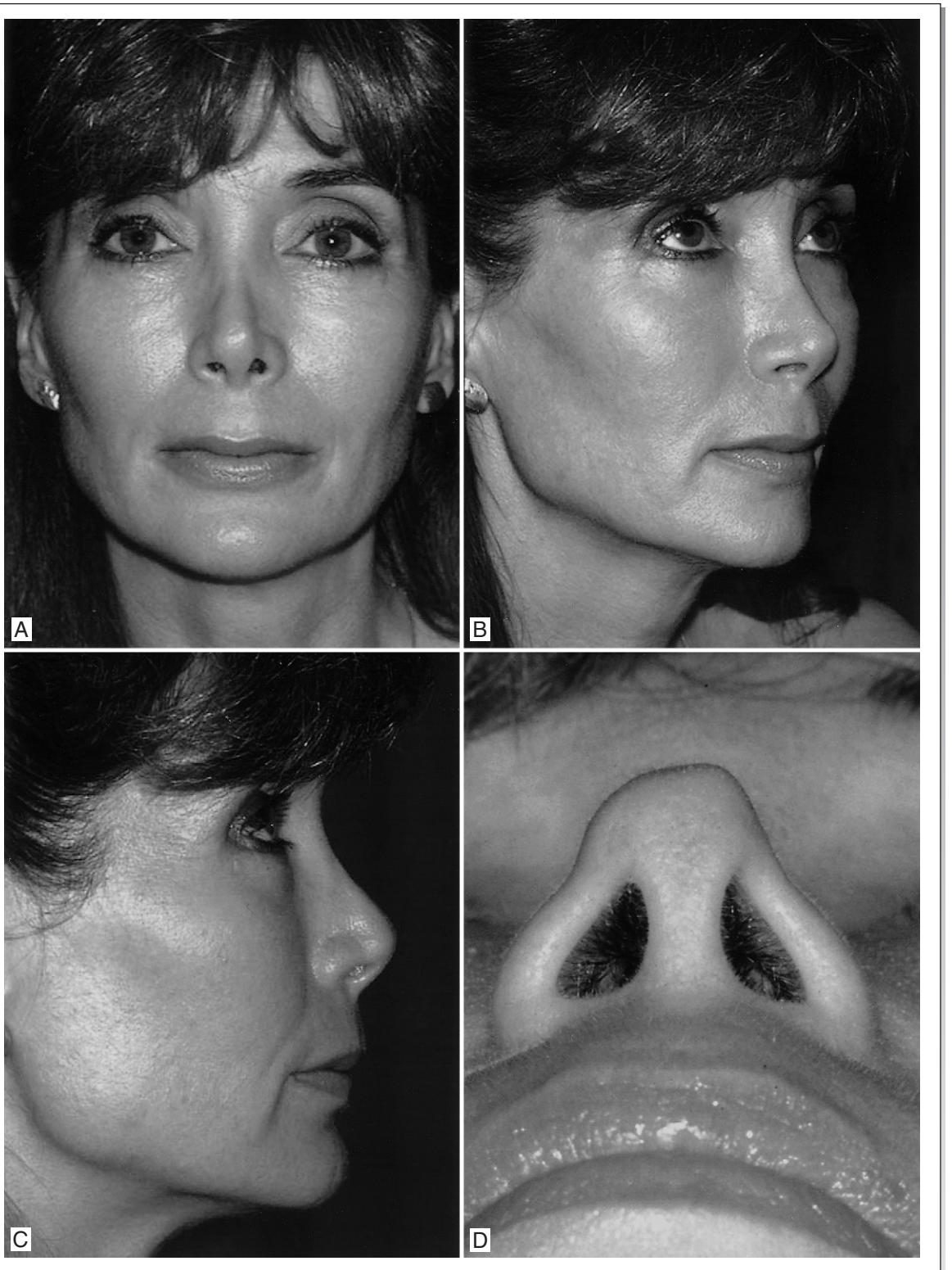

Figure 7 • A-D, Patient who had previously undergone rhinoplasty but has a persistent low radix, poor tip definition with inadequate tip support, and midvault weakness on examination.

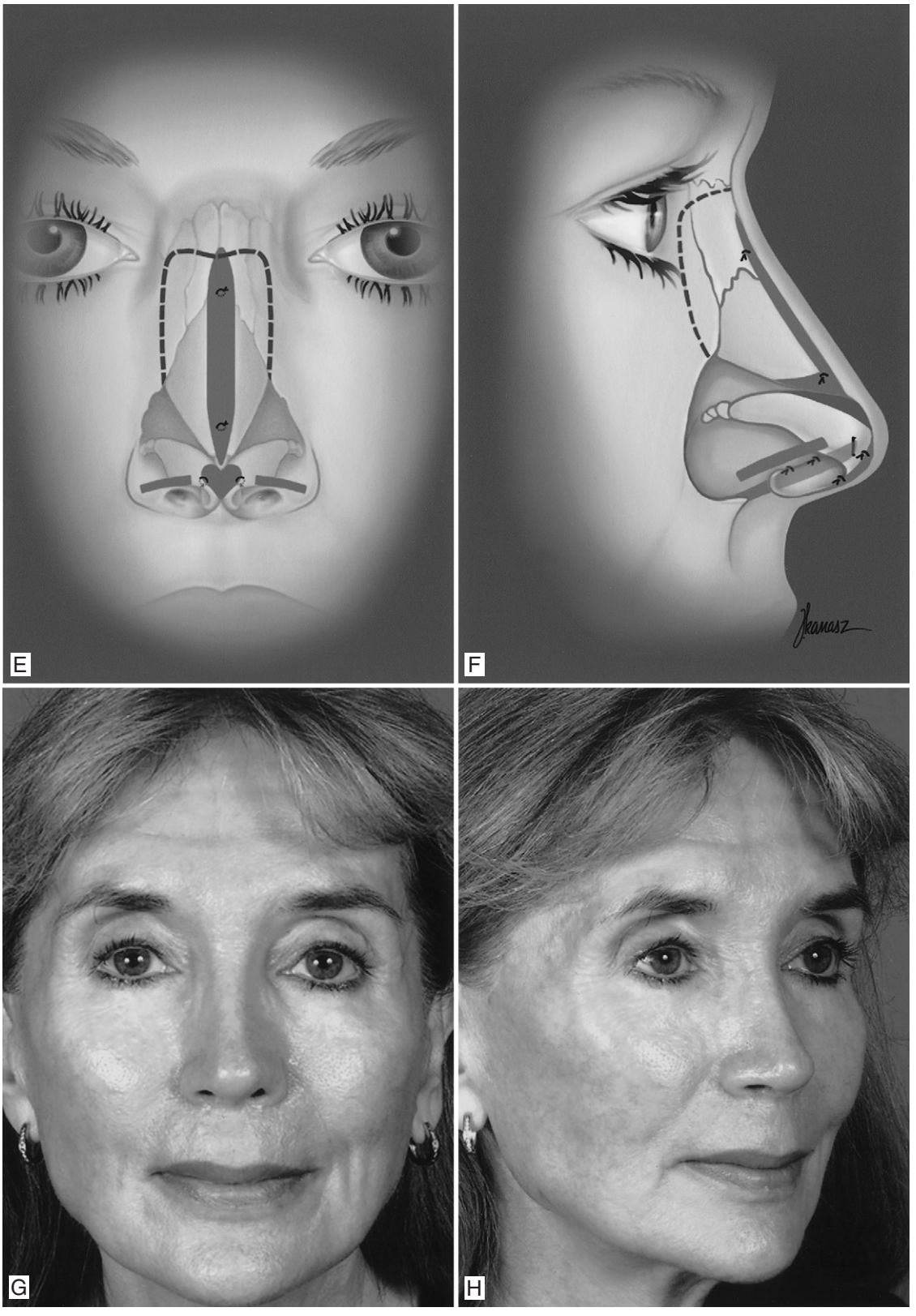

Figure 7, cont'd • E and **F,** A cartilage graft to the radix and upper dorsum, spreader grafts, a tip graft, a columellar strut, and medial crura sutures were placed. **G-J,** Approximately 1 year after secondary open tip rhinoplasty. (From Guyuron B, Varghai A: Lengthening the nose with a tongue-and-groove technique. Plast Reconstr Surg 111:1533-1539, 2003.)

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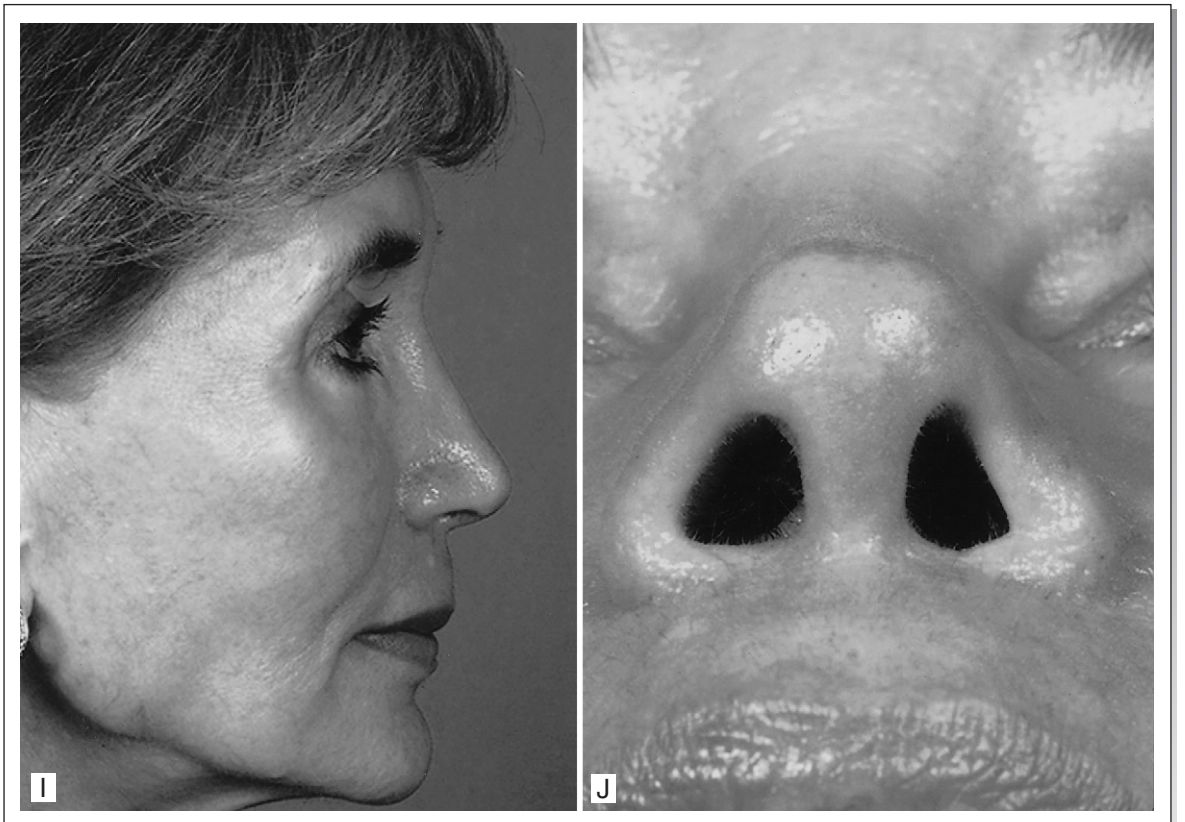

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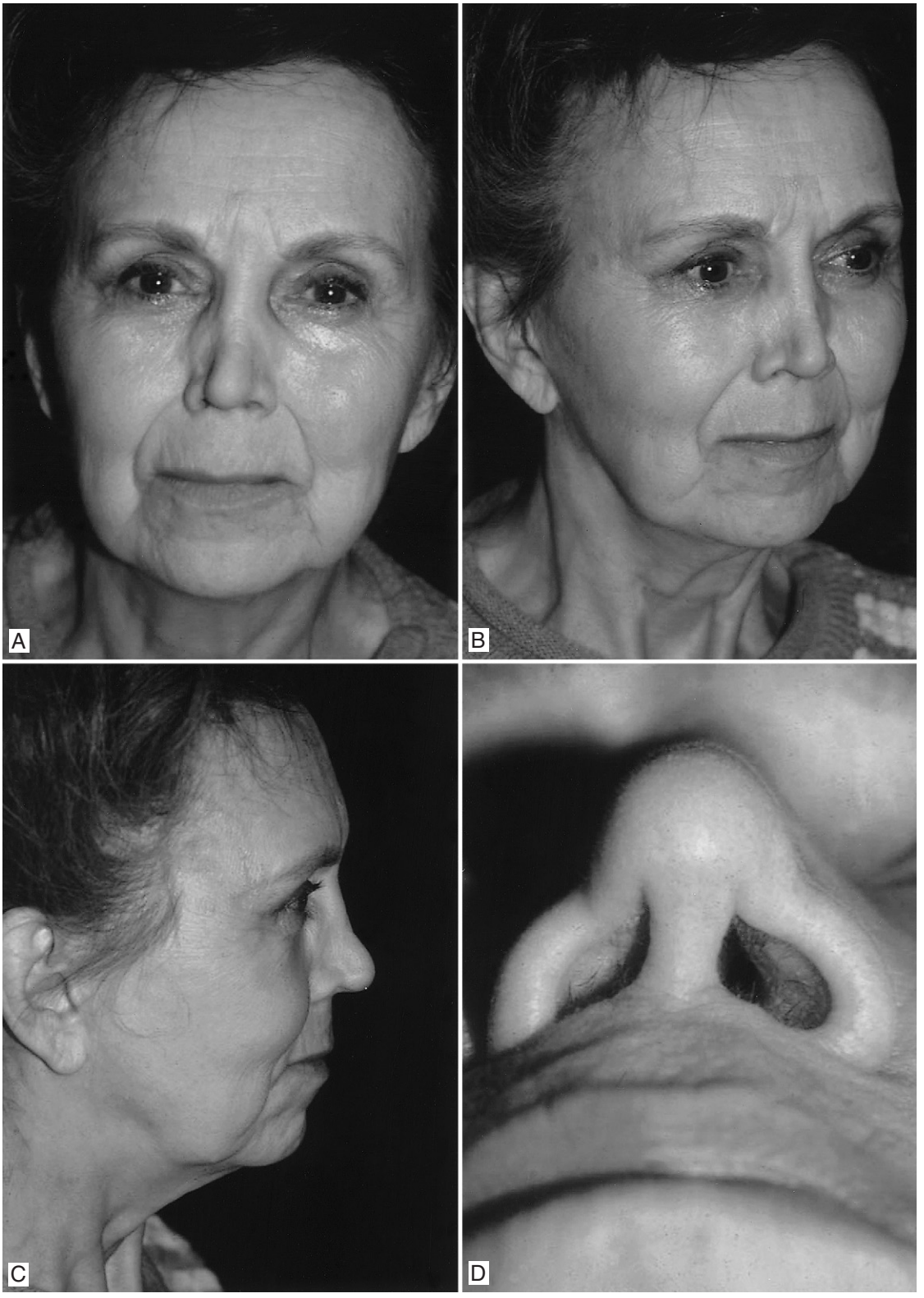

Figure 8 • A-D, Significant deformities of the dorsum, nasal bones, and lower lateral cartilage and nasal length deficiencies are noted after primary rhinoplasty. *Continued*

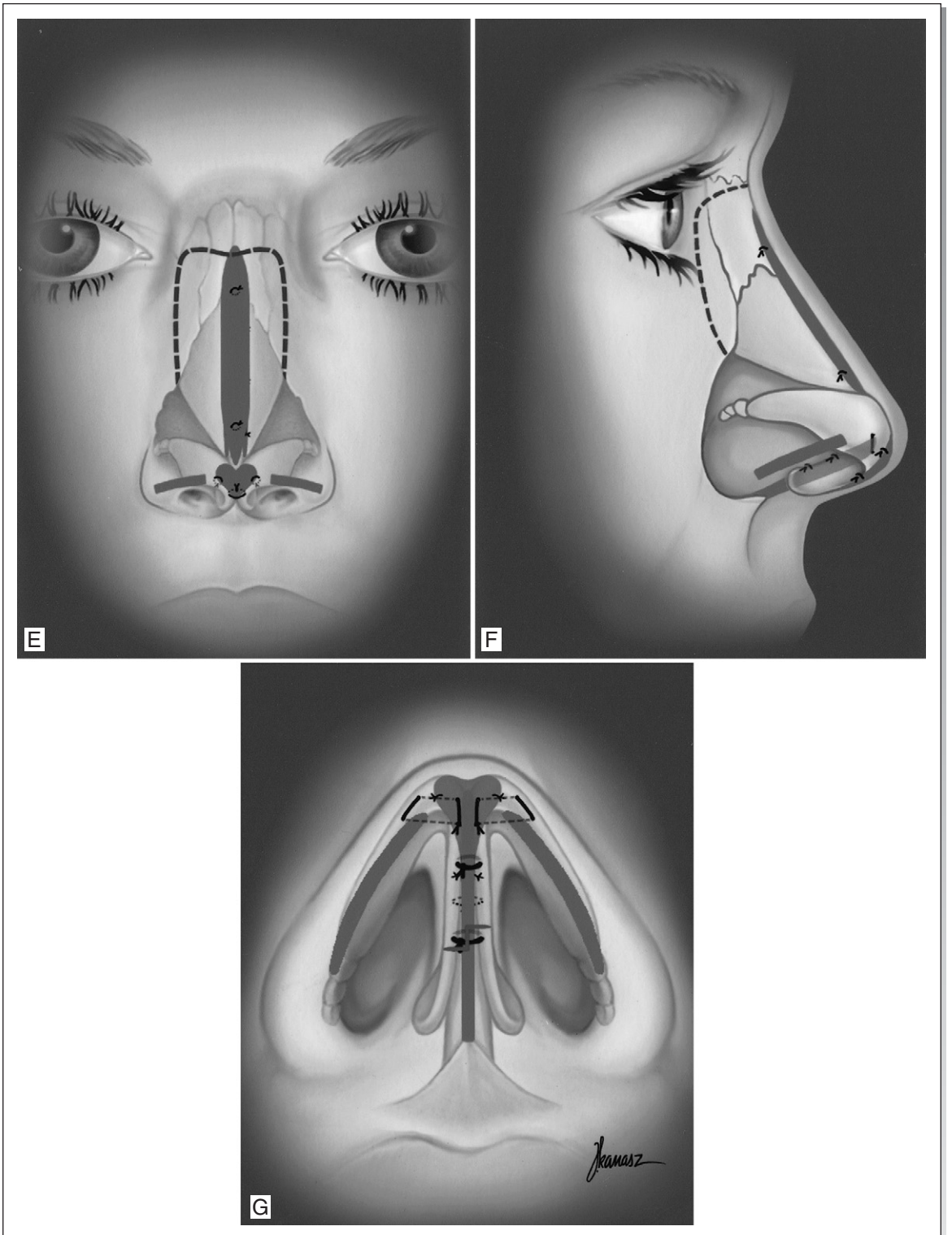

Figure 8, cont'd • E-G, Technique of open secondary rhinoplasty with lateral osteotomies, a dorsal graft, a tip graft with transdomal sutures, and alar rim grafts with the use of a tongue-and-groove technique.

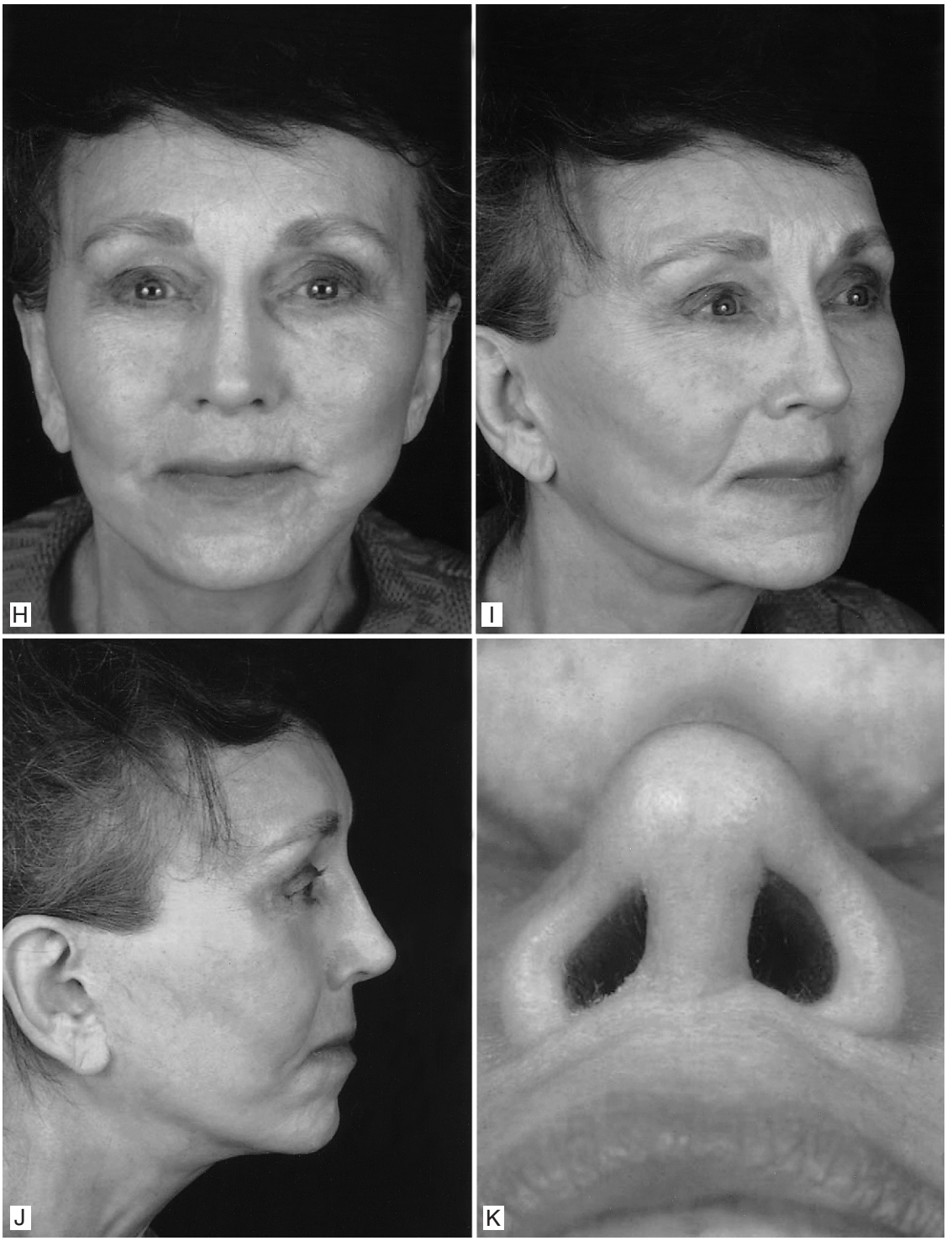

Figure 8, cont'd • H-K, Postoperative appearance. (From Guyuron B, Michelow BJ, Englebardt C: Upper lateral splay graft. Plast Reconstr Surg 102:2169-2188, 1998.)

limited supply of septal cartilage, the potential need to harvest cartilage from other sites (namely, conchal and costal cartilage), and potential complications associated with each site should be discussed with the patient. Patients undergoing secondary rhinoplasty have a higher likelihood of telangiectasia, increased scar tissue, and skin sleeve shrinkage. Abnormal healing should be discussed with the patient, especially those with documented wound-healing abnormalities. Despite all efforts to avoid such an outcome, the potential for decreased nasal-breathing capacity should be discussed with the patient. Challenging secondary rhinoplasties will most likely require future revision to achieve the optimal result.

Pearls And Pitfalls

- Define the nasal deformity and establish goals in a systematic manner.
- Prepare the patient for a less than perfect outcome.
- Previous operative reports and preoperative conjectures may not always correlate with intraoperative findings, and this problem can be compounded by the presence of extensive scar tissue, rigidity, and distorted anatomy.
- Facial structures that impinge on nasal harmony should also be addressed.

SUGGESTED READINGS

Behmand RA, Ghavami A, Guyuron B: Nasal tip sutures part I: The evolution. Plast Reconstr Surg 112:1125-1129, discussion 1146-1149, 2003.

Guyuron B: Secondary Rhinoplasty. Indications, Operations, and Outcomes, vol 5. In Guyuron B (ed): Aesthetic Surgery. St Louis, CV Mosby, 2000, pp 2673-2682.

Guyuron B, Behmand RA: Nasal tip sutures part II: The interplays. Plast Reconstr Surg 112:1130-1145, discussion 1146-1149, 2003.

Guyuron B, DeLuca L, Lash R: Supratip deformity: A close look. Plast Reconstr Surg 105:1140-1151, discussion 1152-1153, 2000.

The Nasal Airway

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The nasal airway is a complex, dynamic anatomic structure. In addition to septal deviation and turbinate hypertrophy, structural integrity of the internal and external nasal valves is a key element of airway patency. Detailed preoperative assessment of these four anatomic areas is critical for successful correction of nasal obstruction.

Etiopathogenesis

Nasal airway obstruction can involve an interplay between several reversible and nonreversible components. Reversible factors are related to the normal nasal cycle or to mucosal edema secondary to allergic or vasomotor rhinitis. Nonreversible anatomic components include a deviated septum, enlarged bony turbinate, nasal polyps, and weakened nasal valves.

The cause of the perceived symptom of nasal obstruction is occasionally related to a patient's poor understanding of the normal nasal cycle. Patients believe that any restriction to airflow from either nasal passage is a symptom of nasal airway obstruction. Educating patients about alternation in the laterality of nasal airflow is important and can be even more important after surgery. Patients with long-standing nasal obstruction may also be unfamiliar with the sensation of restored nasal airflow after successful surgery.

Classic reductive rhinoplasty that emphasizes *removal* or *reduction* of nasal tissue (or both) is a common cause of iatrogenic nasal airway compromise because the support of the nose is weakened. Reductive maneuvers can destabilize the nasal valve and lead to significant nasal valve compromise.

Nasal obstruction was previously considered synonymous with either septal deviation or turbinate hypertrophy. As understanding of nasal function has become more sophisticated, it is now known that these conditions are not the only cause of obstruction. The stability and strength of the lateral nasal wall and external nasal valve can also greatly affect a patient's ability to breathe through the nose. In some patients the patency and integrity of the internal valve may be the most important aspect of the nasal airway. Conversely, a deviated septum may in fact have much less effect on airway patency than previously thought. Moreover, approximately 18% of nasal septa in the general population are "deviated."

In some patients, the external valve has also been shown to be an isolated cause of nasal obstruction, as documented by rhinomanometric studies. In these patients, improvement of external valve function corrects obstructive symptoms.

Pathologic Anatomy

The overall patency of the nasal valve is affected by the septum, turbinates, and internal and external nasal valves. The *internal valve* is defined as the area formed by the junction of the caudal edge of the upper lateral cartilage and the quadrangular cartilage and is bounded inferiorly by the floor of the nose and inferolaterally by the inferior turbinate. The *external valve* is defined as the entrance to the nares and consists of the alar sidewalls and crural cartilage. The size and patency of the nasal airway is dependent on both *static structures* and *dynamic forces*. *Static structures* relate to the position of the septum and the size of the inferior and middle turbinates. *Dynamic elements* include mucosal hypertrophy secondary to allergic or inflammatory rhinitis and the integrity of the internal and external nasal valves. Any congenital or acquired weakness of the upper or lower cartilage or their

investing soft tissue may affect the ability to maintain adequate nasal airflow volume. Understanding the concept of static structures and dynamic forces provides a critical foundation for successful treatment of nasal airway obstruction.

The internal nasal valve is the narrowest cross-sectional passage in the nose. The attachment of the caudal edge of the upper lateral cartilage to the septum forms an angle of approximately 10 to 15 degrees with a resulting cross-sectional area of 55 to 83 mm. During normal respiration, one of the physiologic roles of the nasal valve is to create some element of airflow turbulence. When air is inspired, it is forced to pass through the narrow nasal valve area, which increases its speed and pressure. As the speed increases, laminar flow is disrupted and increasing turbulence results. Some turbulence is necessary for normal nasal function. Turbulence serves to promote contact between air and mucosa and allows the inspired air to be cleansed of particles, humidified, and heated or cooled. When the nasal valve is compromised, exaggerated turbulence occurs and leads to collapse at lower pressure, thereby contributing to the symptom of nasal obstruction.

Diagnostic Studies

Diagnosis of nasal valve obstruction relies heavily on careful consultation. History taking should focus on the laterality of symptoms, association with allergies, patterns of congestion (daytime, nighttime, seasonal, etc.), previous nasal surgery or nasal trauma, and response to medications.

Patients may provide a history of improved nasal breathing after the application of external nasal splints (e.g., Breathe Right). This information can provide partial evidence of internal or external valve collapse because these devices add support to the lateral nasal wall.

The physical examination begins by observation of the patient's breathing. An inexperienced examiner may mistakenly proceed directly to topical decongestion followed by anterior rhinoscopy with a nasal speculum and lose the ability to diagnose airway abnormalities best appreciated by direct observation of the patient's nasal breathing. Observation may also demonstrate valvular dysfunction, which exceeds septal deviation as the primary cause of nasal airflow obstruction. Close inspection of the nose may reveal medial collapse or "pinching" in the supraalar region. Close observation of the supraalar fold (corresponding to the internal nasal valve) often reveals dynamic collapse with exertional nasal breathing. Forceful nasal breathing can serve to identify areas of inherent weakness in the patient's nasal valve. Collapse in the area of the supraalar crease may be observed, and even collapse of the alar rim can be seen. After observing normal nasal respiration and exertional nasal respiration, the patient is instructed to cover one nostril at a time. This maneuver documents which side is predominately contributing to the patient's symptom of nasal obstruction.

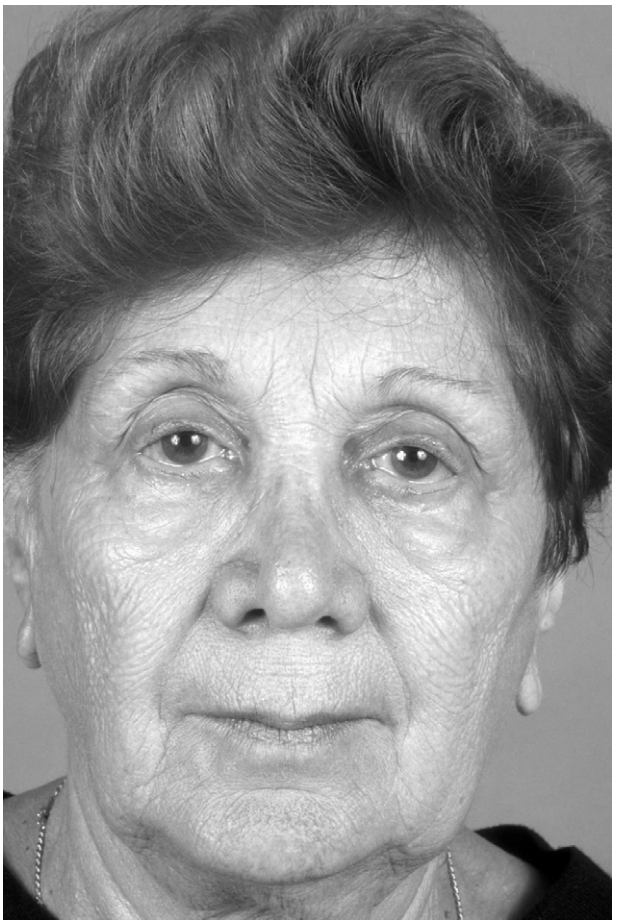

Figure 1 • The classic "inverted V" deformity caused by aggressive dorsal resection and middle vault collapse.

After cosmetic rhinoplasty with overresection of the dorsum and collapse of the middle vault, patients can have an "inverted V" deformity (Fig. 1). This physical sign is strong evidence of internal valve collapse and often indicates patients who will benefit from placement of a spreader graft.

After observation, the intranasal examination should be performed with a light source, nasal speculum, and fine probe. A topical decongestant and anesthetic agent should also be available. The physiologic and structural interrelationships of the intranasal anatomy, especially the four most common culprits of nasal airway obstruction (the septum, turbinates, internal valves, and external valves), are individually examined. An assessment of turbinate size should be made before the application of any decongestant. Routine decongestion of all patients should be performed to avoid missing

Figure 2 • The Cottle maneuver.

pathology such as synechiae or other posterior abnormalities obscured by the turbinates. Either rigid or flexible nasal endoscopy should be performed when necessary to evaluate the more posterior parts of the nasal cavity that can be obscured by significant septal deviation.

Lateralization of the cheek and lateral wall of the nose (the Cottle maneuver) may result in improved nasal airflow and is helpful in distinguishing patients with valvular dysfunction from those with septal or turbinate pathology (Fig. 2).

After the diagnosis of nasal valve collapse has been established, a more accurate determination of the contribution from either the internal or external nasal valves can be obtained by intranasal palpation with a fine probe. During quiet and forced inspiration, lateral displacement of the internal and external valves can produce dramatic improvements in breathing on the affected side and assist in making an accurate diagnosis.

Other techniques such as acoustic rhinometry and rhinomanometry are used occasionally to help diagnose obstruction of the nasal airway. Both have drawbacks and limitations that preclude their routine use.

An evolving problem in the treatment of nasal obstruction is the justification of nasal airway operations to third-party insurance carriers. It is not uncommon for insurance companies to require a preoperative computed tomographic (CT) scan to document septal irregularities. This practice should be discouraged because it adds unnecessary cost to medical care and exposes the patient to a needless dose of radiation. Moreover, the static images generated by CT scanning provide little information about more dynamic problems such as a weak lateral nasal wall or nasal valve collapse.

Intranasal photography is technically difficult, requires costly equipment, and is impractical as a means of photodocumentation.

Goals of Reconstruction

The primary goal of any nasal operation should be to improve the patient's breathing, even in cases undertaken for both functional and aesthetic reasons. The surgeon should accurately diagnose the causative pathology preoperatively and perform the surgery by using a problem-oriented approach.

Treatment

Septoplasty is an often underappreciated operation given its potential complexity. Most septoplasty operations can be successfully performed via an endonasal approach. However, consideration should be given to an external approach for revision septoplasty, correction of severe caudal and high superior deflection, and insertion of spreader grafts.

It is the surgeon's obligation to qualify an operation as a true septoplasty only in patients in whom the operation is performed to improve all deviated parts of the septum (cartilaginous and bony). Incomplete attention to the entire cartilaginous and bony septum is not only surgically inadequate to improve the airway but may also make secondary septal surgery more difficult. Revision septoplasty to correct posterior deflection not addressed in the primary operation is inherently more difficult than primary septoplasty. Dissection between the bilaterally apposed mucoperichondrial flaps can be technically demanding and can place the patient at an increased risk for septal perforation.

If fixed anatomic obstruction is corrected by septoplasty without dynamic forms of nasal obstruction (i.e., valve collapse) being corrected, the patient may experience persistent obstruction. Spreader grafts are particularly useful for correction of internal nasal valve collapse, as demonstrated in patients with an overly narrow middle nasal vault (often seen as an "inverted V deformity" on frontal view). Spreader grafts measure approximately 5 to 15 mm

in length, 3 to 5 mm in width, and 1 to 2 mm in thickness. They are typically positioned from the osseocartilaginous junction to a point just cephalad to the septal angle. In the endonasal approach, precise submucosal tunnels must be developed that are sufficiently tight to prevent shifting of the graft. With an external or "open" approach, the grafts can be directly inserted between the upper lateral cartilage and septal cartilage and sutured in place (Fig. 3). The spreader grafts ensure that adequate middle vault width is established and inferomedial collapse of the internal valve is prevented.

Alar batten grafts are curvilinear cartilage grafts that are placed in a precise pocket at the point of maximal lateral wall collapse or supraalar pinching. The grafts may be fashioned from curved pieces of septal cartilage or from the cavum or cymba concha of the ear. Batten grafts are normally placed in a precise pocket lateral to the lateral crura at the point of maximal lateral wall collapse. This area can be easily identified as a prominent area of lateral wall medialization or pinching (Fig. 4). This is also the area of structural deficiency after rhinoplasty in which excess lower lateral cartilage has been resected. The more concave portion of the graft is positioned medially to counteract the forces of collapse during nasal inspiration. In most cases, alar batten grafts also create fullness or convexity at the site of the graft that tends to decrease as edema resolves and scar contracture compresses and shifts the graft medially.

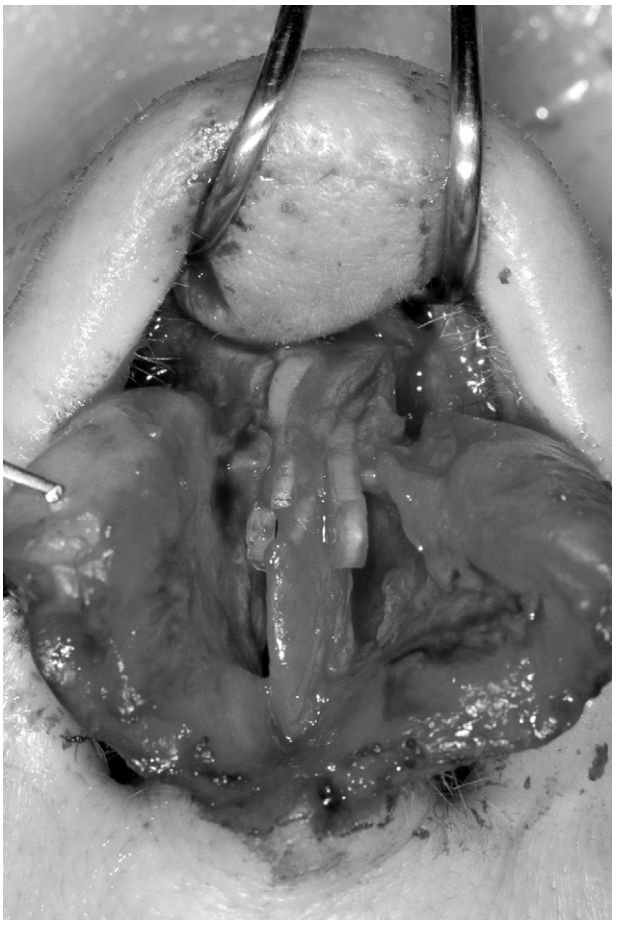

Figure 3 • Spreader grafts: bilateral cartilage strips sutured between the upper lateral cartilage and the quadrangular cartilage.

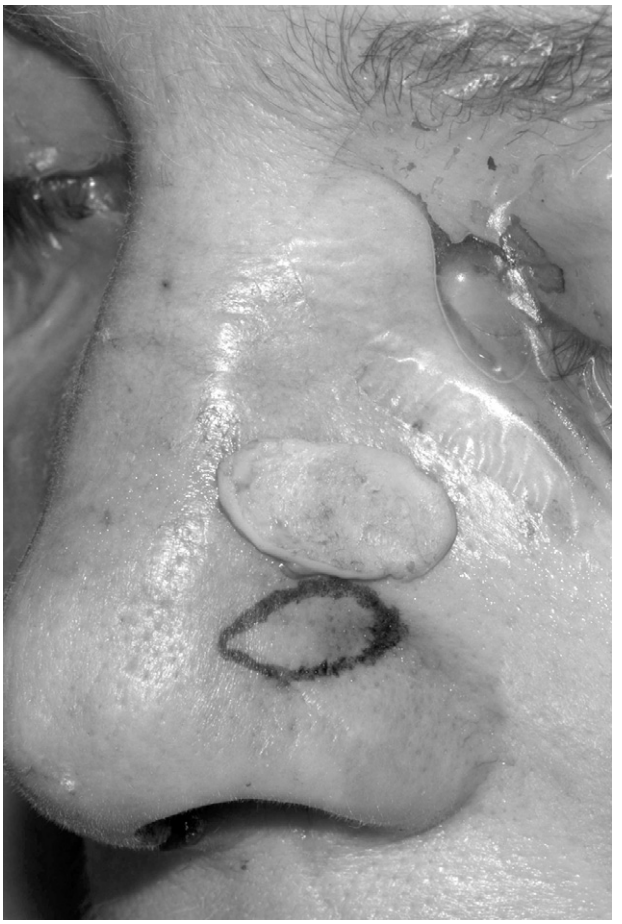

Figure 4 • Alar batten graft and outline of recipient site.

Some patients may present with a widened columella (Fig. 5). Although a widened columella alone is not usually the sole source of nasal obstruction, it can contribute to obstruction when other conditions such as external nasal valve collapse or caudal septal deviation are present. Septoplasty and caudal trimming alone do not correct this condition. There is often excess width of the posterior septal angle; this cartilage can be trimmed to allow the medial crural footplates to medialize. After the soft tissue is removed, the footplates of the medial crura must be brought to the midline with a suture placed bilaterally through the nasal vestibule.

Turbinate hypertrophy may also narrow the nasal airway sufficiently that treatment is required. The

narrowest area of the nasal cavity is in the most anterior aspect of the internal nasal valve. Aggressive inferior turbinate resection beyond its anterior third rarely results in improvement in the cross-sectional area of the nasal cavity. Moreover, resection of the majority of the inferior turbinate places the patient at risk for atrophic rhinitis.

In indicated patients with inferior turbinate enlargement who fail to respond to, or are intolerant of, nasal steroid sprays or oral decongestants, surgical management of the inferior turbinates may be indicated. A number of surgical treatment options exist, but all can be classified into one of three categories, depending on the tissue layer treated. The first is *submucous resection* of the inferior turbinate bone, which often results in expansion of the nasal airway. The turbinate is incised along its anterior head or inferior margin, and the submucosal bone is resected. A second option is *destruction* of the inferior turbinate mucosa, typically performed with one of a variety of lasers or exfoliative chemicals. These treatments destroy the epithelium and underlying glandular structures, thereby causing fibrosis of the layers. Both resection and destruction can put patients at significant risk for atrophic rhinitis, particularly those living in drier, cooler climates. The third and more recent approach is to target the *vascular erectile* tissue of the inferior turbinate while leaving the overlying mucosa largely undisturbed. This technique can be performed either with electrical energy (needle point cautery or radiofrequency ablation) or with a soft tissue shaver. The latter yields a predictable and immediate reduction in size of the inferior turbinate while preserving its humidification and secretory functions.

Middle turbinate enlargement rarely contributes to nasal airway obstruction. The increase in size is typically due to pneumatization of the anterior and inferior portion of the turbinate and is known as concha bullosa (Fig. 6). Such a phenomenon is usually seen in association with septal deflection to the contralateral side, and maximization of the nasal airway may require addressing both conditions. Correction of the septal deflection alone might result in lateralization of the enlarged middle turbinate and compromise of anterior ethmoid and maxillary sinus outflow. Simple crushing of the concha bullosa may instead lead to mucociliary obstruction. Removal of one of the lamellae of the concha is consequently the preferred technique.

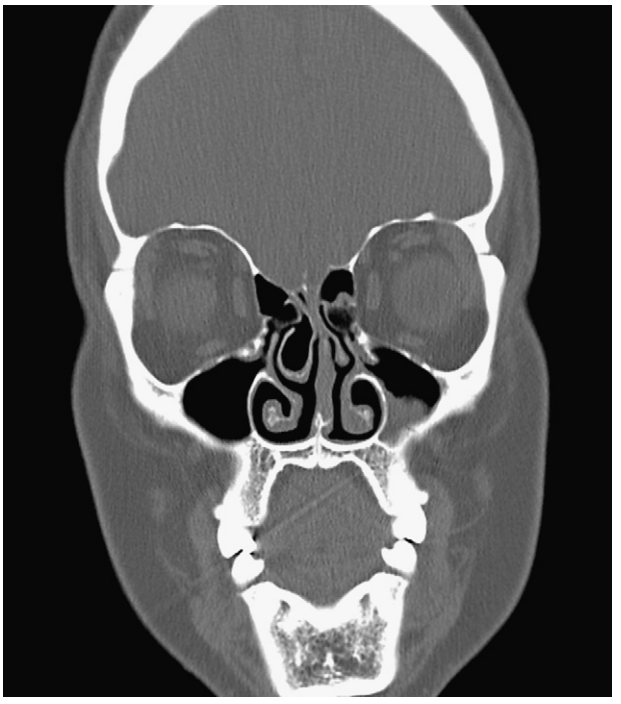

Figure 6 • Coronal computed tomographic image demonstrating concha bullosa involving the right middle turbinate.

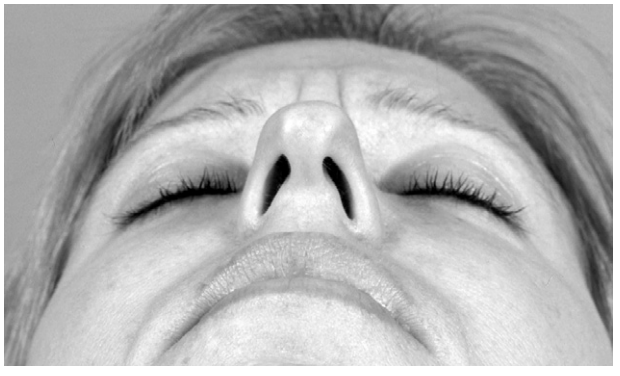

Figure 5 • Widened medial crural footplates contributing to obstruction of the external valve.

Synechiae can be another problematic cause of nasal obstruction. They can result from recurrent intranasal infections or previous trauma, but the majority are secondary to iatrogenic trauma that occurs during septoplasty and nasal surgery. The surgeon should handle the septal flaps delicately at all times to prevent tears in the mucosa. It is critical that one dissect the mucoperichondrial flap below the perichondrium to maximize flap strength and thickness. Whenever there is concern that the raw surfaces of the turbinates are going to be in contact with the septum, it is wise to place a thin intranasal septal splint.

Complications

Complications after nasal airway surgery are not common. Postoperative infection is infrequent, and bleeding is uncommon as well. A small rolled piece of Telfa gauze in each nasal airway helps to stent the airway and absorb any blood from the nasal surgery. A nasal drip pad is routinely applied for the

first 24 hours after surgery. Extensive full packing of the nose is rarely necessary.

Most complications of nasal airway surgery are not immediately apparent but, instead, manifest weeks after surgery when intranasal swelling has decreased and it is evident that the airway has not been improved. This is usually the result of a technical error in the operation. Failure to diagnose the condition preoperatively can also contribute to surgical failure. Often, failure to improve a patient's airway is the result of an inappropriate operation. Placement of inadequate spreader grafts or undersized battens can also lead to poor outcomes. Attention to detail in every step of the patient's preoperative evaluation, operative procedure, and postoperative care is the surgeon's best tool to prevent unsuccessful outcomes.

Pearls and Pitfalls

- Understand that nasal obstruction is secondary not only to deviated septa and enlarged turbinates but also to the more dynamic structures of the internal and external nasal valves. These four structures must be routinely and individually evaluated.
- Perform a complete physical examination. Direct observation of the patient's nasal respirations—both quiet and vigorous—helps to elucidate the causes of nasal obstruction. Avoid the temptation to proceed immediately to nasal speculum anterior rhinoscopy and spend a sufficient amount of time meticulously inspecting the four main anatomic structures that affect the nasal airway.
- During surgery, handle nasal tissues with care and delicacy. Carefully dissect the mucoperichondrial membranes. Expose all septal irregularities (cartilaginous and bony), and close all tissues meticulously.
- Place a supportive dressing on the nose to allow for soft tissue stabilization as the grafts heal.
- Monitor patients long-term because postoperative nasal obstruction may take weeks to manifest.
- Both functional and aesthetic nasal operations should routinely include techniques that maintain or improve the airway.

SUGGESTED READINGS

Constantian MB, Clardy RB: The relative importance of septal surgery and nasal valvular surgery in correcting airway obstruction in primary and secondary rhinoplasty. Plast Reconstr Surg 98:38-54, discussion 55-58, 1996.

Gordon ASD, McCaffrey TV, Kern EB, Pallanch JF: Rhinomanometry for preoperative and post-operative assessment of nasal obstruction. Otolaryngol Head Neck Surg 101:20-26, 1989.

Sheen JH: Spreader graft: A method of reconstructing the roof of the middle nasal vault following rhinoplasty. Plast Reconstr Surg 73:230-239, 1984.

Toriumi DM, Becker DG: Rhinoplasty Dissection Manual. Philadelphia, Lippincott Williams & Wilkins, 1999.

Toriumi DM, Josen J, Weinberger M, Tardy ME Jr: Use of alar batten grafts for correction of nasal valve collapse. Arch Otolaryngol Head Neck Surg 123:802-808, 1997.

The Chin

SEAN G. BOUTROS

The chin is one of the major components of the lower third of the face. It defines the lower facial aesthetic lines and completes overall facial harmony. It is a symbol of strength and intelligence. The purpose of this chapter is to review the aesthetic goals of chin surgery and to demonstrate reproducible surgical techniques that provide optimal results.

Pathologic Anatomy

Problems of the chin are divided into two broad categories: those related to facial skeletal disorders and those related to isolated chin disorders. Skeletal disorders with associated chin deformities, such as those found in Treacher Collins syndrome and craniofacial microsomia, are discussed in other chapters. In addition to syndromic skeletal disorders, chin problems may be associated with mandibular abnormalities such as prognathism and micrognathia. Isolated chin deformities can be subdivided into disorders of vertical or horizontal excess (macrogenia) or deficiency (microgenia).

Facial Analysis Systems

A systematic definition of the ideal projection of the chin has been proposed by many authors. These proposals are rough guidelines, and important variables such as age, stature, gender, and ethnicity must also be considered. A useful guideline for horizontal projection of the chin is the Gonzalez-Ulloa and Stevens line (Fig. 1), which is drawn at right angles to the Frankfort horizontal plane and should touch the soft tissue radix and the most anterior soft tissue point of the chin. The Gonzalez-Ulloa and Stevens line usually gives a strongly projecting chin. Another commonly used guide is the Steiner line, which passes through the nasal inflection point and is tangent to the chin and the upper and lower lips. In the frontal view, the chin should project such that the lower third of the face is divided by the commissure into an upper third and lower two thirds (Fig. 2).

Men and women have different facial aesthetic standards, with a prominent chin being more acceptable in men. In addition, the chin has a complex interplay with the nose such that larger chins are balanced by larger noses and less projecting chins by smaller noses.

The chin is composed of bony and soft tissue components. The bony component represents the most anterior portion of the mandible. It is best defined by the anterior and inferior margin of the mandible and has characteristic prominences, including the mental protuberance and the mental tubercle. The soft tissue of the chin is composed of the paired mentalis muscle, depressor labii inferioris, depressor anguli oris, and orbicularis oris. The mentalis muscle is the most important not only because of its size, but also because of its critical function as the only elevator of the lower lip. The muscle also defines the chin pad and chin cleft, the latter attributed to a diastasis of the mentalis muscle. In most cases, when the mentalis muscle is incised, there is a white central muscle-free zone. In patients with clefts, the muscle-free zone is accentuated, and there is more fibrous tissue between the paired mentalis muscles.

Preoperative Evaluation

Patients should undergo a through examination of the lower part of the face. The occlusion and dental relationships should be noted. Patients with poor dental relationships may benefit from orthognathic surgery in addition to chin surgery. The lip/tooth relationships are noted. The lower lip at rest should be above the lower incisors. An overly recumbent lower lip with incisor show is often associated with poor lip competence and muscular hypertrophy in

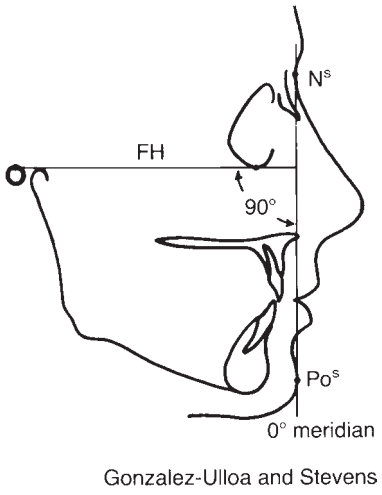

Figure 1 • The Gonzalez-Ulloa line determines the ideal horizontal chin projection. (From McCarthy JG, Ruff G: The chin. Clin Plast Surg 15:125, 1988.)

an attempt to achieve competence. These patients are at increased risk for postoperative lip ptosis unless the lip is supported. They are also at risk for complications with chin implantation. The motor innervation of the lower lip should be documented in addition to evidence of mentalis fasciculation. Fasciculations may indicate poor lip relationships and should alert the surgeon. The sensation of the lower lip should be tested. It is not uncommon to have temporary or even permanent sensory changes postoperatively, and these changes should be differentiated from preoperative sensory disturbances. If chin implants are planned, the mental tubercle should be palpated. If prominent, it may interfere with seating of the implant and may need to be contoured.

All patients desiring chin surgery should be evaluated with standard photographs. They are useful in discussing the goals of surgical correction. The photos should note the position of the lips and the relationship of the lips to the underlying teeth. Some patients have preoperative incisor show. Other patients have evidence of mentalis fasciculation, which must be noted and incorporated into the operative plan.

Cephalometric analysis is indicated for four reasons: to document the position of the mental foramen, to illustrate the interplay of the bone and soft tissue, to show the depth of the canine roots, which can approach the inferior border in an extremely deficient mandible, and to evaluate the overall jaw position.

A combination of studies assists the surgeon in planning the amount of augmentation or reduction. The surgeon must keep in mind that a bony correction of 1 mm is translated to approximately 0.8 mm of soft tissue advancement. For greater advancement, specifically, those greater than 1 cm, the ratio probably drops to 1:0.7. Patients who require only minor augmentation of the chin can forego cephalometric studies.

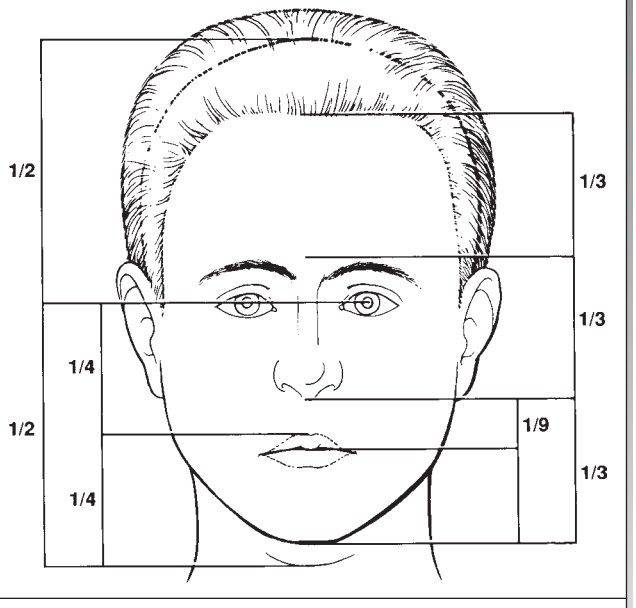

Figure 2 • Ideal vertical projection of the chin. The lower third of the face should be divided into an upper third and lower two thirds by the lips. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990.)

Reconstructive Goals

The goal of aesthetic chin surgery is to provide a pleasing outline of the lower third of the face and restore overall facial harmony. The chin should be positioned so that its greatest projection lies on the facial midline. It should not draw attention away from other facial features. It should support lower lip position, preserve lower lip movement or sensibility, and allow for unconscious lip competence. It is important to accomplish these goals safely and avoid postsurgical nerve insensibility and motor dysfunction.

Surgical Treatment

A *horizontal deficiency with vertical neutrality* is the most common indication for aesthetic chin surgery. This deficiency may be an isolated complaint, but it is often coupled with other facial aesthetic requests such as rhinoplasty or facialplasty.

Patients should be divided into two general groups. The first includes patients requiring only minimal augmentation, which is best accomplished

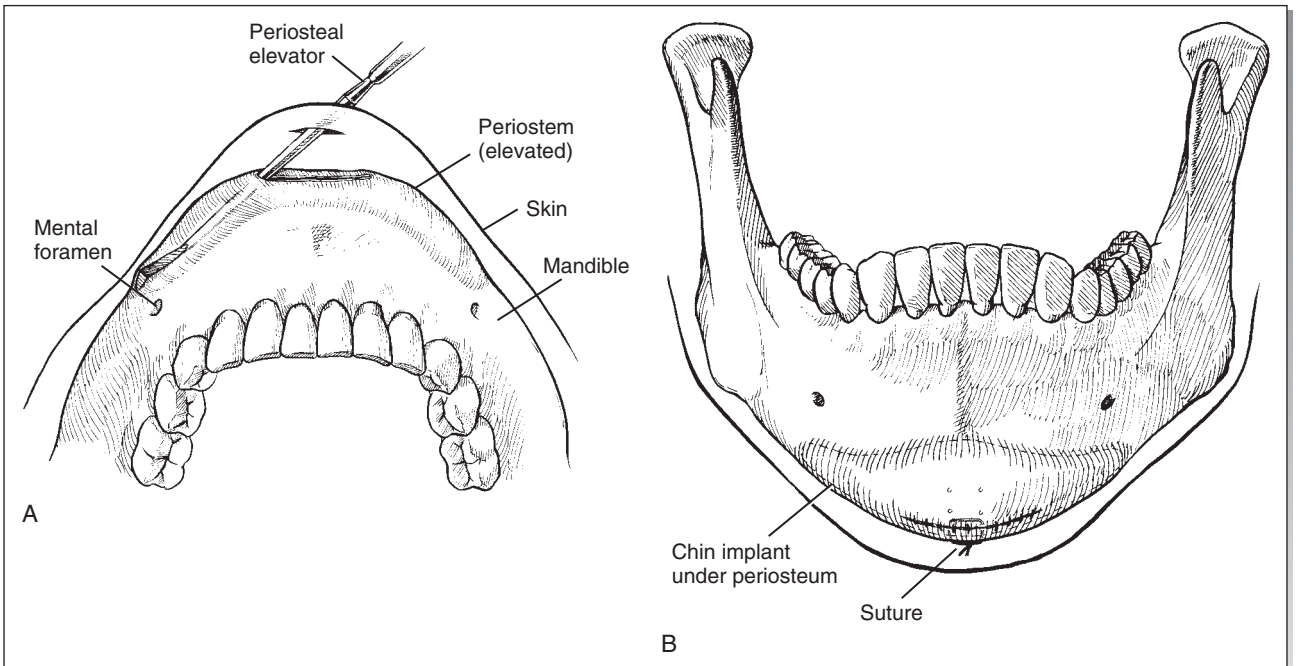

Figure 3 • Placement of a chin implant. The implant should be placed just at the lower border of the mandible. The pocket should be kept as small as possible to avoid implant malpositioning. **A,** Surgeon's view in creation of implant pocket. **B,** Final implant position. Note the midline suture.

by placement of a chin implant. It is preferable to place the implant via the submental approach for three reasons: a lower chance of muscular lip problems because the mentalis muscle is left undisturbed, decreased incidence of infection, and technical ease of placement with a lower rate of implant malposition.

Small to medium-sized silicone implants are preferable to larger ones. Silicone implants are easy to insert and are easier to remove in the event of malpositioning or extrusion/infection. Before implantation, a suture or notch is placed in the midline of the implant both to orient the implant and to stabilize it once implanted. The facial midline is also marked. It is important to note that the midline of the chin and the dental midline can be different from the facial midline. This discrepancy should be noted so that the implant does not accentuate any preexisting asymmetry. A submental incision is made and a subperiosteal implant pocket dissected. A periosteal elevator slightly smaller than the implant is used to avoid creating an oversized pocket. The surgeon's free hand is placed on the mandibular border to keep the pocket as low on the mandible as possible. The pocket is made slightly longer than the implant on the patient's left side because a right-handed surgeon has more control here. It is slid completely on the left side, bent, and advanced into the right side. The sliding motion prevents the tails of the implant from being angulated or kinked. After the implant is in place, it is secured with a suture. The pocket controls the cranial/caudal position, and the suture controls lateral movement of the implant (Fig. 3).

The second group includes younger patients, patients requiring more than a medium-sized implant, or those needing more than 8 mm of advancement. A sliding genioplasty is preferred. In addition, patients with mentalis fasciculation from marginal lip competence and patients who would benefit from orthognathic surgery but refuse are also better suited to a chin osteotomy.

The incision is made on the lingual aspect of the lip approximately 15 mm above the gingivobuccal sulcus. The mucosa is dissected from the underlying muscle to the depth of the sulcus. The mentalis muscle is divided. The lower portion of the mandible is degloved in a subperiosteal plane. It is important to identify the mental foramen below the first or second bicuspid to avoid injury to the mental nerve. The midline of the mandible is scored to establish midfacial identification. The mandible is osteotomized 12 to 15 cm above the inferior border in the midline to a portion just below the mental foramen. It is important to plan the osteotomy on preoperative cephalograms to ensure the safety of the canine roots and the inferior alveolar nerve. The bony segment is advanced and fixated, with the posterior muscle attachments left intact as they provide vascularity to the chin segment. Either lag screws or prebent genioplasty plates can be used for rigid fixation. The plates have the advantage of providing precise, preset advancement. The posterior edge of the chin segment is contoured with a bur so that it

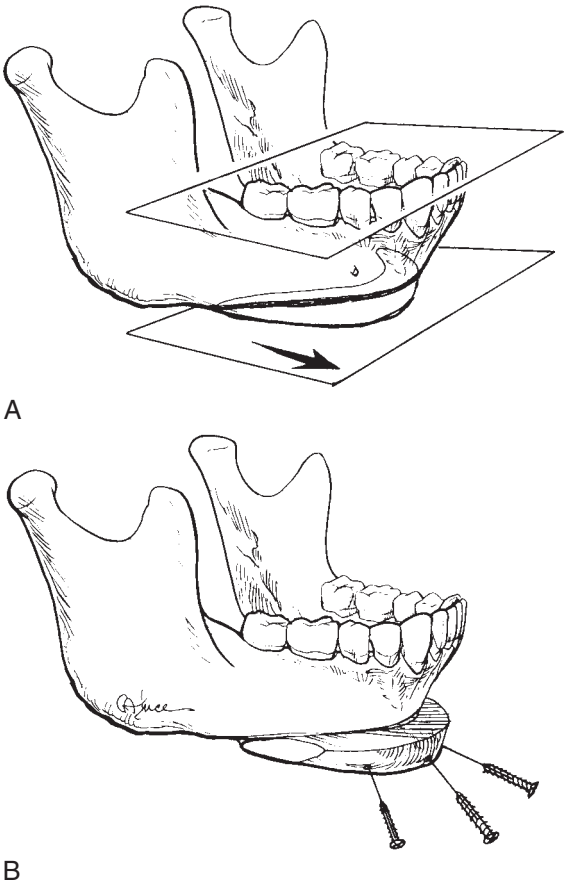

Figure 4 • Sliding genioplasty. **A,** The osteotomy is made in a plane parallel to the occlusal surface. Consequently, there is no change in the vertical dimension. **B,** Lag screws provide fixation. The poles of the advanced segment have been contoured. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990.)

is not palpable (Fig. 4). The mentalis muscle is repaired in a slightly higher position, and the mucosa is closed in an interrupted fashion and chin straps applied to the chin.

In patients with *horizontal deficiency and vertical excess*, a jumping genioplasty is performed (Fig. 5). The surgical approach and line of osteotomy are the same as for the sliding genioplasty; however, the segment is advanced so that the posterior cortex of the chin segment rests on the anterior cortex of the mandible. This maneuver simultaneously decreases the vertical dimension and provides significant horizontal advancement. The horizontal advancement is often excessive, but the osteotomized segment can be burred on its posterior aspect to prevent overprojection of the chin. In this situation, lag screws are the fixation method of choice.

In the case of *combined horizontal and vertical deficiency*, the osteotomy is performed in the same manner (see Fig. 5); however, an interpositional bone graft is placed to increase the vertical dimension at the same time as the horizontal deficiency is addressed. The bone graft source can be the iliac crest or the calvaria. The calvaria has the advantage of ease of harvest and minimal donor site discomfort.

Patients with *horizontal and vertical excess* should undergo reduction genioplasty. This procedure is performed in a similar fashion as other chin osteotomies (Fig. 5). A parallel osteotomy is made and the intervening segment of bone removed. The osteotomized segment is secured with screws or plates in either an advanced or a recessed position. One must note that direct removal of the inferior segment of bone yields poor results because of disruption of the critical bone-muscle interface. In certain patients with isolated *horizontal excess*, the anterior portion of the chin can be directly removed with a bur. To obtain a constant result, it is helpful to cut the most projecting portion of the mandible with a sagittal saw to the depth of the reduction and then use a bur to reduce the bone to this level.

Postoperative Care

As previously mentioned, a chin strap is applied at the conclusion of the operation to provide support and comfort and decrease the edema associated with the procedure. All patients with osteotomies are also instructed to use Peridex oral rinse five times a day. They are kept on a liquid diet for 2 days and transitioned to a soft diet as tolerated. Patients take prophylactic antibiotics for 3 days.

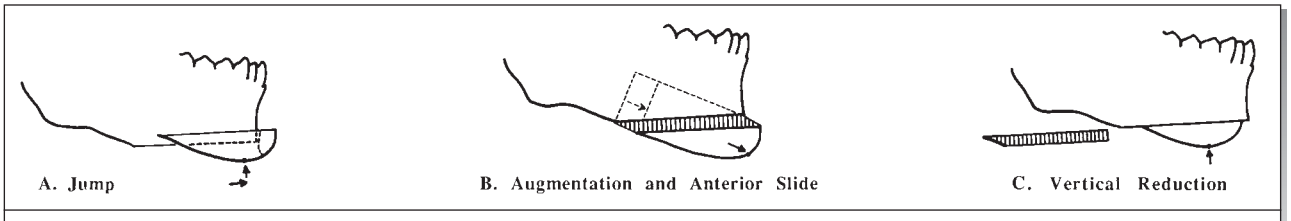

Figure 5 • **A,** Combined vertical excess and horizontal deficiency are best treated by jumping genioplasty. **B,** Combined vertical and horizontal deficiency is treated by interpositional genioplasty. **C,** Vertical excess is treated by reduction genioplasty with either an anterior or posterior slide. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990.)

Pearls and Pitfalls

- Avoid large or extended implants, especially in younger patients, because they can result in long-term bone erosion and are more prone to malposition. These patients are better candidates for osseous genioplasty.
- For chin implants, create a small pocket at the mandibular border. Such a pocket decreases implant mobility and allows for more accurate positioning.
- Leave a generous cuff of mucosa to facilitate suture closure.
- Preoperatively plan the movement of the osteotomy segment on the cephalogram.
- Stabilize the genioplasty segment with either lag screws or plates.

SUGGESTED READINGS

Matarasso A, Elias A, Elias R: Labial incompetence: A marker for progressive bone resorption in Silastic chin augmentation. Plast Reconstr Surg 98:1007-1014, 1996.

Posnick JC: Craniofacial and Maxillofacial Surgery in Children and Young Adults. Philadelphia, WB Saunders, 2000.

Yaremchuk MJ: Improving aesthetic outcomes after alloplastic chin augmentation. Plast Reconstr Surg 112:1423-1432, 2003.

Zide BM, Boutros S: Chin surgery III: Revelations. Plast Reconstr Surg 111:1542-1550, 2003.

Zide BM, Pfeifer TM, Longaker MT: Chin surgery: I. Augmentation—the allures and the alerts. Plast Reconstr Surg 104:1843-1853, 1999.

Botulinum Toxin

ALAN MATARASSO

The cutaneous manifestations of photodamage (extrinsic) and chronologic (intrinsic) aging have an effect on all soft tissue layers and skeletal structures of the face. These cutaneous alterations result in the characteristic appearance of the face as it evolves with advancing age.

Etiopathogenesis

Dermal photoaging causes elastosis, along with increased ground substance and a reduction in type I collagen; as a result, dermal thinning progresses at a rate of approximately 6% per decade. Concomitant changes also occur in the epidermis and subcutaneous adipose tissue. Consequently, treatment objectives should be directed toward preventing, restoring, or reversing the underlying cause. Nonsurgical rejuvenation of the face ideally involves three approaches: (1) resurfacing techniques or chemical peeling agents, which have their greatest effect on the epidermis and superficial dermis; (2) fillers or injectables, which are intended to replace missing dermal components or subcutaneous fat; and (3) paralytic agents, which cause chemodenervation of the underlying muscle responsible for the repetitive motion that results in rhytides and thin, wrinkled skin. The latter technique is discussed in this chapter.

Diagnostic Studies

There are no specific diagnostic studies unless pathologic conditions (e.g., Ehlers-Danlos syndrome) are suspected. A photographic record is valuable, as is an anatomic data sheet that records the process and treatment doses.

Goals of Treatment

The goal of treatment of dynamic facial wrinkles is weakening of the underlying facial musculature. In some cases (e.g., glabella and radix), complete paralysis is desired. In other areas (e.g., brow and orbicularis), partial or segmental weakening is sufficient, as complete paralysis may result in complications. The paralysis will result in a temporary improvement in the overlying mimetic wrinkles. Another goal is control of hyperhidrosis.

Botulinum Toxin

Botulinum neurotoxin type A is one of eight serologically distinct and species-specific toxins produced by the anaerobic bacterium *Clostridium botulinum*. Within 6 to 36 hours of application to striated muscle, the toxin produces chemodenervation by preventing release of acetylcholine at the neuromuscular junction. The result is muscle atrophy with subsequent flaccid paralysis. Muscular function gradually returns after approximately 3 months, and axonal sprouting prevents permanent paralysis of the treated muscle.

Each vial of Botox is a lyophilized complex consisting of 100 U of toxin (10% variation), 0.5 mg of human albumin, and 0.9 mg of sodium chloride. The toxin is received freeze dried and kept frozen until it is reconstituted and used within 4 to 24 hours.

Botulinum toxin is injected into a region of the muscle according to precise anatomic location. Accurate muscle identification can be achieved by asking the patient to contract the muscle targeted for treatment. After isolating the muscle, Botox is injected into the muscle, the underlying periosteum, or the overlying subcutaneous tissue in a specific volume

TABLE 1 Botox: General Dosing Guidelines

AREA	MUSCLE	NUMBER OF UNITS
Glabella	Corrugator supercilii/ procerus/orbicularis oculi (medial fibers)	20-40
Forehead	Frontalis	30-60
Crow's feet	Orbicularis oculi (lateral fibers)	5-15 (per side)
Upper lips	Orbicularis oris	3-10
Chin	Mentalis	5-10
Neck	Platysma	30-60
Hyperhidrosis	Sympathetic blockage of eccrine sweat glands	50-100 (per side)

according to the desired effect and muscle mass (Table 1).

The median lethal dose (LD_{50}) has been estimated to be between 2800 and 3500 U in humans. The maximum total recommended dose is 300 to 400 U at a single session and not more than 400 U over a 3-month period. Intravascular injection does not result in systemic toxicity.

To reduce the most common adverse sequelae (i.e., ecchymosis and erythema), gentle pressure with or without cold compresses can be applied to the treatment site immediately after injection. Patients can resume normal activities immediately after treatment but are advised to remain upright and avoid lifting or straining for 3 to 4 hours after injection to prevent unwanted spread of the toxin. The use of cover-up or camouflaging agents is unrestricted.

The initial effects of treatment can be seen within a few hours, and the full effect can be observed up to 14 days after treatment. Failure can most often be attributed to residual muscle activity resulting in a small area of movement, normal recruitment of adjacent muscles, or residual static lines that cannot be improved by Botox treatment. Muscle activity begins to return gradually at approximately 3 months after injection; ultimately, full muscle mobility returns.

Local complications such as pain, edema, erythema, ecchymosis, headache, and short-term hypoesthesias can be related to the injection site. The most notable and disfiguring local complications occur when the toxin diffuses to surrounding muscles and paralyzes unintended muscles.

General complications include loss of facial expression (mask-like facies), incomplete muscle paralysis with residual rhytides, and unwanted muscle paralysis from spread of neuromuscular blockade to adjacent sites (as in local complications).

Systemic reactions include nausea, fatigue, malaise, flu-like symptoms, and distant rashes. Distant neuromuscular effects have been documented by single-fiber electromyographic studies, but the clinical significance of these findings remains unclear.

Immunologic complications include acute type I reactions and may be attributable to the presence of human serum albumin. Such reactions may result from the presence of circulating antibodies and are reported to occur in 3% to 5% of patients when large volumes of toxin are used. With the reduction in protein content in newer formulations of Botox, no reports of this complication have been noted. The development of antibodies seems to correlate with increasing number of injections, frequency of treatment, and total cumulative dose of toxin. Limiting the total amount of toxin to less than 100 U per session and avoiding booster injections for a minimum of 3 months are recommended measures to prevent the formation of neutralizing antibody. Botulinum toxin type B (Myobloc/Neurobloc) is currently available and may be considered an alternative in patients with immunologic resistance to botulinum toxin type A. It has the advantage of a faster onset of action and is useful when results are needed in 24 to 48 hours.

Therapeutic failure can result from a variety of causes, such as improper mixing, dilution, or storage; incorrect muscle identification; or failure to differentiate a static rhytid from a dynamic wrinkle.

Pearls and Pitfalls

- There are two areas of controversy regarding botulinum toxin that have yet to be determined: the appropriate dilution of the toxin and the method and length of storage of the reconstituted toxin. Large dilutions with lower toxin concentrations require larger injection volumes to achieve the desired result. These large volumes can result in diffusion of the toxin to unwanted surrounding areas. For this reason, the author prefers a low volume and high concentration of Botox. The length of viability of reconstituted toxin is also unresolved, but the best results are seen when the toxin is used within 24 hours of reconstitution.
- Provide analgesia—topical creams, ice compresses, anxiolytics, or any combination of these measures.
- Change needles frequently. Insulin syringes do not have a Luer-lock, and therefore less toxin is inadvertently wasted. The disadvantage of insulin-type syringes is the difficulty of drawing the solution into the syringe because of the unreusable needle. Furthermore, the needle, intended for only a single use, dulls quickly.
- Before injection, examine the patient for preexisting upper eyelid ptosis or brow ptosis. There are three types of "ptosis" that can occur with the use of Botox: (1) drug induced as a

result of diffusion of the toxin to the levator aponeurosis, (2) unmasking of preexisting upper lid ptosis that had been compensated for by the frontalis muscle, and (3) true brow ptosis caused by injecting the lower border of the lateral frontalis muscle.

- "Extended" areas of treatment (e.g., nasolabial folds, upper lip lines, marionette creases, chin dimpling) should be only weakened and not paralyzed to avoid functional imbalance. These areas should be treated with more dilute concentrations of Botox.
- When treating one of an agonist-antagonist muscle group (e.g., frontalis and corrugator) in an attempt to "reshape" (i.e., alter the balance between elevator and depressor muscles), consider the need to counterbalance the opposing muscle with a small amount of toxin to avoid imbalance. When reshaping is not the goal (e.g., orbicularis oris), consider the effect that paralysis of the treated muscle has on its functional use.
- Hyperdynamic facial lines pretreated with Botox in conjunction with laser resurfacing produce improved results by improving collagen reorganization. The effect of paralyzing an area injected with a soft tissue filler also yields an enhanced result because of a reduction in muscle activity and therefore slower breakdown of the filler.
- Precise identification of the muscular anatomy and needle placement is essential for a successful outcome. Consider the effect that Botox has on anatomy altered by previous surgery.
- Evaluate nonresponders who may require additional toxin. Occasionally, they can be responding, but surrounding muscles are being recruited or the remaining rhytides are due to dermal atrophy.
- Match the dose of toxin to the muscle mass, intended outcome, prior experience, and age and gender of the patient.

SUGGESTED READINGS

Carruthers J, Fagien S, Matarasso SL, Botox Consensus Group: Consensus recommendations on the use of botulinum toxin type a in facial aesthetics. Plast Reconstr Surg 114(6 Suppl): 1S–22S, 2004.

Faigen S: Botox for the treatment of dynamic and hyperkinetic facial lines and furrows: Adjunctive use in facial aesthetic surgery. Plast Reconstr Surg 103:701, 1999.

Matarasso A: Clinical usage of Botox. In Aesthetic Surgery Video Journal. New York, American Society for Aesthetic Plastic Surgery, TRT Productions, 1999.

Matarasso A (Guest Ed): Nonoperative techniques for facial rejuvenation. Part I. Clin Plast Surg 27:479–676, 2000.

Matarasso A, Matarasso S (Guest Eds): Nonoperative techniques for facial rejuvenation. Part II. Clin Plast Surg 28:1–252, 2001.

Matarasso A, Matarasso SL: Botulinum A exotoxin for the management of platysma bands. Plast Reconstr Surg 112(5 Suppl):138S–140S, 2003.

Matarasso SL: Complications of botulinum A exotoxin for hyperfunctional lines. Dermatol Surg 24:1249, 1998.

Matarasso SL: Update on the aesthetic uses of botulinum a neurotoxin in facial rejuvenation. Curr Probl Dermatol 13:46, 2001.

Laser Skin Resurfacing and Fillers

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JASON R. LUPTON

This chapter reviews the use of dermal filling agents in combination with laser resurfacing for the improvement of photodamaged facial skin and atrophic facial scars. Emphasis is placed on proven techniques for both light- and dark-skinned patients. In addition, a comprehensive review of dermal filling agents is included because soft tissue augmentation is often combined with laser treatments.

Pathologic Anatomy

Superficial wrinkles are textural changes that result from intrinsic aging and photoaging. Mimetic wrinkles are lines or furrows caused by deep dermal creases from repetitive muscle movement (combined with dermal elastosis). Folds are defined as overlapping, sagging skin.

Topical exfoliating agents primarily affect the epidermal level and, over time, possibly benefit the dermis. Deeper resurfacing procedures (laser resurfacing, chemical peels, dermabrasion) are designed to improve the skin by "sanding down" adjacent tissue so that rhytides and scars are less apparent. They may also produce collateral improvement of the dermal layer. Nonablative technology with cumulative applications is designed to restore the dermis and bypass the epidermis, thereby avoiding the erythema associated with traditional laser technology.

Injectables or soft tissue filler materials are intended to restore a more youthful facial contour by replacing lost dermal elements or subcutaneous fat. A plethora of filling agents are available and fall into five categories: natural human materials (e.g., AlloDerm, autologous fat), sources derived from animals (e.g., bovine collagen), synthetic materials (e.g., hyaluronic acid products), materials of a chemical nature (e.g., silicone), or combined materials (e.g., inner methylmethacrylate beads covered with a collagen surface). Alternatively, fillers can also be classified as temporary or permanent. Almost all fillers are temporary other than silicone. *No ideal filler material yet exists.* However, because of patient demand and manufacturer interest, the field of injectable soft tissue fillers is rapidly evolving.

Goals of Treatment

The goals of restoring the youthful appearance of an aging face are as follows:

1. Replace—replacing atrophic dermal or subcutaneous structures
2. Recontour—reshaping facial volume
3. Resurface—resurfacing the skin surface
4. Relax—improving rhytides by relaxing the causative muscle
5. Reposition/redrape/remove—anatomic repositioning and redraping of loose sagging soft tissue

Diagnostic Studies

There are no specific diagnostic studies unless pathologic conditions (e.g., Ehlers-Danlos syndrome) are suspected. A photographic record is valuable.

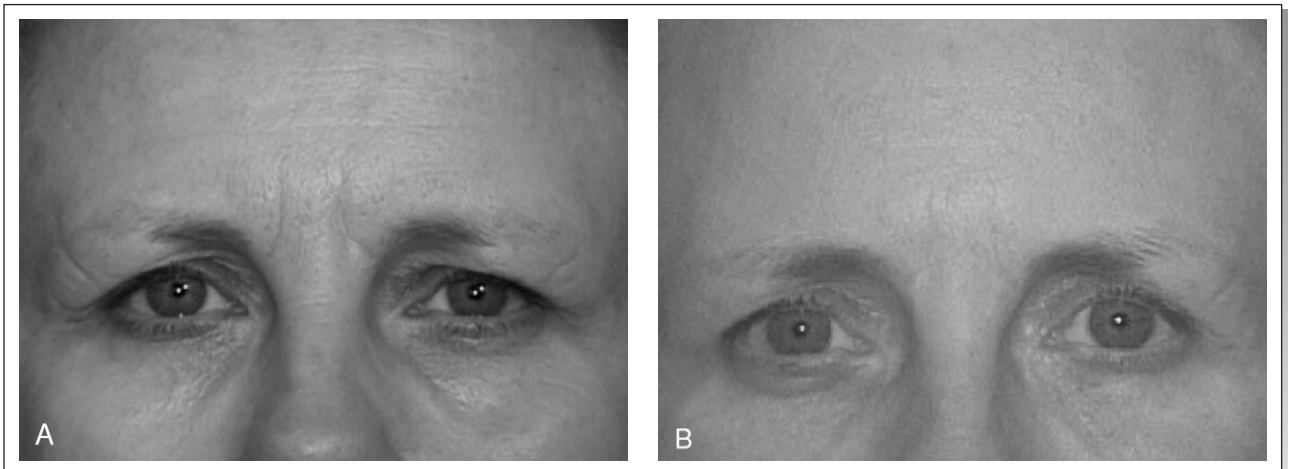


Figure 1 • A, Periorbital and brow rhytides. **B,** Appearance 6 months after CO_2 laser resurfacing.

Cutaneous Laser Resurfacing

Since the mid-1990s, the effectiveness of high-energy, pulsed, and scanned carbon dioxide (CO_2) and erbium:yttrium-aluminum-garnet (Er:YAG) lasers has been established for the treatment of photoinduced facial rhytides, dyschromia, and atrophic scars.

With a wavelength of 10,600 nm, the CO_2 laser uses tissue water as its targeted chromophore and vaporizes 20 to 30 μm of tissue per pulse, thus effectively removing the entire epidermis in a single laser pass using typical treatment parameters. This action produces a limited spread of coagulative necrosis (50 to 150 μm wide) that induces new collagen formation and persistent dermal remodeling —the process responsible for the majority of the long-term benefits of treatment (Fig. 1). Although the CO_2 laser typically produces at least 50% improvement in rhytid severity or scar depth after treatment, it is associated with the highest post-treatment morbidity and risk of adverse sequelae.

The erbium laser, with a wavelength of 2940 nm, has a much higher absorption coefficient than does the CO_2 laser, and its energy is 12 to 18 times more efficiently absorbed by water-containing tissues. As a result, most of this laser's energy is absorbed by water within the superficial epidermis, causing the thermal energy to be ejected within the desiccated tissue during laser irradiation. Each pass of the erbium laser penetrates to a depth of 2 to 5 μm per pulse and produces a zone of thermal necrosis ranging another 20 to 50 μm after a typical multipass procedure. The erbium laser is a precise ablative tool that produces little residual thermal damage and therefore minimal long-term neocollagenesis. The major disadvantage of the erbium laser system is the lack of intraoperative hemostasis because of inadequate vessel coagulation.

Newer long-pulsed erbium laser systems have been developed in an attempt to reduce these shortcomings, most notably effecting collagen shrinkage and improved vessel coagulation. By generating pulse widths up to 500 μm, larger zones of thermal necrosis are created with resultant collagen contraction and remodeling. Although the long-pulsed erbium laser approaches the CO_2 laser in terms of its effect on dermal tissue, it is not associated with as long a postoperative recovery course nor as high a risk for postoperative complications.

The relatively new "nonablative" laser systems have gained in popularity because an increasing number of patients are willing to accept more modest improvement, particularly when only limited or no recovery time is available. Infrared-range laser systems, including the 1320-nm neodymium:yttrium-aluminum-garnet (Nd:YAG) laser, the 1450-nm diode laser, and the 1540-nm erbium:glass laser, are most commonly used for nonablative skin resurfacing. Tissue water is also the targeted chromophore, and heat energy is largely deposited in the upper papillary dermis, where the bulk of solar elastosis is located. Dynamic cryogen spray devices or contact cooling handpieces are used for epidermal preservation. Multiple nonablative treatment sessions are typically recommended at monthly intervals, and the final clinical results may not be apparent for 4 to 6 months after the final treatment session. Clinical results are not as favorable as those observed after ablative laser resurfacing, but a 20% to 50% improvement can be expected (Fig. 2).

Technique

No consensus exists regarding whether pretreatment with topical retinoids, α-hydroxy acids, or hydroquinone-containing compounds enhances

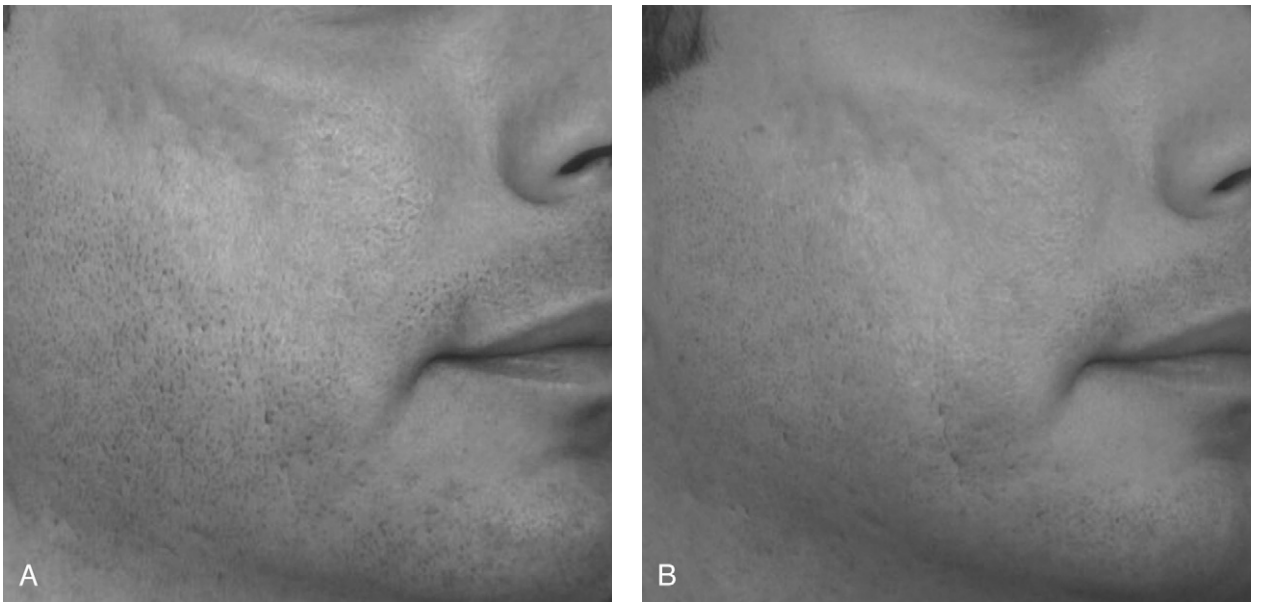


Figure 2 • A, Atrophic facial scars before treatment. **B,** Appearance 6 months after a third 1450-nm diode laser treatment.

post–laser resurfacing results. Antiviral prophylaxis for herpes simplex virus exposure or reactivation is required when treating the perioral area in all patients. In addition, perioperative antibiotic prophylaxis is also recommended for all patients.

CO_2 Laser

When using the CO_2 laser, either an entire aesthetic unit or the full face should be treated to avoid obvious lines of demarcation between treated and untreated skin. A treatment fluence of 300 mJ is most commonly used for the initial pass in patients with lighter skin types and 200 to 250 mJ in those with darker skin. Adjacent scans are directed in a nonoverlapping manner to avoid excessive heat deposition and spread of thermal damage to unintended tissue structures. Because the depth of ablation and degree of residual thermal damage are directly correlated with the number of laser passes performed, stacking of pulses or scans must be strictly avoided to decrease the risk of hypertrophic scar formation or hyperpigmented streaking in darker-skinned individuals. Successive passes (two to three on average) should be performed only after the desiccated tissue has been completely removed from the surgical field. Excessive deposition of heat occurs because desiccated tissue acts as a heat sink for progressive thermal damage. The direction of laser passes should also be alternated to avoid striped patterns on the skin. For atrophic facial scars, the 3-mm handpiece can be used to sculpt scar edges for maximal flattening. Only a single pass of the laser at reduced fluence should be delivered along the mandibular border because of the tendency for hypertrophic scar formation in this area.

An increasing number of surgeons are now performing single-pass CO_2 laser resurfacing to avoid many of the aforementioned complications. In this technique, a single laser pass is delivered with the scanner, followed by freehand use of the 3-mm spot size handpiece to "fill in" any untreated areas. The partially desiccated tissue is left intact after the procedure to serve as a biologic dressing; the tissue typically sloughs off after 3 to 4 days. The single-pass technique is preferred for patients with darker skin tones to avoid postinflammatory hyperpigmentation. Additional laser passes can be applied to focal areas (e.g., cheek scars, upper lip rhytides) as necessary to effect more tissue tightening.

Erbium:YAG Lasers

Short- and long-pulsed Er:YAG lasers can also be used to treat photodamaged facial skin and atrophic scars. With the short-pulsed systems, the main goal is to maintain a uniform cutaneous surface and ablative depth. Because this system has high affinity for water-containing structures, the surgical field must be dry before treatment. Any moisture on the field absorbs the laser energy and renders the treatment less effective. The entire epidermis can be removed after two to three passes at 5 to 10 J/cm^2, whereas focal areas with either more photodamage or deeper scars can receive additional passes. Fewer passes should be applied at the treatment periphery to ensure natural blending between treated and untreated skin. In general, it is more difficult to

perform multiple-pass erbium laser resurfacing because of inadequate vessel coagulation and transudative fluid loss obscuring the operative field. Local injection of 1% lidocaine with epinephrine improves visualization and decreases bleeding.

POSTOPERATIVE MANAGEMENT. Either an open or closed wound care technique can be used postoperatively. The open technique involves the application of topical barrier ointments such as Aquaphor, Catrix 10, or plain petrolatum for the first 3 to 5 postoperative days, whereas a transparent, semioccclusive dressing such as Vigilon or Silon-TSR is left intact on the laser-treated skin in the closed technique. The dressings must be changed at least every 24 hours to avoid bacterial colonization and permit proper wound visualization. Some physicians prescribe the closed technique for the first few days in an attempt to reduce patient discomfort, followed by an open technique for the remainder of the healing period.

Postoperative side effects and complications are similar after either erbium or CO_2 laser skin resurfacing. Transient erythema, edema, and serous discharge are universal findings. The degree of postoperative erythema is directly correlated with the level of ablation and the amount of residual thermal damage produced. Erythema after multipass CO_2 laser skin resurfacing typically persists for 3 to 6 months, whereas it usually resolves within only 1 month after erbium laser treatment. Milia formation, exacerbation of acne, contact allergies, superficial bacterial infections, and reactivation of herpes simplex are other potential postoperative complications. Pigmentary alterations, including hyperpigmentation and delayed-onset hypopigmentation, may also develop. Hyperpigmentation is most common in patients with dark or olive skin tones and can be effectively treated with topical bleaching or peeling agents (e.g., hydroquinone, glycolic acid). Severe complications, including hypertrophic scar formation, ectropion, and systemic infection, are rare.

Soft Tissue Augmentation

Many dermal filling agents are available for soft tissue augmentation. Most of these substances are best used in the lower part of the face and can be combined with other rejuvenative procedures such as cutaneous laser resurfacing or botulinum toxin injection. Requirements for the ideal soft tissue filler are outlined in Table 1.

TABLE 1 Requirements for the Ideal Soft Tissue Filler

SAFETY	EFFICACY
Noninfectious	Easily injected/implanted
Nonimmunogenic	Quick recovery
Nontoxic	Not visible (nonlumpy)
Noncarcinogenic	Nonresorbable (long-lasting)
Nonmigratory	Soft and pliable (natural feel)
FDA approved	

FDA, Food and Drug Administration.

Injectable Bovine Collagen

Bovine collagen implants are the most popular dermal filling agents used. The implants consist of purified, reconstituted, fibrillar bovine type I and type III collagen. Because bovine collagen is xenogeneic, double skin testing is mandatory 1 month before implantation to assess the risk for a hypersensitivity reaction.

Zyderm I is best injected as superficially as possible for treatment of the fine lines around the eyes and mouth with approximately 200% overcorrection. Zyderm II is best suited for scars and glabellar frown lines and should also be placed in the superficial dermis. Zyplast is reserved for deeper lines (e.g., nasolabial folds) and larger defects (e.g., surgical/traumatic scars) and may be used as a base for more superficially placed Zyderm I or II. Zyplast should be placed in the middermis without visual overcorrection (it should be palpable but not seen).

The major disadvantage of collagen implantation is its relatively short duration (3 to 6 months), thereby requiring frequent reimplantation. Side effects are rare but include allergic reactions (3% to 5%) that are manifest clinically as erythema and induration. Systemic steroids may be used to treat this condition, but it may recur after discontinuation of the steroids. Two other serious, albeit rare, complications of collagen implantation include vessel occlusion and abscess formation. The risk for vessel occlusion is highest with the use of Zyplast in the glabellar region. The area immediately becomes painful and dusky colored, whereupon the injection should be discontinued and ice applied. Abscesses may form at any time (even several months) after implantation, and treatment should include the use of intralesional or systemic steroids, as well as incision and drainage. These patients tend to have high levels of anti–bovine collagen antibodies and should not receive further treatment with bovine fillers.

Autologous Filling Agents

ISOLAGEN. Isolagen is an autologous collagen process whereby dermal fibroblasts and type I collagen are obtained from human skin punch biopsy samples and expanded in tissue culture over a 6-week period. A series of injections (1 mL) are delivered to the desired sites at biweekly intervals. The

process should be theoretically associated with a longer duration of correction because increased local production of collagen and decreased degradation by collagenase would be expected.

The thin Isolagen solution is best used for the treatment of fine lines and shallow atrophic scars and is administered through a 30-gauge needle into the upper dermis with 200% to 300% overcorrection. Two to four injection series are necessary to effect improvement of mild to moderate rhytides and scars, whereas five or more sessions are needed for deeper defects. Some studies have shown correction to last as long as 6 months, with the nasolabial folds responding best to treatment. However, other studies have documented its disadvantages, including its subtle treatment effect and greater expense. Because of its low viscosity, most patients find the degree of correction obtained with Isolagen to be less than that obtained with bovine collagen.

AUTOLOGEN. Autologen is another autologous injectable collagen that is harvested directly from the dermis of the recipient patient. Excess skin that is excised during routine surgery is processed so that intact collagen fibers are extracted. Tissue processing takes approximately 3 weeks, and the final product can be stored for up to 6 months.

Autologen is injected into the middermis, and nerve blocks or local infiltration with lidocaine is often required before treatment. Serial injections are made with 20% to 30% overcorrection; most patients undergo three treatment sessions at biweekly intervals. Advantages of Autologen for soft tissue augmentation include a relatively long duration and negligible risk of hypersensitivity. The main disadvantage is the large amount of donor skin required for tissue processing.

Lipotransfer and Lipocytic Dermal Augmentation

Lipotransfer is best suited for correction of larger-volume defects in the subcutis. Lipocytic dermal augmentation is a refinement of the lipotransfer technique that enables the surgeon to inject processed fat into the dermis for correction. With this method, fat is injected intradermally via 23- or 25-gauge needles with slight overcorrection. Dermal thickness is increased over time because of volume expansion and an inflammatory response. Lipotransfer can be used to augment the subcutis in concert with a lipocytic dermal augmentation procedure to fill dermal defects and rhytides. Transferred fat typically lasts 3 to 6 months, although tissue longevity can be enhanced if treatment is repeated 3 to 4 weeks after the initial session.

Allogeneic Human Collagen

DERMALOGEN. Dermalogen is a neutral pH buffer solution of human tissue collagen matrix processed from the dermal layer of deceased donor skin specimens. This Food and Drug Administration (FDA)-approved tissue product consists of intact collagen fibrils, elastin, and glycosaminoglycans and is procured from accredited tissue skin banks. Dermalogen is processed with two viral and prion inactivation steps to ensure safety.

Dermalogen is best used for amelioration of prominent nasolabial folds, glabellar frown lines, perioral rhytides, and atrophic scars. Because of its high viscosity, Dermalogen should not be used for fine perioral or periorbital rhytides. Dermalogen should be injected into the mid to deep dermis by a series of injections with a 30-gauge needle at a 30- to 45-degree angle to the skin surface. One must be careful to avoid placement of the material too superficially to prevent the formation of small, white, beaded elevations that resemble milia. Defects should be overcorrected by 10% to 20%. Most patients require three treatment sessions at biweekly intervals for best results.

Side effects of treatment with Dermalogen include burning and stinging at injection sites, prolonged erythema, and acne-like eruptions. The main disadvantages include concern regarding tissue safety and the significant pain experienced during injection.

ALLODERM. AlloDerm is an acellular dermal graft processed from tissue bank–derived human skin. The graft is processed by removal of epidermal and dermal cells, which are antigenic targets of cell-mediated immunity. The dermal tissue that is implanted serves as a matrix for migration and repopulation by the recipient's own fibroblasts and endothelial cells. Allografts are screened for viruses before use. In addition, transplanted tissue is treated with antiviral agents that inactivate human immunodeficiency virus, and the living cells responsible for propagation of viral diseases are extracted from the graft. Thus far, there have been no reported cases of transmission of viral disease as a result of dermal graft implantation.

AlloDerm implantation requires the use of local anesthetics or nerve blocks, or both. The graft is first rehydrated in saline, and a 1- to 2-mm piece is introduced through small incisions in the skin surface, such as the lips, and sutured into place. AlloDerm implants are particularly useful for the treatment of iatrogenic depressions after liposuction.

Synthetics

HYALURONIC ACID. Derivatives of hyaluronic acid include Hylan B gel and Restylane. Hyaluronic acid is a naturally occurring polysaccharide component of the intercellular matrix that is responsible for hydration and stabilization of the connective tissue–containing cells of the dermis. Hylan B gels are cross-linked hyaluronan polymers with high molecular weight and relatively long duration in

tissue. Side effects of treatment include transient erythema and bruising. There is little risk of allergic reaction with this implant because the natural hyaluronan polymer exhibits no species specificity and does not induce a significant immune reaction.

Restylane has several advantages over other filling agents. It is relatively complication free and delivers a predictable soft tissue result that lasts 4 to 6 months. There is minimal pain with injection and it has a natural feel. It should be injected into the reticular dermis via a threading technique. It is most useful for lip, nasolabial fold, and "marionette" lines.

ARTECOLL. Artecoll is an implant made of polymethylmethacrylate beads suspended in 3.5% atelocollagen with lidocaine. The telopeptide ends of the collagen moiety are removed to reduce antigenicity. Artecoll beads range in size from 30 to 40 μm and are implanted via 27-gauge needles after skin testing to document possible hypersensitivity. Artecoll should be placed between the lower dermis and subcutaneous fat without overcorrection by using a fanning motion and injection on both entering and exiting the skin. After implantation, the collagen moiety is phagocytized over a 1- to 3-month period and is replaced by newly synthesized fibroblasts and collagen fibers. Treatments are repeated at 1- and 3-month intervals for complete correction of defects. Side effects include temporary erythema, edema, pruritus, and moderate pain. Allergic reaction to the bovine collagen portion of the implant is another potential complication, and rates of reaction are similar to those of other injectable bovine products.

POLY-L-LACTIC ACID. Poly-L-lactic acid (Sculptra, Dermik Laboratories) is the latest addition to the expanding armamentarium of filler materials. It is the same material used in absorbable sutures and is indicated for deeper dermal filling to create more durable and greater volume replacement. It is highly biocompatible and nonallergenic; thus it can be used without skin testing. The material is biodegradable, yet the results achieved after a series of 3 to 5 monthly injections are relatively long-lasting, averaging 2 or more years, presumably because of neocollagenesis. It can be used in combination with more superficial filler materials in a layering technique for patients with both volume loss and superficial rhytides.

Poly-L-lactic acid is supplied as a lyophilized powder in a vial that is stored at room temperature. It is reconstituted with sterile water for injection to a volume of 5 mL and left to rehydrate for 2 hours. The contents of the entire vial are used at each session (in 0.1-mL to 0.2-mL aliquots) for cosmetic correction of nasolabial or mesolabial folds. Greater volumes may be needed to achieve the desired results in persons with more profound defects, such as in individuals with HIV-associated lipoatrophy.

SUGGESTED READINGS

Alster TS: Cutaneous resurfacing with CO_2 and erbium:YAG lasers: Preoperative, intraoperative, and postoperative considerations. Plast Reconstr Surg 103:619-632, 1999.

Alster TS, Lupton JR: Prevention and treatment of side effects and complications of cutaneous laser resurfacing. Plast Reconstr Surg 109:308-316, 2002.

Alster TS, West TB: Human-derived and new synthetic injectable materials for soft-tissue augmentation: Current status and role in cosmetic surgery. Plast Reconstr Surg 105:2515-2525, 2000.

Elson ML: Soft tissue augmentation: A review. Dermatol Surg 21:491-500, 2000.

Hardaway CA, Ross EV: Nonablative laser skin remodeling. Dermatol Clin 20:97-111, 2002.

Klein AW, Elson ML: The history of substances for soft tissue augmentation. Dermatol Surg 26:1096-1105, 2000.

Hair Restoration

ALFONSO BARRERA

Over the past decade, significant advances have been made in hair restoration. Micrografts (one- to two-hair follicular unit grafts) and minigrafts (three- to four-hair follicular unit grafts) have revolutionized hair restoration and have become the standard of care. Other previously described techniques such as tissue expansion, scalp flaps, and scalp eduction should certainly be kept in the surgeon's armamentarium.

The use of single-hair grafts is not a new concept. Tumura in 1943 reported the use of single-hair grafts to restore pubic hair, and Fujita in 1953 described their use in eyebrow reconstruction. Their application to the scalp was reported by Nordstrom in 1981 and by Marritt in 1984. They introduced the application of single-hair grafts to the frontal hairline to camouflage plugs and scars from previous hair restoration procedures. This technique modification resulted in a more natural-looking hairline by providing an anterior transition zone.

Uebel in 1991 introduced the concept of micrografts (one- or two-hair grafts) and minigrafts (three- or four-hair grafts) applied in large numbers (1000 to 1200 grafts) to cover large areas of hair loss in male pattern baldness. The author has subsequently increased the number of grafts transplanted in a single session (up to 2500) and has also described multiple other applications for micrografts and minigrafts: reconstruction of natural-appearing sideburns, eyebrows, mustaches, and beards. These techniques can be applied to other body areas as well, including the lower extremities.

Anatomy

In 1984 Headington described the concept of the *follicular unit*. Histologic study of the skin traditionally focused on vertical sections. He studied transverse microscopic sections of the human scalp and observed that hair grows in follicular units of one to four hair follicles with independent neurovascular bundles, sebaceous glands, sweat glands, and piloerectile muscles surrounded by a sheath of collagen (Fig. 1). It was theorized that maintenance of these true physiologic units as intact as possible would increase survival and ultimate hair growth of the grafts.

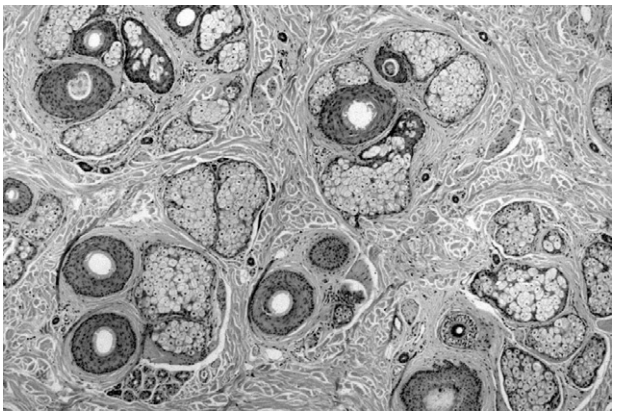


Figure 1 • Transverse histologic section of the scalp demonstrating follicular units.

The life cycle phases of a hair follicle are as follows: *anagen* is the actively growing phase in which follicular cells are multiplying and keratinizing. In a nonbalding scalp, approximately 90% of the hairs are normally in this phase, which lasts approximately 3 years. During the *catagen* phase, the base of the hair becomes keratinized, forms a club, and separates itself from the dermal papilla. It moves toward the surface and is eventually connected to the dermal papilla by only a connective tissue strand. This phase lasts 2 to 3 weeks. During the *telogen* or resting phase, the attachment at the base of the follicle becomes weaker until the hair finally sheds. During this period the follicle is inactive and hair growth ceases. This phase lasts 3 to 4 months and commonly occurs after hair transplantation. For this reason, significant growth of the hair grafts is

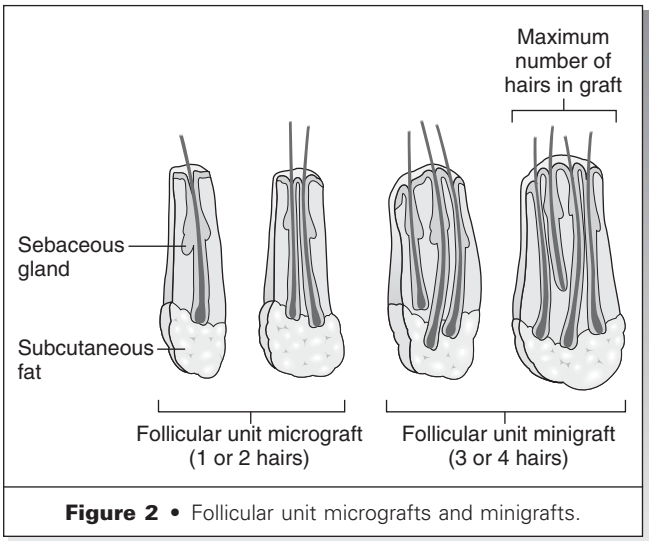


Figure 2 • Follicular unit micrografts and minigrafts.

not seen until the telogen phase is over. In addition, some of the native hair often enters the catagen phase and later the telogen phase as a result of the insult of the surgery, a process termed *telogen effluvium*.

Goals of Reconstruction

The most optimal hair transplantation results are obtained with one- and two-hair follicular units (micrografts) and three- and four-hair follicular units (minigrafts) (Fig. 2). When dissecting grafts, two, three, or four hair follicles are kept together as a unit, and the tissue around the hair shaft of single-hair grafts is preserved to ensure survival of all vital components.

The appearance of hair fullness is determined by hair mass, which is related to the number of hairs, the thickness of the individual hair shafts, and the texture, curliness, and color of the hair. In addition, the contrast in color between the scalp and the hair also has a significant influence on the optical illusion of fullness. An average healthy nonbalding patient has a density of about 200 hairs/cm^2 (range, 130 to 280). Only 50% of this number is needed to give an appearance of normal density, which is approximately 100 hairs/cm^2 (range, 65 to 140). This number can be transplanted in two sessions of micrografting and minigrafting.

Patient Selection

There is no known method to create new hair, and all current techniques for hair restoration involve redistributing the patient's existing hair. Therefore, candidates for hair transplantation are limited to individuals who have a favorable donor site surface area and density relative to the size of the area to be transplanted. Several centers worldwide are attempting to clone hair follicles or culture and multiply hair follicles in the laboratory setting. If successful, patients with limited donor hair (follicularly challenged patients) will be treated by limited harvest of hair follicles, while at the same time eliminating donor site morbidity and discomfort.

Male pattern baldness is a progressive condition. Although the rate of hair loss slows after the age of 40 years, it never stops. Therefore, the preoperative plan must ensure a natural-looking long-term result. It is essential to document realistic expectations by the patient.

Reconstructive Applications of Micrografts and Minigrafts

Micrografts and minigrafts can also be transplanted anywhere on the face and are thus useful for restoring sideburns, the temporal hairline, eyebrows, mustache, and beard. The most common reasons for using micrografts and minigrafts in reconstructive cases include the following:

1. Revision of unfavorable results from previous hair transplantation procedures (the "plug look," cornfield rows, and hairline scars)
2. Correction of hair loss after thermal injuries
3. Posttraumatic injuries
4. Postsurgical conditions (the loss of sideburn and the temporal hairline after a face lift)
5. Certain congenital conditions (absence of hair on the prolabium in bilateral cleft lips, hair loss as a result of excision of congenital hairy nevus or arteriovenous malformations).

Treatment

Male pattern baldness is usually treated with between 1000 and 2500 grafts per session (megasessions), depending on the degree of hair loss. This labor-intensive procedure requires an organized and efficient surgical team.

The surgical team consists of three surgical assistants and the surgeon. The surgeon should remain in the operating room for the duration of the procedure and insert all grafts personally. Efficiency is critical when transplanting a large number of grafts in a single session.

Supraorbital and occipital nerve blocks are performed with 0.5% bupivacaine with 1:200,000 epinephrine (approximately 30 mL). In addition, a ring block is also induced below the proposed hairline. A tumescent solution of 0.5% lidocaine with 1:200,000 epinephrine is infiltrated into the donor site.

The patient's head is turned to the left and the right half of the donor ellipse is harvested. Under

3.5× loupe magnification, thin slices (1.5 to 2.0 mm thick) are dissected from the donor ellipse with Personna Prep blades over a sterile wooden board and handed to the assistants. The assistants prepare the final grafts from these slices while the surgeon closes the right half of the donor strip. Careful dissection of the thin slices into micrografts and minigrafts is the most tedious part of the procedure and one of the most important steps. The incisions must be made precisely parallel to the hair shafts at all times to minimize the loss of hair follicles.

Graft Dissection

To dissect the slices into micrografts and minigrafts, the assistants use 3.5× loupe magnification, No. 10 blades, and jeweler's forceps. The darker and thicker the individual hair shafts, the easier it is to dissect the grafts. Ideal grafts should have intact hair shafts all the way from the subcutaneous fatty tissue to the scalp surface, be somewhat thick, and contain from one to four hairs. The grafts are held atraumatically by the fatty tissue under the hair bulbs or by the tissue around them, not by the hair bulb or dermal hair papilla itself. In patients with light-colored or white hair, an operating microscope is used for a safe dissection of the grafts. The surgeon then turns the patient's head to the right for harvesting the left half of the donor ellipse. The donor site on the left side is subsequently closed, and the surgeon finishes slicing the remaining segment of the donor ellipse.

Several hundred grafts have been dissected at this point. They are lined up in rows on a wet towel in preparation for insertion. The process of graft dissection and insertion continues until all the grafts are transplanted. It is imperative to keep the grafts wet in normal saline solution and ideally chilled to approximately 4° C until inserted.

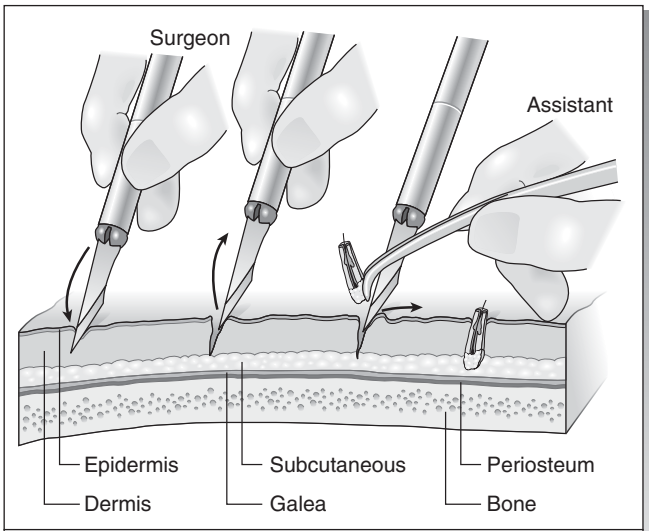
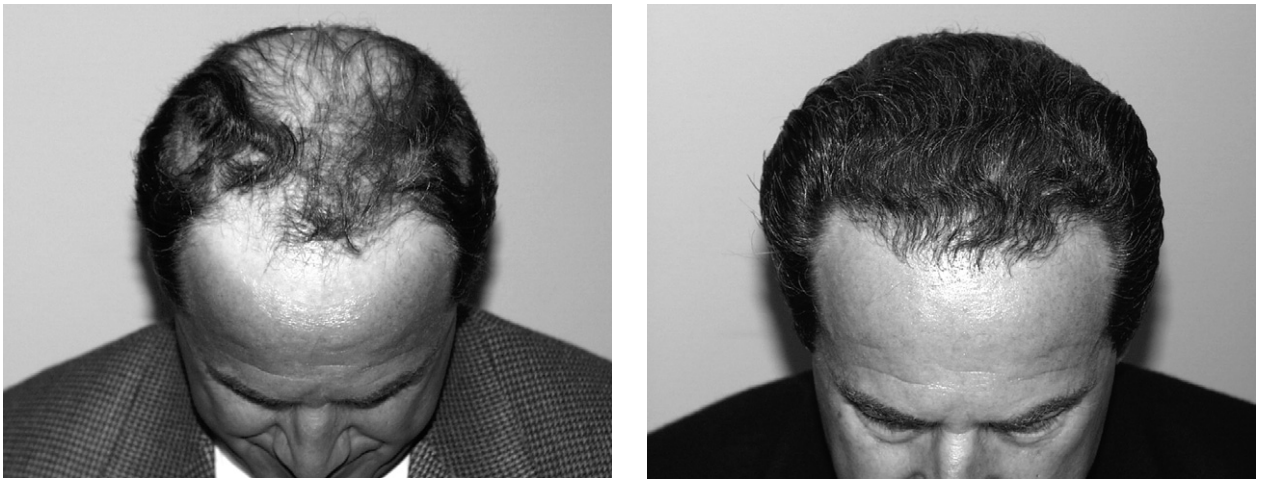


Figure 3 • Technique of micrograft and minigraft transplantation: "slit and slide."

Graft Insertion

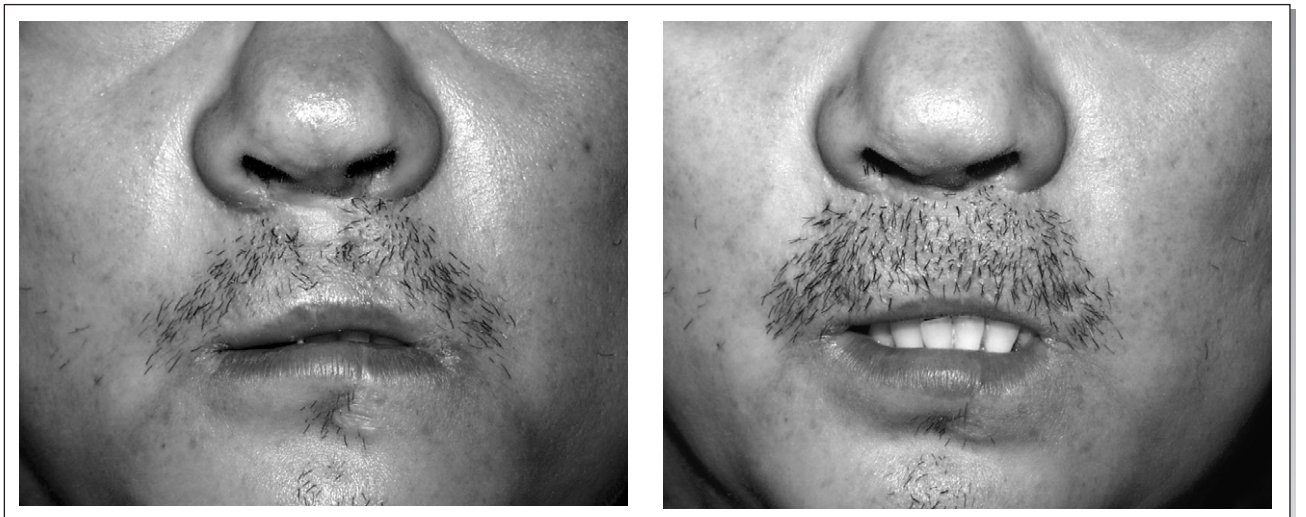
Infiltration of tumescent solution into the recipient area is important to promote hemostasis and produce temporary edema of the scalp, which facilitates graft insertion.

A slit and insert technique eliminates the need for punch removal of scalp or for dilators. As a slit is created, a graft is immediately inserted (Fig. 3). In the anterior part of the scalp, an intentionally irregular 1-cm hairline transition zone (Fig. 4) is



A B

Figure 4 • Severe hair loss (male pattern baldness type VI) in a 54-year-old man. **A,** Before transplantation. **B,** A year after a single megasession of 2070 micrografts and minigrafts.



A B

Figure 5 • Bilateral cleft lip deformity and absent hair in the prolabium of a 40-year-old man. **A,** Before transplantation. **B,** A year after a single session of 200 micrografts and minigrafts.

created by making slits with a No. 22.5 Sharpoint blade. Posterior to the transition zone, No. 11 Feather Personna blades are used to create the slits. The same technique is used elsewhere on the face. When restoring the eyebrows, mustache, or beard, it is best to make most or all of the slits initially and insert the grafts later to minimize graft extrusion.

When a small number of grafts is needed, they are harvested from a donor ellipse extending from the occipital area posterior to the mastoid. The size of the donor strip varies from very small to large, depending on the number of grafts required, as well as donor site hair density. The slits on the face are made with a No. 22.5 Sharpoint blade or a NoKor 18-gauge needle. The blade must be inserted at the angle of the desired direction of hair growth (Fig. 5).

Revisions are occasionally necessary. If large plugs from a previous hair transplant are present, plug reduction (partial removal of the plugs) is performed by coring out the hair follicles with a 2- to 4-mm punch biopsy forceps. These plugs can be recycled as micrografts and minigrafts. Additional grafts (if available) are inserted in front of and between the plugs to obtain an optimal aesthetic result.

Postoperative Care

One or two layers of Adaptic, Kurlex, and 3-inch elastic Ace bandage are used to dress the scalp, whereas nonadherent gauze and ½-inch Steri-Strips are used on the face.

The hair transplanted to the face is usually harvested from the scalp. It has the characteristics of scalp hair and thus needs to be trimmed frequently. Axillary or pubic hair can also be used, but a larger donor ellipse is required because these areas normally have less hair density.

Complications

Complications are few. The author has treated more than 500 patients without a single incident of infection or hematoma.

Keloids are rare but may develop at the donor site. If grafts are inserted too deep, ingrown hairs and small inclusion cysts may develop.

Pearls and Pitfalls

- For hair restoration, micrografts and minigrafts result in a natural-appearing hairline, and scarring is usually undetectable.
- Megasessions may limit the patient to one or two procedures, thus minimizing downtime.
- The direction of hair growth can usually be controlled by angling the surgical blade; this is most useful in difficult areas such as eyelashes and eyebrows.
- Hair transplantation is a useful adjunct for other types of alopecia including posttraumatic and congenital defects, such as those of the eyebrows, lips, and sideburns.

SUGGESTED READINGS

Barrera A: Refinements in hair transplantation: Micro and minigraft megasession. Perspect Plast Surg 11:53, 1998.

Barrera A: The use of micrografts and minigrafts for the correction of the postrhytidectomy lost sideburn. Plast Reconstr Surg 102:2273, 1998.
Barrera A: Hair Transplantation—The Art of Micrografting and Minigrafting. Quality Medical Publishing, St Louis, 2002.
Headington JT: Transverse microscopic anatomy of the human scalp. Arch Dermatol 120:449, 1984.
Marritt E: Single hair transplantation for hairline refinement: A practical solution. J Dermatol Surg Oncol 10:962, 1984.
Nordstrom REA: Micrografts for improvement of the frontal hairline after hair transplantation. Aesthetic Plast Surg 5:97, 1981.
Uebel CO: Micrografts and minigrafts: A new approach to baldness surgery. Ann Plast Surg 27:476, 1991.
Vogel JE: Correction of the cornrow transplant and other common problems in surgical hair restoration. Plast Reconstr Surg 105:1528-1536, discussion 1537-1541, 2000.

Alloplastic Materials for Facial Reconstruction

BARRY L. EPPLEY

The strategic use of implantable biomaterials can play a valuable role in aesthetic facial surgery. Significant advances in materials science and implant designs have made certain types of alloplastic implants useful for the aesthetic augmentation of facial landmarks and contours. The increased frequency of use is a direct result of greater patient demand for improvement in facial appearance, the limitations and morbidity associated with autogenous grafting, and tolerance of well-vascularized facial tissues to alloplastic materials.

Regardless of their chemical composition and structure, alloplastic implants ensure the surgeon of two basic features: simplicity of the operative procedure and predictable postoperative volume stability and facial contour changes. Such features are particularly attractive in patients seeking elective aesthetic facial surgery in which the morbidity of the procedure, length of recovery time, and reliability of the outcome are major factors in the decision to undergo surgery.

Etiopathogenesis

The perception of facial beauty is influenced by the symmetry and balance of its various features. For differing genders and races, the relationships of the nose, cheek, chin, and jaw lines contribute powerfully to the visual perception of an individual. Certain soft tissue facial landmarks, such as the size and proportion of the upper and lower lips and that of the nasal tip, have also become important hallmarks of facial beauty. Underdevelopment and disproportion of facial skeletal relationships are the most common causes of inadequate projection and proportion of facial contours. Although most of the soft tissue envelope of the face is directly proportional to the underlying bony support, the size and shape of the nose and lips are more independent of the underlying facial skeleton.

With aging, the osteocutaneous ligaments weaken, subcutaneous fat loss occurs, and the soft tissue envelope descends. These processes result in loss of a youthful appearance accentuated by varying degrees of orbitomalar ptosis, submalar hollowing, deepening of the nasolabial folds, development of jowls, and loss of sharpness of the cervicomental angle. These changes may be accompanied by resorption of the underlying skeleton, which can be further magnified by the loss of teeth and alveolar bone.

Pathologic Anatomy

The surgeon must develop an appreciation of the relationships between various contours and projections of the face. Much of this knowledge can be learned from the quantitative study of anthropometric and cephalometric values of facial norms.

The anatomic findings associated with deficiency of various facial aesthetic units are as follows:

Chin deficiency

- The most anterior projection of the chin lies posterior to a vertical line drawn down from the glabella
- Vertical/anteroposterior shortening of the lower part of the face
- Increased angle of facial convexity
- Increased (obtuse) cervicomental angle
- Deficient lower third of the face
- Class II malocclusion (if severe)

Malar deficiency

- Zygomatic/malar flattening

Perceived "lack of cheek bones"
Concave facial profile
Class III malocclusion (if severe)

Midfacial (paranasal-premaxillary) deficiency
Acute (less than 90 degrees) nasolabial angle (dependent on the position of the nasal tip and maxilla)
Flat nasal base
Concave facial profile
Class III malocclusion (if severe)

Midfacial (infraorbital) deficiency
Infraorbital rim depression or recess (tear trough deformity)

Mandibular ramus deficiency
Round or oval face
Lower facial asymmetry (body and ramus)

Upper/lower lip deficiency
Deficient upper lip pout (horizontally positioned behind the lower lip in profile)
Inadequate upper/lower lip projection (at or posterior to a line drawn from the subnasale to the pogonion in profile)
Inadequate upper/lower lip projection (posterior to Rickett's E-line, or a line drawn from tip of the nose to the anterior projection of the chin)
Undesirable vertical lip ratio (less than a 1:1 ratio of the upper to the lower lip)

Nasolabial fold depression
Accentuation or deepening of the transition zone between the upper lip and infraorbital/malar tissues

Diagnostic Studies

Photographs are the primary modality for aesthetic analysis of the face. Additional close-up views of the brow, eyes, nose, and lips may be obtained as indicated. Computer video imaging is helpful, particularly in morphing the anticipated contours so that the patient can determine whether the recommended changes are beneficial. Cephalometric radiographic analysis in the profile and frontal view defines the relationships of hard and soft tissue landmarks.

Reconstructive Goals

The basic concept of alloplastic facial rejuvenation is *enhancement* of existing facial contours to create balance, harmony, and symmetry, all of which contribute to facial attractiveness. These enhancements may be specifically listed as follows (Fig. 1):

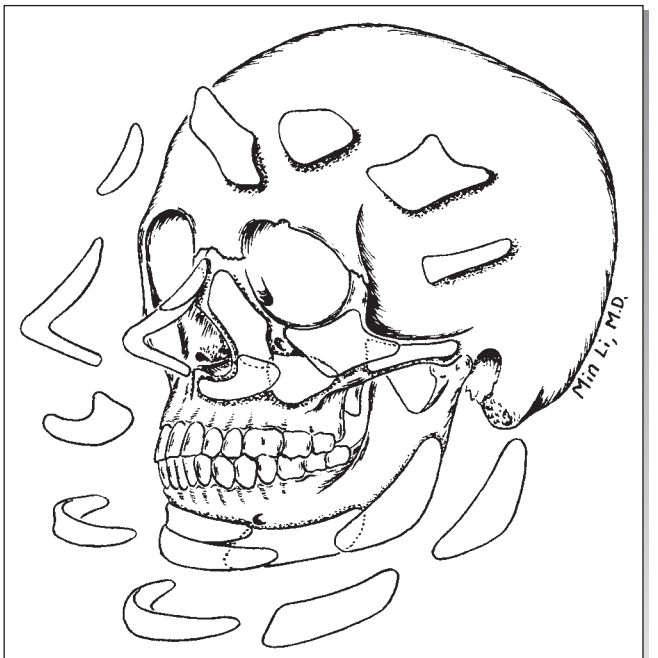


Figure 1 • Potential zones of onlay facial skeletal augmentation.

Augmentation of the malar-midface region
Malar eminence
Zygomatic arch
Palpebral-malar sulcus
Paranasal-premaxillary area
Submalar triangle

Augmentation of the mandibular region
Centrolateral aspect of the chin
Chin-jowl region
Body
Angle-ramus region

Augmentation of the lips
Body of the lips
White roll

Augmentation of the nasolabial region
Nasolabial sulcus
Nasolabial fold

Treatment

Principles of Facial Implant Augmentation

The end-stage healing response to all alloplastic materials is fibrous encapsulation, which serves to separate the body from the foreign material. A fibrous scar forms around all biomaterials im-

TABLE 1 Characteristics of Facial Implant Materials

	SILICONE	MEDPOR	GORE-TEX	MERSILENE
Ease of insertion	++++	++	+++	+
Modifiable	++++	++	++++	+++
Adaptability	++++	+++	++++	++++
Ease of removal	++++	++	++++	+
Implant styles	++++	+++	++	0

planted in the face, regardless of the composition or physical form of the alloplast.

The quality and quantity of soft tissue around a facial implant are the most critical factors for long-term success and are far more important than the chemical composition of the alloplast. Soft tissue coverage over an implant should be as thick as possible. Exposure or extrusion rarely occurs with alloplastic implants that are more deeply placed (i.e., subperiosteal, submuscular). Implants placed immediately under the dermis or in areas of thin overlying subcutaneous fat have a higher incidence of postoperative extrusion or infection.

Placement of alloplastic facial implants should be accompanied by concurrent administration of antibiotics. The rationale for antibiotic therapy is to prevent bacterial inoculation of the implant surface. Additional antibiotic benefit is often sought by washing or soaking the implant before insertion.

Types of Facial Implants

Although many types of materials have been used in the face, few have enjoyed clinical success. Contemporary facial implants are composed of the polymers dimethylsiloxane (silicone), polyethylene (Medpor), polytetrafluoroethylene (PTFE, Gore-Tex), and polyester (Mersilene). Knowledge of the differing materials is essential before their application (Table 1).

SILICONE. Silicone is used primarily as subperiosteal onlay implants for bony augmentation and less frequently at the subcutaneous level, primarily in nasal dorsum placement.

Silicone is a polymer created from the interlinking of silicon and oxygen (with methyl side groups) into alternating monomers of dimethylsiloxane ($SiO[CH_3]_2$); it is resistant to degradation because of the strong and stable silicon-oxygen bonds. When converted into a polymer and vulcanized, a solid rubber is created. Silicone rubber offers the advantages of ease of placement through small incisions, capability of being modified intraoperatively, and low cost.

GORE-TEX. Gore-Tex has been approved as a facial implant material since 1994 and has been widely used for subdermal implantation in the lip, nasolabial folds, glabella, nasal dorsum, and other subcutaneous facial regions, as well as for bony augmentation of the midface, malar, and mandibular areas. Its use as a soft tissue implant, as opposed to a bony onlay, is what distinguishes Gore-Tex from other types of facial implants.

PTFE fluorocarbon has a carbon ethylene backbone to which is attached four fluorine molecules. Bonding of highly reactive fluorine to carbon creates an extremely stable biomaterial that is not biodegradable in the body because of a lack of enzymes capable of disrupting the fluorine-carbon bonds. In addition to its chemical stability, its surface is nonadherent and has antifriction properties. It is flexible, deformable, and easily cut and shaped, with a low tensile strength. It is usually composed of fine expanded PTFE fibrils that are oriented and held together by solid pieces of the same material. The fibrillar composition results in noninterconnected surface openings with pore sizes ranging between 10 and 30 μm. The latter property allows for limited soft tissue ingrowth.

MEDPOR. This type of implant material is available in a wide variety of physical forms for bony onlay augmentation. A variety of facial implants are available in different sizes and shapes.

Polyethylene has a simple carbon chain structure that is differentiated from Gore-Tex by the lack of fluorination of the ethylene monomer. It is currently available for facial use as high-density polyethylene (HDPE). Although HDPE and PTFE are chemically similar, HDPE has a firmer consistency that resists material compression but permits a degree of flexibility. It also has significant intramaterial porosity, with pore sizes ranging between 125 and 250 μm, a property that permits extensive fibrovascular ingrowth throughout the implant. The implants can be shaped intraoperatively, but with more difficulty than with silicone or Gore-Tex. Its porosity enables it to be loaded with antibiotics by syringe vacuum impregnation.

MERSILENE. Mersilene is a linear aromatic thermoplastic polymer created by the establishment of ester linkages between the carbon bonds. The knitted multifilament mesh is commonly used as a

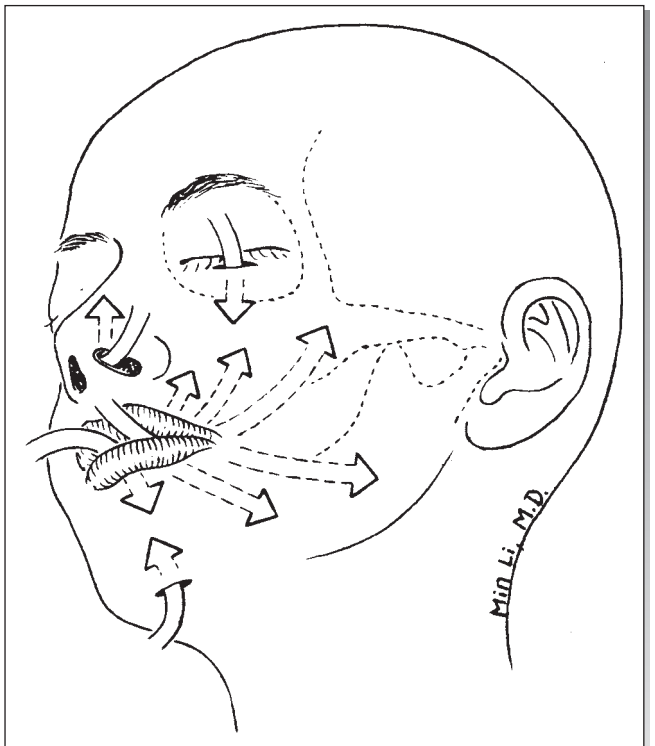


Figure 2 • Surgical approaches for placement of facial implants.

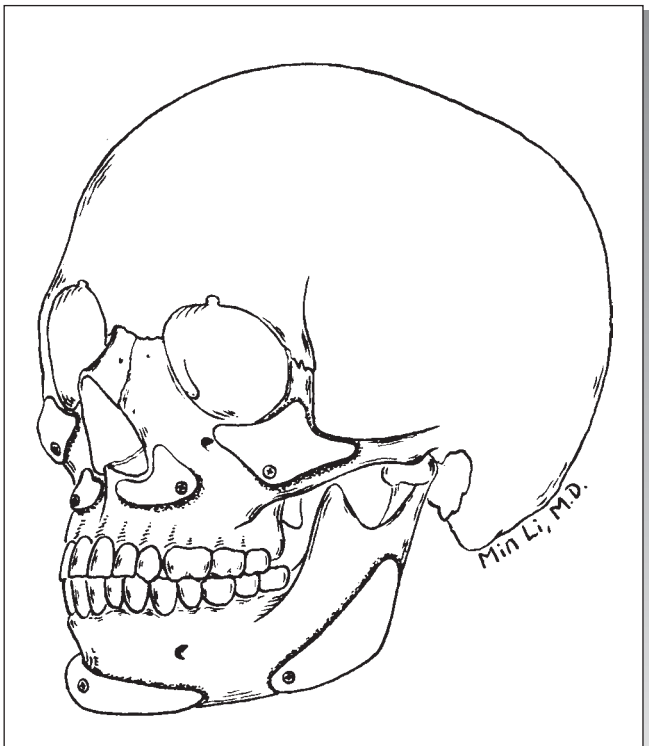


Figure 3 • Technique of screw fixation of onlay implants.

synthetic mesh and is also known chemically as polyethylene terephthalate. The material is essentially nonreactive and becomes encased in an interwoven fibrous tissue matrix. It has been placed as onlay facial and nasal augmentation material.

Surgical Technique

The technical aspects of placing alloplastic implants in the face are fairly simple. However, certain technical guidelines need to be followed to optimize results.

Proper implant selection is the most critical factor in achieving the desired location and amount of facial enhancement. Contemporary facial implants have nearly 20 different styles for augmentation of the midface and mandibular skeleton (see Fig. 1).

All onlay implants are easily placed through an intraoral route, and insertion is rapid and carries little risk of motor nerve injury (Fig. 2). In a face lift patient, the submental route can be used for chin augmentation because of the convenience of the preexisting incision. In experienced hands, malar augmentation can also be accomplished through traditional blepharoplasty or facelift incisions.

Implant fixation to bone is attained intraoperatively by precise pocket dissection and subsequent capsule formation. To ensure maintenance of implant position, titanium screws (1.5 mm) are placed through the implant into bone (Fig. 3).

Closure of intraoral incisions over onlay implants should be performed in two layers to prevent exposure.

Soft tissue implants to enhance the nasolabial fold and lips are most effective if inserted in the shape of oval and round tubes. They are threaded into the recipient site with the aid of trocars or curved alligator forceps (Fig. 4).

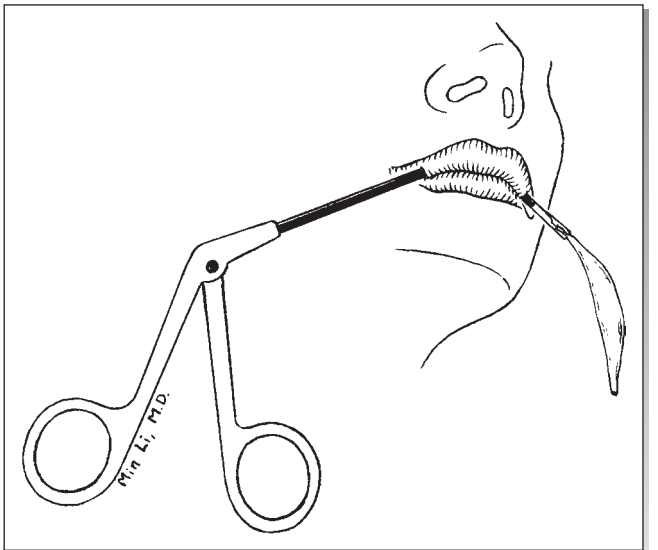


Figure 4 • Upper lip augmentation with a tubular implant.

Postoperative Care

Postoperative oral antibiotics are prescribed. Light compressive facial dressings are recommended for the first 24 hours for edema control. Because the dimensions of the implant pocket or fixation to the underlying bone controls implant position, external implant stability methods are not recommended.

Pearls and Pitfalls

- Dissect the pocket with adequate dimensions. Attempting to make an implant fit into a pocket that is too constricted leads to a higher incidence of external contour deformity and implant palpability.
- For onlay augmentation of the facial skeleton, sufficient subperiosteal undermining should be performed to ensure optimal positioning of the implant.
- Soft tissue implants should be placed as deeply in the tissues as possible while permitting the desired amount of external contour change.
- Skeletal implants may be fixated with titanium screws to prevent malposition or migration.
- Infected implants should be removed. Salvage by administration of antibiotics is rare, especially in soft tissue implants.
- Choose implant materials based on the handling quality of the material. Complications do not seem to differ with the various materials used.

SUGGESTED READINGS

Eppley BL: Alloplastic implantation. Plast Reconstr Surg 104:1761, 1999.

Terino EO, Flowers RS: The Art of Alloplastic Facial Contouring. St Louis, Mosby, 2000.

Breast Cancer

MONICA MORROW
NEIL FINE

Breast cancer is the most common malignancy in American women, with approximately 211,240 new cases and 40,000 deaths projected to occur in 2005. The increased use of screening mammography has resulted in small, node-negative breast cancer or ductal carcinoma in situ (DCIS) being diagnosed in a higher proportion of women than in the past. The increased number has come with a concomitant increase in the number of options available for local therapy. This chapter reviews local therapy for breast cancer, as well as the indications for postoperative irradiation and chemotherapy.

Local Treatment of Breast Cancer

Breast-Conserving Surgery

The majority of women with DCIS or stage I or II breast cancer are eligible for local therapy with breast-conserving surgery (BCS), mastectomy alone, or mastectomy with immediate reconstruction. Contraindications to any type of surgery as the initial treatment of breast cancer are listed in Table 1. These patients should receive primary chemotherapy, with surgery reserved for those who respond to this modality. In patients with early-stage disease (stage 0, I, II), the initial evaluation should identify those with contraindications to BCS or immediate breast reconstruction. Contraindications to BCS fall into three general categories: (1) disease too extensive to be encompassed by limited resection, (2) inability to safely deliver breast radiation therapy (RT), and (3) inability to monitor the patient for local recurrence. These contraindications are summarized in Table 2. Patients with two or more primary tumors in separate quadrants of the breast (multicentricity) and those with persistent tumor at the resection margin are not considered candidates for BCS because of the high likelihood of a significant volume of clinically occult residual tumor that is unlikely to be controlled by RT. Although therapeutic irradiation is contraindicated during any trimester of pregnancy, women in whom breast

TABLE 1 Contraindications to Surgery as the Initial Therapy for Breast Cancer

Distant metastases (stage IV disease)
Extensive nodal involvement manifested by:
Supraclavicular adenopathy
Fixed axillary adenopathy
Edema of the ipsilateral arm
Skin involvement:
Ulceration
Satellite nodules
Inflammatory carcinoma
Fixation to the chest wall, including the:
Ribs
Sternum
Serratus anterior
But *not* the pectoralis major or minor muscles

TABLE 2 Contraindications to Breast-Conserving Surgery

ABSOLUTE
Multicentric carcinoma
Inability to achieve negative margins after reasonable surgical attempts
History of previous therapeutic irradiation to the breast area
First or second trimester of pregnancy
Diffuse indeterminate or suspicious microcalcifications on mammography
RELATIVE
Large tumor in a small breast
Active systemic lupus erythematosus or scleroderma

cancer is diagnosed during the third trimester can undergo local excision with RT delayed until after delivery. For those in whom cancer is diagnosed in the first or second trimester, the delay in treatment is too long for BCS to be routinely recommended. Anecdotal reports of severe fibrotic reaction and tissue necrosis after RT in patients with lupus or scleroderma have made many radiation oncologists reluctant to treat such patients, thus making these conditions relative contraindications to BCS. If a poor cosmetic outcome is anticipated because of the size of the tumor in relation to the size of the breast, the advantage of BCS is lost. However, as discussed later, initial chemotherapy or endocrine therapy can be used in an attempt to shrink the tumor before surgery, thereby potentially allowing the patient to undergo BCS. Partial breast reconstruction is another option in this circumstance.

The frequency of contraindications to BCS is related to tumor stage. A study of 432 consecutive patients with stage 0 (DCIS), I, or II carcinoma demonstrated that only 22.5% had contraindications to BCS. For women with stage I disease, this number fell to 10%, but it was 30% for those with DCIS or stage II disease. Although DCIS is most commonly detected as calcifications on a mammogram when it is still clinically occult, it often occurs as a more diffuse lesion than invasive carcinoma, resulting in a 30% incidence of contraindications in this group. Contraindications to BCS are readily identified by history, physical examination, and a diagnostic mammogram. In contrast to a screening mammogram, which includes only two views of the breast (craniocaudal and mediolateral oblique), a diagnostic mammogram includes magnified views of the tumor to more precisely define its extent, thereby allowing better determination of the amount of breast tissue that must be removed (Fig. 1).

The success of BCS is measured by the incidence of local recurrence in the preserved breast, as well as by cosmetic outcome. Many studies have classified cosmetic outcome by using a rating scale of *excellent* if the treated and the untreated breasts are indistinguishable (except for the surgical scar), *good* when the treatment effect is minimal, and *fair* if there is a significant treatment effect, which may include retraction, contour deformity, a poor surgical scar, loss of ptosis, or radiation effects such as hyperpigmentation or telangiectasia. A rating of *poor* is assigned for major deformities in the breast. In large studies using modern radiation techniques, 85% to 90% of patients rate their cosmetic outcome as good to excellent. Patient evaluations of cosmesis

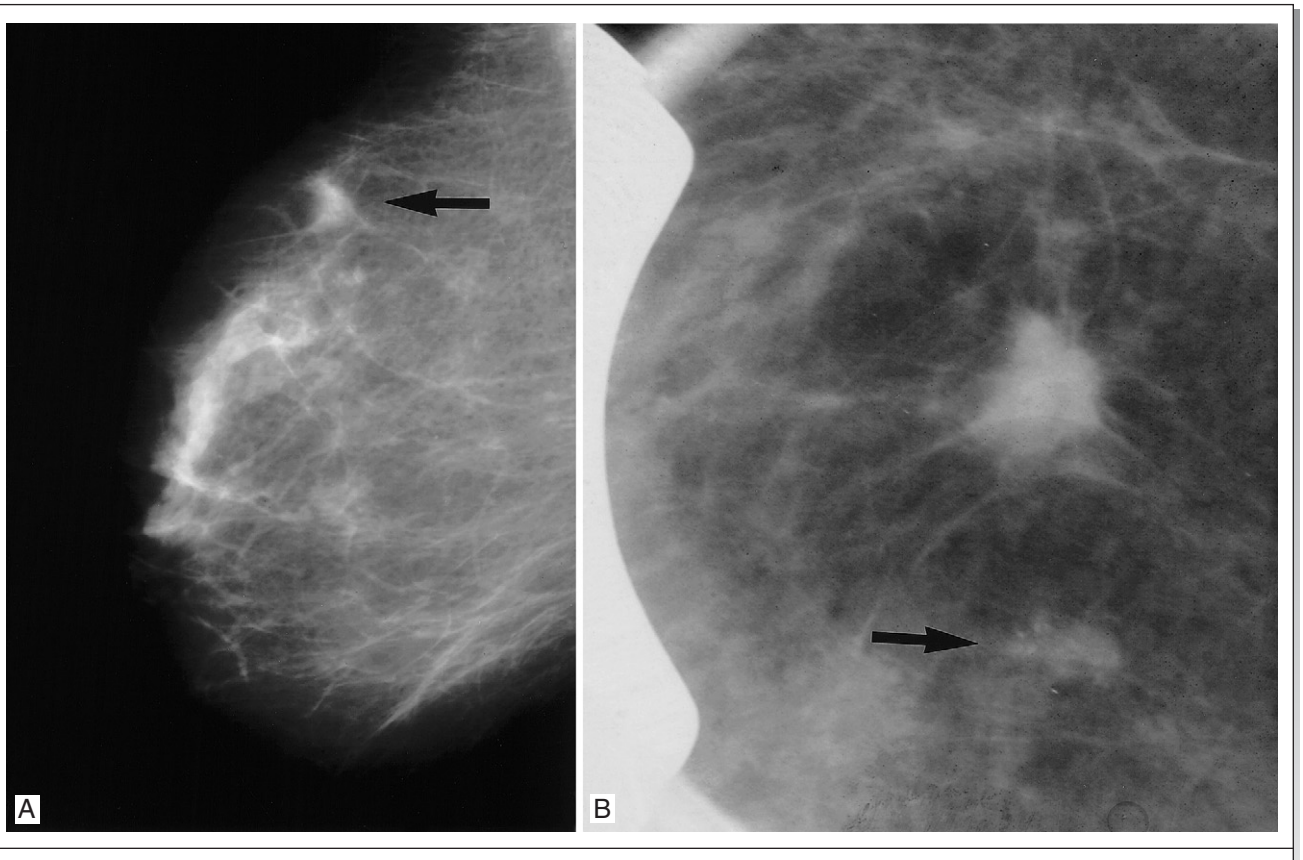


Figure 1 • A, Mediolateral oblique mammogram on which spiculated cancer (*arrow*) is clearly evident. **B,** Magnified view of the area of cancer demonstrating a second separate area of cancer (*arrow*) not well demonstrated on the nonmagnified view.

after BCS tend to be slightly more favorable than those of their physicians.

The final cosmetic outcome after BCS is not usually reached until approximately 3 years after the completion of radiation therapy. During the early posttreatment period, edema may obscure defects in the breast. Fibrosis and retraction continue to occur for the first 3 years after RT. Studies of cosmetic outcome with follow-up for 8 years after treatment suggest that the treated breast remains stable after 3 years, although events that affect the nontreated breast such as pregnancy, major weight gain or loss, and the normal aging process may continue to affect symmetry. Sensation in the treated breast may have a major impact on patient satisfaction. After BCS and RT, the treated breast maintains normal sensation, except perhaps in the area of the surgical scar. For some women this is a major advantage over mastectomy and reconstruction.

Multiple factors have been suggested to influence cosmetic outcome, including initial breast size (with both large and small breasts said to be associated with poor cosmesis), the amount of resection, the use of adjuvant chemotherapy, and the technique and dose of RT. The amount of breast tissue removed appears to be the major determinant of cosmetic outcome, although the extent of deformity varies with the quadrant in which the tumor is located and the distance from the skin surface. In the past, the need to remove the nipple-areolar complex as part of BCS was considered an indication for mastectomy. Although the cosmetic outcome after this procedure is not as optimal as when the nipple and areola are preserved (Fig. 2), the patient is left with a sensate breast mound.

The desire to minimize resection of normal breast tissue must be balanced by the need to minimize the risk of local recurrence in the preserved breast. Early studies suggested that large breast resections (quadrantectomy) were associated with a lower risk of local failure than were more limited resections of normal tissue (lumpectomy). In a randomized trial, local failure rates at 4 years were 2.2% in the quadrantectomy group and 7.0% in the lumpectomy group. Improvements in mammography, the extent of histologic evaluation of the lumpectomy specimen, and the use of adjuvant systemic therapy have resulted in a decrease in the rate of local recurrence after lumpectomy over time, with modern studies reporting local failure rates of 5% to 6% at 10 years. The extent of surgery needed for lumpectomy can be individualized according to the mammographic appearance and the histology of the tumor. Infiltrating ductal carcinomas grow as a cohesive mass, and minimal negative margins (defined as tumor cells not touching an inked surface) reliably indicate removal of the bulk of the tumor. In contrast, infiltrating lobular carcinoma and infiltrating ductal carcinoma with an extensive intraductal component grow in a more diffuse fashion and may have gaps between areas of tumor. For these malignancies, excision with a 5-mm or greater negative margin is prudent.

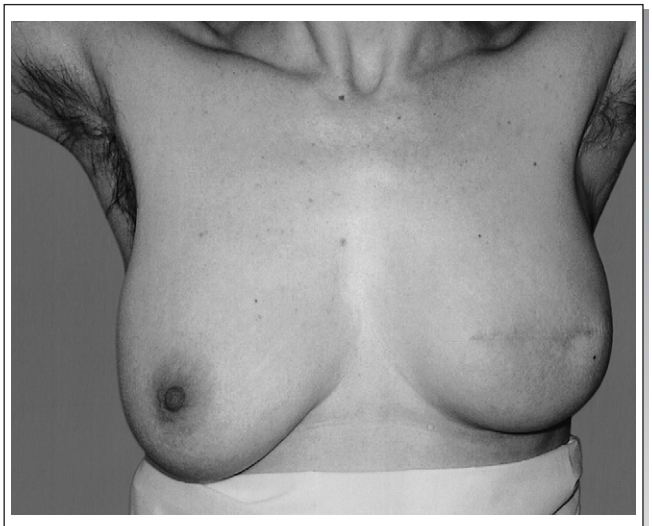


Figure 2 • Cosmetic outcome of breast-conserving surgery with removal of the nipple-areolar complex. The uplifting of the breast is secondary to radiation.

Technique of Lumpectomy

Skin incisions in the upper part of the breast should be placed in lines of minimal tension. Circumareolar incisions, although cosmetically preferred, are not used for tumors in the periphery of the breast because they do not provide adequate exposure to ensure complete tumor removal. In the inferior portion of the breast, radial incisions usually produce less alteration in nipple position than skin crease incisions do. Preservation of subcutaneous fat is a key element in maintaining breast contour. Breast tissue should be incised to a depth of 5 to 10 mm above the tumor before excision is carried out. The specimen is marked with orienting sutures so that if there is tumor at the margin, its precise location will be known and reexcision can be limited to the involved margin surface. The breast cavity is not reapproximated and drains are not placed. Allowing the breast cavity to fill with serum helps to maintain breast contour and makes resection cosmetically acceptable in the majority of women with small breasts (Fig. 3).

Breast Conservation in Women with Large Tumors

For a woman with a large tumor in a small breast who would not be a candidate for conventional BCS, preoperative chemotherapy or endocrine therapy may be indicated to shrink the tumor. In the United

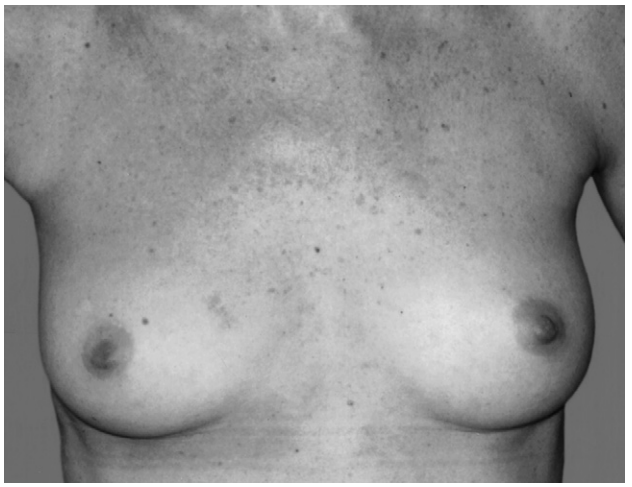


Figure 3 • Outcome of breast-conserving surgery and radiotherapy in a woman with small breasts and a primary tumor in the medial aspect of the breast. No defect is apparent at the lumpectomy site.

States, preoperative chemotherapy has been used much more widely than endocrine therapy. Approximately 80% of tumors respond to preoperative chemotherapy with at least a 50% reduction in the greatest diameter of the tumor. In 10% of cases, pathologic resolution of all invasive cancer is seen. In randomized trials, the use of preoperative chemotherapy increases rates of breast preservation by 8% to 10%.

An alternative approach to a woman with a large tumor that is particularly useful when a significant amount of the tumor is intraductal carcinoma is the use of an endoscopically harvested latissimus flap for partial breast reconstruction. This procedure preserves a sensate breast and nipple, and insertion of the latissimus into the breast does not interfere with follow-up mammography for detection of recurrent tumor. This procedure is ideal for tumors in the upper outer quadrant, but any tumor location other than the lower inner quadrant is amenable to this technique. The procedure is performed in two stages. The initial step is lumpectomy and sentinel node biopsy. After a complete pathology examination verifies negative margins and the absence of metastases in the sentinel node, the reconstruction is performed as a second separate procedure. If axillary dissection is necessary (because of either metastases in the sentinel node or a contraindication to sentinel node biopsy), it is combined with the reconstruction. With this approach, hospitalization is required only for the second stage of the procedure. Margins focally positive for tumor may be safely reexcised at the time of reconstruction. However, if extensive tumor is present at the margin after the initial excision, reexcision should be carried out as a separate procedure to ensure that negative margins can be obtained before harvesting the flap.

Technique of Endoscopic Latissimus Harvest

The patient is placed in the lateral decubitus position and the arm is prepared and draped in the field to allow for full movement and repositioning during the operative procedure. The axillary incision for removal of the sentinel node or axillary node dissection is extended to a length sufficiently long to admit the surgeon's hand (usually 8 to 9 cm). Care is taken to place the incision between the anterior and posterior axillary lines so that it is not visible with the arm in a down position. The border of the latissimus is identified and elevated to expose the thoracodorsal neurovascular pedicle. The serratus branch is ligated and divided to avoid traction injury to the thoracodorsal pedicle. The latissimus muscle is elevated from the chest wall, primarily by blunt dissection. The endoscope is used sparingly, both for lighted retraction and for true endoscopic visualization. Lumbar perforating vessels are cauterized during the dissection if they are clearly visualized. The superficial border of the muscle and the superior/medial and inferior/lateral borders are rapidly dissected free with a combination of scissors and cautery dissection. The origin of the latissimus muscle is divided from the paraspinous border with the use of endoscopic scissors and tactile guidance from the surgeon's inserted hand. The muscle is delivered into the axillary incision and all visible vessels identified and cauterized (Fig. 4). Division of the insertion into the humerus allows the muscle to be transferred via a submammary tunnel into the breast defect. The endoscope is inserted into the back cavity through the axillary incision, and all blood vessels are identified and cauterized. Routine closure and drainage of the back wound are performed (Fig. 5).

Mastectomy with or without Reconstruction

Mastectomy, alone or with immediate reconstruction, should be discussed preoperatively with all patients. The presence of severe comorbid conditions that would preclude the prolongation of general anesthesia is the only absolute contraindication to immediate reconstruction. In the United States, immediate reconstruction continues to be performed infrequently. Between 1985 and 1990, only 3.4% of patients undergoing mastectomy underwent reconstruction within 3 months of their cancer diagnosis. In 1994, this figure had increased to 8.3%. Factors associated with an increased use of reconstruction include noninvasive disease, younger patient age, higher income, and geographic location.

Technique of Mastectomy

When mastectomy is performed without a planned immediate reconstruction, the excess skin of the

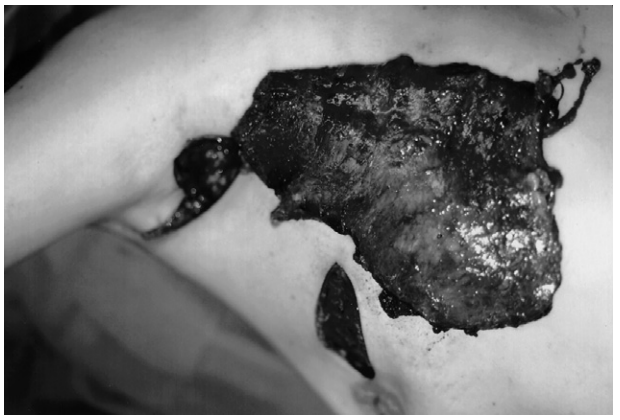


Figure 4 • The latissimus flap has been liberated and tunneled through the right axillary incision.

breast and the nipple-areolar complex are excised. Attempts to preserve excess skin in the event that reconstruction is desired in the future may result in unsightly skin folds that may make wearing a prosthesis difficult, in addition to the skin folds being troublesome to keep clean. After planning the skin incision, the breast is infiltrated with tumescent solution. Flaps are elevated superiorly to the clavicle, medially to the sternum, inferiorly to the rectus sheath, and laterally to the latissimus dorsi. The fascia of the pectoralis major is the deep margin of the resection unless an implant/expander reconstruction is planned. In that case, the fascia can be preserved to facilitate the development of a pocket for the implant. In the past, the pectoral fascia was thought to serve as a barrier to the lymphatic dissemination of tumor cells, and its removal was thought to be important. The demonstration that lymphatic channels pass from the breast through the pectoral fascia and into the pectoral muscles has disproved this theory, thus making fascial preservation a safe practice.

The other modification of mastectomy when immediate reconstruction is being performed is the use of a skin-sparing approach. The only skin that must be removed for oncologic purposes is the nipple-areolar complex and the surgical biopsy site (if present). Diagnostic needle biopsy techniques avoid the problem of incorporating biopsy site incisions. Exposure to perform the mastectomy is obtained by skin incision rather than excision, a strategy resulting in smaller scars and an improved cosmetic appearance (Fig. 6).

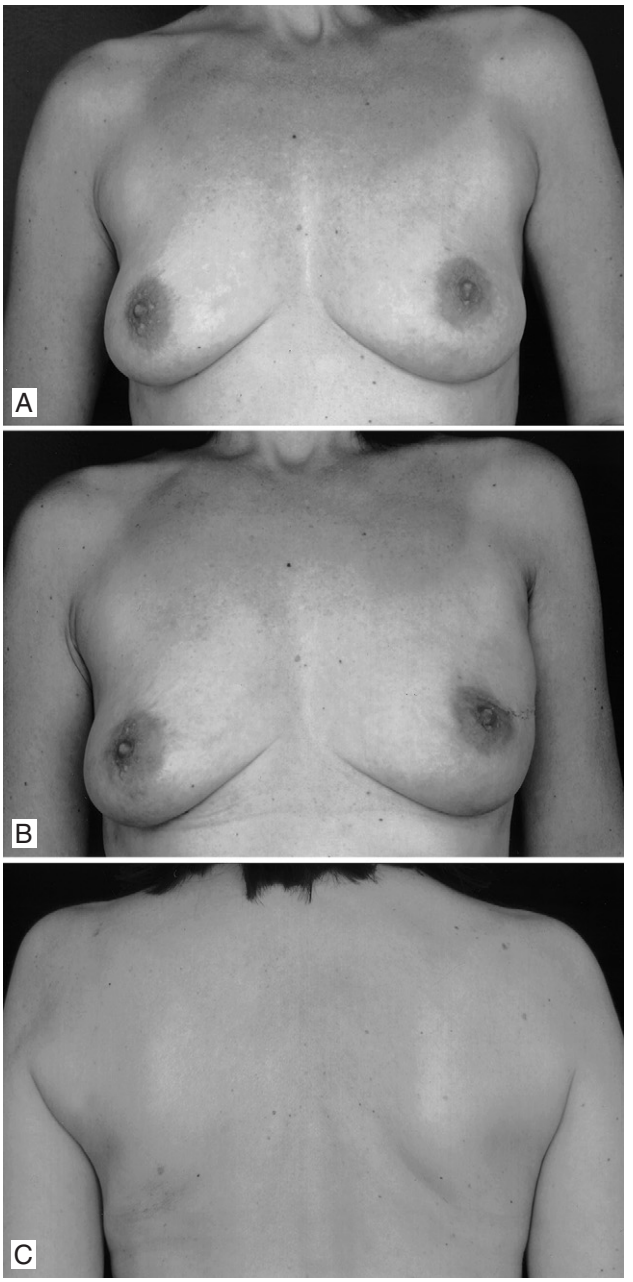


Figure 5 • **A,** Preoperative appearance of the breasts. **B,** Postoperative appearance after reconstruction with an endoscopically harvested latissimus flap (left side). **C,** Postoperative view of the back donor site. Note the minimal asymmetry.

Management of Axillary Lymph Nodes

Therapeutic management of the axilla is currently in a state of transition. For many years, removal of level I and level II axillary nodes was standard practice for invasive carcinoma. Lymphatic mapping and sentinel node biopsy are now replacing axillary dissection as the initial staging procedure for the majority of clinically node-negative patients. The sentinel node, defined as the first node to receive lymphatic drainage from a tumor, is identified by injecting the breast parenchyma around the tumor, the skin overlying the tumor, or the subareolar

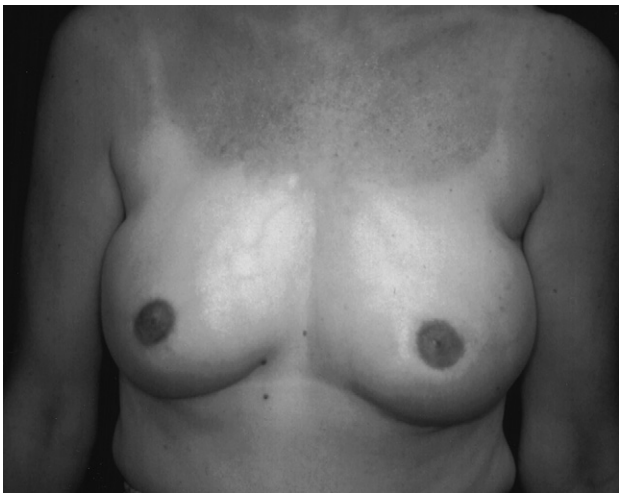


Figure 6 • Postoperative appearance after bilateral mastectomy with transverse rectus abdominis myocutaneous flap reconstruction in association with a skin-sparing approach. The scars from the nipple-areolar excision are incorporated into the areolar reconstruction. The lateral skin incisions, placed to provide exposure for complete breast excision, are faintly visible.

region with a radioactive colloid, 1% isosulfan blue dye, or a combination of the two. A sentinel node can be identified in approximately 90% of women with breast cancer. If the sentinel node is free of metastasis, the remaining axillary nodes are free of metastasis in 95% or more patients. Follow-up studies indicate that isolated axillary recurrence is present in fewer than 1% of patients after sentinel node biopsy alone. The advantages of the procedure are a decrease in both short-term (pain, decreased range of motion) and long-term (lymphedema) morbidity in comparison to axillary dissection. Contraindications to sentinel node biopsy include suspicious palpable adenopathy, previous axillary surgery, and pregnancy. The procedure can be performed whether the breast is treated by BCS or mastectomy.

Adjuvant Radiotherapy

When BCS is performed for invasive cancer, the use of postoperative RT is routine. Nine prospective randomized trials have compared outcomes in patients treated with and without RT. Although individual studies have failed to demonstrate a survival benefit for RT, the use of RT reduces the risk of breast recurrence by an average of 84%. A meta-analysis of these studies does show a small improvement in survival with the addition of RT. The standard method of irradiation had been to treat the entire breast with a dose of 4500 to 5000 cGy, with a boost dose of an additional 1000 to 1500 cGy given to the area of the tumor. For the first 6 years after treatment, the majority of local recurrences develop at or around the site of the primary tumor. After that time, cancer most commonly occurs in other quadrants of the breast and probably represents a new primary tumor rather than local recurrence.

The pattern of local recurrence near the initial tumor site, coupled with the need to perform mastectomy to treat new second cancers after the entire breast has been irradiated, has stimulated interest in the use of breast irradiation limited to the quadrant of the breast in which the tumor is located. It is delivered either through the implantation of catheters in the tumor bed, which allows the radiation to be delivered in a short time, or by external beam technique. Additional follow-up is needed to determine whether long-term local control and aesthetic outcome are equal to that reported after whole-breast RT.

Until recently, the benefits of postmastectomy RT were thought to be limited to reduction of local recurrence in the chest wall and nodal areas. For this reason, postmastectomy RT was reserved for the relatively small subset of women with metastases to four or more axillary nodes and for those with locally advanced breast cancer, and it had little impact on the selection of local therapy. Three recent prospective randomized trials have suggested that RT improves survival in women with nodal metastases and that this benefit extends to those with involvement of only one to three nodes. Postmastectomy RT is now being offered to a larger group of women, and this treatment change has potential implications for the use of immediate reconstruction. Studies have shown that like native breast tissue, flap reconstruction tissue undergoes fibrosis and fat necrosis after RT, and these effects may not be evident until 3 years after treatment. The use of RT after implant reconstruction is associated with a greater rate of implant loss and capsular contracture than observed in nonirradiated patients. The authors' approach to a patient with palpable suspicious nodes and a high likelihood of requiring postmastectomy RT is to place a tissue expander at the time of the mastectomy, thereby allowing skin preservation and maintaining a small scar. Tissue expansion is undertaken during chemotherapy. At the completion of RT, if the patient is not satisfied with the aesthetic outcome, a secondary flap reconstruction can be considered.

Adjuvant Systemic Therapy

The majority of women with invasive breast carcinoma receive some form of systemic therapy: chemotherapy, endocrine therapy, or both. The absolute benefit of adjuvant therapy is based on the risk of recurrence. Adjuvant chemotherapy or tamoxifen reduces the risk of recurrence by approximately a third. For a patient with several positive

nodes who has a 60% risk of relapse, therapy decreases the risk by 20%. The same therapy in a patient with a 10% risk of relapse results in only a 3% absolute benefit. For this reason, adjuvant therapy is recommended for all patients with nodal metastases. For patients with negative nodes, treatment is usually given for tumors larger than 1 cm in diameter. When both chemotherapy and RT are indicated, chemotherapy is administered first. Tamoxifen is given for 5 years, either concurrently with RT or after its completion. Some data suggest that chemotherapy should be initiated within 6 weeks of surgery for maximum effect; it is important that adequate wound healing be achieved within this period. Studies suggest that in the hands of experienced surgeons, the use of immediate reconstruction does not delay the institution of chemotherapy.

SUGGESTED READINGS

Kroll SA, Schusterman MA, Tadjalli HE, et al: Risk of recurrence after treatment of early breast cancer with skin-sparing mastectomy. Ann Surg Oncol 4:193-197, 1997.

Monticciolo DL, Ross D, Bostwick J 3rd, et al: Autologous breast reconstruction with endoscopic latissimus dorsi musculosubcutaneous flaps in patients choosing breast-conserving therapy: Mammographic appearance. AJR Am J Roentgenol 167:385-389, 1996.

Morrow M, Harris JR: Local management of invasive breast cancer: Breast. In Harris JR, Lippman ME, Morrow M, Osborne CK (eds): Diseases of the Breast, 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2004, pp 719–744.

Morrow M, Bucci C, Rademaker A: Medical contraindications are not a major factor in the underutilization of breast conserving therapy. J Am Coll Surg 186:269-274, 1998.

Morrow M, Scott SK, Menck HR, et al: Factors influencing the use of breast reconstruction post mastectomy: A National Cancer Data Base study. J Am Coll Surg 1:1-7, 2001.

Overgaard M, Hansen PS, Overgaard J, et al: Postoperative radiotherapy in high-risk premenopausal women with breast cancer who receive adjuvant chemotherapy. N Engl J Med 337:949-955, 1997.

Overgaard M, Jensen BM, Overgaard J, et al: Postoperative radiotherapy in high-risk postmenopausal breast cancer patients given adjuvant tamoxifen: Danish Breast Cancer Cooperative Group DBCG 82c randomized trial. Lancet 353:1641-1648, 1999.

Veronisi U, Volterrani F, Luini A, et al: Quadrantectomy versus lumpectomy for small size breast cancer. Eur J Cancer 26:671-673, 1990.

Wolff AC, Davidson NE: Primary systemic therapy in operable breast cancer. J Clin Oncol 18:1558-1569, 2000.

Breast Reconstruction

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The breast is an important symbol of femininity. Patients with breast deformities often experiences loss of self-confidence that may affect their everyday life. Breast reconstruction can restore a "sense of self" and help the patient return to a normal life.

This chapter details the surgical techniques used in breast reconstruction, including implants, expanders, local tissue transfers, and microvascular free flaps.

Etiopathogenesis

The causative factors that necessitate breast reconstruction can be divided into two broad categories (1) *congenital*—including those not evident until puberty, and (2) *acquired*—resulting from an insult to the breast (e.g., mastectomy, trauma, burns).

Two common congenital disorders are Poland's syndrome and the tuberous breast deformity. *Poland's syndrome* consists of a spectrum of hypoplastic elements confined unilaterally to the thorax and upper extremity, the most consistent of which is partial or total absence of the pectoralis major muscle. The mildest form of the anomaly includes mild breast hypoplasia and superolateral displacement of the nipple-areola complex. In the most severe form, there may be partial agenesis of the ribs and sternum; brachysyndactyly; mammary aplasia; absence of the latissimus dorsi, serratus anterior, and pectoralis major muscles; scoliosis; and rib deformities. *Tuberous breast deformity* can also consist of a spectrum of soft tissue deformities and in its full expression is characterized by a deficient diameter of the inframammary base, a constricting ring that herniates the nipple-areola complex, hypoplasia of the lower mammary quadrants, and a high location of the inframammary fold.

Acquired breast deformities have multiple causes, including trauma, burns, and recurrent infectious breast disease or mastitis. The most common acquired breast deformity results from the local treatment of breast cancer.

Preoperative Evaluation

Physical examination is the most important component of the preoperative consultation. Three anatomic observations should be made to devise a reconstructive plan:

1. A thorough evaluation of the *breast defect* must be performed. The surgeon must consider the components of the breast that require restoration, including the chest wall, pectoralis major muscle, breast tissue, and nipple-areola complex. It is also paramount to evaluate the quality of the tissues to be reconstructed, particularly if the patient has received radiotherapy.
2. The *contralateral breast* must be examined. It is important to evaluate not only the *size* but also the amount of ptosis present to perform a procedure that will most likely recreate its *shape*.
3. In cases of autologous tissue reconstruction, the *donor site* should be evaluated. Assessment includes an evaluation of any abdominal scars or previous surgery. Previous subcostal incisions or laparoscopic entry sites may divide the superior epigastric pedicle and preclude pedicled transverse rectus abdominis myocutaneous (TRAM) flaps. If the latissimus dorsi muscle is paralyzed or weak, or scapular winging is noted, the thoracodorsal pedicle has most likely been divided. If the thoracodorsal

pedicle is divided proximal to the serratus branch, the latissimus dorsi flap can still be used because reversal of blood flow in the serratus branch allows the muscle to remain vascularized.

At the initial consultation, all reconstructive options should be discussed with any patient considering elective breast reconstructive surgery. The goals and realistic expectations of the patient and surgeon alike should be set forth to ensure the optimal treatment plan.

Imaging studies are necessary only in select cases. Preoperative duplex Doppler examination is advocated by some reconstructive surgeons before free tissue transfer. Other studies might include a magnetic resonance angiogram to evaluate the thoracodorsal pedicle in the case of delayed breast reconstruction.

Reconstructive Goals

The reconstructive goals are to re-create the dimensions, position, and contours of a healthy breast, with adequate projection and a well-defined inframammary fold. Nipple-areolar reconstruction should create an areola of the appropriate size with a projecting nipple. To improve the final aesthetic outcome of the breast, an additional surgical procedure on the contralateral breast may be necessary to achieve symmetry.

Treatment

Poland's Syndrome

Reconstruction of the breast in Poland's syndrome usually consists of a subcutaneous breast implant with nipple-areola reconstruction. Some cases may require chest wall reconstruction with a latissimus dorsi pedicled flap to replace the absent pectoralis major muscle. In cases of severe breast aplasia/hypoplasia with a high nipple-areolar position, expansion is usually necessary before placement of a definitive prosthesis. Some surgeons have advocated the use of autologous tissue techniques to re-create a natural-appearing breast. For example, a free TRAM flap may be necessary to achieve an optimal result (Fig. 1). Because vascular anomalies may be present in these patients, preoperative vascular studies are justified.

Tuberous Breast Deformity

Augmentation with a subglandular implant, spreading of the breast tissue by incision and unfurling, a combination of placement of a subglandular implant with a spreading technique, Z-plasty at the inferior pole of the breast, and reconstruction with autologous tissue are options that can be used to treat tuberous breast deformities. The choice depends on the extent of the deformity.

Breast-Conserving Reconstructive Surgery

Breast conservation therapy usually consists of either local wide excision, lumpectomy, or quadrantectomy.

1. *Reduction mammaplasty.* In patients with large breasts and partial defects, reduction mammaplasty with various pedicles (inferior, medial, superior) that include the nipple-areola complex can give a natural shape and satisfactory results.
2. *Latissimus dorsi muscle filling.* In patients with small breasts, correction of partial mastectomy defects may be achieved by using a pedicled latissimus dorsi flap to restore the tissue deficiency. If the tissue has significant radiation injury, it may be necessary to replace the skin with a myocutaneous latissimus dorsi flap. This procedure can be performed with endoscopic assistance.
3. *Local flap reconstruction.* A skin paddle from the local chest wall or deepithelialized, pedicled flaps may be used to reconstruct partial breast defects. The thoracoepigastric flap based on a lateral branch of the superior epigastric artery is the most widely used option. A thoracodorsal artery perforator flap rotated from the back to the breast is another local reconstructive option.

Total Mastectomy Reconstructive Surgery

The skin markings should be made before mastectomy. In general, the sternal notch, chest midline, and inframammary fold are marked to delineate the extent of dissection. After mastectomy it is important for the reconstructive surgeon to be aware of the viability of the skin flaps. Skin flaps that are thin, especially in patients with potentially compromised vasculature (e.g., smoker, diabetic, irradiated), may result in necrosis and should be trimmed before placement of a tissue expander or insetting of the flap.

Expander Implant Reconstruction

Candidates for implant reconstruction include women with small breasts who do not desire autologous tissue reconstruction or require bilateral mastectomies. Ideal patients have lax skin of good quality and an intact pectoralis muscle. Through previous incisions, the lateral borders of the pectoralis major and serratus anterior muscles are identified. A subpectoral plane is established, and

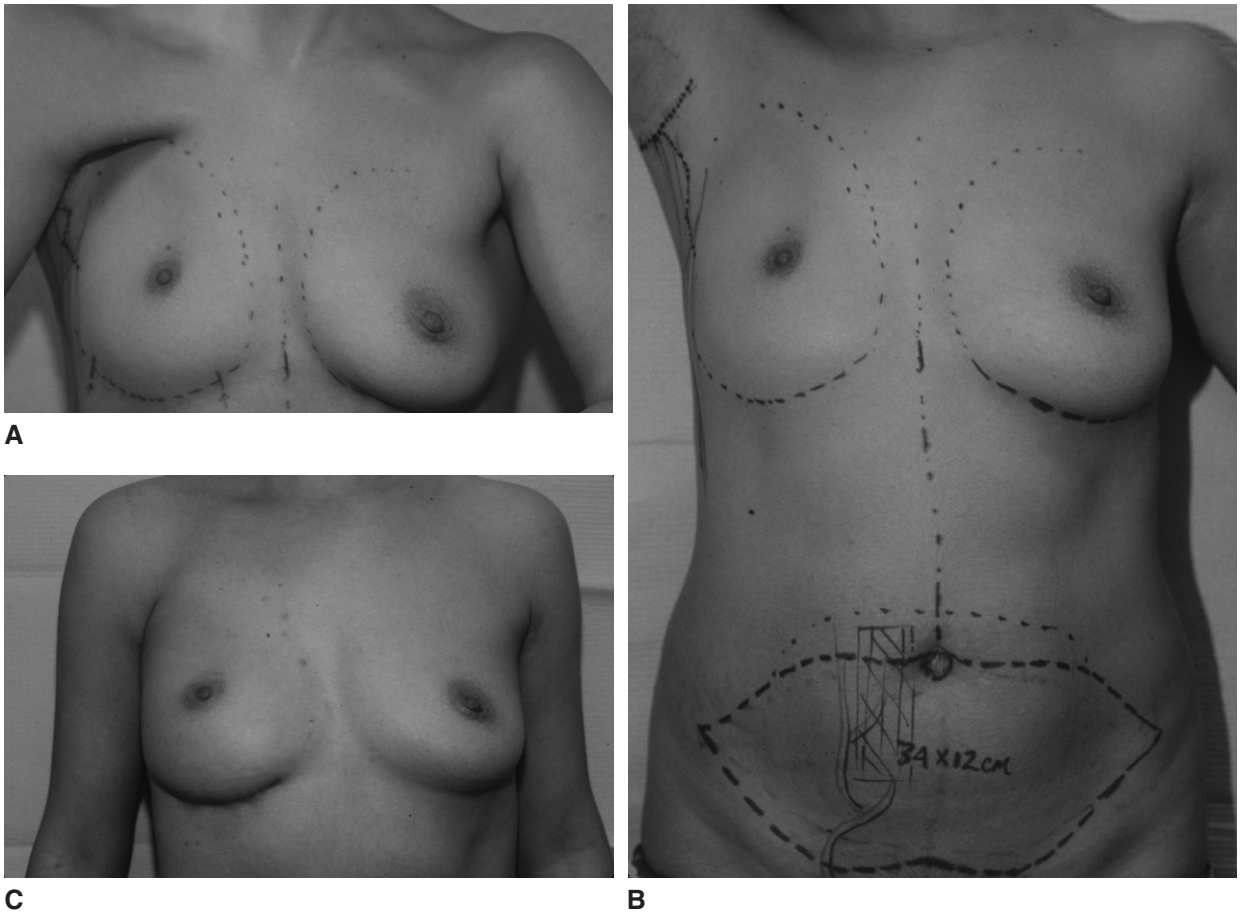


Figure 1 • A, Patient with Poland's syndrome of the right side of the chest. **B,** Outline of the planned free microvascular transverse rectus abdominis myocutaneous flap reconstruction. **C,** Postoperative result.

partial release of the inferior border of the pectoralis major is performed. Limited elevation of serratus muscle fascia along the anterior axillary line limits potential lateral migration of the implant. Textured expanders allow ingrowth of collagen from the periprosthetic capsule, which assists in fixing an implant's position.

Weekly tissue expansion is performed to fill the expander 20% to 30% greater than the predicted implant volume. Second-stage tissue expander implant exchange is performed approximately 3 to 6 months later after complete expansion. The final reconstruction should be performed at least 1 month after completion of chemotherapy. The skin flaps are elevated and an incision is made through the pectoralis muscle in the direction of the muscle fibers for removal of the tissue expander and access to the inframammary fold. Definition of the inframammary fold with 2–0 silk suture can assist in the final outcome. The position of the implant pocket at this time plays an important role in the final result. Either a saline or silicone implant may be used as the permanent implant. Most patients choose saline implants, which are effective for mound projection; however, if a large volume (>500 mL) is required, a silicone implant is preferred to avoid superior pole rippling. Occasionally, nipple reconstruction can be performed at this time, but it is most often reserved for a third procedure.

Autologous Reconstruction

LATISSIMUS DORSI FLAP. Reconstruction with a latissimus dorsi flap usually requires the inclusion of a breast implant to achieve adequate volume and projection (Fig. 2). However, a "fleur-de-lis" flap can be used to avoid the use of an implant. A tradeoff is a more extensive donor scar on the back. It is a reliable technique with a low incidence of flap failure. Indications for a latissimus dorsi flap include patients with loose back skin and generous fat, large ptotic breasts that cannot be easily created by an implant alone, and irradiated breast cancer patients seeking reconstruction when skin quality is poor and the blood supply is unreliable. This subset of patients is not usually suitable for a TRAM flap or

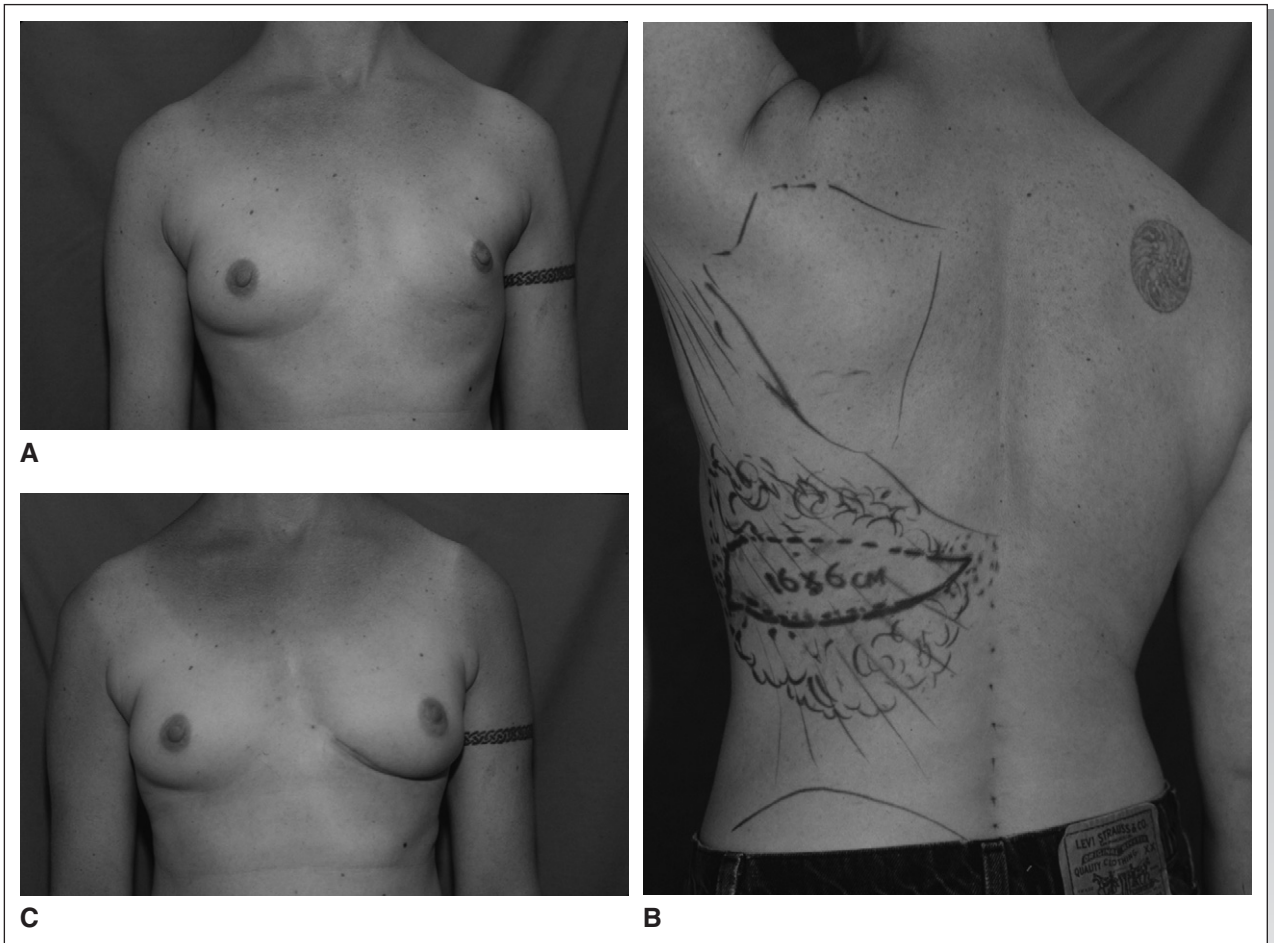


Figure 2 • A, Patient with congenital left breast hypoplasia and failed implant reconstruction. **B,** Outline of the planned pedicled latissimus dorsi flap reconstruction. **C,** Postoperative result.

more extensive surgery. Skin islands can be designed in a variety of positions over the latissimus dorsi muscle. The entire muscle is usually harvested and tunneled across the axilla. If soft tissue bulk is desired, the thoracodorsal nerve should be preserved. Conversely, the thoracodorsal nerve can be divided after anterior transposition of the flap in patients in whom muscle atrophy is not a concern. The muscle is sutured to the pectoralis major, and the implant is placed underneath both muscles. A low, oblique skin island can easily be inset in the inferior lateral quadrant of the reconstructed breast to allow for ptosis and to define the inframammary fold. The latissimus muscle insertion on the humerus can be transected and anteriorly transposed to decrease a lateral contour ("bulging") deformity.

TRAM FLAP. The TRAM flap is the workhorse of autologous breast reconstruction. It can be used as a pedicled flap, free flap, or perforator flap. Ideal patients are moderately built with generous abdominal fat and a lax abdominal wall from previous pregnancy. Patients must also be willing to accept the obligate abdominal scar and minor muscle loss. The main advantages of a TRAM flap are the permanent, naturally soft contour that can be achieved without the need for an implant, as well as the removal of excess abdominal tissue and tightening of the abdomen. The TRAM flap receives its main blood supply from perforators of the distal deep inferior epigastric artery. The perforators fill from the deep superior epigastric artery through "choke" vessels within the muscle above the umbilicus. For this reason, it is important to design the skin island so that it is centered just below the umbilicus to recruit the maximum number of "choke" vessels and thereby ensure the necessary reverse blood flow for the TRAM flap to be pedicled superiorly. Four vascular zones with different degrees of vascularity are present in the skin paddle. *Zone I* lies directly over the ipsilateral rectus muscle being raised and is well perfused, whereas *zone IV* is the tissue farthest from the pedicle and is the least reliable.

PEDICLED TRAM FLAP. Flap dissection is carried out in a lateral-to-medial direction until the lateral

row of perforators is localized. The flap is elevated from the opposite side across the midline until the medial perforators are visualized. The perforators should be maximally preserved before incising the fascia, and the muscle is divided inferiorly and mobilized out of the rectus sheath. Care is taken to divide the eighth intercostal nerve at the costal margin to reduce postoperative muscle fullness in the epigastrium. The flap is brought through a subcutaneous tunnel that connects the abdomen to the mastectomy site and it is rotated 90 to 180 degrees as needed.

Microsurgical Reconstruction

Advances in the field of microsurgery have allowed plastic surgeons to broaden their armamentarium for autologous breast reconstruction and have made microvascular free flaps the primary choice for a growing number of breast cancer patients. The theoretical advantages of a free flap are an enhanced blood supply to the autogenous breast tissue, greater flexibility in shaping the tissue, and aesthetically acceptable donor sites in the case of TRAM or gluteal flaps.

FREE TRAM FLAP. The free TRAM flap operation allows the transfer of a large volume of vascularized skin and subcutaneous tissue to the mastectomy site (Figs. 3 and 4). Modifications of the flap-harvesting technique, including muscle-sparing and perforator flaps, are designed to minimize abdominal wall hernias and preserve rectus muscle strength and function. The flap pedicle is based on the deep inferior epigastric vessels, and the preferred recipient vessels are the thoracodorsal or internal mammary artery and vein. If the thoracodorsal vessels are chosen, injury to the long thoracic nerve should be avoided, and the serratus branch of the thoracodorsal artery should be preserved for a possible latissimus flap in the future. Contralateral versus ipsilateral harvest of the flap depends on the goals of shaping the flap to restore the skin and volume requirements of the mastectomy defect.

Safety is always a primary concern when choosing a free TRAM flap procedure because of the length of the procedure and potential microvascular complications. Severe atherosclerosis, congestive heart failure, and pulmonary disease can increase risk and impede oxygen delivery to the flap. A history of deep vein thrombosis of the lower extremities and pulmonary emboli may also be a contraindication to a free TRAM flap.

A two-team microsurgical approach is preferred. Identification plus preparation of recipient vessels is helpful before harvesting the free flap. Preoperatively, the flap pedicle (left versus right) should be determined for breast reconstruction. In general, if the patient has a narrow breast base with signifi-

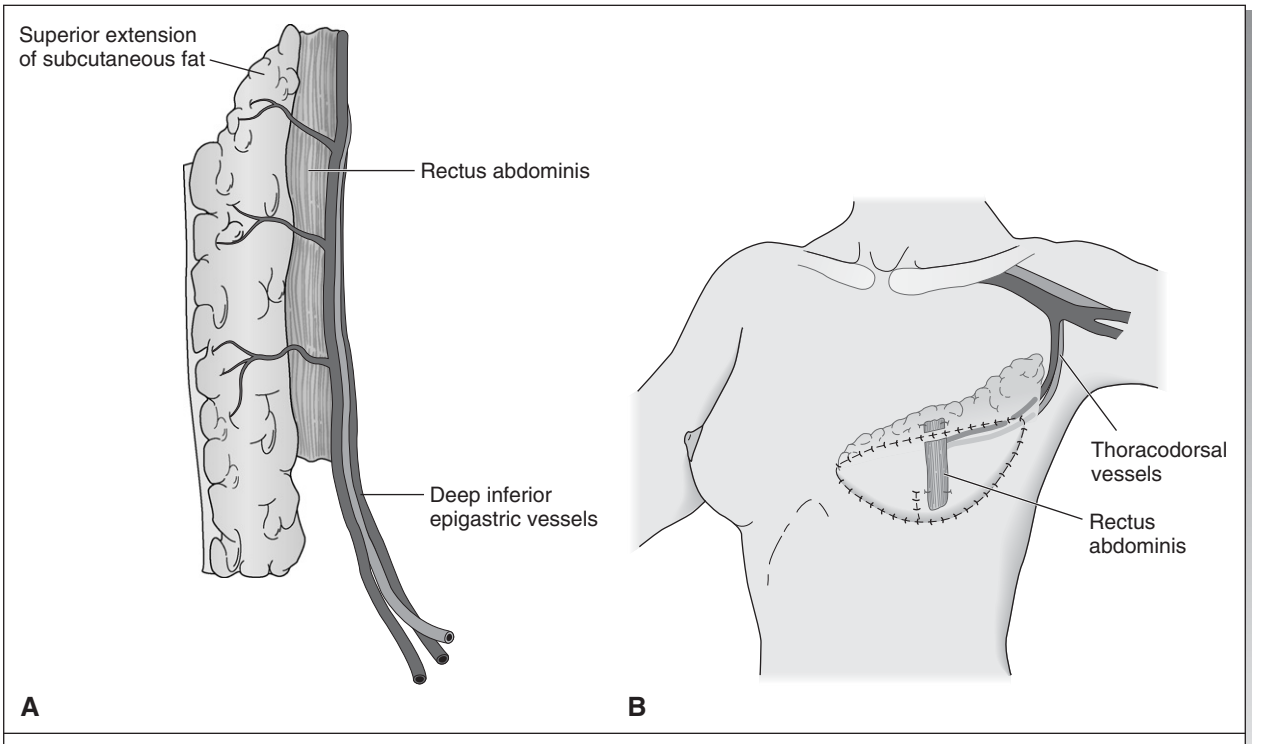
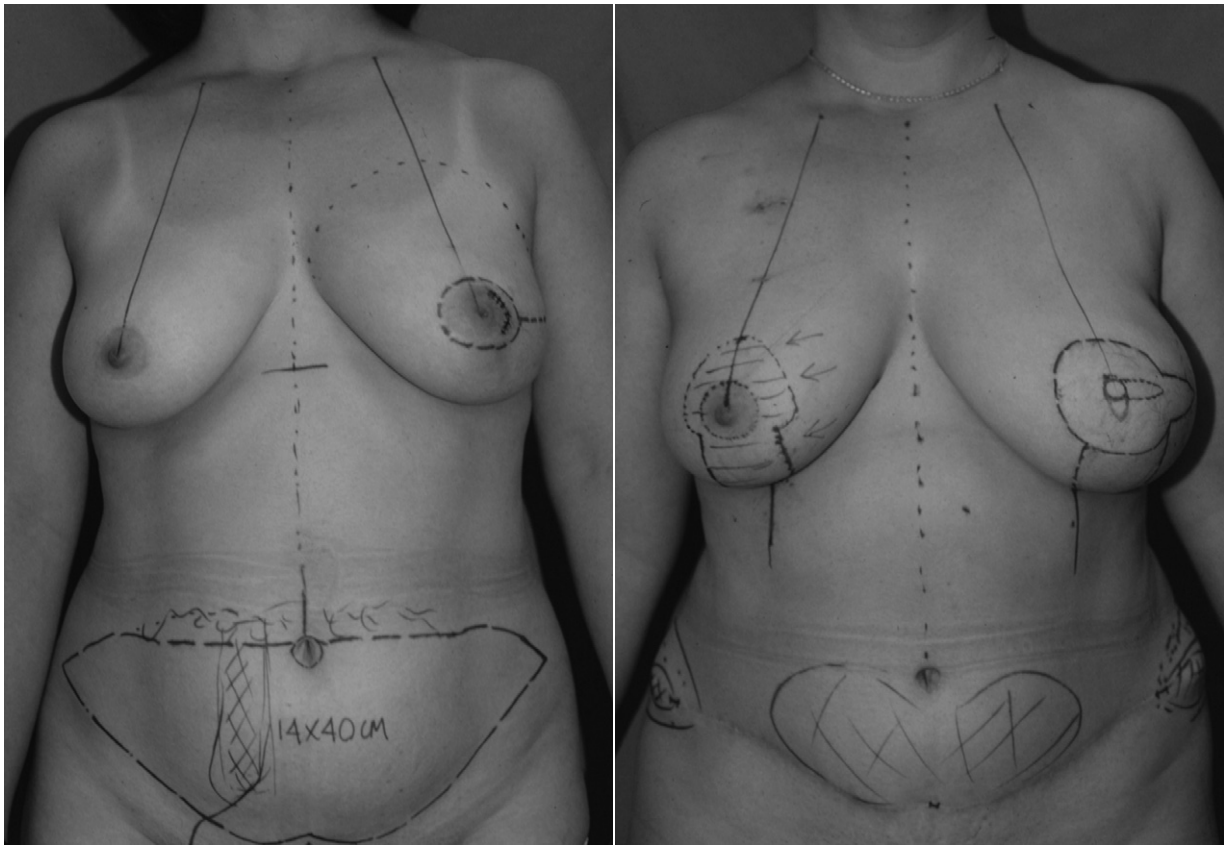


Figure 3 • A, Technical details of free transverse rectus abdominis myocutaneous (TRAM) flap dissection. Note the superior extension of subcutaneous fat and the limited harvest of fascia and muscle. **B,** The long vascular pedicle of the free TRAM flap allows ease of anastomosis with the recipient axillary vessels and versatility in molding the breast mound.



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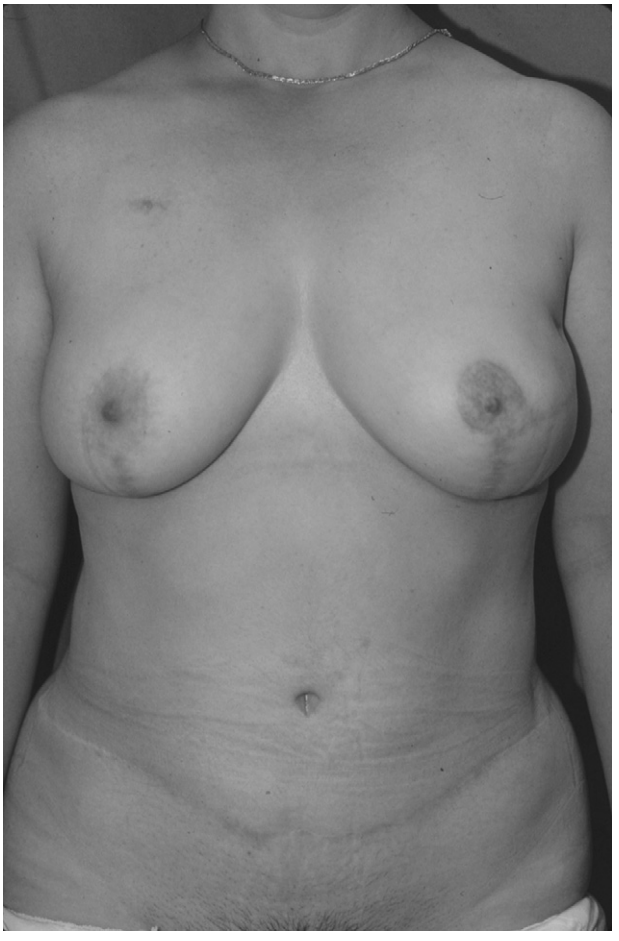

C

Figure 4 • A, Patient with cancer of the left breast who is to undergo left skin-sparing mastectomy with immediate reconstruction by a free transverse rectus abdominis myocutaneous flap. **B,** After breast reconstruction, left nipple-areola reconstruction with right vertical mammaplasty and abdominal liposuction/scar revision is to be performed. **C**, Final result.

cant ptosis, an ipsilateral flap yields the best result. A wide breast is better reconstructed with a contralateral TRAM flap. Similar to harvest of a pedicled TRAM flap, the medial and lateral rows of perforators are identified. The anterior abdominal wall fascia is incised with preservation of the medial and lateral rectus muscles. The flap is harvested when the recipient vessels are adequately prepared and the pedicle divided.

PERFORATOR FLAPS. Dissection of the musculocutaneous perforator vessels that provide the blood supply to a myocutaneous flap allows harvesting of the flap without the need to incorporate any muscle. The result is a perforator flap consisting of only skin and fat. Although this technique is tedious and technically demanding, many surgeons believe that the advantages of added strength and better contour of the donor site are considerable.

The *deep inferior epigastric perforator (DIEP) flap* implies the dissection of one to three perforator vessels through the rectus abdominis muscle, down to their origin from the deep inferior epigastric artery. The DIEP flap maintains the advantages of the free TRAM flap while reducing the lower abdominal wall contour deformities sometimes associated with the TRAM donor site.

The *superior gluteal artery perforator flap* is used as an alternative to the DIEP flap. By following the perforator through the gluteus maximus muscle, the length of the pedicle (6 to 7 cm) of the superior gluteal flap is considerably increased, thereby allowing for more insetting options and facilitating microvascular anastomosis. The main advantages of perforator flaps are reduced postoperative pain and a faster recovery period.

OTHER FREE FLAPS. Other free flaps may be used as alternatives to the free TRAM or DIEP flap when these flaps are not available (e.g., in very thin patients, postabdominoplasty patients, or patients with scars within the planned flap).

The *superficial inferior epigastric artery (SIEA) flap* is an option for breast reconstruction when the vessels are of sufficient caliber (less then 15% of cases). It is ideal in the sense that it does not produce morbidity of the abdominal wall and allows a concealed scar similar to a free TRAM flap. The major drawbacks of a free SIEA flap are the short length and small vessel diameter. The SIEA is absent approximately 40% of the time, and the vessels may be unusable (<1.5 mm in diameter) 20% of the time. Many surgeons routinely dissect these vessels in all TRAM cases and, if suitable, proceed with an SIEA flap.

The *Rubens flap (periiliac flap)* is based on the deep circumflex iliac artery. Although the vascular anatomy is highly reliable, the main disadvantage of this flap is violation of the abdominal wall with an increased risk for flank hernias if adequate attention is not directed to abdominal wall closure.

The *superior gluteal flap* is a fasciocutaneous flap based on the superior gluteal artery (Figs. 5 and 6). The ideal patient is an active, young middle-aged patient desiring permanent reconstruction with excellent projection. She usually refuses implants and the use of abdominal muscles, or other reconstructive efforts have failed.

The flap dissection is technically difficult, and the flap has a short pedicle. The tissue may also be difficult to mold into a natural-appearing ptotic breast.

The *anterolateral thigh flap* is a fasciocutaneous flap and another alternative for breast reconstruction in patients with abundant lateral thigh tissue. It is based on the lateral femoral circumflex artery and is more amenable to molding than the gluteal flap. However, it should be emphasized that there is potentially a highly visible donor site scar. Lateral femoral cutaneous neurapraxia and seroma formation have also been described.

Nipple-Areola Reconstruction

AREOLA. The optimal method for areola reconstruction is intradermal pigment tattooing because this technique yields satisfactory long-term results without the necessity for additional donor sites.

NIPPLE GRAFTING. Also known as nipple sharing, this technique yields a natural-appearing nipple and consists of grafting part of the normal nipple into the reconstructed breast mound. Depending on the length of the donor nipple, the graft may be taken as a wedge excision or a cap amputation.

SKATE FLAP. This technique produces a projectile nipple. A local flap is raised and wrapped on itself. It leaves a raw donor site that usually requires skin grafting from the groin or the axillary portion of the mastectomy scar.

C-V FLAP. This technique uses two long triangular V flaps extending horizontally and one semicircular C flap originating from the most inferior ends of the V flaps. The triangular flaps are wrapped around the C flap, which acts as a core. It also allows direct closure of the donor sites. Overcorrection in length is necessary to allow for subsequent settling and loss of height.

Postoperative Care

Postoperative care of the patient is individualized and based on the complexity of the procedure. Drains are placed for 3 to 5 days after mastectomy to decrease the likelihood of hematoma and seroma formation. Patients who have been treated by free tissue transfer undergo standard postoperative free flap protocol monitoring. Free flap loss can generally be avoided by using meticulous microsurgical

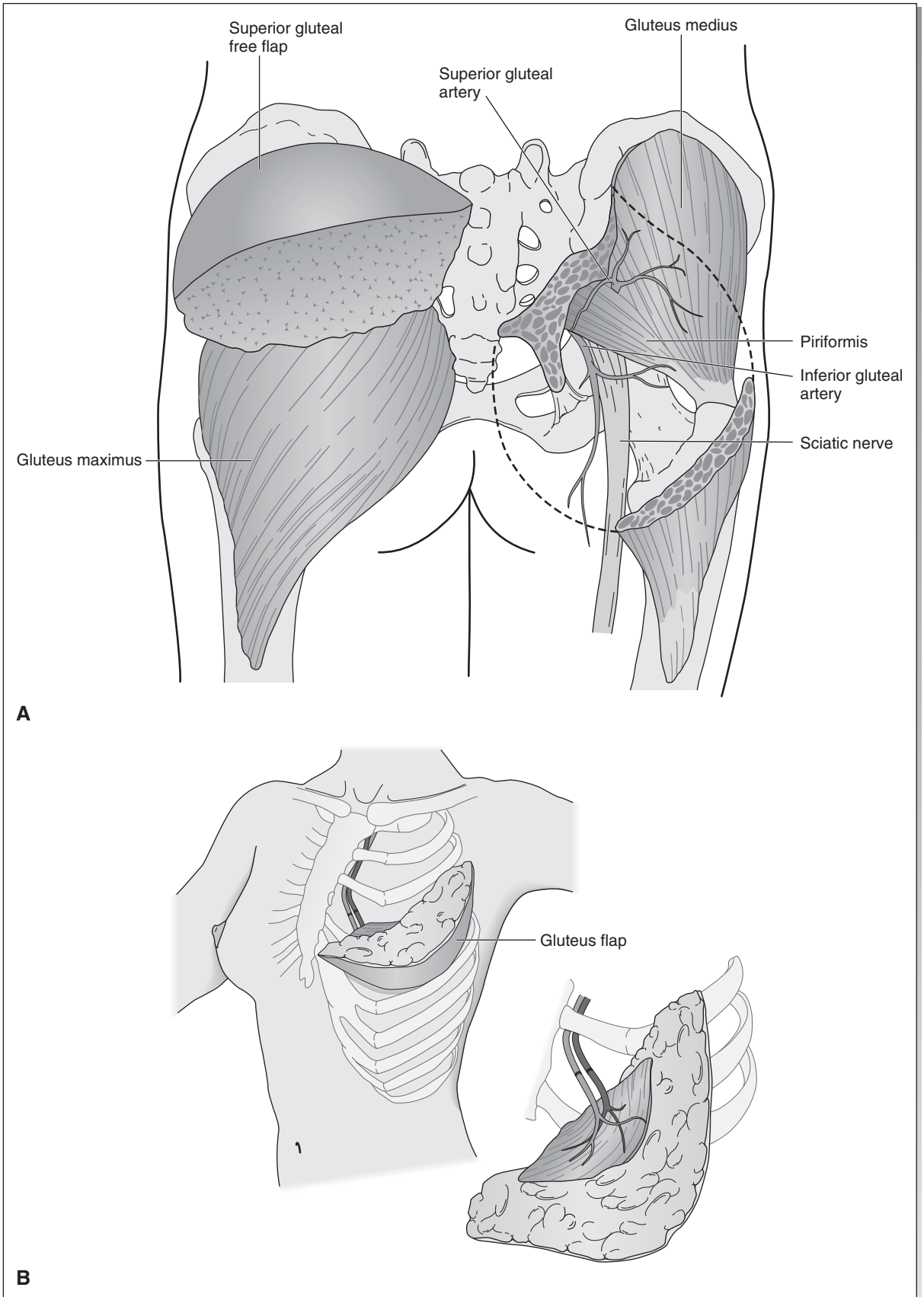

Figure 5 • A, The superior gluteal free flap is supplied by the superior gluteal vessels and supported by a portion of the superior gluteus muscle. Design of the flap includes dissection of the skin island and a wedge of subcutaneous fat sufficient to match the contralateral breast as closely as possible. Careful design minimizes the donor defect. **B,** Insetting of the gluteal free flap is more easily done when the internal mammary vessels are used as recipient vessels.

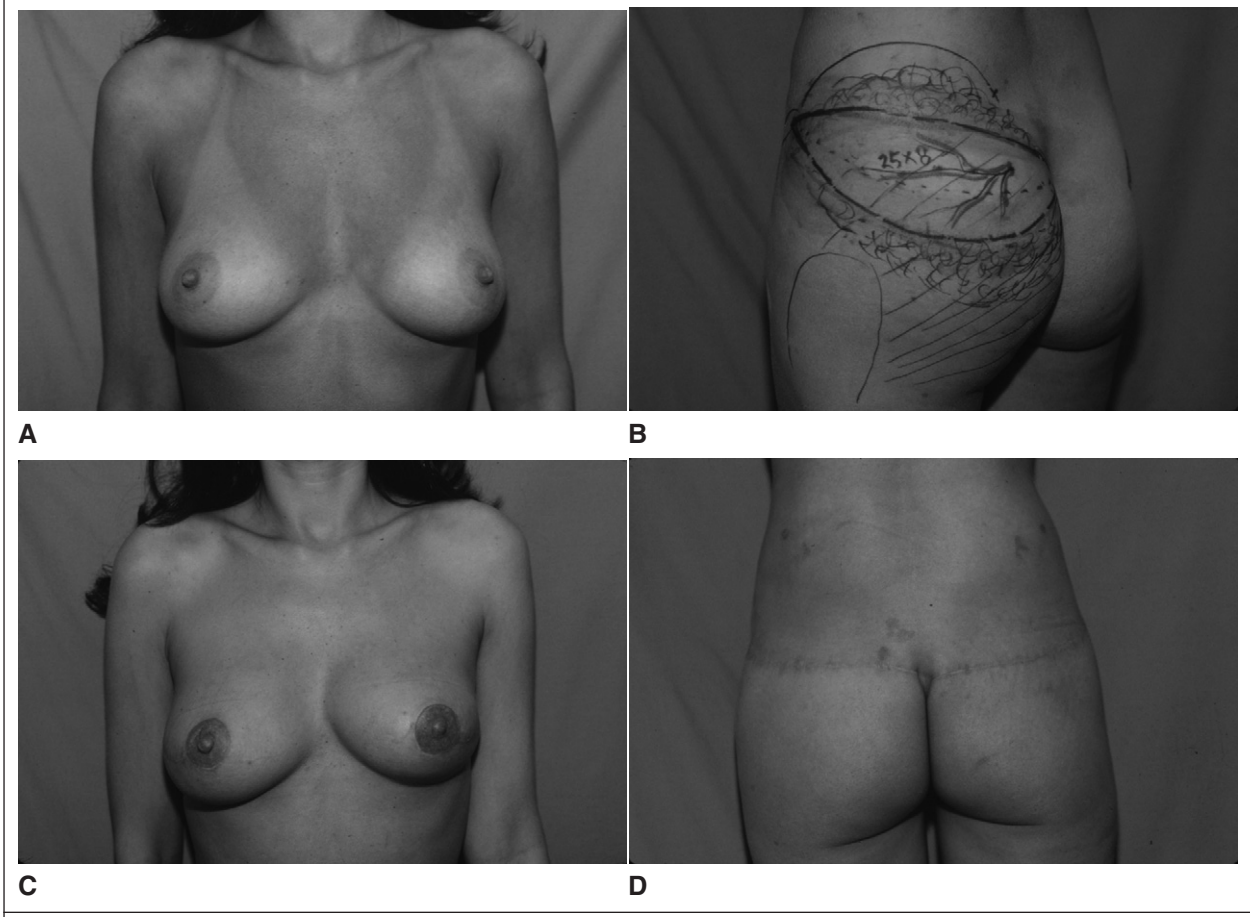

Figure 6 • A, Patient for prophylactic bilateral mastectomy (breast cancer positive). **B,** Outline of the superior gluteal artery flap. **C,** Final result. **D,** Donor site.

techniques, properly selecting patients for a specific type of reconstruction, and implementing attentive postoperative monitoring and care.

Complications

Expander Implant Reconstruction

Early complications of tissue expansion reconstruction are a function of the wound, the device, the process of expansion, and the quality of tissue cover at the site of the mastectomy. Wound complications (hematoma, seroma, and infection) may occur. Skin necrosis and wound breakdown occur more frequently after mastectomy as a result of pressure applied to the skin flaps. Complications from the device are usually due to expander failure, which may occur secondary to needle puncture at the time of suturing or periodic refilling. Mastectomy skin flaps that are thin may have inadequate blood supply along the wound edges, thus requiring additional débridement before wound closure.

Late complications of implant reconstruction include capsular contracture, superior pole rippling, and implant rupture. Capsular contracture rates have been reported to be as high as 35%, and capsulectomy/capsulotomy and implant exchange or removal may be required in the event of contracture. Superior pole rippling is more commonly seen in underfilled saline implants. Silicone implants have the advantage of less noticeable rippling and more natural shape and feel. The rate of rupture of silicone gel implants approximates 50% (follow-up of more than 10 years after insertion). Saline devices have a 1% per year cumulative rupture rate after 5 years.

Autologous Reconstruction

TRAM Flap. Abdominal bulging, fat necrosis, partial flap loss, and total flap loss are potential complications. The complication rate has been reduced by using improved flap-harvesting techniques for free TRAM reconstructions. Partial flap loss occurs more commonly than total flap loss. Skin envelope complications may also occur. In the setting of immediate reconstruction, some surgeons

advocate the use of intravenous fluorescein and a Wood lamp to avoid leaving a compromised mastectomy flap behind and thereby reduce the chance of native breast flap necrosis. Abdominal hernias occur at varying rates, depending on the method of closure and patient selection. Wound complications such as infection and dehiscence occur at an incidence similar to that of other surgical procedures, but with a higher frequency in smokers and diabetics. Size mismatch, an unsatisfactory aesthetic result, and the need for revision surgery may also be considered complications.

Latissimus Dorsi Flap. Major complications are rare. However, minor complications such as seroma formation at the donor site are common and occur regularly in obese patients. Prolonged placement of drains is usually necessary. Some surgeons advocate quilting sutures to plicate and decrease the space.

Pearls and Pitfalls

- Expander implant reconstruction may result in a high-riding implant with an inadequately defined inframammary fold. Lowering the inframammary fold may be necessary along with restriction of superior pocket dissection. A compression band worn above the breast 4 to 6 weeks after implant placement may also be helpful.
- When a latissimus implant reconstruction is performed, it is important to avoid placing the skin island high on the breast. The skin paddle should rest in the inferior pole of the breast to allow an increased amount of soft tissue volume in the inferior aspect of the new breast mound.
- In women with narrow, ptotic breasts, vertical or diagonal orientation of the flap might yield a more symmetrical reconstruction, whereas in more overweight patients with wider breasts, horizontal placement might be more effective. Proper use of flap-folding techniques can also improve the final aesthetic result.
- Folding the TRAM flap on itself allows for more projection of the lower pole, as well as improved definition of the inframammary fold in delayed reconstructions.
- At the time of initial TRAM flap dissection, beveling of the superior flap helps to reduce excessive upper pole fullness and provides a more natural contour.
- With TRAM flap reconstruction, maximal preservation of the anterior rectus sheath results in fewer abdominal wall hernias or bulges. Lateral abdominal wall myofascial release should be used in patients with excessive tension at the midline closure.

SUGGESTED READINGS

Bostwick J: Plastic and Reconstructive Breast Surgery, 2nd ed. St Louis, Quality Medical Publishing, 1999.

Elliot LF: Options for donor sites for autologous tissue breast reconstruction. Clin Plast Surg 21:177, 1994.

Kroll SS: Breast Reconstruction with Autologous Tissue: Art and Artistry. New York, Springer-Verlag, 2000.

Spear SL: Surgery of the Breast: Principles and Art. Philadelphia, Lippincott Williams & Wilkins, 1998.

Chest Wall Reconstruction

ADNAN M. DIN
GREGORY R. D. EVANS

Since its beginnings, thoracic surgery has been associated with significant mortality. Patients would often succumb to respiratory failure, infection, or their underlying disease process. A significant factor in outcome was the thoracic wound and the resulting instability of the chest. Furthermore, resection of chest wall tumors was not an option because reconstructive techniques were limited. Over the past 30 years, the development of ventilators, antibiotics, and synthetic mesh, along with a detailed understanding of muscle flap anatomy, has enabled surgeons to reconstruct chest wall defects. This chapter reviews the etiology of chest wall defects and the reconstructive techniques currently available for chest wall reconstruction.

Etiopathogenesis/Causative Factors

Chest wall defects can result from infection, resection of tumors, radiation injury, trauma, or congenital defects. The most common cause of chest wall defects is an infected or unstable median sternotomy wound (Fig. 1). Median sternotomy wound infections occur in up to 5% of cardiac surgery patients. These infections pose a major risk that significantly increases morbidity and mortality. Risk factors include diabetes, obesity, distant infections, emergency surgery, prolonged cardiopulmonary bypass, and tracheotomy. In addition, the use of both internal mammary arteries devascularizes the sternum and increases the risk. The most common organisms include *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Pseudomonas*, *Serratia marcescens*, and enterococci. Historically, these wound were left to granulate and heal by secondary intention; however, such treatment is associated with significant cost and mortality.

Chest wall malignancies can be benign, malignant, metastatic, or the result of direct extension of tumor. Benign primary chest wall neoplasms include osteochondroma and fibrous dysplasia. They usually require local excision alone. Chondrosarcoma, the most common primary malignant chest wall tumor, requires wide resection. Metastases and direct extension are most commonly due to advanced breast or lung cancer.

Radiation therapy has become a mainstay of treatment of many types of malignancy. However, radiation damage can lead to microvascular fibrosis with thrombosis, delay in wound healing, wound breakdown, and infection. In addition, radiation-induced changes most often worsen with time. Areas with significant radiation changes may be painful, unstable, and unsightly and thus require wide resection and reconstruction.

Congenital chest wall defects are less common, the most frequent being Poland's syndrome. In addition to the breast deformities (mammary aplasia/hypoplasia) in this condition, partial agenesis of the ribs and sternum may also be present and may require treatment.

Pathologic Anatomy

The ribs, sternum, and spine are the structural support of the chest. They form a cone with the apex directed superiorly. They are rigid to protect the underlying organs while simultaneously remaining flexible to facilitate respiration. This framework allows for muscular attachment to the clavicle, humerus, and scapula and supports the upper extremity.

The muscles of the chest wall are divided into respiratory muscles, arm muscles, muscles joining the chest and abdomen, muscles joining the chest and head, and muscles supporting the spine. The respiratory muscles are the diaphragm and the intercostals. They form the limits of the chest and divide the chest from the abdominal cavity and the more superficial muscles. The intercostals link the ribs and add dynamic stability in addition to

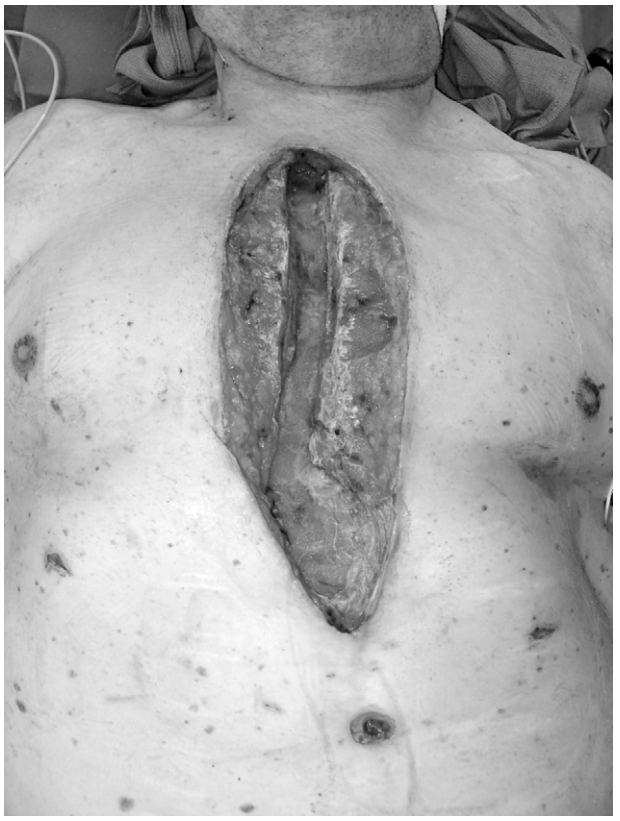

Figure 1 • Patient with sternal infection after coronary artery bypass graft surgery. The open wound requires flap closure and adequate débridement.

assisting with respiration. The important arm muscles include the pectoralis, latissimus, serratus, trapezius, and shoulder girdle muscles. They provide shoulder stabilization and arm movement. The actions of these muscles are redundant, and therefore muscles can be sacrificed and used for reconstruction. The important muscles joining the chest and abdomen are the rectus abdominis and the obliques. The rectus is a workhorse in chest wall reconstruction because it is easily harvested and provides excellent reconstructive tissue.

The blood supply of the chest is abundant. It can be divided into a posterior, an anterior, and a lateral supply. The posterior supply is a segmental supply directly from the aorta and is not generally used for chest wall reconstruction. It is rarely interrupted and it reliably supplies the ribs and intercostals. The anterior supply is derived from the internal thoracic or internal mammary vessels. It joins with the posterior supply through the intercostals, but unlike the posterior supply, it is often used in reconstructive procedures because this vessel supplies the rectus abdominis muscle and the medial row of perforators of the pectoralis muscle. The two internal mammary vessels, however, also supply the sternum, and when both are sacrificed in cardiac revascularization, the sternum is left relatively avascular. The lateral blood supply consists of the branches of the subclavian and axillary arteries. The important vessels include the thoracoacromial artery (supplying the pectoralis), the thoracodorsal artery (supplying the latissimus and the serratus), and the lateral thoracic artery.

Diagnostic Studies

Basic preoperative assessment of patients with chest wall defects includes chest radiographs, complete blood counts, and arterial blood gas values. Nutritional evaluation by determination of serum albumin and transferrin levels and anthropometric measurements are considered in selected patients. In addition, pulmonary function tests document the patient's ability to withstand any degree of chest wall flaccidity necessitating reconstruction. Computed tomography is useful for assessment of tumors or infections. Cardiac evaluation is limited to patients with risk factors; these patients are screened for underlying coronary disease.

Reconstructive Goals

Chest wall resection does not routinely require complex reconstruction because of the abundant local muscular tissue and redundant bony support. Superficial soft tissue defects that do not involve the ribs and sternum can often be reconstructed with local soft tissue flaps. Despite lack of consensus, reconstruction of bony chest wall defects is only necessary if three or more ribs are resected or if the defect is larger than 5 cm. These large defects can result in paradoxical movement of the chest with inspiration and can thus lead to pulmonary insufficiency. In patients undergoing radiation therapy, up to five ribs may be resected before reconstruction is considered because of the increased fibrosis and resulting stiffness of the chest wall in these patients. Overall, the primary goals of chest wall reconstruction are as follows:

1. Débridement of infected or necrotic tissue
2. Coverage of vital structures
3. Restoration of function
4. Stabilization of the bony skeleton
5. Obliteration of dead space
6. Aesthetic considerations

Surgical Treatment

Because of the availability of abundant local tissue, most chest wall defects are amenable to reconstruc-

tion with local tissue. The latissimus dorsi and pectoralis muscles are most commonly used for most chest wall reconstructions. In addition, rectus muscle flaps, with or without a cutaneous component, are useful in cases in which the pectoralis or latissimus is not available. Overall, a variety of options can be used, and planning must consider areas of previous irradiation or surgery. Microvascular free flaps for reconstruction are occasionally used when regional flaps are unavailable or have failed.

Reconstruction often requires establishing rigidity in the chest wall. Ribs, iliac crest, and fibula bone grafts can be used, but they are associated with donor site morbidity. Alternatively, Gore-Tex, Prolene, or Marlex mesh can be useful for establishing stability of the chest wall. The patch is tailored to the chest wall defect and sutured to the edge of the defect in taut fashion. A composite Marlex mesh and methylmethacrylate sandwich can provide additional rigidity in patients with significant defects. To construct this composite, a Marlex mesh layer is sutured to the edges of the chest wall defect. Methylmethacrylate is applied to the Marlex and covered with an additional piece of Marlex mesh to create a "sandwich." The composite graft is irrigated with chilled saline because of the exothermic reaction of methylmethacrylate. It is important to use caution with prosthetic materials in areas of previous radiation therapy because these areas are prone to infection and exposure. Well-vascularized tissue must cover the prosthetic reconstructions to limit potential complications. Moreover, in cases of radiation injury, autogenous reconstruction of the structural stability of the chest wall with bone or cartilage grafts is preferred.

Pectoralis Major

The pectoralis muscle flap is a versatile workhorse for anterior chest wall defects. It is especially useful for median sternotomy wound infections. These infections require débridement of devitalized or infected tissues, followed by coverage of the defect with viable tissue. This is best achieved by using the pectoralis muscle flap. It is important to débride adequately the costal cartilage because exposed cartilage has little defense against recurrent infection.

Advancement of the muscle is based on the thoracoacromial pedicle. The subcutaneous tissue is undermined over the anterior surface of the pectoralis fascia, and the muscle is dissected medially off the chest wall. The thoracoacromial pedicle enters the muscle laterally on its undersurface. While preserving this pedicle, the muscle is divided from its humeral and clavicular attachments to allow rotation into the wound. This disinsertion from the humerus can be facilitated by making a separate incision over the deltopectoral groove. After the muscle is rotated and sutured in place, closed suction drains are inserted and the skin is closed primarily. Mobilization of one pectoralis muscle is usually sufficient, and the nondominant muscle is preferred to minimize donor site morbidity. In some instances, both pectoralis muscles must be mobilized to cover larger defects.

Alternatively, the muscle can be turned over and based on the perforators from the internal mammary vessels. This technique is useful for a lower defect because turnover maximizes reach to the lower portion of the sternal defect. With this technique, the dissection is performed over the anterior surface of the pectoralis fascia. Dissection should proceed to the humeral limits of the pectoralis. It is disinserted from its humeral and clavicular attachments. The thoracoacromial artery is identified and ligated. It again can be helpful to make a separate incision over the deltopectoral groove for the disinsertion. The undersurface of the muscle is freed from the chest wall to the medial row of perforators. It can be split along the fibers to facilitate coverage of lower defects. Over time, scarring and fibrosis of the pectoralis muscle often lead to adequate chest wall stability and protection. The shortcoming of the pectoralis flap is its inability to reach the lowest portion of the sternum and xiphoid. For low defects, alternative options may be required.

Latissimus Dorsi

Soft tissue closure of the anterior, posterior, or lateral chest wall is usually achieved with the versatile latissimus dorsi flap. The latissimus muscle is an excellent choice for chest wall reconstruction because of its bulk, wide arc of rotation, and reliable vascularity. Its wide arc of rotation can be based on either the thoracodorsal artery or the 9th to 11th intercostal arteries; however, the latter is less reliable. An optional and reliable skin island may also be designed up to 10 cm in width and can be oriented in a horizontal, oblique, or vertical direction.

Posterior chest wall defects are less frequent. However, defects of the lower two thirds of the chest are also closed preferentially with the latissimus dorsi flap. The latissimus muscle is divided from its humeral attachment and can be mobilized based on blood supply from the lumbar and intercostal arteries to cover the lower two thirds of the back. An alternative and perhaps more reliable option is to place vein grafts into the thoracodorsal vessels to allow further extension of the muscle to the lower part of the thorax.

Rectus Abdominis

The rectus abdominis flap is useful for lower anterior chest wall reconstruction. The flap can even be used after internal mammary artery harvest based on collateral circulation through the intercostal vessels, although in these cases the skin island is not as reliable. In general, skin paddles may be oriented in a variety of patterns, depending on the

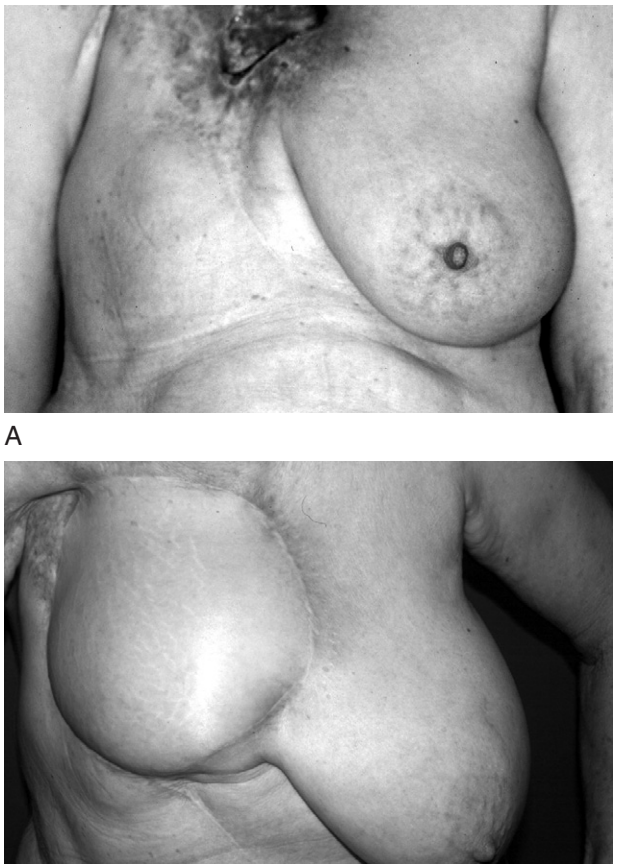

Figure 2 • A, Fifty-five-year-old woman with recurrent breast cancer after right mastectomy. An ulcerative lesion is identified on the anterior chest wall. **B,** The surgical resection site was covered with the rectus abdominis muscle and a skin paddle (transverse rectus abdominis myocutaneous flap). Changes consistent with previous radiation therapy can be noted on the chest wall skin.

defect. Extension of the skin pattern beyond the border of the muscle allows for coverage of the midthorax. When possible, the paraumbilical perforators should be incorporated to increase the vascularity of the skin island (Fig. 2).

Omental Flap

The omental flap is occasionally used to close chest wall defects. Its pliability permits adequate soft tissue fill in both external thoracic cavity and internal pleural defects. Harvest of the flap does carry a risk of introducing infection into the abdominal cavity, bowel perforation, and possible herniation. A history of previous intraabdominal infection, surgery, or dense adhesions is a contraindication to the use of this flap.

The greater omentum is mobilized based on either the left or right gastroepiploic artery by blunt dissection from the greater curvature of the stomach and the transverse colon. A pedicle based on the right gastroepiploic vessels is usually preferable because of its increased vessel diameter and length, which can be extended from 9 to 12 cm. The short branches extending from the gastroepiploic arch to the greater curvature of the stomach are ligated and divided. The flap can be brought out through a diaphragmatic defect for lesions within the thoracic cavity or exteriorized through the anterior abdominal wall. Depending on the size and location of the associated defect, the gastroepiploic artery and vein can be isolated on either side and divided to optimize flap length. Care must be taken to avoid tension, which can damage omental tissue and vessels. Recipient vessels may be prepared in the event of free tissue transfer.

Trapezius

Defects in the posterior upper midline or cervical areas are preferentially closed with a trapezius muscle flap. The flap is based on the transverse cervical artery. The ascending branch runs parallel to the anterior border of the trapezius, and the descending branch passes beneath the trapezius muscle at the base of the neck. This distinct division of the transverse cervical artery can support the separate territories.

The lower portion of the muscle is elevated from the skin and detached from the scapula. A skin island may also be designed over the muscle. The muscle is generally used as either a horizontal shoulder fasciocutaneous flap or a vertical island myocutaneous flap. Care must be taken during dissection of the flap to prevent injury to the spinal accessory nerve.

Serratus

Located on the lateral aspect of the chest, this muscle is occasionally used for local soft tissue closure. Taking its origin from the medial border of the scapula, the muscle attaches to the lateral external borders of ribs 1 to 8, with its innervation and blood supply derived from the long thoracic nerve and artery. Its use in chest wall reconstruction is limited by its location, size, and arc of rotation. Elevation is similar to harvest of the latissimus dorsi flap. In addition, the serratus and latissimus dorsi may be harvested as two separate flaps based on the same thoracodorsal pedicle.

Poland's Syndrome

Treatment of Poland's syndrome remains controversial. Some surgeons advocate early surgery to prevent psychological repercussions. In young girls, the operation is often delayed until full breast maturity. Others advocate insertion of a tissue expander in early adolescence with periodic expansion throughout puberty. Most reconstructions involve harvesting the ipsilateral latissimus to cover the anterior chest wall after skeletal stabilization with

Marlex or Prolene mesh. Occasionally, patients with marked rib deformity may require subperichondrial cartilage resection or sternal osteotomy and rotation, or both.

Postoperative Care

Mortality rates after chest wall reconstruction are less than 5%. Most patients have little to no functional impairment after flap harvest. Patients are encouraged to ambulate early to prevent any postoperative pulmonary complications. Most deaths in the first year after surgery are usually due to the underlying disease process.

Postoperative patients should be monitored closely to check flap viability. Constriction of the reconstructed flap through a subcutaneous tunnel may lead to flap failure from arterial insufficiency or venous outflow obstruction. In areas that have previously undergone radiation therapy, subcutaneous tunnels are avoided. Drains are used in all cases and are subsequently removed when total drainage has decreased to less than 30 mL per 24 hours. Patients are also instructed to abstain from strenuous activity or lifting for 6 weeks. Mesh fractures have been noted, but the overlying soft tissue flap often prevents complications.

Pearls and Pitfalls

- Wide resection of all tumors and areas of infection should be undertaken before reconstruction.
- Marlex, Prolene, or methylmethacrylate/Marlex composite patches are useful for stabilizing chest wall defects larger than 5 cm or defects resulting from removal of three or more ribs.
- Prostheses are best avoided in irradiated or infected tissue.
- A satisfactory blood supply and obliteration of dead space are central to success in reconstruction.

SUGGESTED READINGS

Cohen M: Reconstruction of the chest wall. In Cohen M (ed): Mastery of Plastic and Reconstructive Surgery. Boston, Little, Brown, 1994, pp 1248-1267.

Mathes SJ: Chest wall reconstruction. Clin Plast Surg 22:187-198, 1995.

Parham FW: Thoracic resection for tumors growing from the bony wall of the chest. Trans South Surg Gynecol Assoc 11:223, 1898.

Sabanathan S, Shah R, Mearns AJ, Richardson J: Chest wall resection and reconstruction. Br J Hosp Med 57:255-259, 1997.

Seyfer A, Graeber G, Wind G: Atlas of Chest Wall Reconstruction. Rockville, MD, Royal Tunbridge Wells, 1986.

Abdominal Wall Reconstruction

NOLAN S. KARP

Abdominal wall hernias and defects are a common clinical entity, and most are treated by a general surgeon. Occasionally, however, these defects are not amenable to simple closure, and more complex procedures are required. Understanding of the techniques of moving tissues enables the plastic surgeon to assist in these difficult cases. This chapter reviews the principles of abdominal wall reconstruction and highlights proven reconstructive techniques.

Etiopathogenesis

Abdominal wall defects can result from many clinical situations, including incisional hernias, traumatic wounds, tumor resection, and infection. Incisional hernias are the most common cause of abdominal wall defects. Most hernias are closed by a general surgeon. Wide defects and recurrent hernias present a significant challenge, and plastic surgeons are often consulted for assistance. Traumatic wounds of the abdomen may be the most difficult to treat because of the haphazard, acute, and frequently contaminated nature of the wounds.

Resection of abdominal wall tumors is a less common cause of abdominal defects, but it often results in defects of multiple layers. Sarcomas, including rhabdomyosarcoma and liposarcoma, are the most common primary malignancy, although metastatic tumors are also seen. Desmoid tumors are locally invasive, benign tumors that tend to recur and require wide resection for cure. Dermatofibrosarcoma protuberans is a dermal neoplasm that is also resected with wide margins.

Postsurgical infections of the abdominal wall are fairly common, and mild infections lead to an increased rate of incisional hernias. Primary necrotizing infections of the abdominal wall are rare, and a delay in diagnosis is common. This delay often results in devastating consequences. These cases require aggressive débridement to healthy tissue, diagnosis of the infecting organisms, and administration of intravenous antibiotics.

Goals of Reconstruction

Reconstruction of the abdominal wall depends on the nature of the tissue loss. The priority in abdominal wall reconstruction is wound coverage or closure to achieve protection of vital visceral organs, fascial support, muscular function, and aesthetic appearance. Attaining all of these goals is frequently not possible. The timing and priority of the reconstruction depend on the clinical situation and the individual patient.

Anatomy

The skin of the abdominal wall is generally quite lax. This laxity allows undermining to recruit excess skin and close most defects. Alternatively, the underlying abdominal wall (fascia and muscle) is a favorable bed for skin grafting. Varying amounts of fat are present in the abdominal wall, but the fatty layer is not usually a significant factor when abdominal wall reconstruction is required, although it may become a factor for aesthetic defects.

The fascia of the anterior of the abdomen is the primary support structure and is complex. The most superficial fascia is the superficial fascial system (SFS). First described by Lockwood, the SFS is composed of thin fascial layers between the abdominal wall fat units. The most discrete layer of the SFS is Scarpa's fascia. The SFS provides substantial support to the abdominal wall and it serves as a

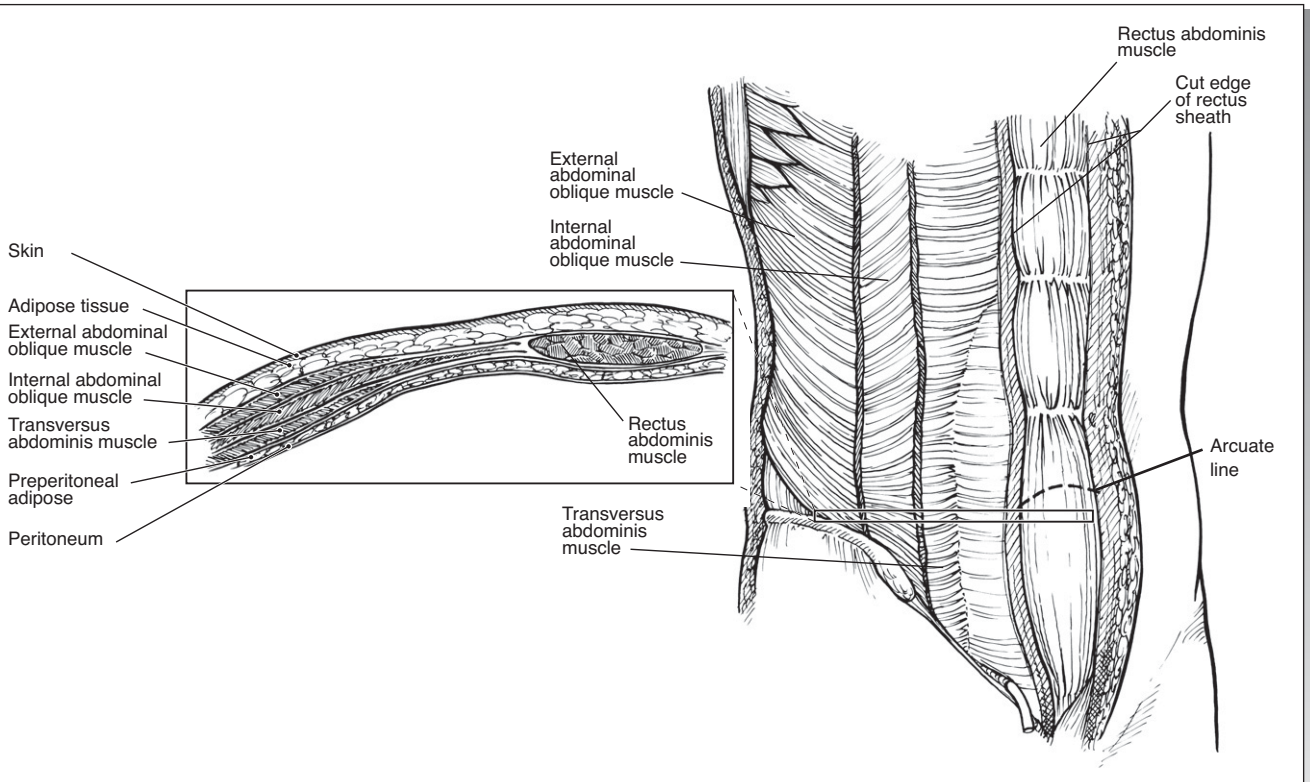

Figure 1 • Diagram of the layers of the abdominal wall. **Inset,** Cross section of the abdominal wall.

junctional complex between the musculofascial layers and the overlying skin.

The musculofascial layers provide the main support to the anterior of the abdomen. The fascia that surrounds the external oblique, internal oblique, and transversus abdominis muscles fuses in the midline to form the linea alba (Fig. 1).

The rectus abdominis muscles originate on the pubic crest and insert on the costal cartilage of the fifth through seventh ribs. The fascia of the external oblique muscles is anterior to the rectus muscles. The fascia of the internal oblique muscles splits and inserts both anterior and posterior to the rectus abdominis muscles above the arcuate line. The arcuate line is located midway between the umbilicus and the pubis. Below the arcuate line, the internal oblique muscle inserts only anterior to the rectus abdominis muscle. This arrangement creates an area of weakness below the arcuate line, where the posterior support of the abdomen consists of only the fascia of the transversus abdominis muscle.

The external oblique, internal oblique, and transversus abdominis muscles are flat, with their fibers oriented in different directions (see Fig. 1). The muscles function in a coordinated manner to increase intraabdominal pressure to allow activities such as defecation and childbirth.

The rectus abdominis muscles typically have three tendinous inscriptions. The first is usually below the umbilicus and is the weakest. The other two inscriptions are located at equal intervals between the xyphoid and the umbilicus.

The rectus abdominis muscles provide contraction to achieve function similar to that of the more lateral muscles. The rectus abdominis muscles also serve as vertical pillars of the anterior abdominal wall to balance the function of the posterior paraspinous muscles.

Treatment

For traumatic defects, the intraabdominal injuries are always addressed first and the abdominal wall is débrided to viable tissue. Primary closure is attempted when possible. If second-look procedures are planned, it is beneficial to achieve temporary closure of not only skin but also fascia. Such closure prevents contracture of the muscular abdominal wall and decreases edema of the viscera. Both of these goals are crucial to future closure. If the fascia will not close, temporary closure is achieved by any means possible. Most commonly, it is achieved with mesh or some other prosthetic material. When the patient is stabilized, the abdomen is reconstructed in stages.

When possible, immediate closure of the abdominal wall is preferred. Primary closure reestablishes

the normal functions of the abdominal muscles and in most cases leads to uneventful healing. In cases of prolonged abdominal surgery, extensive surgery, or wound inflammation, there may be edema of the viscera. Primary closure may not be possible because of the increased volume of the abdominal contents. Tension in the abdominal closure, as tension in any closure, often leads acutely to dehiscence or in the long-term to herniation. In addition, excess tension may lead to intraabdominal compartment syndrome.

In cases of excess tension, mesh, other prosthetic material, or cadaver dermis may be used as temporary or permanent fascial closure. In these cases, undermining of the skin can recruit the laxity in the skin and subcutaneous tissue to allow primary skin closure over the prosthetic fascial closure. If such closure is not possible, the exposed mesh can be allowed to granulate and may be skin grafted at a later time. Alternatively, when the edema of the bowel subsides, the prosthetic material can be removed and the abdominal fascia closed.

Prosthetic materials for permanent implantation should generally be placed on the inner aspect of the abdominal wall and not on the outer abdominal wall. Hernia sacs often dissect through the tissue/prosthetic interface when the prosthetic material is placed on the outer surface, whereas when placed on the inner surface, the rate of success is higher.

For selection of prosthetic material, several options are available, each with its advantages and disadvantages. Marlex mesh is readily incorporated into surrounding tissue. It handles well and forms a thick layer of scar that attaches it to the fascial margins. Because it is well incorporated, it is resistant to infection. However, Marlex can lead to extensive adhesions and promote bowel fistulas and thus is not the preferred material for direct contact with the gut. Gore-Tex does not become as well incorporated, does not form extensive adhesions to the fascial margins, and is more prone to infection than Marlex. It has the advantage of less adhesion formation and decreased risk of fistula formation. Sandwiches of Gore-Tex and Marlex can be made or layered constructs used. These constructs place the Gore-Tex layer in contact with the viscera and the Marlex layer against the fascia. Other materials such as Vicryl mesh and dermis can also be used. They are not permanent and are replaced with scar over time. These materials are extremely useful in temporary circumstances or to bolster primary closure, but they are not generally the preferred method of permanent reconstruction of defects.

In cases in which closure with prosthetic materials is in not indicated, abdominal wall closure is often possible with repositioning, partition, and separation of tissues of the abdomen. The two most common techniques are *component separation* and *myofascial release*, also called *fascial partition*.

Component separation can be used to close midabdominal defects of up to about 10 cm. The width of the defect that can be closed with this technique is less in the upper and lower portions of the abdomen. In this technique, the rectus abdominis muscle and fascia are mobilized medially to the midline. The internal oblique fascia and the transversus abdominis fascia are attached posteriorly, whereas the external oblique fascia is completely incised and the muscle separated and allowed to retract laterally (Fig. 2).

The method of myofascial release or fascial partition allows more extensive release and can achieve closure of wounds up to 20 cm in the midabdomen. As with the component separation method, the width of defect that can be closed is less in the upper and lower portions of the abdomen. The procedure

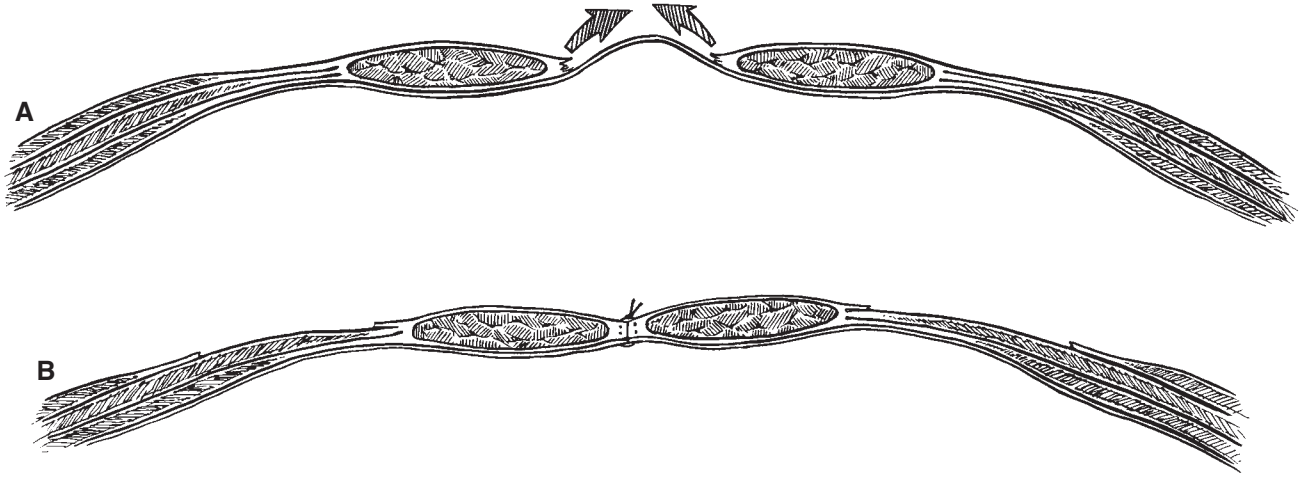

Figure 2 • Component separation method of abdominal closure. The external oblique muscle is completely incised and mobilized from the rectus sheath.

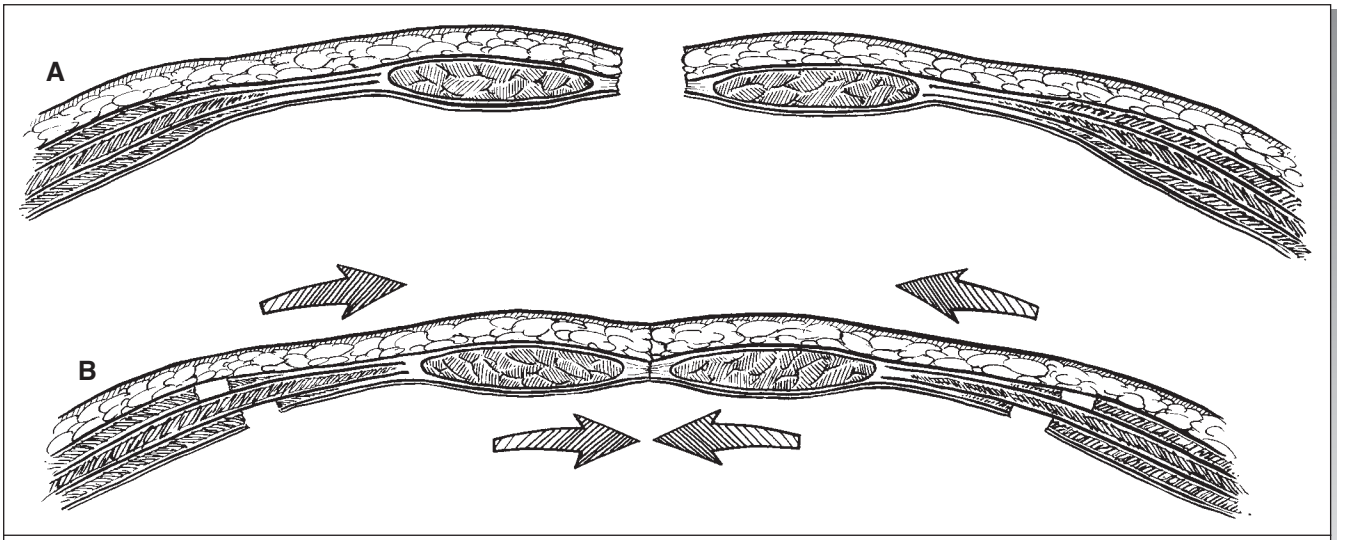

Figure 3 • Myofascial release method of abdominal closure. The external oblique muscle is incised. If more advancement is needed, the transversus abdominis is released. Alternatively, the transversus abdominis is left intact, and the internal oblique is incised externally.

of myofascial release consists of progressive release of the lateral abdominal wall muscles until closure is achieved. Usually, skin flaps are elevated and the external oblique is released laterally, where herniation or bulging is less likely. If further release is required, the transversus abdominis muscle may be released from inside the abdomen (Fig. 3), with the internal oblique left as the only intact lateral muscle. If an intraabdominal approach to the lateral transversus abdominis is not possible, a lateral release of the internal oblique may be performed. Not only does muscle release allow medial movement of the rectus abdominis muscles, but the pull of the lateral abdominal muscles against the closure is also less and disruption of the wound is less likely. The vascular and more important nerve supply of the rectus and oblique muscles traverses between the internal oblique and the transversalis muscles. Attempts should be made to preserve the innervation. Division of the nerves could lead to weakening of the already tenuous abdominal wall.

Tissue expansion is occasionally indicated in delayed reconstruction of large abdominal wall defects. There is no indication for tissue expansion in the acute setting or with signs of infection. Tissue expansion is more commonly used in the pediatric population to close congenital defects in the abdominal wall.

In cases in which a fascial defect is not able to be closed with prosthetic material, component separation, or myofascial release, a local or microvascular free flap may be used. The most common local flaps are the tensor fascia lata (TFL), rectus femoris, and rectus abdominis.

The TFL is supplied by the lateral femoral circumflex vessels. The vessels enter the muscle 10 cm below the anterior superior iliac spine. This becomes the pivot point for rotation of the flap. The fascia may be harvested beyond the end of the muscle to the level of the knee. The flap will reach up to or just above the umbilicus for coverage of fascial defects. As a myocutaneous flap, the skin paddle that is distal to the end of the muscle is somewhat unreliable and may not reach above the umbilicus.

The rectus femoris (RF) is also supplied by the lateral femoral circumflex vessels. The flap has an axial blood supply and a better arc of rotation than a TFL flap does. The rectus femoris flap will reach the upper part of the abdomen as a myofascial or myocutaneous flap. The RF contributes to the last 15 to 20 degrees of terminal extension of the knee. The knee extensor mechanism needs to be repaired by centralizing the vastus lateralis and medialis muscles.

The rectus abdominis may be rotated on either the superior epigastric or deep inferior epigastric vessels. The deep inferior epigastric vessels are the primary blood supply to this muscle. The flap is particularly useful as a vertical rectus abdominis myocutaneous (VRAM) flap and may reach nearly the entire surface of the anterior abdominal wall.

Microvascular free flaps are occasionally required for abdominal wall reconstruction when no other reconstructive options are available. Omental or mesenteric vessels may be used for vascular access. These vessels provide considerable flexibility in flap inset and positioning. Fascial, muscle, or musculocutaneous flaps may be used, depending on the clinical situation.

Postoperative Care

Patients are placed in a light abdominal binder postoperatively. This provides comfort, especially with

the necessary pulmonary toilet. Drains are placed under the skin flaps and removed when output falls to less than 30 mL/24 hr. Gastric drainage is important until the return of bowel function because increased pressure from abdominal distention can stress the abdominal repair. The diet is slowly advanced as tolerated. Antibiotics are used, especially in repairs with prosthetic materials.

Pulmonary toilet is critical after closure of abdominal defects. The increased abdominal pressure can lead to atelectasis at the lung bases and predispose to pulmonary infection. The postoperative pain and resulting immobility, along with the frequently long nature of these procedures, can increase the risk of deep venous thrombosis. Patients should always be fitted with compression boots in the operating room. Early mobilization should be encouraged. Enoxaparin (Lovenox) should be considered if early mobilization is not possible.

Pearls and Pitfalls

- The plastic surgeon should be called early for the treatment of abdominal defects. Closure is best achieved in the primary situation even if it entails unplanned procedures such as myofascial release or component separation. Defects are easier to handle acutely than after contraction of tissues.
- The reconstructive surgeon must know the anatomy of the abdominal wall and be ready to provide the wide gamut of appropriate surgical solutions, from simple skin grafts to complex free flap reconstructions.
- Primary closure with tension increases the risk for recurrent herniation.
- Myofascial release is beneficial not only for closure of defects but also for removal of the antagonist forces of the external oblique, and thus it decreases recurrence rates.

SUGGESTED READINGS

Carlson G, Bostwick J: Abdominal wall reconstruction. In Achauer BM, Eriksson E, Guyuron B, et al (eds): Plastic Surgery. St Louis, Mosby, 2000.

Levine JP, Karp NS: Restoration of abdominal wall integrity as a salvage procedure in difficult recurrent abdominal wall hernias using a method of wide myofascial release. Plast Reconstr Surg 107:707-716, discussion 717-718, 2001.

Ramirez OM, Ruas E, Dellon AL: "Component separation" method for closure of abdominal wall defects: An anatomical and clinical study. Plast Reconstr Surg 86:519-526, 1990.

Roth DA: Thoracic and abdominal wall reconstruction. In Aston SJ, Beasley RW, Thorne CHM (eds): Grabb and Smith's Plastic Surgery, 5th ed. Philadelphia, Lippincott-Raven, 1997.

Shaw WW, Aston SJ, Zide BM: Reconstruction of the trunk. In McCarthy JG (ed): Plastic Surgery. Philadelphia, Saunders, 1990.

Reconstruction of Posterior Trunk Defects

SAI S. RAMASASTRY

Posterior trunk defects can occur in both pediatric and adult patients. Although such defects are most commonly congenital in nature in the pediatric population, acquired defects can also occur in infants and children. In the adult population, almost all posterior trunk lesions are acquired. This chapter addresses the posterior trunk defects commonly seen in both children and adults, with the exception of pressure sores and giant congenital nevi, which are covered in other chapters.

Etiopathogenesis

Congenital Defects

In the pediatric population, midline defects involving the spine and defects of the lateral aspect of the trunk are congenital, secondary to tumor extirpation or trauma (Table 1). In this age group, soft tissue tumors of the trunk are primarily related to congenital abnormalities: neural tube anomalies, teratomas, dermal sinuses, and postanal pits.

Aplasia cutis congenita, or congenital absence of skin, is a rare entity. It may appear as a congenital ulcer, and the defect is generally characterized by a patch devoid of skin or hair. The area is often covered by a thin, transparent membrane or a necrotic ulcer. The defect may be small or large, single or multiple. Although it most commonly affects the scalp, typically the vertex of the skull, it can also occur on the trunk and the extremities, occasionally with lesions on both the ventral and dorsal aspects of the trunk.

Congenital defects of the chest wall include Poland's syndrome and defects of the anterior aspect of the rib cage. In Poland's syndrome, portions of the serratus anterior and other posterior trunk muscles may be missing, in addition to the pectoral muscles, thereby leading to a variable degree of scapular hypoplasia, elevation, and winging. The chest wall deformities generally cause no major physical disability, only psychological embarrassment. In most cases, however, the latissimus dorsi muscle is normal and provides a reliable method for soft tissue augmentation of the anterior of the chest.

Neural tube anomalies range from spina bifida occulta to gross spinal rachischisis with anencephaly, which is incompatible with life. The term *spina bifida* refers to a developmental dorsal defect in the vertebral column through which the contents of the spinal cord may protrude. Except in its simplest form (*spina bifida occulta*), the anomaly is usually grave and associated with paralysis of the lower limbs and perineal sphincters. Because of multiple associated developmental abnormalities, patients with neural tube anomalies have a mortality rate of over 40% that is attributed to meningitis, progressive hydrocephalus, or complications related to paraplegia.

Myelomeningocele refers to all lesions involving dysplasia of the spinal cord and meninges. The dorsal part of the cord may herniate into the sac and give the appearance of a dilated central canal in cross section; in this case, it is called a *syringomyelocele*. Usually, however, multiple cystic spaces are present in the dysplastic cord. An unenclosed neural tube that appears on the surface is termed a *myelocele*.

In *meningocele*, herniation of the meninges occurs through a gap between the vertebral arches. Meningoceles constitute about 14% of all cases of spina bifida. The lesions manifest as a midline swelling in the back, commonly in the lumbosacral region. The defect is usually, but not always, midline. A meningocele is usually covered completely with skin; however, the skin cover may be thin, atrophic, and prone to ulceration and rupture. A meningocele may also be associated with lipomatous or angiomatous tissue.

TABLE 1 Major Defects of the Posterior Aspect of the Trunk in the Pediatric Population

- Defects of the integument
  - Congenital
    - Aplasia cutis congenita
    - Giant hairy nevus
  - Acquired
    - Skin loss: necrotizing infections or posttraumatic defects
- Defects of the thoracic wall
  - Congenital
    - Poland syndrome with absence of the latissimus dorsi and/or serratus anterior muscles
  - Acquired
    - After tumor resection
    - After trauma
    - Burns and radiation injury
- Defects of the spine and lower part of the back
  - Congenital
    - Myelomeningocele
  - Acquired
    - Pressure sores
    - After tumor resection
    - Burns and radiation injury

Sacrococcygeal teratomas are the most common congenital tumors of the posterior aspect of the trunk in the pediatric population, with an incidence of 1 in 400,000 births. Other tumors include lipomas, lipoblastomas, and hemangiomas. The majority of teratomas are benign at birth; however, the incidence of malignancy increases with age, reaching a 50% incidence at 2 years. Therefore, prompt treatment is critical. The tumor is usually attached to the coccyx, occasionally with extension into the abdomen. Large tumors may cause difficulty during delivery.

Acquired defects of the posterior trunk in children are less common than congenital defects. Management of these problems in the pediatric population is similar to that in adult patients.

Acquired Defects in Adults

Acquired defects of the back in adults result from radiation injury, tumor ablation, or wound dehiscence and infection. Acquired defects may be located from the neck to the sacrum, but they are commonly seen in the cervicodorsal and sacral regions.

Midline defects of the back secondary to radiation injury, wound dehiscence with exposure of spinal stabilization devices, and postoperative wound infection with dural leakage pose difficult reconstructive challenges. The combination of previous neurosurgical procedures and extensive radiation therapy frequently results in unstable posterior midline wounds, particularly in the cervical region. Primary treatment of skin and soft tissue tumors often requires surgical ablation and adjunctive radiation therapy to prevent local recurrence or limb amputation. Late recurrences may present difficult wound problems because of radiation damage to the skin, scarring at the surgical site, or dural injury from previous resection. Chronic osteomyelitis of the spine is a rare complication. Aggressive débridement followed by immediate muscle flap coverage in conjunction with a 6-week course of culture-specific antibiotic therapy is currently the treatment of choice.

Flap Considerations

Flaps for Posterior Trunk Reconstruction

A variety of flaps are available for trunk reconstruction (Table 2). The three major pairs of poste-

TABLE 2 Systematic Regional Approach for the Reconstruction of Posterior Trunk Defects

WOUND LOCATION	MUSCLE FLAP
Cervical area	Trapezius muscle or musculocutaneous flap
Upper thoracic area	Trapezius muscle or musculocutaneous flap Latissimus dorsi muscle or musculocutaneous flap
Midthoracic area	Trapezius muscle or musculocutaneous flap Latissimus dorsi musculocutaneous flap Reverse latissimus dorsi muscle flap Paraspinous turnover flap
Lower thoracic and thoracolumbar area	Latissimus dorsi musculocutaneous flap Reverse latissimus dorsi muscle flap Paraspinous turnover flap
Lumbosacral area	Reverse latissimus dorsi muscle flap Latissimus dorsi musculocutaneous flap Greater omentum flap Latissimus dorsi muscle free flap (vein graft elongation of the pedicle) Latissimus dorsi muscle free flap
Sacral area	Gluteus maximus musculocutaneous flap Latissimus dorsi muscle free flap with vein graft elongation of the pedicle

From Ramasastry SS, Schlechter B, Cohen M: Reconstruction of posterior trunk defects. Clin Plast Surg 22:167-185, 1995.

rior trunk muscles available for reconstruction of midline and lateral posterior trunk defects are the trapezius, latissimus dorsi, and gluteus maximus (Figs. 1 and 2). Because of their location and extensive arc of rotation, the trapezius and latissimus dorsi flaps are particularly useful for reconstruction of trunk defects. All three muscles also have large cutaneous territories that permit mobilization as musculocutaneous flaps as well.

TRAPEZIUS FLAP. The trapezius muscle is a flat muscle of the upper posterior aspect of the trunk that arises from the superior nuchal line, the external occipital protuberance at the nuchal ligament, the thoracic vertebrae, and the supraspinous ligaments (see Fig. 1). It inserts on the lateral third of the clavicle, the spine of the scapula, and the acromion.

The muscle is innervated by the spinal accessory nerve and branches of C3-C4. The trapezius elevates the shoulder and rotates the scapula. Loss of function of the entire muscle causes shoulder drooping. Use of only the middle and lower portion of the muscle, however, creates minimal functional deficit. The trapezius muscle is useful for coverage of defects in the high thoracic and cervical areas.

The blood supply to the trapezius muscle comes from branches of the transverse cervical artery at the base of the neck. The ascending branch of the transverse cervical artery supplies the superior portion of the muscle, and the descending branch

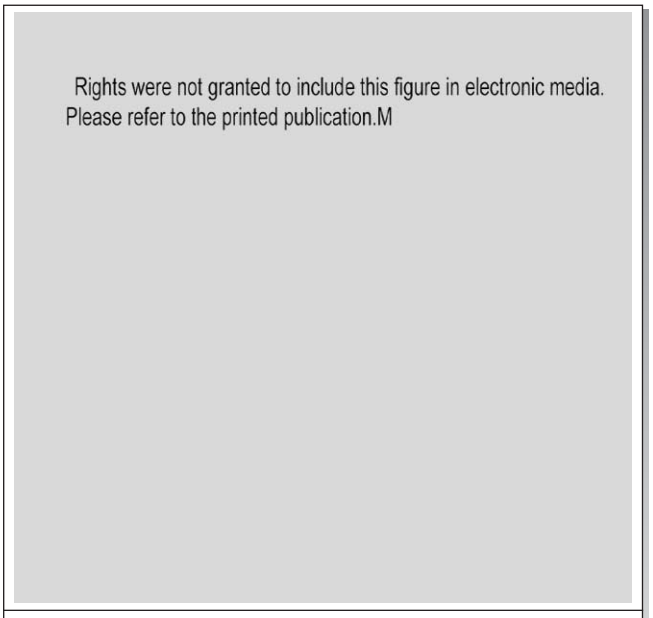

Figure 1 • Muscle flap availability for reconstruction of cervical, thoracic, and lumbar defects. **A,** Latissimus dorsi muscle based on the thoracodorsal vessels. **B,** Reverse latissimus dorsi muscle flap based on perforating vessels. **C,** Trapezius muscle flap based on the descending branch of the transverse cervical artery. (From Cohen M [ed]: Mastery of Plastic and Reconstructive Surgery. Boston, Little, Brown, 1994, p 1251.)

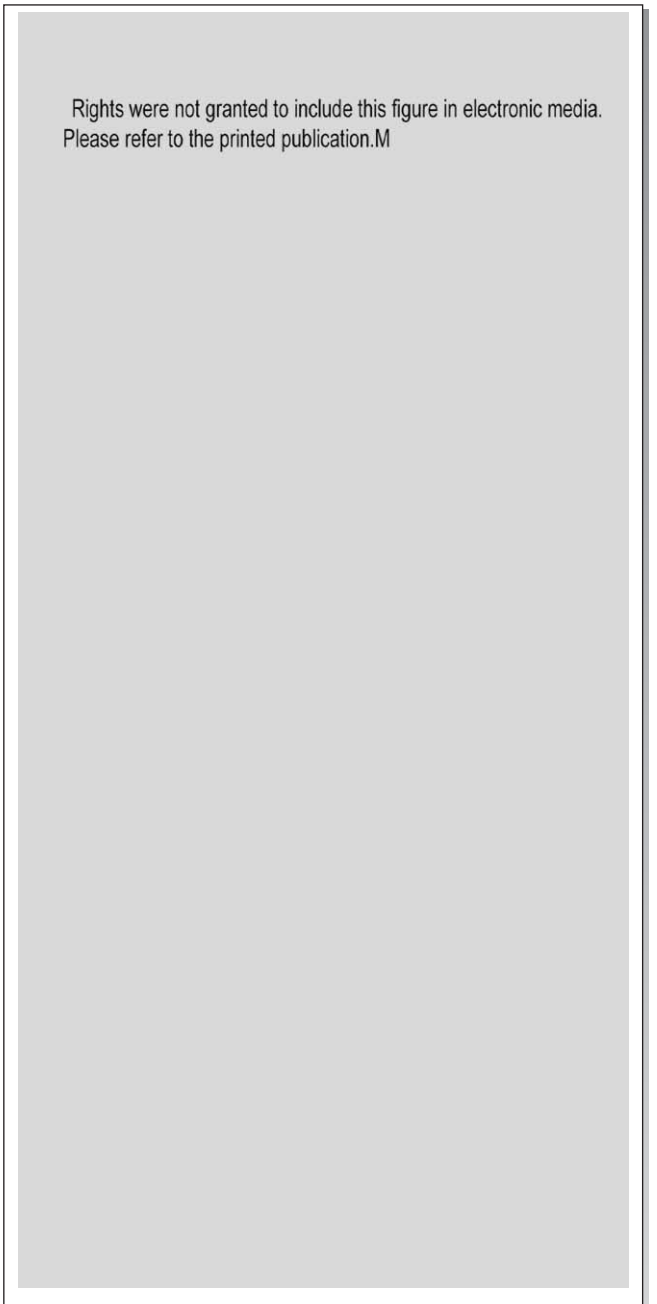

Figure 2 • **A,** Schematic of the anatomic basis of using bilateral interconnected latissimus dorsi–gluteus maximus musculocutaneous flaps. The latissimus dorsi and gluteus maximus muscles are shown with the major vascular pedicles: the thoracodorsal artery and the superior and inferior gluteal arteries. (The paravertebral perforating vessels are not shown.) **B,** Cross-sectional schematic showing rich anastomoses between the latissimus dorsi vessels and the superior gluteal vessels supplying the lumbar skin maintained by submuscular dissection without skin undermining. IGA, inferior gluteal artery; GMM, gluteus maximus muscle; SGA, superior gluteal artery. (From Ramirez OM, Ramasastry SS, Granick MD, et al: A new surgical closure of large lumbosacral meningomyelocele defects. Plast Reconstr Surg 80:804-805, 1987.)

supplies the inferior portion of the muscle. The inferior portion of the muscle is used for reconstruction of cervical and upper thoracic defects.

LATISSIMUS DORSI FLAP. The latissimus dorsi is a broad flat muscle covering almost half of the back. Its extensive origin extends from the lower thoracic, lumbar, and sacral regions of the spine through the thoracolumbar fascia to the crest of the ilium. It attaches laterally to the intertubercular groove of the humerus and forms the posterior axillary fold. The latissimus dorsi is innervated by the thoracodorsal nerve (C6-C8). The muscle, although contributing to shoulder adduction, extension, and medial rotation, is largely expendable. Even though muscle-sparing techniques have been described to preserve local mass and function, the entire muscle is generally used for reconstruction of the trunk without causing a significant functional deficit.

The latissimus dorsi has a dual blood supply. The thoracodorsal artery, the major vascular pedicle, enters the muscle on the deep surface in the posterior axillary fold, approximately 10 cm from the muscle insertion. By dividing the broad attachment to the chest wall, the muscle can be rotated (based on the major pedicle) to cover the vertebrae from the lumbar to the cervical region. It provides excellent blood supply to the overlying skin and adequate muscle bulk. The extensive arc of rotation of the vascular pedicle and the large muscle size make the latissimus dorsi flap the workhorse of trunk reconstruction. The large-diameter thoracodorsal vessels are also suitable for microvascular tissue transfer, and the pedicle, if lengthened with a vein graft, enables the muscle to reach the lumbosacral area.

In addition to the major pedicle, perforating branches of the intercostal and lumbar arteries pierce the latissimus dorsi muscle in a segmental fashion to supply the posteromedial aspect of the muscle and the overlying skin. These vessels form the vascular basis of the transverse lumbosacral back flap and the "reverse" or medially based musculocutaneous flap. The insertion of the muscle can be transected from the humerus; the entire muscle is elevated from the chest wall to the level of the paraspinous muscles, thus preserving the perforator blood supply, and transposed to cover defects of the lumbar and lumbosacral regions. Further modifications using triangular and rhomboid flap techniques based on one or two lumbar perforators supplying the distal latissimus dorsi muscle have been described both as musculocutaneous and as fasciocutaneous flaps.

Previous injury from radiation or surgery or ligation of the thoracodorsal pedicle, as during radical mastectomy, is not an absolute contraindication to use of the latissimus dorsi muscle. It has been shown that after such ligation, the latissimus dorsi muscle receives its blood supply in a retrograde manner from the serratus branch of the thoracodorsal artery via the intercostal system. As long as collateral flow is left intact, one can safely elevate the latissimus dorsi muscle in selected patients.

PARASPINOUS FLAP. The paraspinal musculature lies in the paravertebral gutter and extends from the thoracic to the lumbosacral region. The muscles, segmented in nature, arise from the laminae and transverse processes of the vertebrae and attach to the posterior aspect of the ribs and the iliac crest. Their blood supply is segmental and arises from the perforators extending from the dorsal segmental branches of the aorta. Thus, these muscles have a limited arc of rotation. Although the paravertebral muscles can be mobilized by fracturing the transverse processes and using them as osteomuscular flaps, this technique is rarely used today. Currently used modifications include bipedicle or turnover muscle flaps.

Turnover flaps of the paraspinous muscles have been used for thoracic and lumbar wound closure, but the bipedicle flap design limits flap excursion and utility. Superiorly based unipedicled paraspinous muscle flaps can be used for the coverage of lumbar wounds. The paraspinous muscles receive their blood supply from segmental perforating lumbar vessels; after flap elevation, the distal parts of the muscle are nourished by longitudinal intramuscular vessels. It is recommended that the superiorly based paraspinous muscle flap be mobilized from the thoracolumbar fascia laterally, the posterior superior iliac crest caudally, and the quadratus lumborum muscle anteriorly. The flaps can be elevated bilaterally up to T10 and can be transposed to cover midline lumbar defects.

GLUTEUS MAXIMUS FLAP. The gluteus maximus has the shape of a parallelogram. Its attachments include the ilium, sacrum, coccyx, and sacroiliac and sacrotuberous ligaments. The muscle attaches laterally to the greater trochanter of the femur and the iliotibial tract. The vascular territory of the gluteus maximus extends from the T12 level inferiorly to the distal third of the posterior aspect of the thigh. The muscle has four vessels that enter its deep surface. The superior and inferior gluteal vessels enter approximately 5 cm from the pelvic origin of the muscle, and the medial circumflex and first perforating branches of the femoral artery enter the muscle close to the femoral attachment. There are extensive anastomoses between the gluteal system and the lumbar perforators (see Fig. 2).

The relatively lateral position and deep location of the gluteal vessels, in addition to the robust collateral circulation, allows safe use of the gluteus maximus flap in cases of radiation necrosis in the sacral area. The muscle is innervated by the inferior gluteal nerve, which accompanies the inferior gluteal artery. The neurovascular pedicle should be

preserved to maintain vascularity and function. This flap can be used safely in nonparalyzed ambulatory patients because structural integrity and function of the muscle unit are preserved.

Flap design depends on the location and size of the defect. For large lumbosacral defects, the triangular skin paddle overlying the sacral portion of the muscle is extended to the lumbar area on the lumbar fascia. For sacral and coccygeal defects, adjacent triangular skin islands overlying the corresponding muscles are used. For defects up to 10 cm in diameter, modified sliding flaps can be used successfully without skin islands and V-Y advancement. The muscle and the skin are elevated en bloc and advanced to the midline.

OMENTAL FLAP. The greater omentum is an expansive intraabdominal fibrofatty apron that is attached to the greater curvature of the stomach and the transverse colon. It is supplied by the right and left gastroepiploic vessels, which arise from the celiac and superior mesenteric vessels. Knowledge of the vascular anatomy allows the surgeon to selectively divide the vascular arcade and lengthen the flap for more distant recipient sites. The greater omentum can be dissected from the transverse mesocolon and the greater curvature of the stomach and mobilized as a vascularized flap based on the right or the left gastroepiploic pedicle, depending on the location of the defect (Fig. 3). Division of one of the gastroepiploic vessels is usually the simplest and most commonly used method of omental elongation. This flap is tunneled retroperitoneally through a defect in the lumbar fascia/quadratus lumborum muscle to reach the posterior aspect of the trunk.

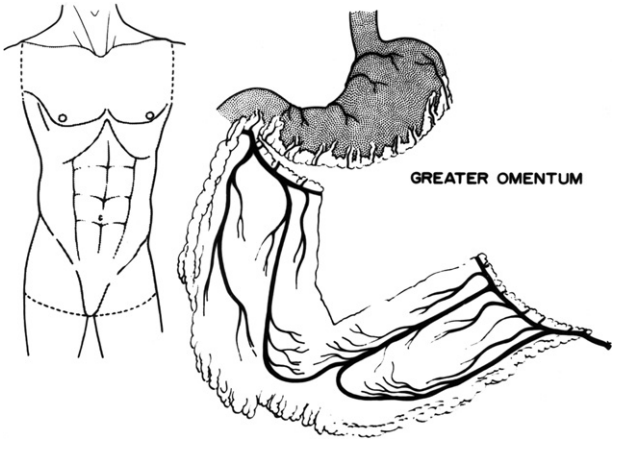

Figure 3 • Elongated omental flap based on the right gastroepiploic vessels and the resulting arc of rotation (*dashed lines*).

Surgical Goals

Certain general reconstructive principles should be followed to achieve successful coverage and minimize the incidence of complications and failures:

1. Control of infection with appropriate antibiotics
2. Local wound care
3. Extensive débridement of all devitalized soft tissue, cartilage, and bone
4. Reestablishment or maintenance of skeletal stability when needed
5. Preservation of neural function
6. Obliteration of dead space with well-vascularized tissue
7. Early definitive coverage of the defect to minimize infectious complications

The choice of flap for reconstruction varies according to location, size, and extent of the defect; previous radiation therapy; previous incisions; tissue availability; and the surgeon's preference and experience.

If there is no evidence of infection, large defects of the spine can be stabilized immediately with autologous bone grafts and hardware. Dural defects are repaired with dural grafts before soft tissue coverage with muscle flaps is undertaken. Bone grafts and prosthetic material are contraindicated in the presence of infection. Flaps can also be mobilized to reach the intrathoracic cavity to cover the bronchial stumps for the treatment of empyema and bronchopleural fistulas. In contrast to anterior chest defects, posterolateral chest wall defects do not require skeletal reconstruction for bone stability because the scapula and surrounding soft tissue provide the necessary support.

One-stage reconstruction of congenital and acquired back wounds requires either one- or two-layer muscle closure, depending on the size and depth of the wound. In myelomeningocele patients, the initial step before flap closure is closure of the dural defect and débridement of the devitalized cutaneous margins. In the acquired category, débridement of scarred, irradiated, and infected tissue is mandatory. Adequate tumor-free margins should have been ensured previously either by frozen section or by permanent section when treating malignant disease. Traditional wound closure of back defects by primary closure under tension or the use of skin grafts or local transposition flaps is quite often a poor choice because of inadequate blood supply to the area, especially after irradiation. Skin grafts are a poor choice if chemotherapy is planned after extensive soft tissue resection for tumor treatment. Although skin grafts may provide temporary coverage, the use of immediate flap coverage for

wound closure permits rapid treatment with adjunctive chemotherapy and radiotherapy if needed.

Treatment

Treatment usually involves surgical correction of the defect and soft tissue coverage, preferably by a multidisciplinary team, including a neurosurgeon, pediatric surgeon, and plastic surgeon.

The treatment of choice for teratomas is operative removal of the entire mass as soon as possible. The usual surgical approach is posterior or perineal. Large intrapelvic or intraabdominal extensions may require a combined abdominosacral operation. To minimize recurrence, the coccyx is removed with the tumor. After the tumor is removed, the levator ani raphe should be reattached to the sacrum to restore the levator sling.

Most often the defect of aplasia cutis congenita manifests as a small ulcer, which will heal spontaneously with moist dressing changes and conservative wound care. If an eschar is present, it is maintained as a biologic dressing until the defect heals or surgery is performed. Larger areas are best managed by débridement and closure by primary wound approximation, skin grafting, or local skin flaps for larger defects.

Reconstructive Surgical Procedures in Spina Bifida

Surgical closure of open myelomeningoceles increases the 5-year survival rate from approximately 10% (untreated) to approximately 50% (treated). Survivors require management of related problems such as locomotion, incontinence of urine and stool, severe spinal deformities, pressure sores, and neuropathic ulcers of the extremities.

Reconstructive procedures should be tailored to the defect. Desiccation of the spinal cord should be prevented as soon as the diagnosis is established to avoid deterioration in neurologic function. The exposed cord should be protected and kept moist with sterile isotonic solution, and the wound should be closed as soon as possible. In infants with a simple meningocele and adequate skin cover, surgery can be deferred until the patient is 3 months of age.

After the spinal cord is covered with well-vascularized tissue, the hydrocephalus needs to be decompressed either with temporary measures, such as a ventricular tap, or permanently with a ventriculoperitoneal shunt. Although lower limb deformities such as clubfoot and paralytic dislocation of the hip are common, every effort should be made to render the child as ambulatory as possible.

No treatment is required for the bony defect in spina bifida occulta, but the overlying triangle of hair-bearing skin may be excised for aesthetic reasons. If the skin is thin and atrophic or absent, as in the open variety of spina bifida (myelomeningocele), the neural defect must be covered with well-vascularized tissue. In infants with pronounced kyphosis, it may be necessary to remove some of the prominent laminae (kyphectomy) with bone-cutting forceps. Such removal helps to eliminate tension on the soft tissue closure and minimizes skin breakdown.

Soft tissue coverage is needed as soon as the dura is covered. The majority of defects are sufficiently small that they can be closed primarily with simple undermining of the skin edges to achieve a tension-free approximation in the midline. However, approximately 25% of defects are sufficiently large that primary closure cannot be accomplished with simple skin undermining. Larger myelomeningocele defects can be difficult to repair and are associated with wound breakdown and infection. These considerations are important because primary healing has been shown to be a significant determinant of neurologic outcome by preserving functioning neural tissue and preventing infectious complications.

Various methods are reported in the literature for the closure of large, open myelomeningocele defects (Table 3). Closure of myelomeningocele defects with a split-thickness skin graft is an option. Soon after the neural repair, a thin skin graft is applied to the

TABLE 3 Documented Methods of Repair of Myelomeningocele Defects

Primary closure
Wide undermining with skin advancement
Cutaneous flaps without skin grafting
Limberg flap or flaps
Double rotation
Double transposition
Cutaneous flaps with skin grafting of the donor site
Bipedicle—vertical orientation
Bipedicle—horizontal orientation
Muscle flaps with split-thickness skin grafting of the primary defect
Latissimus dorsi
Gluteus maximus
Trapezius
Musculocutaneous flaps with split-thickness skin grafting of the donor defect
Vertical bipedicle latissimus–gluteus maximus
Vertical bipedicle latissimus-gluteus fasciocutaneous
Reversed latissimus island flap
Bilateral sliding muscle flaps
Latissimus dorsi
Latissimus dorsi–gluteus maximus*
Paraspinous osteomuscular flap or flaps
Split-thickness skin graft

*Author's preferred method.
From Ramasastry SS, Cohen M: Soft tissue closure and plastic surgical aspects of myelomeningocele defects. Neurosurg Clin N Am 6:280, 1995.)

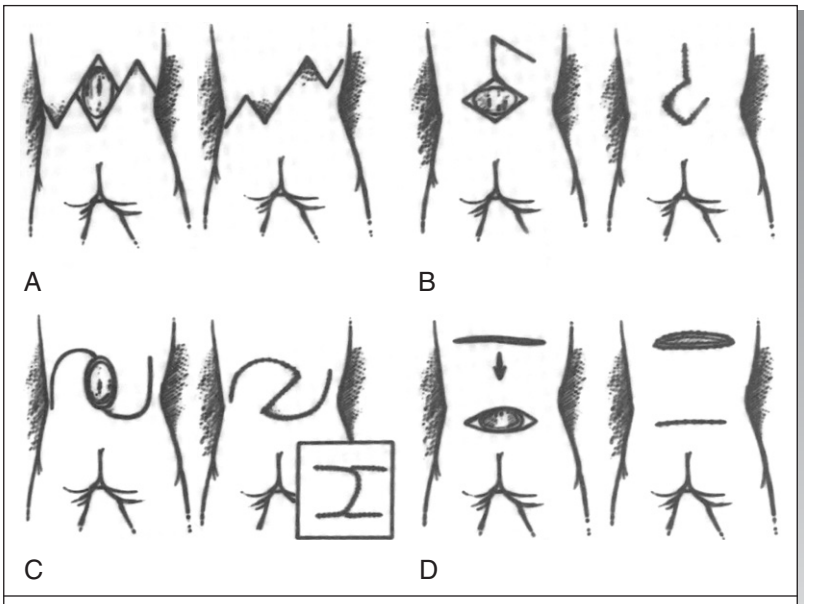

Figure 4 • Overview of surgical techniques for repair of medium to large myelomeningocele defects. **A,** Double Z-plasty. **B,** Limberg type. **C,** Bilateral transposition flaps. **D,** Bipedicle lumbosacral flaps. All are skin flaps with random blood supply. (From Zide BM: Spina bifida. In McCarthy J (ed): Plastic Surgery, vol 6. Philadelphia, WB Saunders, 1990, p 3784.)

neural plaque. Leakage of cerebrospinal fluid (CSF) requires frequent aspiration to facilitate graft adherence to the bed. However, a skin-grafted myelomeningocele is vulnerable to repeated trauma, functional impairment, or meningitis from CSF leakage and is therefore not a satisfactory long-term solution. It is best to use the skin graft as a temporary biologic dressing and perform a definitive repair within the first 2 weeks of age and within the same hospitalization because removal of the skin graft at a late stage is difficult and attempts at removal enhance the risk of damaging functional neural tissue and increasing neurologic impairment.

Several variations of skin flaps have been described for closure of larger myelomeningocele defects to avoid wound problems associated with wide skin undermining and primary closure (Figs. 4 and 5; see also Table 3). Local suprafascial skin flaps include double transposition flaps, rhomboid flaps, double Z-plasty technique, bipedicle flaps with lateral relaxing incisions, and bipedicle lumbosacral flaps. All of these skin flaps have a random blood supply and require extensive undermining. Tension at the suture line, local pressure from underlying bony prominences, a marginal blood supply, or subclinical infection (or any combination of these problems) often results in wound breakdown, thus necessitating coverage with a more reliable flap reconstruction.

Musculocutaneous flaps have the benefit of transferring compound tissue with a dependable blood supply to achieve primary closure of myelomeningocele defects in a safe and reliable manner. The considerable bulk of the muscle flaps helps to obliterate residual dead space and provides immediate coverage. Because of their rich vascular supply, the flaps enhance wound healing and assist in the elimination of residual local infection. They provide protection for underlying vital structures such as the spinal cord or the lungs, as well as stable coverage of bone grafts, dural patches, and orthopedic hardware.

The author's preferred method for closure of large myelomeningocele defects is en bloc advancement of bilateral interconnected latissimus dorsi–gluteus maximus musculocutaneous flaps without lateral relaxing incisions (Fig. 6). A rich anastomotic plexus exists between the vasculature of the skin overlying the gluteus maximus and the latissimus dorsi muscles. Adequate circulation to the lumbar area skin between the latissimus dorsi and the gluteus maximus musculocutaneous territories is maintained if dissection of both muscles is performed in continuity, even if the paraspinous segmental perforators are sacrificed (see Fig. 2). Mobilization of both musculocutaneous units as one composite unit out to the midaxillary line achieves three-layer (muscle, subcutaneous tissue, and skin) coverage without tension and provides ample soft tissue padding over the neural repair that is durable with minimal risk of late skin breakdown. Kyphectomy can be performed safely to correct the gibbus deformity at the time of flap closure. No relaxing incisions or back cuts are used, and no skin grafts are needed.

During initial closure of the dural defect, desiccation of the cord is avoided by frequent warm saline irrigation. Undermining of the skin should be

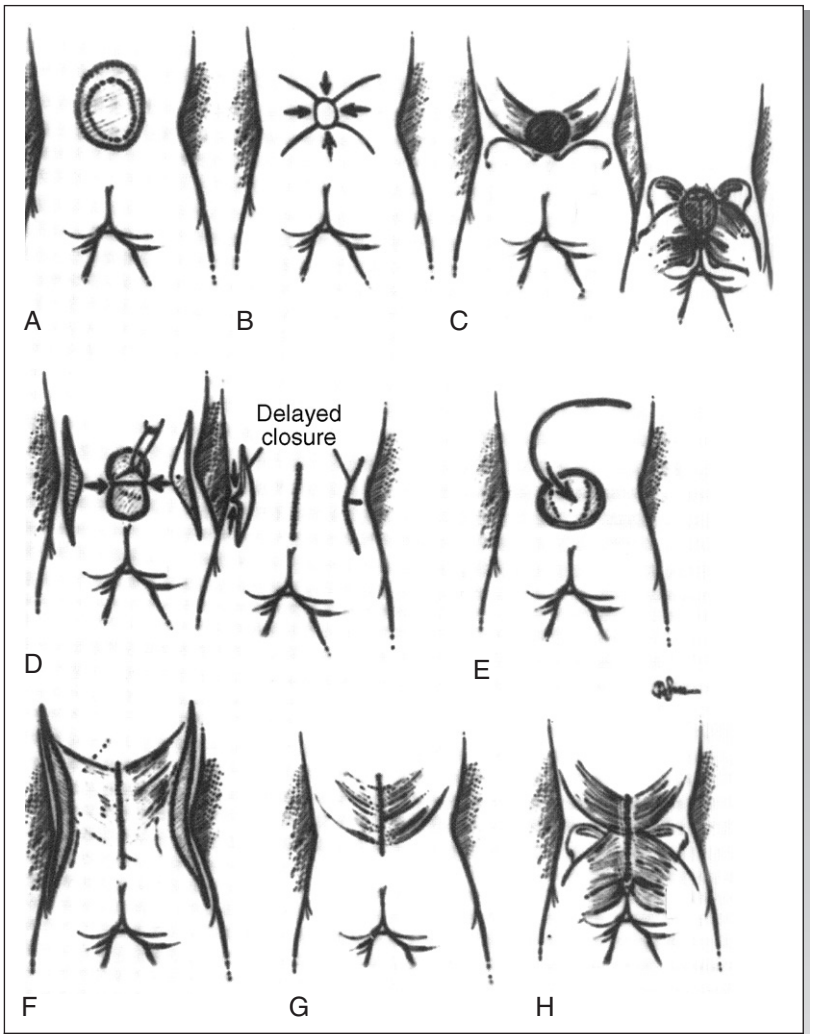

Figure 5 • Overview of the techniques used for larger myelomeningocele defects. **A,** Delayed split-thickness skin graft (STSG) on the dura. **B,** Partial flap and STSG on muscle. **C,** Bilateral muscle advancement and STSG. **D,** Bipedicle skin flap. **E,** Lumbosacral back flap. **F,** Latissimus myocutaneous (MC) flaps with relaxing incisions. **G,** Latissimus MC flaps without relaxing incisions. **H,** Bilateral double MC flaps. (From Zide BM: Spina bifida. In McCarthy J (ed): Plastic Surgery, vol 6. Philadelphia, WB Saunders, 1990, p 3786.)

avoided and the neural closure should be meticulous and watertight. After dural repair, flap dissection is begun by incising the thoracolumbar fascia over the paraspinous muscles and carrying the dissection under the latissimus dorsi muscle to its free lateral border bilaterally. The perforating paravertebral vessels are cauterized and divided in the process to achieve adequate medial advancement. The entire latissimus dorsi muscle is thus based on the thoracodorsal vessels and nerve, which should be preserved. The latissimus dorsi muscle is freed from its attachment to the external oblique and serratus posterior muscles by sharp dissection. The gluteus maximus muscle is detached from the iliac crest and the sacrum. The dissection is carried in the plane between the gluteus maximus and medius muscles. Care is taken to preserve the superior and inferior gluteal vessels. One should be aware that the quadratus lumborum muscle is often poorly developed in infants, and thus the retroperitoneal structures, including the ureter, must be safeguarded. No flank relaxing incisions are used, and the lumbar area skin between the latissimus dorsi and the gluteus maximus musculocutaneous units is maintained intact. Both flaps meet in the midline with minimal tension and can be approximated in three layers. Meticulous hemostasis is mandatory and drains are unnecessary.

Advantages of this technique include: (1) no relaxing incisions or back cuts are used; (2) no skin grafts are needed; (3) the blood supply of the intervening skin between the latissimus dorsi and gluteus maximus muscles is reliable; (4) inclusion of the gluteus muscle as a musculocutaneous unit pro-

Figure 6 • A, Submuscular dissection of the latissimus dorsi and gluteus maximus myocutaneous units bilaterally. *Dotted lines* indicate the area of undermining at the submuscular plane beneath the latissimus dorsi muscle (LDM) and the gluteus maximus muscle (GMM). **B,** Schematic showing approximation of the LDM and GMM myocutaneous units in the midline. (For the sake of clarity, the muscle approximation is shown through a cutaneous window.) No back cuts are made in the skin. (From Ramirez OM, Ramasastry SS, Granick MD, et al: A new surgical approach to closure of large lumbosacral meningomyelocele defects. Plast Reconstr Surg 80:804-805, 1987.)

vides significant superomedial advancement, thereby facilitating closure of large defects of the low thoracolumbar and lumbosacral areas; (5) transfer of the gluteus maximus muscle with this design does not cause functional deficit; and (6) this composite muscle flap provides satisfactory soft tissue padding over the neural repair. Theoretical disadvantages associated with dissection of such an extensive nature include significant blood loss and prolonged operative time. The use of electrocautery and meticulous dissection with attention to hemostasis helps to eliminate any significant blood loss and the need for blood transfusion. The procedure is well tolerated by newborn infants. Dural closure and soft tissue coverage can be achieved in one stage, within 12 to 24 hours of birth of the infant. Satisfactory healing can be expected, and no deterioration in neurologic function is noted after this technique.

Regional Flap Approach to Trunk Defects

Cervical defects of small to medium size are reconstructed reliably with a trapezius flap based on the dominant vascular pedicle, the descending branch of the transverse cervical artery (see Table 2).

Small upper thoracic defects are reconstructed with the trapezius flap. Large upper thoracic defects are reconstructed satisfactorily with the trapezius flap or the latissimus dorsi flap. If a deep cavity is present that requires obliteration, the trapezius flap can be transposed to fill the dead space and the latissimus dorsi musculocutaneous flap can be used to close the superficial aspect of the wound. However, one muscle flap is usually adequate. Proximally based flaps on the thoracodorsal pedicle are more versatile and reliable than the reverse.

Midthoracic defects can be closed with the lower portion of the trapezius, the latissimus dorsi, or a reverse latissimus dorsi flap. If closure of a deep wound is necessary, turnover flaps of the paraspinous muscle with thoracolumbar fascia are also used. The flap is designed by incising the muscle and fascia along their length, approximately 2 to 3 cm from the spinous processes. The muscle and fascia unit, based on the lumbar perforators, is turned over and sutured together in the midline to close the deep wound. The superficial wound is closed with the latissimus dorsi muscle designed as an "advancement flap" or as a distally based "reverse flap." Lateral relaxing incisions are not generally necessary to achieve tension-free closure when advancing the latissimus muscle flap based on the dominant vascular pedicle.

Thoracolumbar and *lumbar defects* are reconstructed with (1) latissimus dorsi musculocutaneous advancement flaps, (2) a latissimus dorsi musculocutaneous flap with thoracolumbar fasciocutaneous extension, or (3) composite latissimus dorsi and gluteus maximus musculocutaneous interconnected

flaps. The thoracolumbar fascia and paraspinous turnover flaps are useful in obliterating the residual dead space. In the thoracic and lumbar regions, the paraspinous muscles are useful to cover narrow, deep defects, but they are not sufficiently large to cover extensive defects. The paravertebral muscles can be used as turnover flaps or bipedicled flaps to assist in reconstruction of defects in this area. For large defects with deep cavities in the thoracic and lumbar regions, paravertebral turnover flaps help to fill the deep dead space, and the repair can be reinforced with unilateral or bilateral latissimus dorsi muscle or musculocutaneous flaps to achieve multilayer, durable coverage. The latissimus dorsi musculocutaneous flap with thoracolumbar fasciocutaneous extension is based on the thoracodorsal artery. The cutaneous territory overlying the thoracolumbar fascia represents the random extension. To elevate the flap, the distal margins of the latissimus dorsi muscle need to be freed from the ilium and the oblique muscles. The lateral donor defect usually requires a skin graft for closure.

Sacral defects can be covered with either a single or bilateral gluteus maximus musculocutaneous flaps. Defects up to 10 cm can be covered with bilateral sliding flaps without resorting to a V-Y technique for closure. Larger defects, greater than 10 cm, require V-Y advancement flaps; care should be taken to identify and preserve the inferior gluteal vessels and nerve to maintain hip stability in an ambulatory patient.

Role of Tissue Expansion

Tissue expansion is an excellent reconstructive method for the management of superficial defects such as giant congenital nevi, superficial defects secondary to extirpated skin and soft tissue tumors, and defects after débridement of skin and soft tissue infections/burn wounds and traumatic scar deformities in which there is no exposure of vital structures such as the dura, spinal cord, or neural elements, especially when immediate soft tissue coverage is not required. While some have recommended the use of tissue expansion for the treatment of myelomeningocele, this approach is more reasonable for previously skin-grafted myelomeningocele defects, providing reliable soft tissue cover in place of an unstable scar. In the acute management of a large open myelomeningocele in the newborn, rapid tissue expansion does not provide reliable, well-padded, and durable tissue cover. Additionally, a 3- to 4-week delay associated with traditional tissue expansion and with the dural repair left exposed or inadequately covered and the attendant risk of CSF leakage is not recommended.

Microvascular Free Transfer

Most defects of the posterior of the trunk can be managed with pedicled muscle flaps. In the case of irradiation of the spine, extensive trauma, or extensive débridement, however, local pedicle flaps may not be available. In these unusual cases, reconstruction with microvascular free tissue transfer may be necessary (Fig. 7). For example, the latissimus dorsi musculocutaneous flap can be used with staged or immediate extension of the thoracodorsal pedicle and interposition vein grafts to cover large defects in the lumbosacral region. When local flaps are not available, the dorsal branch of the fourth lumbar artery is a satisfactory recipient vessel for latissimus dorsi free muscle flap coverage of large lumbosacral and sacral defects.

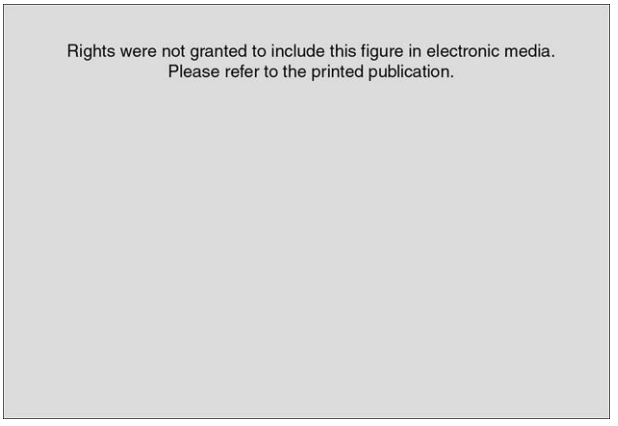

Figure 7 • Sacral defect repaired by one-stage microvascular transfer of a latissimus flap to the sacrum with vein grafts. **A,** The planned operative procedure, a latissimus dorsi myocutaneous flap, is outlined. Note the outline of the margin of débridement of the sacral ulcer. **B,** Interpositional vein grafts 24 cm in length to extend the flap to the sacral area. (From Nahai F, Hagerty R: One-stage microvascular transfer of a latissimus flap to the sacrum using vein grafts. Plast Reconstr Surg 77:312, 1986.)

Postoperative Care and Complications

Complications of flap reconstruction include partial flap loss, persistent dead space from lack of adequate muscle bulk, and persistent infection. Débridement and readvancement of the flap are adequate in most cases. If there is significant or total flap loss, however, a second flap reconstruction is often necessary to obliterate the dead space and protect vital structures.

Pearls and Pitfalls

- Timely soft tissue closure with well-vascularized tissue, especially when the dura is exposed, significantly minimizes wound com-

plications and prevents deterioration of neurologic function.

- The surgeon must adhere closely to the basic reconstructive principles of achieving adequate débridement of all necrotic or devitalized tissues and tumor-free margins whenever possible; management of infection with local wound care and appropriate antibiotic therapy; and coverage with well-vascularized tissues to obliterate any residual dead space and to cover bone grafts, orthopedic hardware, and vital structures such as the dura and spinal cord.
- Flap selection is of paramount importance for success, and only muscles with an appropriate arc of rotation, vascularity (the vascular pedicle should be outside the field of injury after irradiation, trauma, or previous operative procedures), and adequate bulk should be used.
- Adequate flap mobilization to obtain tension-free closure and judicious use of drains and postoperative antibiotics are essential.
- Occasionally, microvascular free tissue transfer may be necessary if local flaps are unavailable.

SUGGESTED READINGS

Casas LA, Lewis VL Jr: A reliable approach to the closure of large acquired midline defects of the back. Surgery 84:632, 1989.

Mathes SJ, Nahai F: Trunk: Posterior trunk. In Clinical Atlas of Muscle and Musculocutaneous Flaps. St Louis, Mosby–Year Book, 1979, p 91.

Ramasastry SS, Cohen M, Radhakrishnan J: Congenital back defects. In Bentz ML (ed): Pediatric Plastic Surgery. Appleton & Lange, Stanford, Conn, 1998, pp 757-781.

Ramasastry SS, Schlechter B, Cohen M: Reconstruction of posterior trunk defects. Clin Plast Surg 22:167-185, 1995.

Pressure Sores

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JAMIE P. LEVINE

The problem of pressure sores has plagued humanity for centuries. Significant advances in surgical treatment were not made until the middle of the 20th century, when both scar and osseous excision with flap coverage was described. However, even now, recognition and treatment of pressure sores often do not receive appropriate medical attention or surgical intervention.

Pathogenesis

The most common sites for pressure sores are the sacral, ischial, and trochanteric areas; other sites include the occipital, malleolar, and heel areas. The anatomic distribution depends on patient positioning and the associated pressure distribution. Five percent to 10% of hospitalized patients have a preexisting pressure sore, and a new pressure sore will develop in nearly 3% during a hospitalization. Approximately two thirds of pressure sores occur in patients older than 70 years. Comorbid conditions include cardiovascular disease, orthopedic injuries, and neurologic disorders. The growth in the elderly segment of the population has led to an increase in the number of pressure sores, along with an attendant increase in the economic impact (approximately $3 billion in the U.S. economy alone).

In chronic care facilities, the incidence of pressure sores ranges from 5% to 40%. In the acute care setting, other disease processes may direct attention away from prevention and diagnosis of pressure sores. Consequently, these ulcers may occur in otherwise ambulatory and sensate patients and tend to be more severe than those seen in a chronic care facility. The advent of dedicated rehabilitation centers specifically geared toward the care of patients with spinal cord injury has greatly improved awareness, prevention, and timely treatment of pressure sores. In general, patients with pressure sores require significantly more nursing care, have lengthy hospital stays, and incur higher hospital charges. Measures to stem the impact of this disease process rest primarily on increased awareness and prevention. The impact of this disease is considerable, as seen by the fact that 8% of paraplegic patients die as a result of a pressure sore.

Etiopathogenesis

Pressure

Constant pressure sufficient to impair local blood flow to soft tissues is the most important determinant leading to the development of pressure sores. The external pressure must be greater than the arterial capillary pressure of 32 mm Hg to impair inflow and must exceed the venous capillary closing pressure of 12 mm Hg to impair venous return. Experimental studies in animal models have shown that constant external pressure for 2 hours produces irreversible tissue injury. When the tissue was examined histologically, no change was seen when pressure was released at 5-minute intervals.

The supine position produces the greatest pressure on the sacrum, buttocks, heels, and occiput—ranging from 40 to 60 mm Hg. The sitting position produces pressure in excess of 75 mm Hg over the ischial tuberosities. In the prone position, the knees and chest are exposed to approximately 50 mm Hg. These variables are extremely important to keep in mind when planning surgical positioning for long surgical procedures.

Epidermal cells are able to withstand the absence of oxygen longer than the more metabolically active

myocytes. Pathologic analysis reveals that muscle is more sensitive to ischemia than are skin and subcutaneous tissues. Although early damage can be observed in the dermal layer and subcutaneous adipose tissue, the epidermis does not show signs of necrosis until late. Some of the first pathologic changes include dilation of capillaries and venules, as well as separation of endothelial cells. Muscle overlying a bony prominence is the most susceptible to ischemia. This concept is important—when skin changes are seen, there must be damage to the underlying muscle.

Infection

Bacterial contamination from inadequate local skin care (urinary/fecal incontinence) can delay wound healing and extend the zone of tissue necrosis. In addition, bacterial infections have an associated collagenolytic process that contributes to the accelerated rate of tissue necrosis. Compressed skin consequently has decreased resistance to bacterial invasion. Causes for this decreased resistance probably include diminished lymphatic drainage, denervation, ischemia, poor vascular recruitment, and impaired immune function.

Shearing Forces

Shearing forces can significantly increase tissue ischemia. When a patient is moved or repositioned in bed, the area of body that is in contact with the bed is subject to shearing forces. While the skin and subcutaneous tissues adhere to the bed sheets, the underlying fascia, muscle, and bone are pulled in the direction of the force applied. The subsequent stretching and compression of the muscle and perforating vessels to the skin can result in ischemic necrosis in compromised anatomic sites.

Edema

Preexisting edema, whether from hypoproteinemia, cardiac failure, hepatic dysfunction, or a septic state, plays a significant role in the infectious process. When a compressive force in excess of capillary pressure (12 mm Hg) is applied to the skin, the venules become engorged, and as a result of outflow obstruction, total tissue pressure increases. Consequently, arterial pressure increases to maintain perfusion, and eventually extravasation occurs and leads to edema in the surrounding tissues. If the local tissues are denervated (e.g., paraplegia), sympathetic tone is lost and the resultant vasodilation increases edema and vascular stasis.

Loss of the lymphatic pump (a skeletal muscle function) in paraplegic/quadriplegic patients contributes to lymphatic edema. Local mediators may also be responsible for edema formation inasmuch as various inflammatory mediators are released in response to the trauma of compression. The normal homeostatic balance among various prostaglandins is altered, and this imbalance results in increased leakage of fluid through cell gap junctions and accumulation of interstitial fluid.

Pathologic Anatomy

Stage 1 pressure sores are characterized only by persistent skin erythema. The process of ulceration is completely reversed by removal of pressure because there is no evidence of skin breakdown. *Stage 2* pressure sores are accompanied by skin breakdown and ulceration of subcutaneous fat. The ulcers often heal with avoidance of pressure and adequate wound care. They are, however, prone to infection, a variable that increases tissue necrosis and extends the sore to a deeper level. *Stage 3* sores have ulceration to the level of muscle. *Stage 4* ulcers are characterized by breakdown to the underlying bony prominence. Osteomyelitis with chronic bone exposure is invariably present.

After pressure sores have progressed from the acute phase to involve deeper structures, there is destruction of skin, subcutaneous tissue, fat, muscle, and even bone. Osteitis or osteomyelitis may exist, and pathologic fractures along with extraosseous calcifications (myositis ossificans) are not uncommon. The actual extent of bone involvement is frequently determined only during operative débridement. Soft tissue infection may be seen in patients with inadequate urinary or fecal diversion when the pressure sore is exposed to these body fluids. Infections may be polymicrobial; among the more common bacteria are staphylococci, streptococci, *Pseudomonas aeruginosa*, *Proteus mirabilis*, and *Escherichia coli*.

Pressure sores that are long-standing and have gone through periods of healing and recurrence have marginal scar epithelium. Granulation tissue may be pale because of thrombosed vessels, a byproduct of chronic inflammation. The infectious process may also result in the formation of sinus tracts, which may readily enter bursal cavities and lead to septic arthritis or joint destruction.

The pathologic process of early pressure sores can often be reversed with simple, conservative measures—relief of pressure and enhanced oxygen delivery. After skin has broken down over a bony prominence, widespread tissue destruction is invariably present in deeper tissue planes, and definitive treatment mandates surgical débridement with reconstruction as indicated.

Diagnostic Studies

Pressure sores are best evaluated by history and physical examination. Active infection is suspected

in the presence of wound edge cellulitis, purulent discharge, and a foul odor. Quantitative wound culture confirms infection if the bacterial count is greater than 10^6 organisms per gram of tissue. Osteomyelitis is best evaluated by bone biopsy. Generally, if bone is palpable, infection is present. If evidence of clinically significant osteomyelitis is noted, long-term intravenous antibiotics alone should not be the planned therapy and should not be considered a substitute for complete wound débridement. Magnetic resonance imaging (MRI) can be a useful, noninvasive tool for the diagnosis of osteomyelitis. It is, however, expensive and rarely provides more information than physical examination alone.

Reconstructive Goals

The goals for the treatment of pressure sores can be summarized as follows:

1. Reversal of the pattern of pressure
2. Treatment of associated conditions (malnutrition, spasticity, contracture, anemia, incontinence)
3. Débridement of necrotic tissue
4. Treatment of infection
5. Closure of the wound

Treatment

Nonsurgical Treatment

ASSESSMENT AND TREATMENT OF ASSOCIATED CONDITIONS. Adequate nutrition must be achieved in all patients with pressure ulcers. Albumin (half-life of 21 days), prealbumin (half-life of 2 days), and retinol binding protein (half-life of 11 hours) are indicators of malnutrition and should be measured in patients with pressure sores. Complete nutritional support, including replacement of vitamins, minerals, and trace elements, is necessary for normal wound healing. Oral supplements, tube feeding, or total parenteral nutrition may be necessary to meet nutritional needs. Furthermore, patients frequently have anemia of chronic disease, which should also be addressed.

Spasticity is common in patients with spinal cord injuries. Medical treatment of muscular spasm should to be instituted early in management. Drugs such as baclofen, diazepam, and dantrolene have proven efficacy in controlling muscle spasms. Surgical treatment of spasm includes nerve blocks, epidural stimulators, rhizotomy, and administration of baclofen by continuous infusion pumps.

Long-standing contracture in patients with denervation is frequently encountered and should be addressed. A physical rehabilitation program is the first step in reversing the contracture. In patients who fail this approach, surgical release is indicated. Tenotomy, bony resection, or closed release is performed, depending on the severity. Without relief of the contracture, the pressure sore will invariably recur because the pressure pattern has not been changed.

Only after these goals have been sufficiently met can an open wound heal by secondary intention. After all necrotic material has been removed by débridement, diligent dressing changes can remove minimal residual necrotic tissue. Dakin's solution (short duration with gross contamination), normal saline, or other wound care agents may be used during dressing changes. Silver sulfadiazine (Silvadene) may be used after the wound is grossly clean because it further reduces the bacterial burden. Another modality that has provided successful treatment and closure of the wounds has been negative pressure dressing therapy. Application of the dressings, however, follows the same rules as that of any of the other type of dressings mentioned earlier, and frequent monitoring is required.

Most importantly, pressure on the skin can be reduced by regularly turning the patient in bed every 2 hours or by the use of air or pressure-reducing mattresses (Clinitron bed). If patients are in the sitting position for most of the day, their weight should be shifted several times every hour.

Surgical Treatment

Surgical treatment of pressure sores requires (1) excision of the ulcer, associated bursa, and necrotic tissue; (2) partial/complete ostectomy to reduce the bony prominence and remove infected bone; and (3) wound closure with healthy, well-vascularized tissue that is of adequate bulk to provide sufficient padding over the residual bony skeleton.

SOFT TISSUE DÉBRIDEMENT AND OSTECTOMY. Although some form of temporizing débridement may be accomplished at the bedside, it is preferable to débride pressure sores in the operating room because optimal anesthesia, patient positioning, and hemostasis can be achieved. The ulcer edges should be excised. Methylene blue can be used to stain the bursa to define its boundaries. After removing the bursa cavity in its entirety, a pulsed irrigation system can be used to reduce the bacterial load.

Removal of the bony prominence is an essential part of the surgical treatment. Infected bone, determined intraoperatively, should be removed from the ulcer base; excessively aggressive débridement of bone should be avoided because it can often lead to bleeding, structural support problems, creation of unnecessary dead space, and redistribution of

pressure to adjacent areas postoperatively. If one-stage reconstruction is not indicated, a sterile moist-to-dry dressing can be packed in the open wound at the conclusion of the operative débridement.

PRESSURE SORE CLOSURE. Selection of a local flap for closure of a pressure sore depends on the site of the sore. Various options exist for each type of pressure sore. The following sections outline the flap of choice for sacral, ischial, and trochanteric pressure sores. It is not a strict or comprehensive set of guidelines as many variables affect the decision-making process.

Sacrum. Sacral pressure sores occur most commonly in patients who are in the supine position. Although these wounds may be closed primarily (if small) or skin grafted, both of these approaches result in unacceptably high rates of recurrence. The gluteus maximus myocutaneous flap is the best option for closure of these wounds. It has the advantage of being a sensate flap with a type III blood supply that can survive with either one of its two dominant pedicles and thus provides versatile coverage in this area (Fig. 1).

Design of the flap should take into account the ulcer size, history of previous surgery, and the ambulatory status of the patient. It is prudent to elevate a larger flap (by several centimeters) than anticipated. The flap can be based on either the superior or inferior gluteal artery and on part of or the entire ipsilateral gluteus maximus. In select situations, bilateral gluteus maximus muscles can be used. The flap can be rotated, advanced, or turned over to fill the sacral defect. Sufficient muscle should be mobilized to allow tension-free closure. In general, it is best to preserve both vascular supplies to maximize the volume of tissue survival and to maintain options if a secondary readvancement procedure is required. Maintenance of both vascular supplies is usually possible, but not absolutely critical, and the muscle is generally able to be advanced to the periphery of the defect from the contralateral side.

Anatomic landmarks routinely marked before incision include the posterior superior iliac spine, ischium, and greater trochanter. The superior gluteal artery can usually be identified a third of the distance from the posterior iliac spine to the greater trochanter. The inferior gluteal artery can be found approximately halfway between the posterior iliac spine and the ischial tuberosity.

The gluteus maximus muscle is mobilized by dividing the superomedial portion of the muscle at its sacral origin and controlling the multiple vascular perforators at this level. The muscle is elevated in an avascular plane above the gluteus medius muscle and the dissection continued inferiorly to the level at which the superior gluteal vasculature exits above the piriformis muscle. This is also the point of rotation for the muscle. The insertion of the muscle to the iliotibial tract should be divided to permit tension-free rotation into the defect. The remainder of the muscle rotation technique, including additional dissection from its origin or insertion, should be tailored to the defect and extended as much as needed to cover the defect with muscle in a tension-free fashion. The inferior portion of the muscle can be elevated to allow for easier rotation. Care should be taken to maintain attachment of the overlying fasciocutaneous portion to the underlying muscle. In general, the inferior half of the gluteus maximus muscle can be left intact (the portion that sits inferior to the piriformis muscle and usually inferior to the ulcer itself); however, if needed, elevation of the muscle can also be incorporated within the rotation flap to cover large defects. The inferior gluteal vasculature can provide additional vascular supply to the flap and, in some cases, even the entire supply.

Identification of the piriformis muscle is critical during the dissection because it is the key landmark for the superior and inferior gluteal vessels, as well as the sciatic nerve. Although these structures do

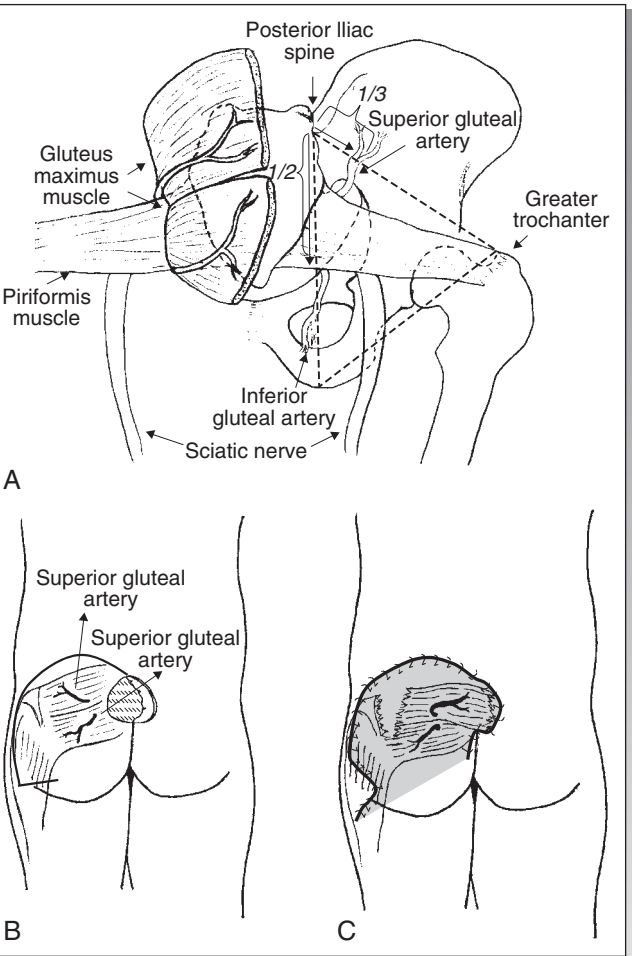

Figure 1 • A–C, The gluteus maximus myocutaneous flap (see text for details).

not need to be visualized after the piriformis is identified, the flap is mobilized appropriately to allow for proper rotation to fill the base of the defect with muscle. A back-cut of the same dimension as the width of the defect is generally necessary to make rotation easier and permit tension-free cutaneous closure. Division of both the origin and insertion of the gluteus maximus should be avoided in ambulatory patients because of the potential for hip instability.

Ischium. The most common location for pressure sores in paraplegic patients is the ischium. When débriding the ulcer, it is important to avoid overly aggressive resection of the ischial tuberosity because such resection will increase pressure on the contralateral tuberosity; bilateral débridement will shift pressure to the perineum. These pitfalls contribute to the high rate of recurrence after treatment of ischial pressure sores. The gluteal thigh fasciocutaneous flap is the first choice for reconstruction because it leaves the gluteus maximus for possible future use and provides a large volume of tissue for wound closure.

The posterior thigh flap (or gluteal thigh rotation flap) is a hearty fasciocutaneous flap (Fig. 2). It provides excellent sensation over critical areas with minimal donor site morbidity. The flap is based on the inferior gluteal artery, with the central axis of the flap located between the ischial tuberosity and the greater trochanter. To allow the donor site to be closed primarily, flap width should be less than 10 to 12 cm. Its length may be up to 32 to 34 cm. The descending branch of the inferior gluteal artery allows the flap to be extended inferiorly toward the popliteal fossa; the entire posterior aspect of the thigh may be raised from the iliotibial tract to the intermuscular septum. The full range of flap

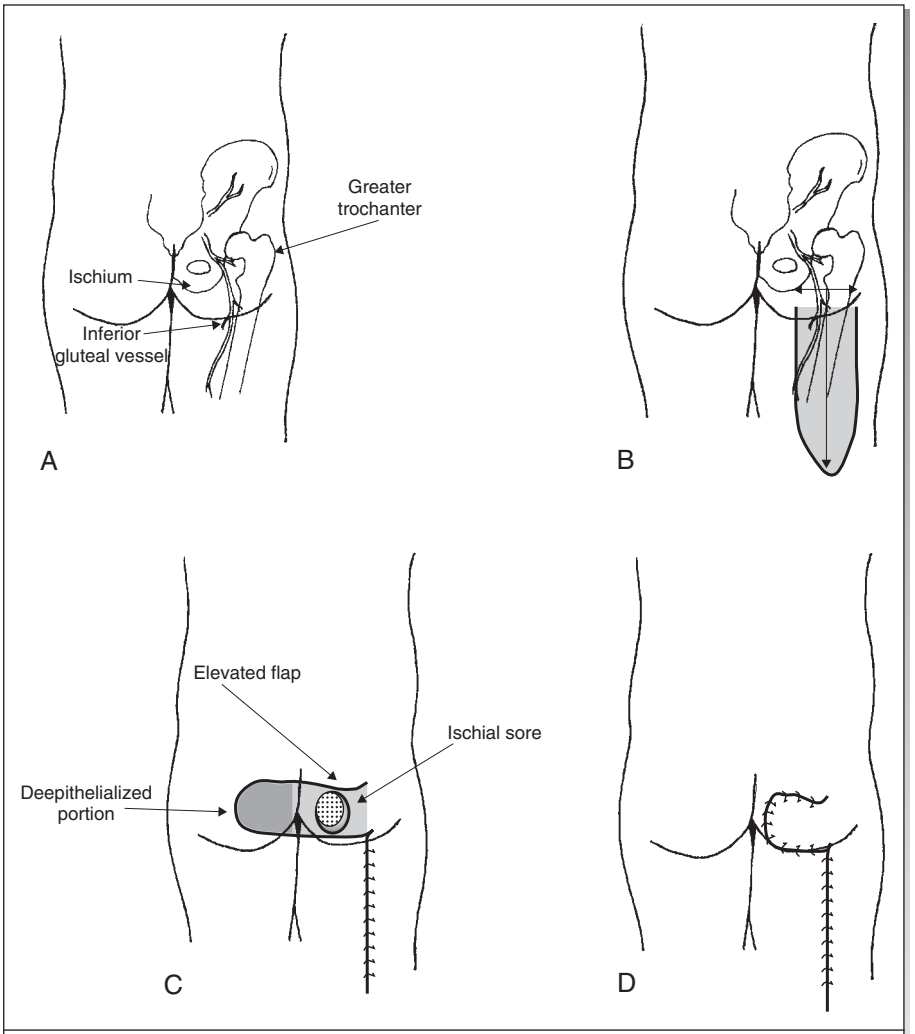

Figure 2 • A–D, The posterior thigh flap or gluteal thigh rotation flap.

mobility includes the sacrum and anterior superior iliac spine superiorly, the pubis, and the greater trochanter.

The plane for elevation of the flap is entered by identifying the posterior femoral cutaneous nerve and the inferior gluteal vessels. Dissection continues proximally until the inferior border of the gluteus maximus muscle is reached, at which point its attachments to the iliotibial tract, intermuscular septum, and femur are divided. After mobilization, the flap can be rotated to fill the defect or used as an island flap. Minimal treatment of the expected dog-ear is performed so that the vascular supply is not compromised. Skin grafts can be used to close the donor site if it cannot be closed primarily. For ischial wounds, the distal portion of the flap can be deepithelialized and buried beyond the defect to prevent marginal breakdown.

Trochanter. Trochanteric pressure sores are less common and generally have minimal skin involvement but extensive bursa formation. Because of the mobility of the greater trochanter and femur, extensive undermining of the soft tissues occurs. Complete excision of the greater trochanter (Girdlestone procedure) is usually necessary to prevent recurrence.

The tensor fascia lata (TFL) is the flap of choice for reconstructing trochanteric defects (Fig. 3). The gluteal thigh fasciocutaneous flap can also be used for smaller defects. The TFL flap is a myocutaneous flap based on the lateral femoral vascular pedicle (type I flap). The muscle originates on the anterior superior iliac spine and inserts onto the iliotibial tract; it measures 10 to 12 cm long, 2 to 3 cm wide, and 1 to 2 cm thick. The skin paddle that is harvested with the flap is 10 cm wide and may be up to 30 cm in length. The inferior limit of the skin flap is approximately 6 cm above the knee. The TFL flap can be raised as a sensate flap because it receives sensory innervation from the second and third lumbar nerves via the lateral femoral cutaneous nerve, as well as a lateral branch of T12.

The lateral femoral circumflex artery is located approximately 7 to 9 cm below the anterior superior iliac spine. The anterior border of the flap is marked by a line connecting the anterior superior iliac spine to the lateral condyle of the tibia. The greater trochanter of the femur is generally the posterior landmark. The subsequent posterior arc of rotation allows for reliable coverage of trochanteric as well as ischial pressure sores.

Flap dissection is carried out after the skin paddle is designed to reach and cover the defect. The flap is divided distally (through the thin fascia lata). It is elevated in a distal-to-proximal direction deep to the fascia lata (which overlies the vastus lateralis). Enough of the flap is elevated in this plane to cover the defect without tension. Identification and visualization of the vasculature are not necessary, and elevation of the flap should be tailored to limit vascular exposure and maximize coverage of the defect.

Modifications of the TFL as a V-Y flap, a bipedicle flap, and a sensory flap have been described. The advancement flap variations do not provide the large amount of bulk that is sometimes required to fill defects. In addition, the vastus lateralis (noted earlier) may be used in conjunction with the TFL flap when the soft tissue defect is considerable. The TFL flap may also be used in the treatment of ischial sores (see Fig. 3C and D).

Postoperative Care

Postoperative care is important for successful surgical treatment of pressure sores. There should be nearly total relief of pressure at the surgical site. Appropriate positioning in a cushioned bed (air mattress) is essential, as is avoidance of shearing forces during patient repositioning. Patients are maintained on bedrest for 2 to 3 weeks postoperatively, after which physical therapy with range-of-motion exercises is initiated. Fecal and urinary diversion is necessary to prevent infection in the postoperative period. This can be accomplished with rectal bags and condom catheters. Surgical diversion is rarely required.

Nutritional support continues, and drains are kept in place for an extended period to reduce the incidence of seroma and hematoma formation and to eliminate dead space, often as long as the patient is kept on bedrest. The initial postoperative dressing is maintained for the first 3 to 5 days. In cases in which fecal contamination is likely, a nonpermeable dressing is preferred. This barrier must be maintained until the wound margins are appropriately sealed/epithelialized to avoid bacterial contamination, which may cause wound dehiscence, infection, or flap loss.

Pearls and Pitfalls

- There is no life-threatening condition associated with a clean, open pressure sore. Patients who are poor candidates should not undergo flap procedures because the chance for long-term success is minimal.
- Flap coverage should be performed only on fully débrided wounds. Single-stage “débridement and coverage” procedures should be avoided.
- Postoperative pressure management is critical to prevent flap loss or recurrence as a result of continued pressure-induced ischemia.

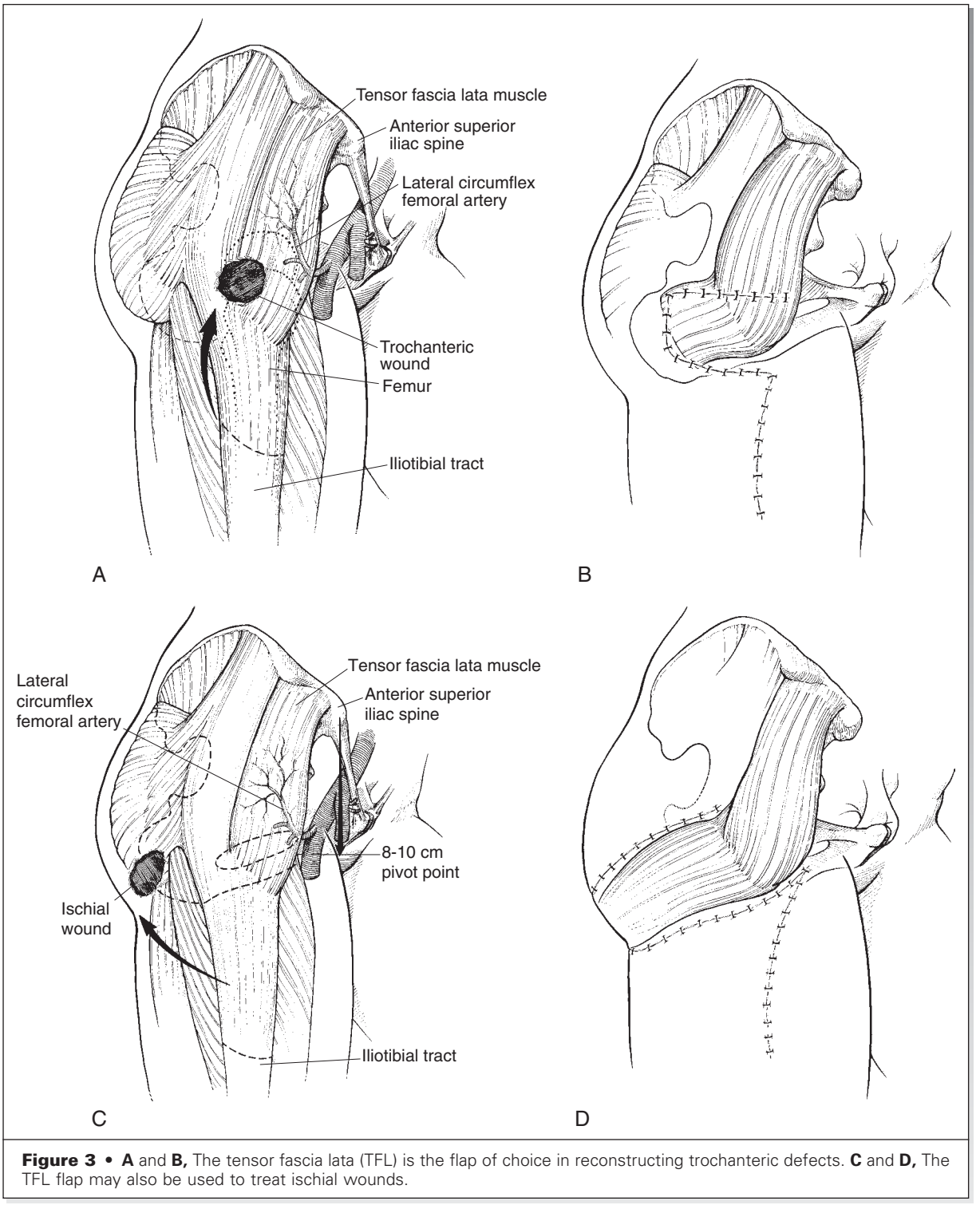

Figure 3 • A and **B,** The tensor fascia lata (TFL) is the flap of choice in reconstructing trochanteric defects. **C** and **D,** The TFL flap may also be used to treat ischial wounds.

SUGGESTED READINGS

Bruck JC, Buttemeyer R, Grabosch A, Gruhl L: More arguments in favor of myocutaneous flaps for the treatment of pelvic pressure sores. Ann Plast Surg 26:85-88, 1991.

Daniel RK, Faibisoff B: Muscle coverage of pressure points—the role of myocutaneous flaps. Ann Plast Surg 8:446-452, 1982.

Gusenoff JA, Redett RJ, Nahabedian MY: Outcomes for surgical coverage of pressure sores in nonambulatory, nonparaplegic, elderly patients. Ann Plast Surg 48:633-640, 2002.

Mancoll J, Phillips L: Pressure sores. In Achauer BM, Eriksson E, Vander Kolk C, et al (eds): Plastic Surgery: Indications, Operations, and Outcomes. St. Louis, CV Mosby, 2000, pp 447-462.

Nahai F: The tensor fascia lata musculocutaneous flap. Ann Plast Surg 6:788, 1979.

Breast Augmentation

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Over the past 10 years, following the Food and Drug Administration's restrictions on the use of silicone gel–filled breast implants, there have been improvements in breast implant design. Low-bleed silicone elastomer shells, implant shell surface texturing, and more durable, thicker materials have led to devices with a lower risk of capsular contracture and probably greater longevity. These developments have allowed a shift in the focus of breast augmentation away from avoidance of capsular contracture toward improvements in surgical technique and aesthetic outcome. Total muscle coverage had been recommended to reduce implant palpability and decrease the incidence of capsular contracture. However, such coverage was accomplished at the expense of optimal lower pole shape and inframammary fold definition. As a result, either late superior migration of the implants or pseudoptosis developed in a significant number of patients because of the gravitational effects on the breast tissue, in contrast to an implant that remains in a more cephalic position because of support from the inferior muscle attachments. The development of newer devices has permitted use of the partly subpectoral plane or "dual-plane" approach, which has become popular and has proved to provide a low rate of contracture, less implant distortion with muscle activity, satisfactory mammographic evaluation, and a more attractive inferior breast contour.

The choice of access incision is an additional decision. The periareolar approach has certain advantages over the inframammary, transaxillary, and transumbilical methods. It provides a central point of access to both the subglandular and subpectoral planes, as well as all quadrants of the breast. It allows for a variety of different types of mastopexy either simultaneously or later; in addition, it permits manipulation of breast parenchyma, such as radial incisions that may be required for a tuberous breast deformity. Various implant sizes, shapes, and types work well with the periareolar approach, even with the smallest areola. The periareolar approach is particularly helpful in patients with a high inframammary fold in whom lowering of the fold makes positioning of an inframammary incision imprecise because of the slight variability in the location of the new fold. Finally, the resultant scar with a periareolar incision is usually inconspicuous.

Etiopathogenesis

Patients seek breast augmentation for various pathologies. Many have minimal breast tissue because of simple hypoplastic development. Other patients have postpartum atrophy as a result of involutional changes in the breast after pregnancy or nursing, and these women often require an increase in volume as well as correction of ptosis. Some patients have congenital deformities and asymmetries characterized by tuberous breast deformity, Poland's syndrome, or chest wall abnormalities such as pectus excavatum or carinatum.

Pathologic Anatomy

A comprehensive preoperative evaluation is important to obtain satisfactory results. Breast size is commonly graded by bra and cup size, which is however highly variable from manufacturer to manufacturer. In addition, the individual patient's perception is a critical factor. An approximately 100-cc increase in volume is equivalent to an increase of one cup size.

Dimensional planning is helpful even when using round implants. Critical measurements include the nipple-to-fold distance, base width of the existing

breast, intermammary distance, desired breast width, desired intermammary distance, breast soft tissue envelope compliance, upper and lower breast soft tissue thickness, nipple ptosis, and amount of glandular ptosis below the inframammary fold. Patients with up to a grade I ptosis can usually be augmented with acceptable aesthetic results by placement of an implant alone. However, with greater degrees of nipple and glandular ptosis, it is less likely that an implant alone will be satisfactory.

Although not covered in this chapter, breast asymmetry and chest wall deformity (noted earlier) can be approached by choosing from the vast array of implant sizes and shapes either to increase tissue volume or to mask a contour deformity.

Diagnostic Studies

A preoperative mammogram is an appropriate study in accordance with the clinical guidelines of breast cancer screening established by the American Cancer Society. A follow-up mammogram approximately 6 months after surgery is advisable to obtain a new baseline.

Goals of Reconstruction

The goal of breast augmentation is to achieve an improved, fuller breast contour while producing a more harmonious balance between the breast, chest wall, and overall stature of the patient.

Treatment

The patient is marked in the preoperative holding area in the sitting or standing position with the arms symmetrically resting on the thighs or at the sides. The chest midline and inframammary folds are marked. Additional markings are made for any alterations to lower the fold or correct an asymmetry. The procedure can be performed under local anesthesia with sedation (with or without intercostal blocks) or under general anesthesia, depending on patient preference. The patient is positioned in the akimbo position with special attention paid to proper padding of the elbows, wrists, and intravenous sites.

In the operating room, before infiltration of local anesthetic solution, the periareolar incision is marked precisely at the inferior areolar/mammary skin border centered at the 6-o'clock position. If the incision is not made perfectly at the areola-skin junction, visibility of the scar is dramatically increased. The skin incision is made through the dermis with a scalpel, and electrocautery is used to incise the underlying tissue (Fig. 1). The dissection continues in a gland-splitting plane with beveling away from the nipple down to the underlying pectoralis muscle fascia. This technique leaves adequate breast tissue superiorly and inferiorly to facilitate closure. When the pectoralis fascia is reached, the dissection proceeds in a subglandular plane downward to the inframammary fold and upward to the level of the inferior border of the areola. In patients with more breast soft tissue leading to ptosis, the subglandular dissection may continue as high as the level of the superior border of the areola. If minimal ptosis is present, as in a juvenile breast, the subglandular dissection can be less extensive and consists of only the lower few centimeters. The dissection creates a standard subglandular pocket inferiorly while exposing the inferior edge of the pectoralis major muscle (Fig. 2). Allis clamps are used to retract the muscle, which is then dissected along its inferior border (Fig. 3). After the loose areolar plane deep to the muscle is entered, the superior pocket is created by using a combination of mostly cautery and minimal blunt dissection with urethral dilators under the pectoralis major muscle and above the pectoralis minor and serratus muscles (Fig. 4). The lateral and superior portions of the dissection should be precise and proportional to the size of the implant to avoid lateral migration of the latter. The lateral

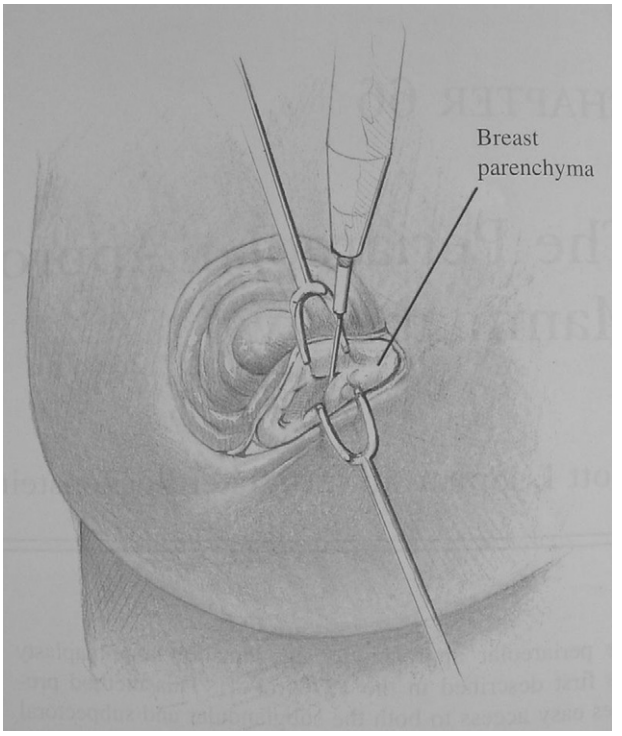


Figure 1 • After the skin incision, dissection proceeds with electrocautery in a breast-splitting plane. Careful attention is directed to preservation of adequate tissue superiorly and inferiorly.

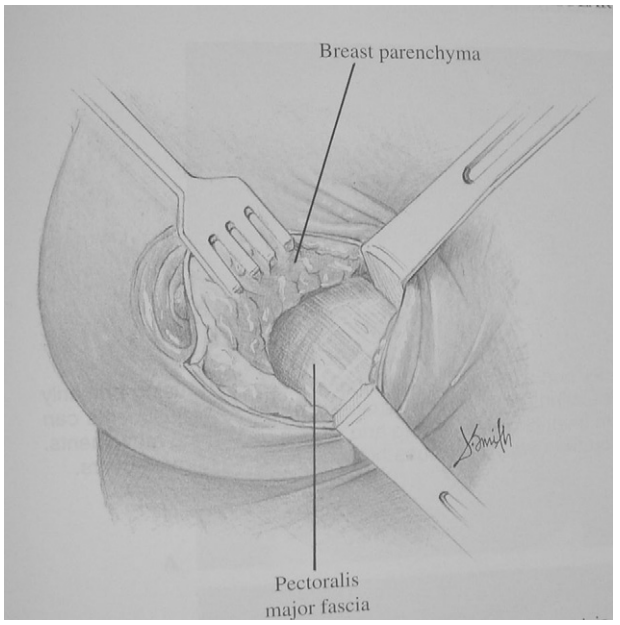


Figure 2 • After the pectoralis fascia is reached and the inferior border of the pectoralis muscle identified, subglandular dissection is undertaken inferiorly. Depending on the amount of ptosis and preexisting breast tissue, the subglandular dissection is continued superiorly over the free border of the pectoralis major muscle, either a few centimeters or occasionally as far as the superior border of the areola in more ptotic patients.

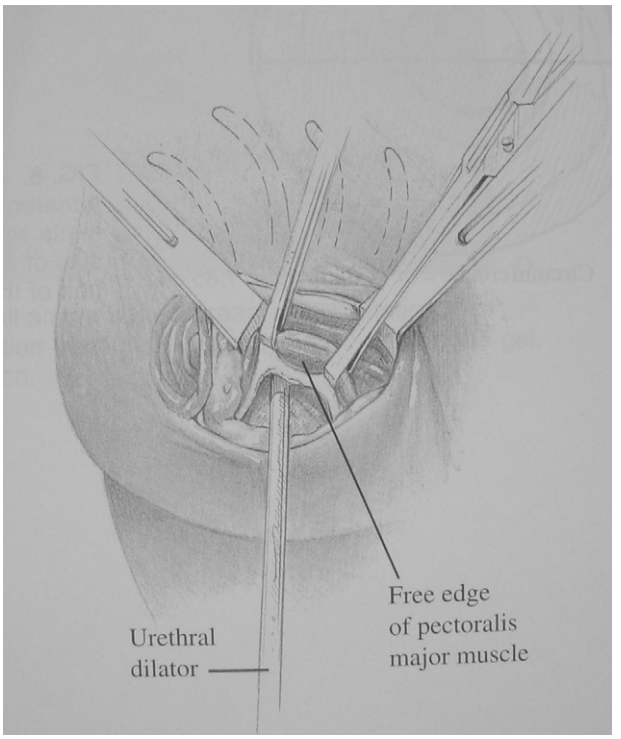


Figure 4 • The most superior portion of the subpectoral dissection can be completed by passing a blunt urethral dilator through the areolar tissue.

and superior portions are also the areas most likely to encounter bleeding and are most at risk for nerve injury. The medial inferior edge of the pectoralis muscle origin should be released to avoid a step off deformity between the subglandular and subpectoral pockets (Fig. 5). The pockets are irrigated with antibiotic solution and hemostasis is achieved. The "dual-plane" pocket allows for implant placement

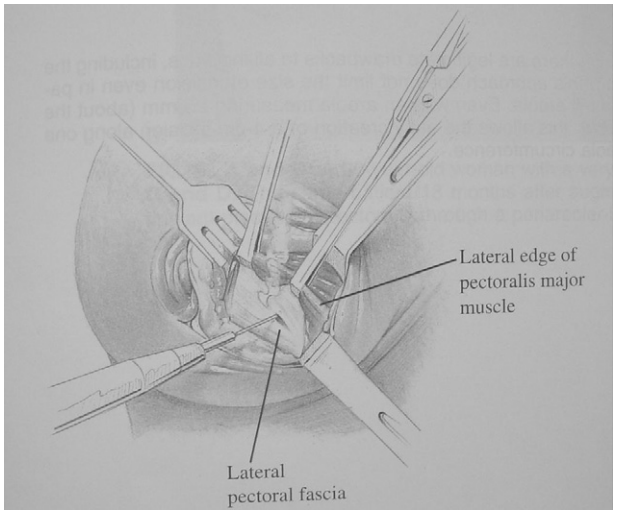


Figure 3 • The inferior border of the pectoralis major is grasped with Allis clamps and elevated. The muscle is incised in the direction of its fibers to allow access into the areolar plane. The dissection is continued subpectorally.

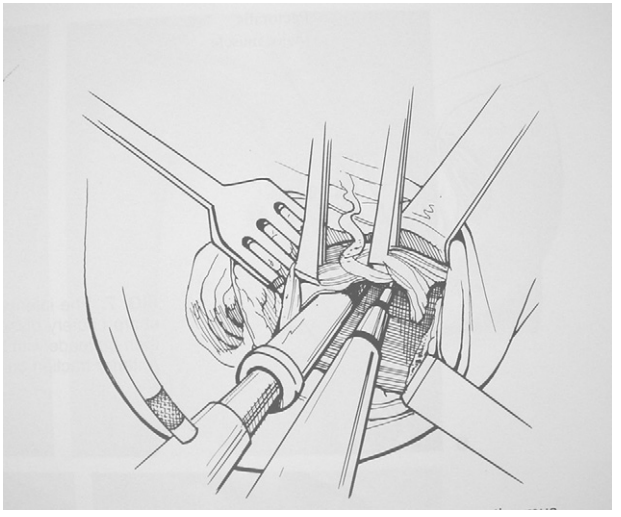


Figure 5 • The medial inferior attachments of the pectoralis major are released to prevent a step off deformity between the subpectoral and subglandular dissections.

and soft tissue redraping to create a more aesthetic lower pole contour and to avoid a "double-bubble" deformity.

The implant is placed in the pocket and, in the case of saline devices, filled to the appropriate volume. The patient is seated upright on the operating room table and the breasts are assessed for symmetry with respect to size, contour, position of the implant, inframammary fold, and nipple height. If asymmetry is observed, the implants can be left in position and a finger or a blunt urethral dilator used to make pocket and fold adjustments.

Closure begins with three or more interrupted sutures to reapproximate the split breast tissue over the lower pole of the implant. The skin edges are scrutinized and, if significantly traumatized, are trimmed. Although up to 2 or 3 mm of breast skin is routinely resected, the areola is always conserved. The skin is approximated with interrupted intradermal sutures, followed by running intradermal closure.

Postoperative Care

After completion of the procedure, the patient is placed in a bra. In patients receiving shaped implants, a small binder is placed across the superior pole of the breasts to discourage implant rotation. Bras and binders are worn continuously for 2 weeks. Strenuous activity is discouraged for 2 weeks; however, most patients return to normal activities in a day or two. During the first postoperative visit at 7 days, patients are instructed in massage techniques to help prevent capsule formation. Gentle massage is prescribed for shaped, textured implants and more vigorous massage with displacement exercises for round, smooth implants.

Pearls and Pitfalls

- The periareolar approach to "dual-plane" augmentation produces an attractive breast shape in both the short and long term.
- The most significant drawback of dual-plane augmentation is that it leaves less soft tissue coverage along the inferior pole of the implant, thus increasing the likelihood of lower pole visibility, palpability, rippling, and bottoming out, particularly with saline implants.
- Patients must be warned that the periareolar approach may have an increased risk of loss of nipple sensibility and lactation ability.
- Avoid excessive superolateral dissection.

SUGGESTED READINGS

Hurst NM: Lactation after augmentation mammoplasty. Obstet Gynecol 87:30, 1996.

Neifert M, DeMarzo S, Seacat J, et al: The influence of breast surgery, breast appearance, and pregnancy-induced breast changes on lactation sufficiency as measured by infant weight gain. Birth 17:31, 1990.

Regnault P, Baker TJ, Geason MC, et al: Clinical trial and evaluation of a proposed new inflatable mammary prosthesis. Plast Reconstr Surg 50:220, 1972.

Spear SL, Beckenstein M: The periareolar approach to augmentation mammaplasty. In Spear SL (ed): Surgery of the Breast: Principles and Art. Philadelphia, Lippincott-Raven, 1998.

Spear SL, Bulan EJ: The medial periareolar approach to submuscular augmentation mammaplasty under local anesthesia: A 10-year follow-up. Plast Reconstr Surg 108:771, 2001.

Spear SL, Giese SY: Simultaneous breast augmentation and mastopexy. Aesthetic Surg J 20:155, 2000.

Tebbetts JB: Dual plane breast augmentation: Optimizing implant–soft tissue relationships in a wide range of breast types. Plast Reconstr Surg 107:255, 2001.

Management of Breast Implant Complications

DAVID T. NETSCHER

Women may seek assistance from plastic surgeons for a variety of concerns about their breast implants. With the large number of breast implants, it is expected that some patients will have problems that require implant removal, whereas others may simply request removal out of their own concerns or desires. Although certain risks are associated with breast implants, one can conclude, after a number of studies, that breast implants do not cause breast cancer or collagen vascular disorders and that rupture of silicone gel implants does not lead to any systemic consequences. Nonetheless, severe capsular contracture can cause breast and shoulder pain, sleepless nights, and a myofascial type of traction pain around the shoulder girdle. Periprosthetic infections are rare, but are more commonly of the acute pyogenic type. However, more indolent infections can be caused by fungi and atypical mycobacteria. Infections can cause both systemic and local symptoms. In addition, breast implant shadows may conceal breast cancer despite special mammographic views (i.e., Eklund views).

The purpose of this chapter is to review the management of local breast implant complications, including periprosthetic infections, implant rupture, and capsular contracture. In general, these complications are treated by implant removal and, occasionally, reconstruction (either with another implant or with autogenous tissue). Reconstruction can be performed immediately or in delayed fashion; alternatively, it can consist of mastopexy alone. This chapter presents a rational approach to best manage the patients appropriately.

Etiopathogenesis

Implant Rupture

The incidence of *saline-filled* implant rupture has not been fully evaluated, whereas that of *silicone gel–filled* implant rupture appears to be related to the duration of implant placement. The overall rupture rate of silicone gel implants is approximately 30%, and there is a clear correlation with duration after implantation. The median life span of silicone gel implants from a meta-analysis of more than 1099 implants in several studies was 16.4 years. Of these implants, 79% were intact after 10 years, but only 49% were intact by 15 years (Fig. 1). Neither anatomic placement of breast implants (prepectoral or subpectoral) nor the reason for implantation (reconstruction or cosmetic augmentation) appear to be related to implant rupture. There is a significant negative correlation between the duration of implantation and shell strength, toughness, and elasticity. This association suggests that exposure to the in vivo environment weakens the silicone breast shells over time.

Capsular Contracture

The incidence of symptomatic capsular contracture (Baker grades 3 and 4; Table 1) has been reported to be between 0% and 50% (Fig. 2). Contracture rates for breast implants vary according to implant type: the old sponge types, 100%; gel-filled with a patch, 70%; patchless gel filled, 55%; and saline inflatable, 35%. Saline-filled implants appear to reduce the incidence of contracture in almost every reported study.

The incidence of grade 3 or 4 capsular contracture is approximately 75% for single-lumen silicone gel implants, 12% for double-lumen gel implants, 4% for polyurethane implants, and 5% for saline-filled implants. The incidence of capsular contracture rises significantly with time. There may be an initial rapid rise in the contracture rate (to approximately 30%) in the early years after implantation, followed by a more gradual increase. A limitation of most studies is that interpretation of data is made difficult because of changes in implant design over time.

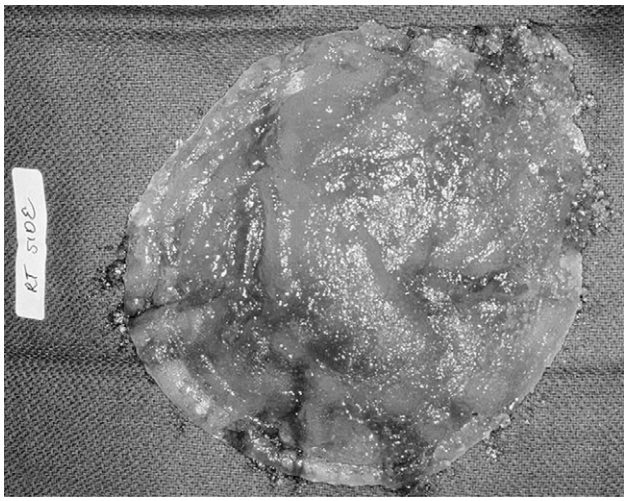


Figure 1 • Ruptured silicone gel implant.

Another factor influencing capsular contracture is the anatomic placement of the implant. A prosthesis placed in a subcutaneous pocket after subcutaneous mastectomy has a significantly higher incidence of capsular contracture than an implant placed subpectorally. Many believe that subpectoral implant placement reduces the incidence of capsular contracture in cosmetic augmentation, but this finding is not borne out by all studies. Subclinical bacterial colonization of mammary implants appears to be etiologically related to the development of capsular contracture. In vitro studies and experimental animal studies support the use of antiseptic irrigation of the breast pocket or impregnation of devices with antibiotics. Whether these measures interfere with the mechanical integrity of breast implants has not been fully investigated.

TABLE 1 Baker Classification of Breast Capsular Contraction

Grade I: No palpable capsule. (The augmented breast feels as soft as a breast that has not been augmented.)
Grade II: Minimal firmness. (The breast is less soft, and the implant can be palpated but is not visible.)
Grade III: Moderate firmness. (The breast is harder, and the implant can be palpated easily and is visible.)
Grade IV: Severe contracture. (The breast is hard, tender.)

Periprosthetic Infection

Major infections of saline-filled tissue expanders occur in up to 10% of cases. However, the incidence of overt infection with mammary implants is only 1.5%, although subclinical periprosthetic positive culture rates from breast implants are as high as 23.5%. The positive cultures correlate statistically with symptomatic capsular contracture, but not with implant rupture or with implant placement (prepectoral or subpectoral). If clinical infection of one mammary implant necessitates removal, the contralateral mammary implant may have to be removed as well for aesthetic reasons.

It is interesting to note that when an overtly infected implant (generally with *Staphylococcus aureus*) is salvaged and retained by the aggressive use of antibiotics, subsequent capsular contracture frequently occurs. Coagulase-negative staphylococci (which colonize breast ducts) have been associated with a symptom complex of myalgia and arthralgia in the absence of overt breast infection. A problem particularly related to the pathogenicity of coagulase-negative staphylococci is their relative protection in

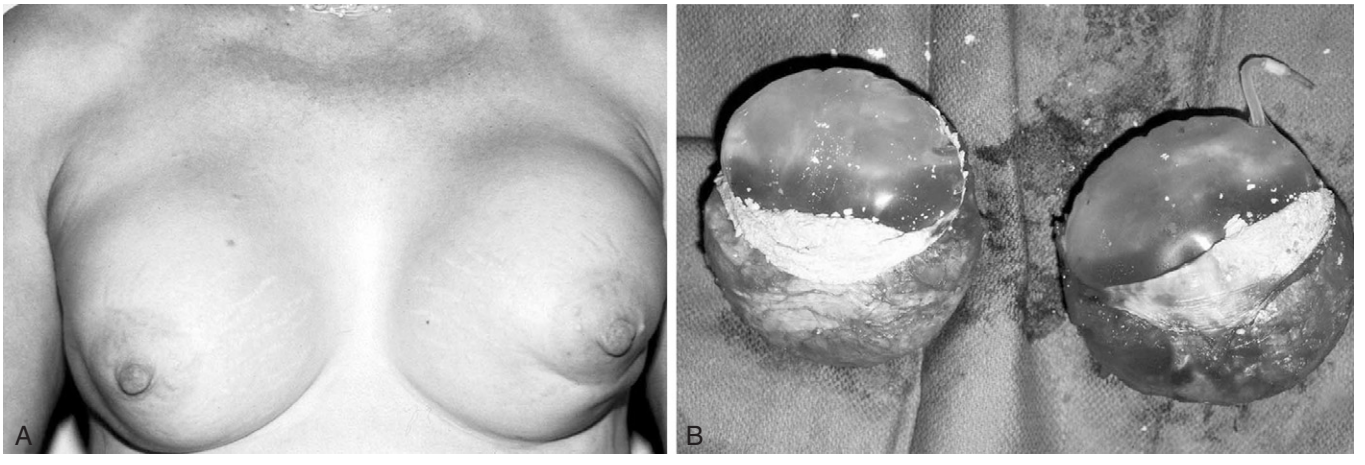


Figure 2 • **A,** Patient with the characteristic deformity secondary to severe capsular contracture. **B,** The contracted capsules around the implants. Note the calcification.

a biofilm layer. Bacteria may adhere to silicone and produce extracellular polysaccharides and glycoprotein that form a "slime" layer. Encased in this biofilm, they remain in a dormant, viable state, but they do not multiply. In addition, fungi (particularly *Aspergillus*) have been reported as colonizers of silicone saline-filled breast implants in the absence of overt external clinical breast infection.

Pathologic Anatomy

Saline-filled breast implant rupture leads to implant deflation with a decrease in breast size/volume on the affected side. The deflated shell may occasionally be palpated as a mass. The deflation may occur secondary to leakage at a fill valve or seam, or it may be due to a true fold flaw and tear in the silicone shell. An external force of sufficient magnitude may directly rupture an implant. In contrast, most silicone gel implant ruptures remain intracapsular and are more difficult to diagnose clinically. Extracapsular rupture may result in a granuloma and can be confused with a breast tumor.

Periprosthetic capsular contracture causes breast deformity in its more severe stages. There is superior displacement of the implant and rounding of the superior pole with asymmetry and irregularity.

When breast implants are removed in a patient with a previous cosmetic augmentation, changes occur in both breast volume and shape. Surgical treatment options are based on the patient's needs and the surgeon's advice. In many patients, implant removal results in an exaggerated degree of ptosis, loss of upper pole cleavage, diminished breast volume relative to chest stature, and breast asymmetry. It is difficult to predict which patients will return most to their preaugmentation state and which patients will have the aforementioned deformities after implant removal and will require reconstruction (Fig. 3).

In patients requiring implant extirpation, it is probably more prudent for both technical and economic reasons to minimize deformities at the time of implant removal than to attempt to correct them at a later date. For example, a patient with a previous subcutaneous mastectomy who has implants removed for painful capsular contractures is going to experience progressive contracture of the skin envelope after implant removal. Reconstruction thus becomes more complex and may require preexpansion if the reconstruction is performed in a delayed manner. A more favorable result is achieved, however, if the existing skin envelope is used immediately after implant removal.

In a patient with a previous cosmetic augmentation who is unhappy with her breast shape and form after the implants are removed, the problem may be a financial rather than a technical one. Often, the aesthetic problem can be readily solved by simple augmentation with a saline implant, but the patient may now be unable to afford a second operation. Although financial concerns should not cloud surgical judgment, whenever surgically prudent it is better to simultaneously combine removal of implants with some type of reconstruction such as mastopexy or augmentation. In a patient who is initially opposed to implant replacement or in whom it is discovered intraoperatively that mastopexy is not possible, it might be prudent to defer surgery and let the patient evaluate her appearance in order to make a decision jointly with the plastic surgeon about the need for further management.

Diagnostic Studies and Preoperative Evaluation

The clinical examination must highlight each of the following to determine whether mastopexy or a replacement implant is needed and to evaluate potential outcomes:

1. *Estimation of the current volume of breast tissue.* The change in bra cup size, records of the implant size, and previous photographs are helpful to determine how much breast tissue will remain after an implant is removed. Mammography may also help to determine the volume of breast tissue relative to implant size.
2. *Skin elasticity.* If breast skin tone is elastic, it is more likely that implant removal alone will mimic the preaugmentation state. The patient's age, presence of striae, number of pregnancies, and degree of existing breast ptosis are helpful in evaluating the potential appearance after breast implant removal.
3. *Breast asymmetry.* Removal of breast implants may unmask previously existing breast asymmetry. Patients may report a significant preexisting breast asymmetry that may indeed have been the original indication for breast augmentation. Breast asymmetry may have been present before breast augmentation, or it may have been acquired as a result of multiple breast biopsies, infections, ruptured gel implants, or multiple capsulectomies. In women with true extracapsular implant rupture resulting in extrusion of silicone into the breast tissue with granuloma formation, removal may result in loss of breast tissue as well.
4. *Patient's view of breast aesthetics.* The patient may now have personal desires that are very different from those when she first sought breast augmentation. Acceptance of additional scars (especially those of mastopexy) and willingness to receive a new implant must therefore be evaluated.

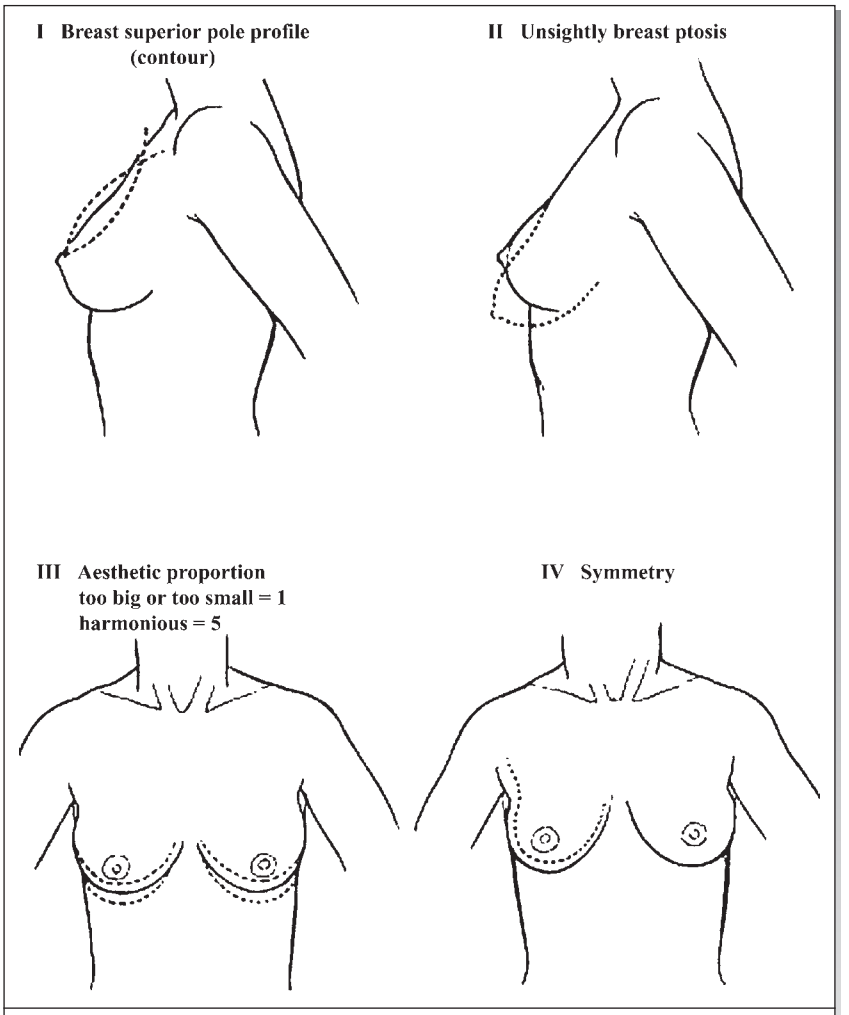


Figure 3 • The undesirable effects of implant removal in patients who had undergone breast augmentation include loss of upper pole fullness with a caved-in appearance, exaggerated ptosis, diminished breast volume relative to torso dimensions, and unmasking of the original breast asymmetry. Dotted line indicates breast contour after implant removal.

5 *The role of breast-imaging techniques.* Breast imaging (mammography, magnetic resonance imaging [MRI], and ultrasonography) does not play a role in the preoperative evaluation of a patient who is going to have an implant removed. When a screening mammogram is indicated to evaluate breast parenchymal disease, it should be performed preoperatively. It may be helpful in giving an idea of the volume of breast tissue relative to implant size (Fig. 4). Mammography may occasionally detect extracapsular silicone gel implant rupture (Fig. 5). In cases in which rupture is uncertain, MRI remains the most accurate diagnostic tool.

Goals of Reconstruction

The goals of reconstruction are as follows:

1 Minimization of breast contour deformity
2 Restoration of breast volume, shape, and aesthetic appearance
3 Creation of skin flaps to accommodate the appropriate breast volume
4 Resolution of infection
5 Minimization of external scars
6 Provision of emotional support to patients

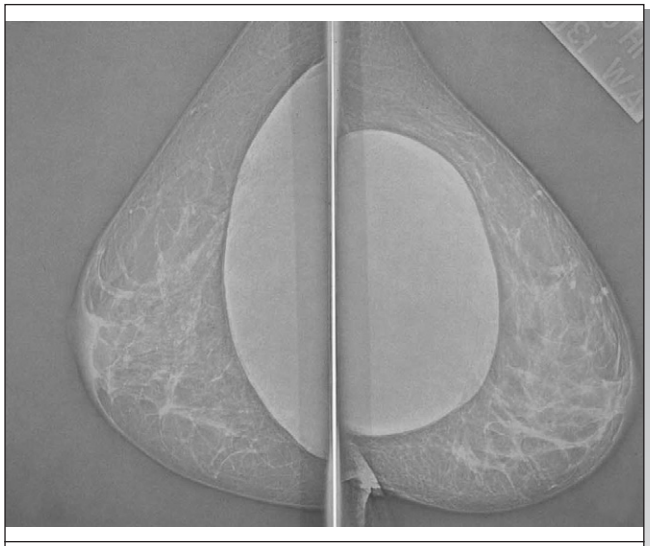


Figure 4 • Mammogram taken before implant removal shows a large volume of breast parenchyma relative to implant size.

Surgical options after a decision has been made to remove a breast implant are as follows:

1. Implant removal alone
2. Implant removal with mastopexy
3. Removal or replacement with an implant (usually saline)
4. Removal or reconstruction with autogenous tissue

Treatment

Management of Patients Previously Reconstructed with Implants

If reconstruction is not performed in a patient who has previously undergone mastectomy and has had the implants removed, the only option available to the patient is to wear a prosthetic bra.

For a patient who has previously undergone subcutaneous mastectomy and has a noncontracted skin envelope with a well-defined inframammary crease, implant removal with simultaneous implant replacement is an ideal treatment. If body habitus allows, autogenous tissue reconstruction using a deepithelialized flap is also an excellent option in a patient who has had a previous subcutaneous mastectomy and implant reconstruction. Because the skin envelope and inframammary crease are already formed, little additional breast contouring is required. In many patients, the volume of the hemi-TRAM (transverse rectus abdominis myocutaneous) flap equals the volume of the implant removed and can therefore be used for reconstruction. If there is significant adipose tissue in the back and flank region, deepithelialized extended latissimus dorsi flaps also provide satisfactory results, particularly in patients with previous abdominal surgical procedures. With autogenous tissue, one is frequently limited in volume for bilateral reconstructions, and occasionally, two different flaps are required. Microvascular free tissue transfer may also have a role in reconstruction of these patients.

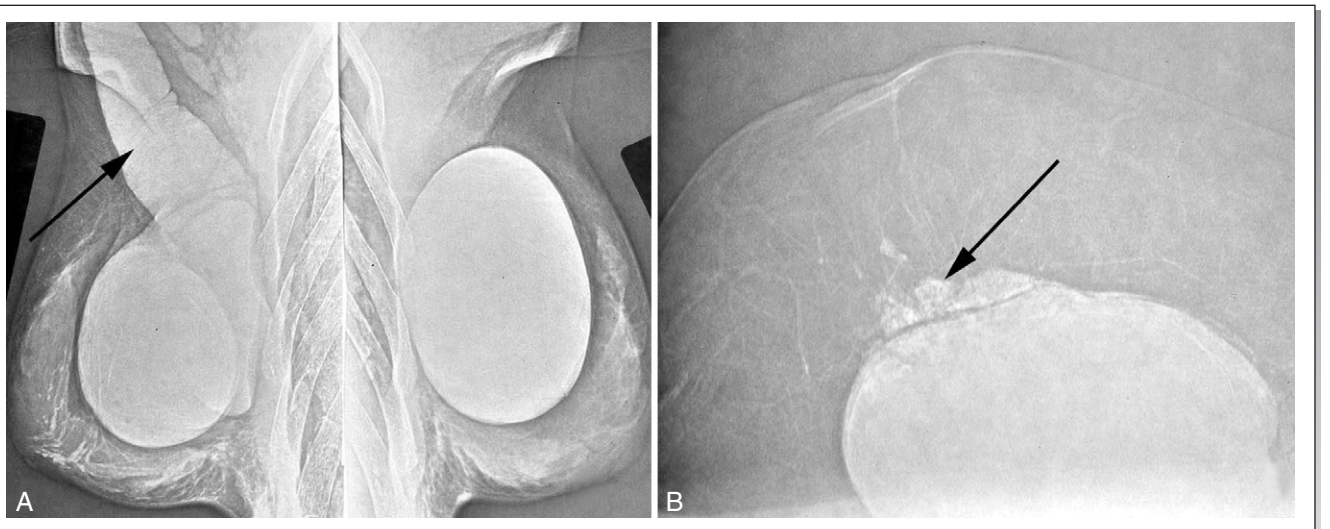


Figure 5 • A, Mammogram showing extrusion of silicone gel (*arrow*) along the intermuscular tissue planes up to the axilla. **B,** A patient with a clinically palpable breast mass has a silicone granuloma (*arrow*), seen mammographically, as a result of extracapsular gel implant rupture.

Management of Patients Who Have Previously Undergone Cosmetic Breast Augmentation

Autogenous tissue reconstruction after implantation in a previously augmented patient for cosmetic reasons is often impractical because of the expense and extent of the surgery involved. However, bilateral TRAM flaps are occasionally required in patients who have not had a previous mastectomy, as well as in patients in whom the original augmentation was performed for significant chest wall asymmetry, such as Poland's syndrome.

In reality, however, most patients who have previously undergone cosmetic augmentation have three choices if the breast implants are to be removed: implant removal alone, implant removal with mastopexy, or implant removal with saline implant replacement. If the patient is to be reaugmented and the breast is soft, an implant of similar size to that previously used is appropriate. However, if the patient has a severe Baker 3 or 4 capsular contracture associated with skin striae and ptosis, a new implant should preferably be of larger size and should be placed prepectorally.

Older, thin patients who have breast striae are more likely to have adverse aesthetic outcomes when implants are removed without additional surgery. Thus, simultaneous mastopexy is advised. Even in slender patients with a small amount of breast tissue and inelastic skin, a pleasing breast contour can be achieved with mastopexy despite loss of breast volume. In contrast, in a full-framed, full-breasted patient who has previously undergone only modest augmentation, who might have gained weight since the initial surgery, and who may perhaps have only a mild degree of capsular contracture with minimal ptosis, one can expect a satisfactory aesthetic outcome after implant removal alone. Similarly, youthful women with no pregnancies, no evidence of striae, a thin body habitus and elastic skin, and a history of only modest augmentation will have satisfactory results after implant removal alone. The ideal candidate for mastopexy associated with implant removal is one with a relatively full body habitus, a substantial amount of breast tissue, and breast ptosis with skin striae.

Pearls and Pitfalls

- Immediate breast reconstruction is favored over delayed reconstruction following breast implant removal. This principle applies particularly to patients who previously had implants placed for reconstruction after mastectomy. Removal of implants without immediate reconstruction results in a contracted skin envelope. It is difficult to recreate a smoothly contoured skin envelope if reconstruction is delayed, and preexpansion may be required for a satisfactory result.
- Implant removal should be performed in conjunction with total capsulectomy for several reasons. There is a slightly increased risk for a contained postoperative seroma if the capsule is retained. Removal of a thickened capsule also leaves behind a more pliable breast envelope. Extracapsular dissection enables one to contain an unrecognized intracapsular rupture of a gel implant and to minimize the chance of spillage into surrounding breast tissue, in addition to avoidance of silicone granulomas.
- Meticulous hemostasis must be achieved by removing the capsule within a relatively easily defined extracapsular surgical plane.
- In the case of subpectoral implant placement, a portion of the posterior wall capsule is retained against the ribs to minimize the risk for pneumothorax.
- If a mastopexy is selected, the "tailor-tacking" technique is recommended. Presumptive markings are placed preoperatively, and the future nipple site is identified. It is important to sit the patient upright on the operating table when mastopexy with "tailor-tacking" sutures is performed.
- Most patients who have previously undergone breast reconstruction had their implants placed subpectorally. For optimal aesthetic results, the new breast pocket must be created in a subcutaneous plane when an autogenous tissue replacement is performed.

SUGGESTED READINGS

Burkhardt BR: Capsular contracture: Hard breasts, soft data. Clin Plast Surg 15:521-532, 1988.

Goodman C, Cohen V, Thornby J, Netscher DT: The life-span of silicone gel breast implants and comparison of mammography, ultrasonography, and MRI in detecting implant rupture: A meta-analysis. Ann Plast Surg 41:577-585, discussion 585-586, 1998.

Netscher DT, Sharma S, Thornby J, et al: Aesthetic outcome of breast implant removal in 85 consecutive patients. Plast Reconstr Surg 100:206-219, 1997.

Netscher DT, Walker LE, Spira S: Avoidance and treatment of breast contour deformities encountered with implant removal. Perspect Plast Surg 11(2):35-54, 1998.

Rohrich RJ, Beran SJ, Restifo RJ, Copit SE: Aesthetic management of the breast following explantation: Evaluation and mastopexy options. Plast Reconstr Surg 101:827-837, 1998.

Breast Reduction

ELIZABETH J. HALL-FINDLAY

Breast reduction presents a challenge to surgeons: how to preserve the blood supply and sensation to the nipple-areola complex while resecting the surrounding skin and parenchyma.

Most North American plastic surgeons have been trained in the inverted "T," inferior pedicle technique because it is a safe, reliable approach to breast reduction. Unfortunately, this method results in a long inframammary scar, a boxy shape, poor projection, and long-term bottoming out or pseudoptosis.

In contrast, European and South American surgeons were achieving satisfactory results with shorter-scar techniques. In addition to reduced scarring, shape and longevity were improved. The procedure took less time to perform and was associated with easier recovery. However, these procedures had a higher revision rate. Some surgeons were reluctant to abandon a procedure that gave acceptable results for one that potentially promised better results but was associated with a difficult learning curve.

Etiopathogenesis

In most patients with a normal body mass index, breast hypertrophy results from an abnormal end-organ response to circulating hormones rather than an abnormal level of hormones. The cause of the increased end-organ response is unknown. In these patients, weight gain or loss does not change the size of the breasts. In obese patients, macromastia is often the result of a combination of fatty infiltration, increased end-organ response, and elevated levels of hormones associated with obesity. The patient will benefit from weight reduction before surgery because surgery alone gives suboptimal aesthetic results. Surgery is indicated for symptomatic relief.

Pathologic Anatomy

Breast hypertrophy usually results from an increase in the glandular, fibrous, and fatty elements of the breast. In younger patients, fibrous elements predominate, whereas in older patients, the breasts have more fat. In all breasts, the most fibrous tissue is located in the upper outer quadrant. In addition, there is often hypertrophy of the areola, and this hypertrophy most often also requires reduction.

Surgical Anatomy

Breast reduction surgery must be performed with an understanding of the normal anatomy of the breast. The nipple-areolar complex must be transposed while maintaining an adequate blood supply. The breast parenchyma needs to be resected without interfering with the blood supply not only to the areola but also to the remaining tissue and skin flaps. Maintaining the innervation of the skin and nipple is necessary for the preservation of sensation. It is important to keep as many connections as possible between the parenchyma and the nipple to preserve the ability to breast-feed.

Arterial

The internal mammary (thoracic) artery, the lateral thoracic artery, and to a lesser degree, the thoracoacromial artery provide the arterial supply to the breast (Fig. 1). They pass through subcutaneous tissue into the breast and then descend into the parenchyma. There is consistently one branch from the internal mammary artery that enters the breast at the second or third intercostal space and angles obliquely downward toward the nipple. At the breast meridian, it is approximately 1 cm deep to the skin and is the basis of the superior or superomedial pedicle.

Branches from the lateral thoracic system are more variable, but also become more superficial as they proceed medially. They supply the lateral pedicle.

The internal thoracic vessels give a consistent branch, which enters the breast on its deep surface. It penetrates the pectoralis muscle at about the level

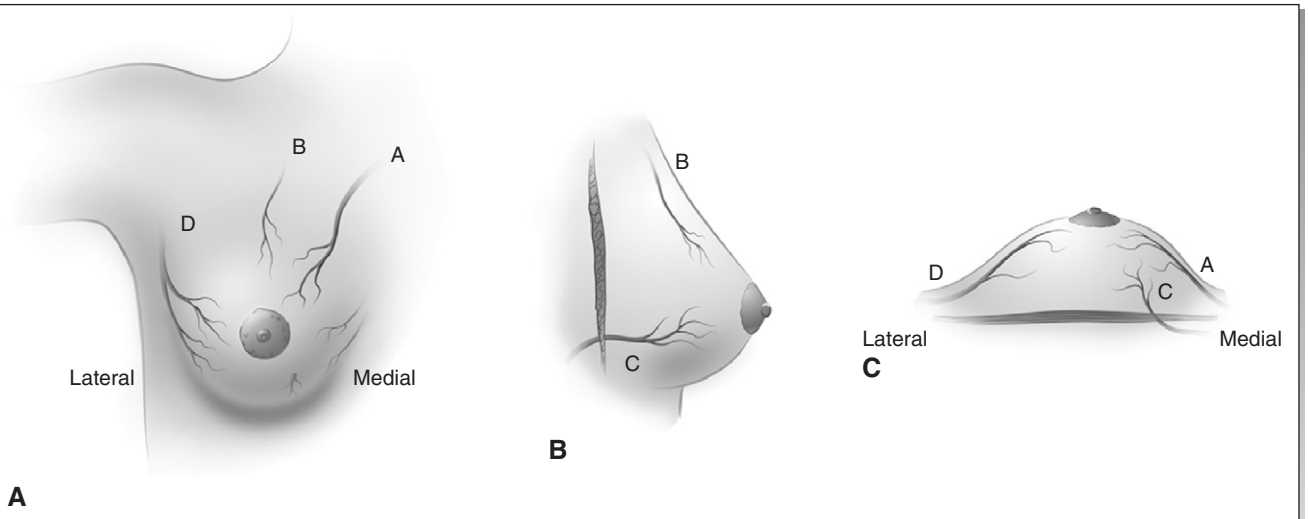


Figure 1 • Anatomy—arterial vascular supply. **A,** The arterial supply consists mainly of branches from the internal mammary (thoracic) system. This system supplies a superficial branch (A) that enters the breast obliquely and a perforator that enters the breast inferiorly (C) through the pectoralis muscle. "A" provides adequate arterial supply to superior and medial pedicles, whereas "C" provides satisfactory arterial supply to inferior and central pedicles. The lateral thoracic system provides input for a lateral pedicle (D). Minor input comes from supraclavicular vessels (B). **B,** Lateral view of the arterial supply. **C,** Craniocaudal view of the arterial supply.

of the fifth or sixth intercostal space medially. It is a large vessel that is usually located just medial to the breast meridian approximately 2 to 4 cm above the inframammary fold. It supplies an inferior or central pedicle.

Venous

Venous drainage of the breast is primarily through a venous plexus that does not follow the arterial supply. The venous plexus is superficial and concentrated around the areola, and circumferentially incising the areola may damage the venous system. In addition, any surgical design (such as a long superior pedicle) that necessitates folding of the pedicle is likely to compromise venous return. Although arterial inflow might be sufficient to overcome folding, venous return could suffer from the compression.

Innervation

The lateral cutaneous branch of the fourth intercostal nerve is the most common nerve supplying sensation to the nipple (Fig. 2). One branch of the nerve travels superficially; the other runs along the pectoralis fascia and takes an almost 90 degree turn at the breast meridian to run vertically toward the nipple. It is for this reason that any full-thickness pedicle (where tissue is left over the pectoralis fascia) is likely to preserve sensation to the nipple-areola complex. In addition, the anterior cutaneous branches take a superficial course and terminate at the medial aspect of the areola. A medially based pedicle would therefore not need to be full thickness to preserve sensation.

There are also supraclavicular branches that supply some sensation; these nerve branches are small and far less important (see Fig. 2).

Diagnostic Studies

Few diagnostic studies need to be performed to determine whether a breast reduction is indicated. Outcomes research has definitively shown the value of breast reduction in improving symptoms of back pain, neck pain, shoulder grooving, rashes, posture problems, and even headaches and tingling in the arms and hands. Unfortunately, most health care insurers are looking for any excuse to deny coverage, and they are demanding—despite evidence to the contrary—either that patients lose weight or that the surgeon remove a minimum weight (often substantial) of tissue per breast.

In addition to standard preoperative laboratory studies, patients in the United States who are older than 35 years should have mammograms performed according to the American College of Surgeons' guidelines.

Reconstructive Goals

The main goal of breast reduction surgery is volume (weight) reduction, but preservation or restoration of breast aesthetics is paramount. Preservation of function (breast-feeding) may be an important goal in young women. The breast should be proportional to body size. It should have an attractive shape with

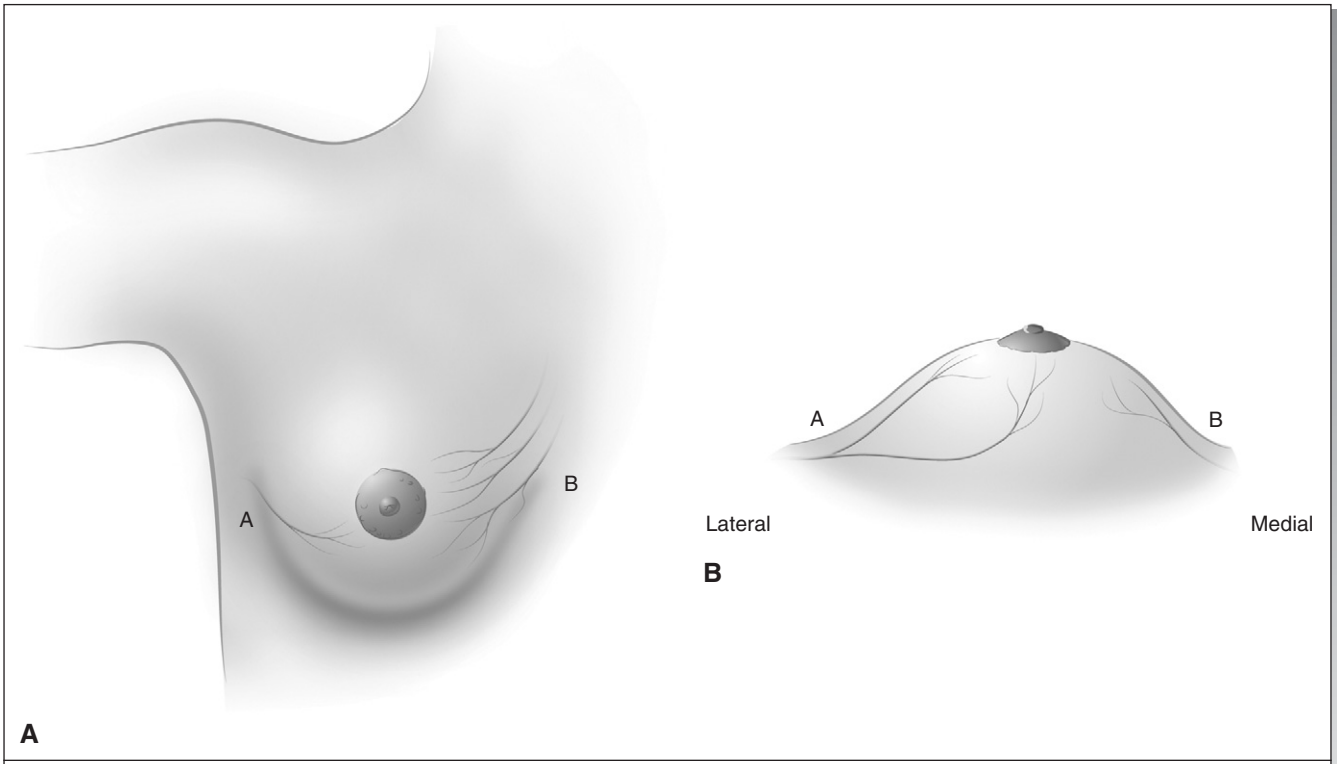


Figure 2 • Anatomy—innervation. **A,** The main sensory innervation to the nipple-areolar complex comes principally through the lateral fourth intercostal nerve (A). There is innervation from the medial intercostal branches as well (B). **B,** A craniocaudal view shows that the fourth intercostal nerve sends a branch superficially, but it also sends a branch just over the pectoralis fascia until it reaches the breast meridian, where it proceeds superficially to supply the nipple-areolar complex.

adequate projection, elegant curves, and a nipple-areola complex that is pleasing in shape and position. The result should also be as long lasting as possible.

Treatment

For the purposes of this chapter, the common methods of breast reduction will be reviewed with special attention to the author's preferred method.

Skin Resection Pattern: Vertical

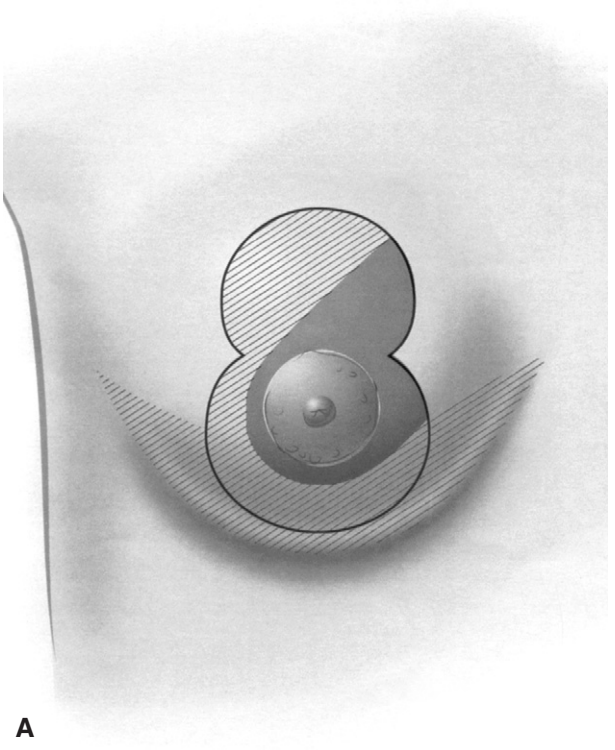
Pedicle: Medial, Superomedial

Parenchyma Resection Pattern: Wise Pattern

The medial pedicle vertical reduction offers several advantages when compared with the other techniques (Fig. 3). The medial pedicle is reliable, provides adequate sensation, and preserves significant glandular-areolar attachment, thereby allowing the possibility of future breast-feeding. In addition, the design maintains maximal medial and superior fullness, elevates the inframammary fold, and allows resection of significant lateral breast tissue. It is reliable and safe and has a shorter learning curve than other short-scar techniques do.

Preoperative marking is critical to ensure postoperative symmetry. Marks are made with the patient standing. The markings include the midline, inframammary fold, breast meridian, and new nipple position. The nipple position is placed at the level of or just below the inframammary fold and is usually between 21 and 26 cm from the sternal notch. It is important not to place the nipple too high because this postoperative problem is difficult to correct. An open circle-shaped pattern for the areolar opening is designed with a 16- to 18-cm circumference. The vertical limbs are created by displacing the breast medially and laterally and marking the transposed breast meridian. The vertical limbs are joined with a curve 2 to 4 cm above the inframammary fold. The areola is designed with a 40- to 50-mm diameter. The base of the medial pedicle should measure between 6 and 10 cm. It should start halfway above the areolar opening and halfway below the areolar opening.

Lidocaine-epinephrine solution is injected at the base of the breast and in the areas to be liposuctioned. The medial pedicle is deepithelialized. The first maneuver is creation of the pedicle. The pedicle is cut straight down to the chest wall, but care should be taken to avoid undermining or resecting



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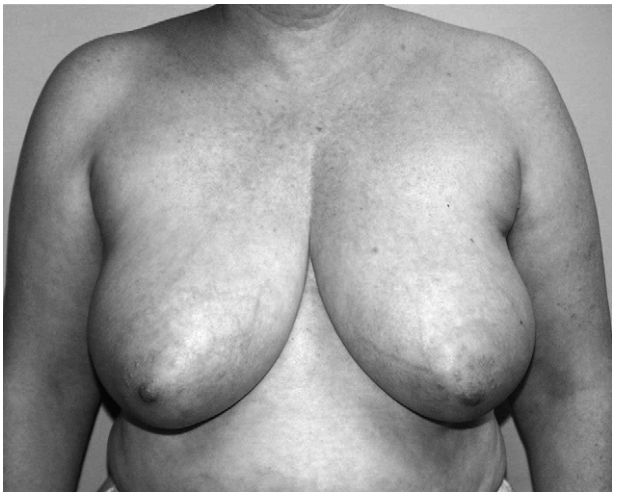

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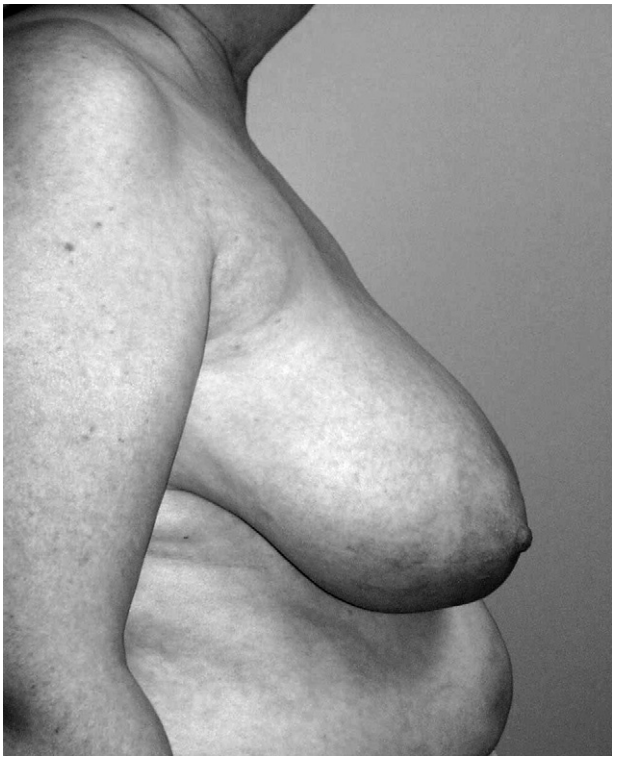

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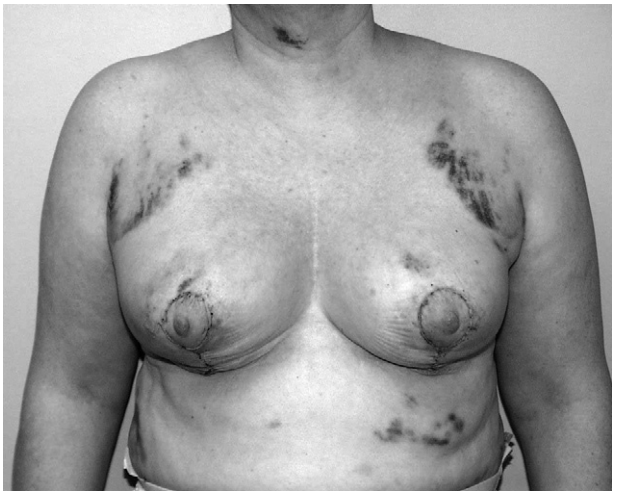

D

Figure 3 • A, The author's preferred method involves the use of a medial pedicle associated with a vertical skin resection pattern. **B,** Frontal preoperative view. **C,** Lateral preoperative view. **D,** Frontal view 10 days postoperatively.

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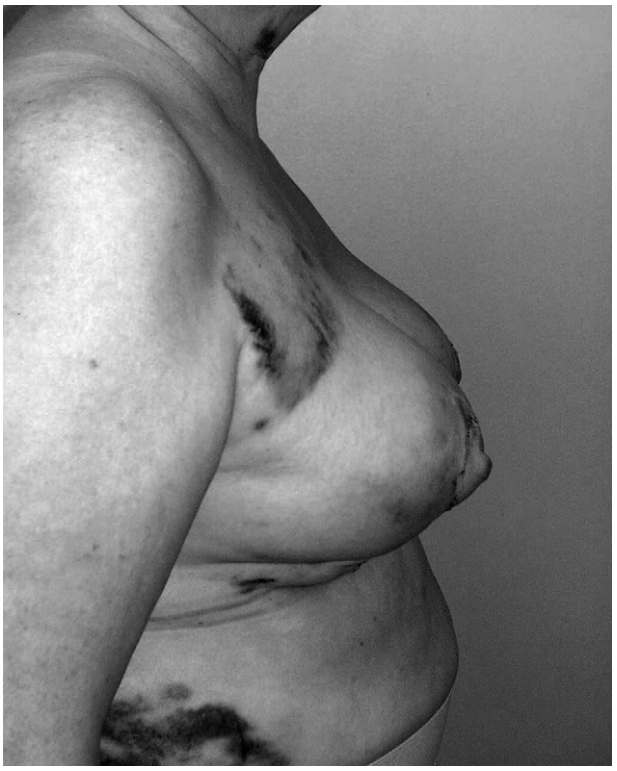

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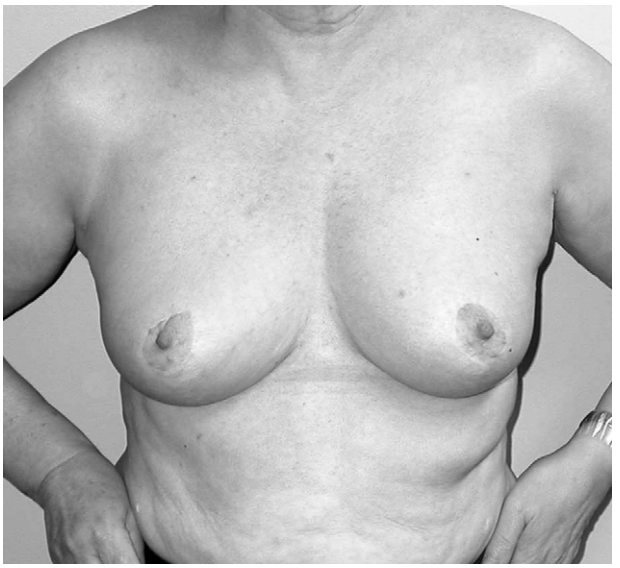

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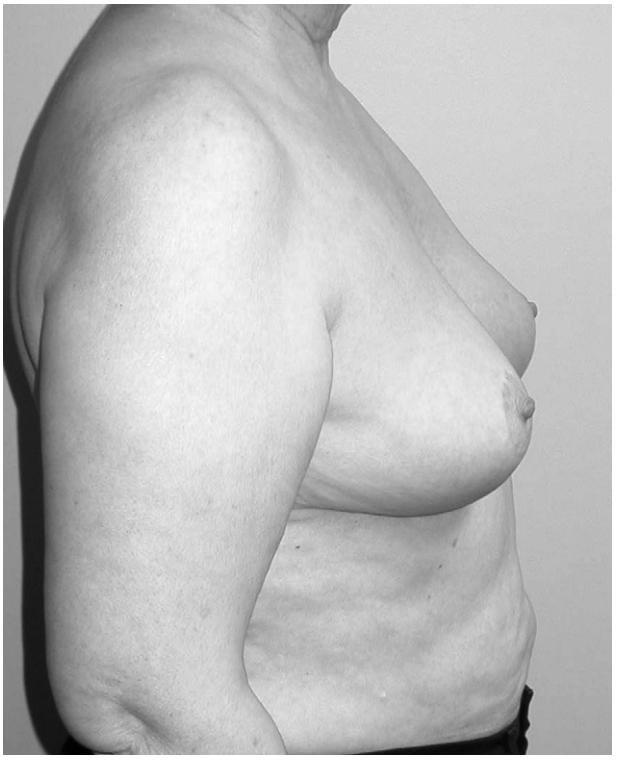

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Figure 3, cont'd • E, Lateral view 10 days postoperatively. **F,** Frontal view 4.5 years postoperatively. **G,** Lateral view 4.5 years postoperatively.

tissue under the pedicle. It is also important to stay above the pectoralis fascia to avoid disrupting the sensory nerve supply to the nipple.

The glandular resection is performed with attention to creation of the medial and lateral pillars. The length of the pillars should be marked. The length varies, depending on the size of the reduced breast, but the pillars should be approximately 7 cm long. The inferior border of the medial pedicle is now the medial pillar. Below the pillar, the resection should be superficial and include the tissue to the deep fascia above the inframammary fold. The pillars become thicker as they extend out medially and laterally. Superiorly, the amount of tissue resected can vary. It is necessary to resect some tissue to allow the pedicle to be rotated into its new location. Some

tissue is left superiorly to provide a platform for the areola, but tissue cannot be pushed into the upper pole. The remainder of the breast tissue can be resected to give a breast of adequate size.

The pedicle is rotated superiorly and sutured to the areolar opening. The pillars are approximated with three to five sutures, and the skin incisions are closed (see Fig. 3).

Skin Resection Pattern: Inverted "T"

Pedicle: Inferior

Parenchyma Resection Pattern: Superior, Superolateral, Superomedial

This design is the most common approach to breast reduction surgery in North America: a Wise pattern for the skin resection and an inferiorly based pedicle (Fig. 4A). The perforator vessels coming through the pectoralis muscle are strong and reliable, thus making this the workhorse technique for all degrees of breast reduction. It relies, however, on a skin brassiere to hold the shape of the breast. The design keeps the vertical incision short to prevent bottoming out and consequently results in a flattened, boxy breast. The effect of gravity causes both the breast tissue and the inframammary fold to descend with time. The result is pseudoptosis and a breast in which the nipples look as though they were placed too high.

Skin Resection Pattern: Vertical

Pedicle: Superior

Parenchyma Resection Pattern: Vertical

Numerous variations on this pattern have been devised (see Fig. 4B). The blood supply is reliable. It comes in as a strong, obliquely oriented vessel from the internal mammary system, and it runs superficially—approximately 1 cm deep to the surface of the skin and usually just medial to the meridian. There are superficial veins that do not travel with the artery, but generally follow a similar pattern. They are often easy to identify and mark preoperatively.

Skin Resection Pattern: Circumareolar

Pedicle: Central

Parenchyma Resection Pattern: Wise Pattern

The circumareolar reduction technique uses a "round block" design without a vertical skin resection scar (see Fig. 4C), undermines the skin all around the breast parenchyma, and uses a permanent suture to close the large circular skin opening to the areola. It is useful for small reductions but may result in a widened scar.

Skin Resection Pattern: None

Pedicle: None

Parenchyma Resection Pattern: Liposuction Only

Liposuction-only breast reduction has definite appeal, but it is limited in application. It would be ideal in young patients with adequate skin tone so that they would not have extensive scarring and the ability to breast-feed would be preserved. Unfortunately, these patients also have thick fibrous breasts with very little fat and they are poor candidates for liposuction.

At the other end of the spectrum, perimenopausal patients have very fatty breasts, but the nipple elevation and skin retraction that can be achieved with liposuction-only reduction will not be sufficient to avoid the development of ptosis. Some patients state that they are satisfied with a shape that is not ideal to avoid the scars associated with skin resection. Liposuction-only breast reduction has a low risk for nipple-areola necrosis, wound-healing problems, loss of sensation, and inability to breast-feed. For some patients, shape is a less important issue. It has been claimed that liposuction is an alternative in patients with medical problems, but it is not truly a less invasive procedure than standard (vertical) breast reduction.

It is more difficult to examine the tissue pathologically when liposuction-only breast reduction is performed. Most of the tissue removed is fat, and even when parenchyma is removed in the standard fashion, little of the tissue is actually subject to microscopic examination.

General Considerations

There is still controversy regarding whether postoperative skin taping helps the shape settle in either the short or the long term. Most surgeons believe that a skin brassiere can shape the breast initially, but it has a variable effect in the long term. In patients with adequate skin elasticity, a skin brassiere may be effective, but in patients with poor skin elasticity, especially in the face of weight changes or pregnancy, skin laxity will determine the longevity of the result.

The Wise skin resection patterns tend to achieve a better initial shape at the end of the procedure, but the vertical approaches result in breasts with more projection and fewer problems with a boxy shape. The skin and breast tissue are brought in from the lateral aspects, whereas the inverted "T" pulls the skin inferiorly. The closure of a horizontal ellipse results in problems with lateral and medial dog-ears.

The techniques that use a lateral, superior, or medial pedicle tend to maintain shape better with time. The inferior pedicle is reliable, but it creates a heaviness at the inferior pole of the breast. This

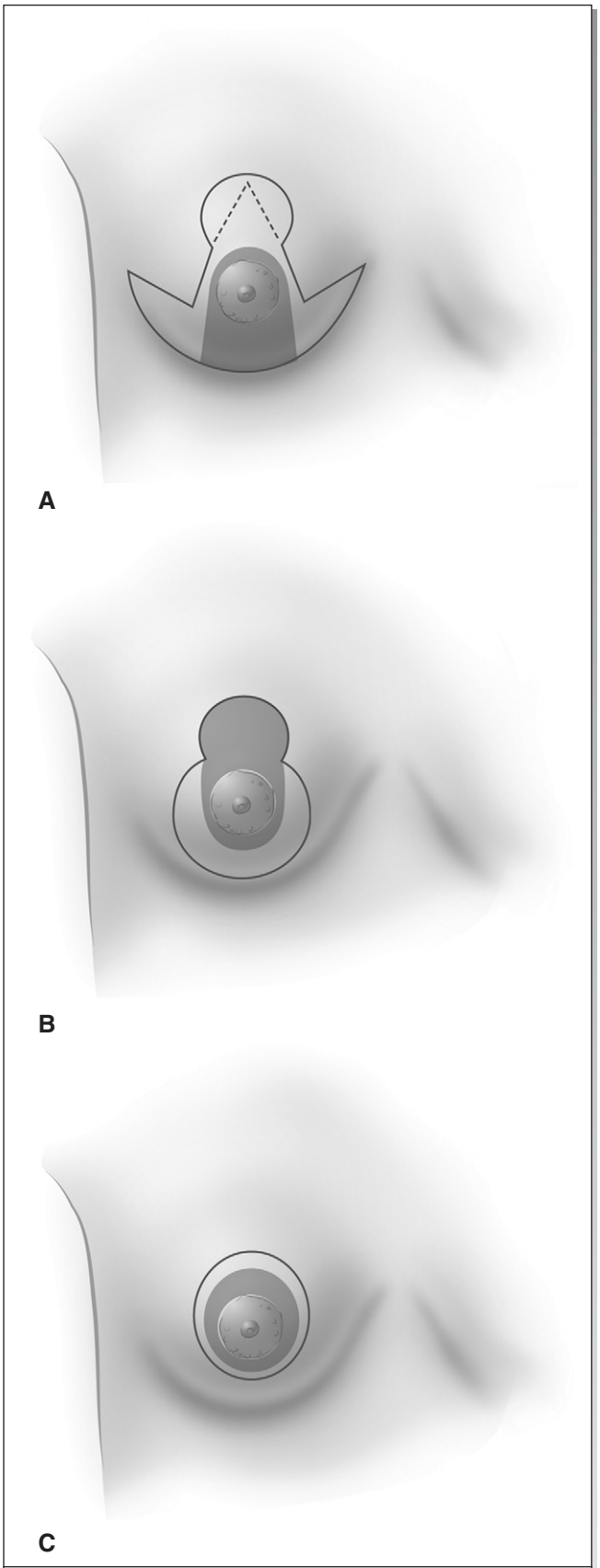

Figure 4 • Skin resection patterns and pedicle designs. **A,** Inverted "T" skin/inferior pedicle. **B,** Vertical skin/superior pedicle. **C,** Circumareolar skin/central pedicle.

heaviness allows gravity to have more of an effect by stretching the inferior skin and causing pseudoptosis and descent of the inframammary fold.

Postoperative Care

The use of drains and antibiotics is controversial. Drains do not prevent hematomas, but they may aid healing by removing fluid from the dead space. With the author's preferred technique, there is significant dead space. Seromas develop, but often do not need to be treated. The routine use of antibiotics may prevent seromas from becoming infected.

As with any breast reduction technique, there is a risk of loss of sensibility or viability in the nipple-areolar complex. If this loss is due to congestion, removal of sutures may improve blood flow. Patients should always be forewarned of the occasional need for revisionary surgery.

Pearls and Pitfalls

- The learning curve for all breast reduction procedures is not an easy one.
- Shorter-scar approaches give a better, more long-lasting shape, but they have a higher revision rate. The revision rate decreases as surgeon experience grows. It is best to discuss the possibility of revisions with the patient preoperatively.
- The inferior pucker with vertical breast reduction improves with time, unlike the lateral or medial pucker (or dog-ear) associated with an inverted "T" approach. Adding a small horizontal component to vertical reductions may be indicated, but it may not change the overall revision rate. A large horizontal scar will pull the skin vertically, thus negating the elegant inferior curve of the breast that can be achieved with the vertical designs. Waiting for the pucker to resolve can be difficult for both the surgeon and patient, but a patient informed preoperatively can be a surgeon's best ally.
- Not all short-scar breast reduction procedures are the same. Not all vertical approaches are the same. Plastic surgeons need to keep the skin resection pattern, the pedicle design, and the parenchymal resection pattern clear in their minds.
- The skin resection pattern should stay well above the inframammary fold so that the scar does not extend onto the chest wall.
- Vertical techniques result in coning of the parenchyma and more projection than is the case with the inferior pedicle, inverted "T"

approach. As a result, the new nipple position must be lowered by at least 2 cm. This maneuver is necessary to accommodate projection and compensate for the fact that the skin (and areola) are not pulled downward with the closure.

- The increased length of the vertical incision is taken up with the increased projection.
- The pillars should be approximately 7 to 8 cm in length, but the skin can be much longer. The skin adapts to the shape of the breast rather than vice versa.

SUGGESTED READINGS

Benelli L: A new periareolar mammaplasty: The "round block" technique. Aesthetic Plast Surg 14:93, 1990.

Georgiade NG, Serafin D, Morris R, Georgiade G: Reduction mammaplasty utilizing an inferior pedicle nipple-areolar flap. Ann Plast Surg 3:211, 1979.

Hall-Findlay EJ: A simplified vertical reduction mammaplasty: Shortening the learning curve. Plast Reconstr Surg 104:748, 1999.

Hall-Findlay EJ: Pedicles in vertical reduction and mastopexy. Clin Plast Surg 20:379-391, 2002.

Lassus C: A 30-year experience with vertical mammaplasty. Plast Reconstr Surg 97:373-380, 1996.

Lejour M: Vertical Mammaplasty and Liposuction of the Breast. St Louis, Quality Medical Publishing, 1993.

Gynecomastia

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Gynecomastia is generalized enlargement of male breasts. It is the most common benign breast condition and affects up to 60% of males. It is observed in newborns as a result of transplacental passage of estrogens, in pubertal boys, and in older men. Gynecomastia is a normal physical finding in the majority of patients; however, it may be the initial sign of a more serious underlying disease process such as malignancy.

Etiopathogenesis

Gynecomastia represents an imbalance in the ratio of circulating estrogens and androgens. The underlying cause of the imbalance is often idiopathic. It may, however, be the result of an associated condition or drug (Tables 1 and 2). It is imperative that malignancy, especially breast and testicular carcinoma, be ruled out when evaluating patients. Gynecomastia must also be differentiated from pseudogynecomastia, which is simply fatty enlargement of the breasts without glandular proliferation.

Pathologic Anatomy

Gynecomastia usually manifests as a firm, mobile disk of tissue underlying the nipple-areolar complex and may be unilateral or bilateral. It can be differentiated from pseudogynecomastia on physical examination as the latter lacks a palpable disk of tissue. On microscopic examination, gynecomastia of recent onset is associated with ductal epithelial proliferation and an increase in vascularity and edema of the surrounding stromal and periductal connective tissue. Long-standing gynecomastia is characterized by a decrease in epithelial proliferation and an increase in stromal hyalinization and fibrosis.

TABLE 1 Conditions Associated with Gynecomastia

PHYSIOLOGIC
Neonatal
Pubertal
Involutional
PATHOLOGIC
Neoplasms
Testicular (germ cell, Leydig cell, or Sertoli cell)
Adrenal (adenoma or carcinoma)
Ectopic production of human chorionic gonadotropin (especially lung, liver, and kidney cancer)
Primary gonadal failure
Secondary hypogonadism
Enzymatic defects in testosterone production
Androgen insensitivity syndromes
True hermaphroditism
Liver disease
Starvation, especially during the recovery phase
Renal disease and dialysis
Hyperthyroidism
Excessive extraglandular aromatase activity
Drugs
Idiopathic gynecomastia

From Braunstein GD: Gynecomastia. N Engl J Med 328:490-495, 1993.

Patient Evaluation/Diagnostic Studies

The most important tools in the evaluation of a patient with gynecomastia are the history and physical examination. Questions should focus on the duration and timing of the breast changes and symptoms, their progression, and medication and alcohol use. Patients who are asymptomatic with a

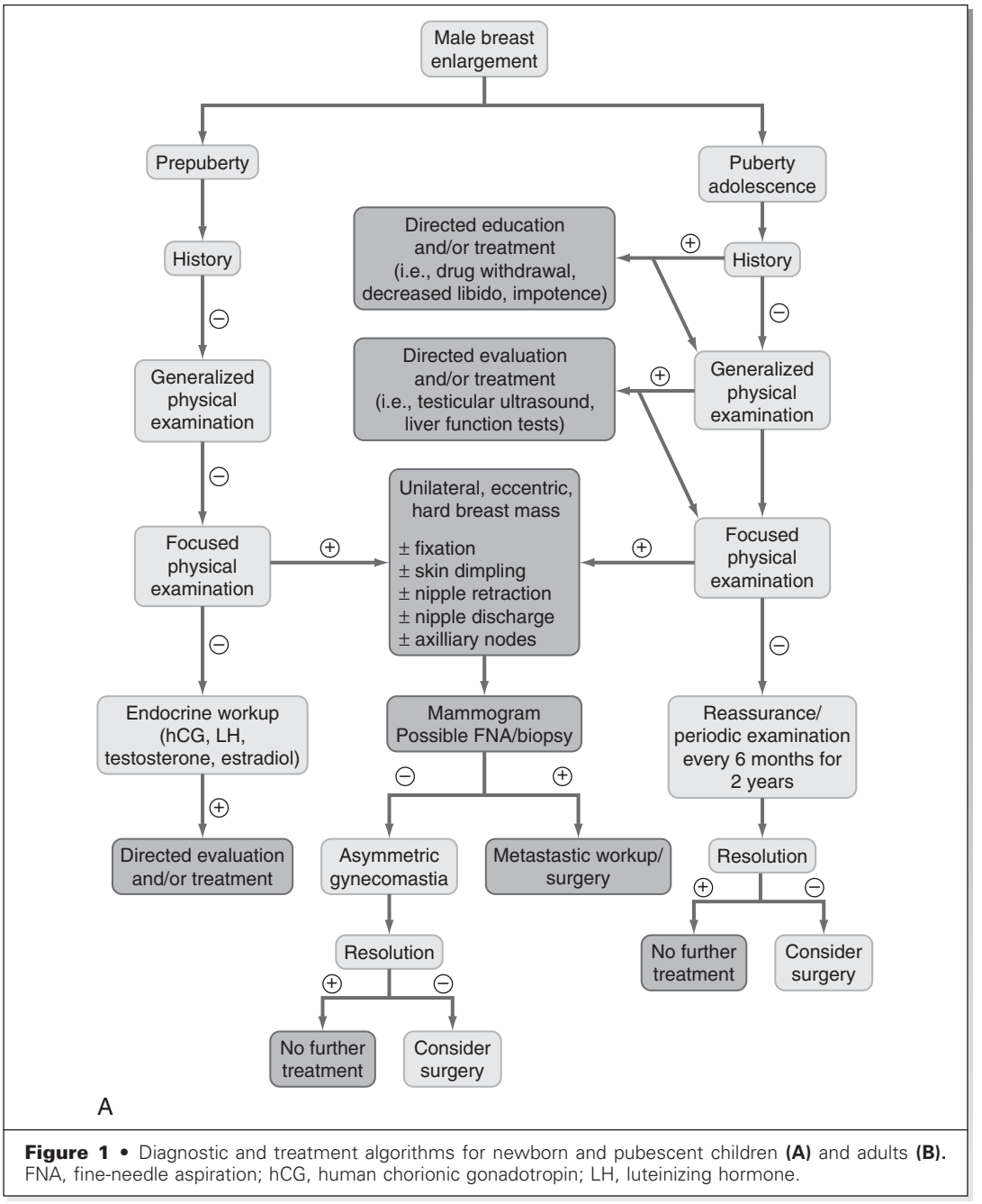

Figure 1 • Diagnostic and treatment algorithms for newborn and pubescent children **(A)** and adults **(B).** FNA, fine-needle aspiration; hCG, human chorionic gonadotropin; LH, luteinizing hormone.

long-standing history of breast enlargement pose less concern than a patient with rapidly increasing, painful breast enlargement. A complete review of symptoms should be undertaken, with particular attention paid to hypogonadism, as well as liver, thyroid, renal, and lung disease.

Masses that are hard, eccentric, or nonmobile are not consistent with gynecomastia, and malignancy should be ruled out. Other disturbing signs include nipple retraction or discharge, skin dimpling, and axillary adenopathy. Thorough examination of the testes, thyroid, and abdomen should also be documented. Laboratory workup should include a basic metabolic profile, as well as liver, and thyroid function studies.

Additional diagnostic workup is dictated by the findings on history and physical examination, as well as by the age of the patient (Fig. 1). If gynecomastia is suspected, reassurance that it is a normal physiologic finding is recommended, with follow-up in 6 months. Adult patients with a suspicious breast mass should be referred for a mammogram, whereas pediatric patients should have the mass evaluated by ultrasound. Further evaluation by fine-needle aspiration can be performed. Testicular ultrasound is recommended for signs of hypogonadism or

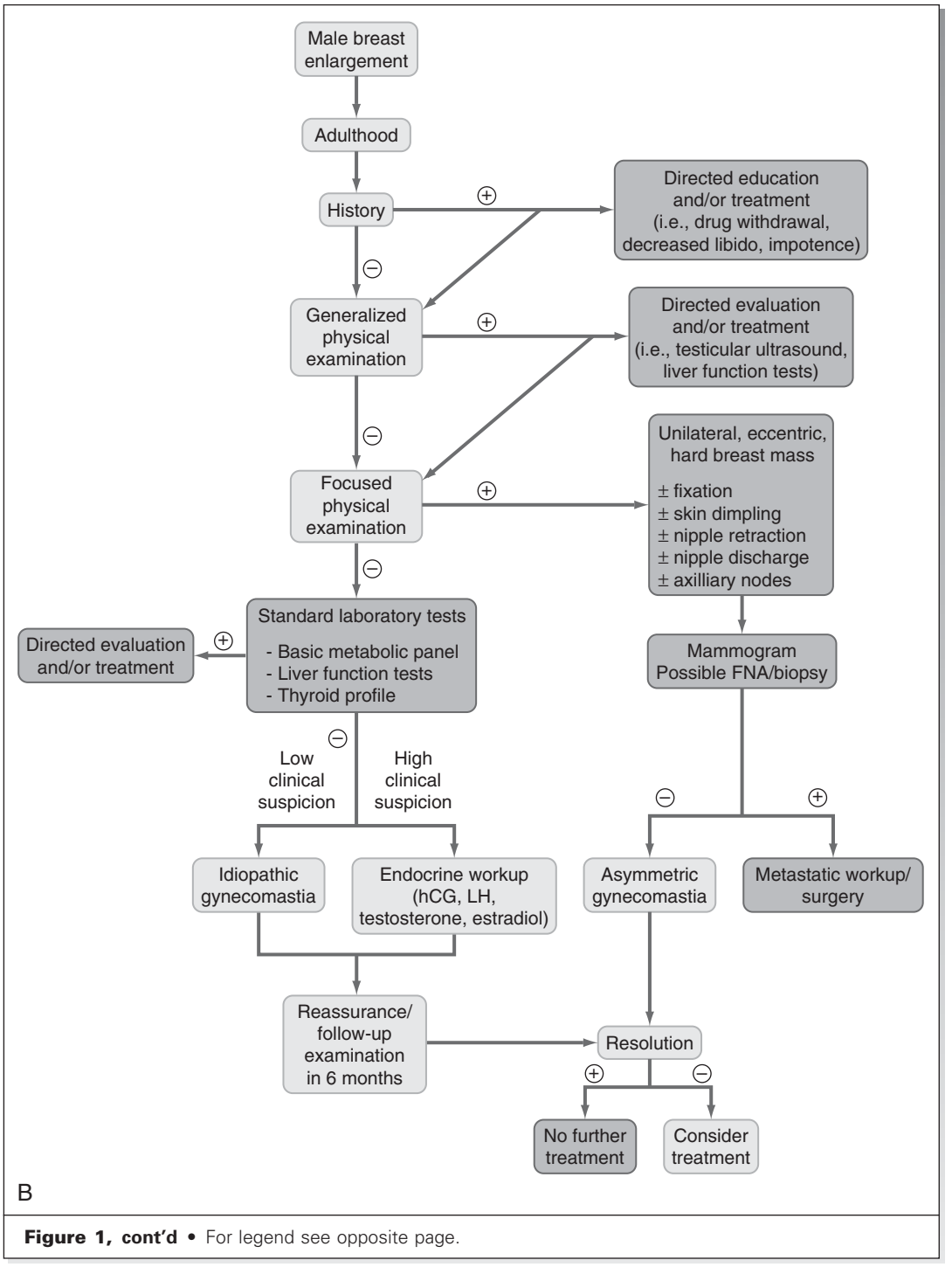

Figure 1, cont'd • For legend see opposite page.

observed testicular abnormality, abdominal computed tomography (CT) for suspected liver disease or adrenal tumor, and chest radiography/CT if signs or symptoms of lung cancer are present. Hormone studies (follicle-stimulating hormone, luteinizing hormone, and β-human chorionic gonadotropin) are recommended for young males with recent onset of breast hypertrophy because this age group has the highest prevalence of underlying testicular tumors.

Indications for Surgery/Reconstructive Goals

If no underlying pathologic process is identified, patient reassurance, with follow-up in 6 months, or surgical intervention is recommended. In the majority of pubertal boys, gynecomastia usually resolves without intervention within 2 years. For this reason, surgery is not the first-line treatment

TABLE 2 Drugs Implicated as Causes of Gynecomastia

CATEGORY	DRUG
Hormones	Androgens and anabolic steroids
	Chorionic gonadotropin
	Estrogens and estrogen agonists
Antiandrogens or inhibitors of androgen synthesis	Cyproterone
	Flutamide
Antibiotics	Isoniazid
	Ketoconazole
	Metronidazole
Antiulcer medications	Cimetidine
	Omeprazole
Cancer chemotherapeutic agents (especially alkylating agents)	
Cardiovascular drugs	Amiodarone
	Captopril
	Digoxin
	Enalapril
	Methyldopa
	Nifedipine
	Reserpine
	Verapamil
Psychoactive drugs	Diazepam
	Haloperidol
	Phenothiazines
	Tricyclic antidepressants
Drugs of abuse	Alcohol
	Amphetamines
	Heroin
	Marijuana
Other	Phenytoin
	Penicillamine

From Braunstein GD: Gynecomastia. N Engl J Med 328:490-495, 1993.

in this age group unless the gynecomastia is long-standing. Surgery is indicated in patients with severe pain and in those afflicted with significant emotional distress. Psychological distress is one of the most common reasons that patients seek medical attention. This factor is very important for the treating surgeon to consider when counseling patients. The reconstructive goal of surgery is to decrease the size of the breast, improve its contour and symmetry, and leave minimal scars.

Treatment

Treatment is identical for both gynecomastia and pseudogynecomastia. From the initial consultation, most patients are approached with the possibility of a two-stage treatment. Liposuction alone, either power or ultrasound assisted, is the first stage. The breast is injected with tumescent solution and aggressively suctioned. Two ports are made, one on the lateral side and the other on the inferior margin of the areola. The suctioning is performed with particular attention to the subareolar region, although the entire breast must be suctioned thoroughly. After initial suctioning, it is important to make a second pass in the areolar region. After fatty tissue is removed, the hard glandular tissue in the areolar region remains. The hard breast tissue should be firmly grasped and a cutting cannula passed in the center. The initial resistance gradually subsides as suctioning is continued in the firm area.

The patient is observed over the next 6 months. In younger patients with tight skin, the contraction is often adequate and a second stage is not usually necessary. Even enlarged areolas contract in size. In patients with severe gynecomastia or loose skin, a secondary procedure may be required, including removal of the remaining subareolar glandular tissue, areola reduction, and skin excision. The two-stage approach leaves less scarring than if primary excision were performed.

Postoperative Care

After liposuction procedures, patients are wrapped with a compressive bandage for 3 weeks to improve contraction of the skin. Antibiotics are not necessary, and patients are discharged the same day. The most common complications are seromas or hematomas, although their incidence is reduced by compression bandages. Skin dimpling may also be observed. With secondary procedures, it is common to overresect subareolar tissue and leave a contour deformity at the edge of the resection.

Pearls and Pitfalls

- Make the correct diagnosis (specifically, associated conditions or malignancy) in cases of male breast enlargement.
- Prepare the patient for two treatment stages. Although adequate results are often obtained with one procedure, the patient should be informed that a secondary procedure may be necessary.
- Liposuction should be performed aggressively with a cutting cannula in the subareolar area. The cannula is difficult to pass in this area, and special attention should be directed to reduction of this tissue.
- Maintain at least 1 cm of tissue under the areola with an open resection to avoid a contour deformity.

SUGGESTED READINGS

Bowers SP, Pearlman NW, McIntyre RC Jr, et al: Cost-effective management of gynecomastia. Am J Surg 176:638-641, 1998.

Braunstein GD: Gynecomastia. N Engl J Med 328:490-495, 1993.

Glass AR: Gynecomastia. Endocrinol Metab Clin North Am 23:825-837, 1994.

Mathur R, Braunstein GD: Gynecomastia: Pathomechanisms and treatment strategies. Horm Res 48:95-102, 1997.

Liposuction and Body Contouring

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Liposuction is the most frequently performed aesthetic surgery procedure. Liposuction and excisional surgery for body contouring are performed to satisfy yearnings for beauty, youth, athleticism, and vigor.

Liposuction consists of surgical aspiration of fat through small incisions with the use of small-caliber cannulas connected to aspiration devices. With the cannula attached to a powerful vacuum source, the surgeon moves the cannula back and forth in a to-and-fro motion. The fat adheres to the aperture at the end of the cannula, and the to-and-fro motion of the cannula through the fat avulses small fragments of tissue that are evacuated into a collection device.

Fat is less dense and structurally weaker than adjacent nerves and blood vessels; therefore, it is preferentially evacuated, whereas nerves, blood vessels, and fascia remain intact. The fascial framework that contains the neurovascular bundles and connects the deep muscle fascia to the dermis is left behind. Removal of subcutaneous fat reduces body contour as the subcutaneous tissues and skin contract to accommodate the diminished volume of the subcutaneous space.

Excisional body contour surgery removes loose, redundant skin and subcutaneous tissue and tightens the adjacent skin and subcutaneous tissue envelope. Abdominoplasty and thigh lift are the most commonly performed excisional procedures for the lower part of the torso and the thigh-buttock area. The excisional procedures are highly effective but are also more physiologically disruptive, painful, and associated with a higher complication rate than liposuction.

Patient Selection and Evaluation

Liposuction and body contour surgery often attract a group of patients who mistakenly believe that these procedures are a cure for obesity. Although liposuction works well in treating figure faults, it is not a proven method of permanent weight loss. Overweight patients are not only unlikely to have long-term benefits from liposuction and body contouring surgery but are also at increased risk for complications from the surgery. Patients with a body mass index higher than 30 should be rejected for surgery and referred for weight loss counseling.

SELECTING FIGURE FAULTS. Patients with disproportionately localized fat can obtain satisfactory results from liposuction. Patients with disproportionately large thighs or buttocks (or both), despite being at normal weight, are especially good candidates. For most body areas, a skin pinch thickness of 3 cm or more indicates enough subcutaneous fat to benefit from liposuction.

Patients with more generalized subcutaneous excess fat (mildly or moderately overweight) can also have satisfactory results from liposuction, but fat removal in these patients usually involves more extensive surgery with more risk. The long-term efficacy of large-volume fat removal remains to be proved. Patients with loose or excessive skin are better treated by excisional surgery. Abdominoplasty, either confined to the anterior of the abdomen or applied circumferentially, is an effective method of excising excess skin and tightening the lower part of the trunk. Skin laxity of the buttocks

and thighs benefits from a thigh lift. Liposuction is frequently performed as an adjunct to the excisional procedures.

Abdominal fascial laxity should be assessed preoperatively. Plicating loose fascia can substantially improve abdominal contour. If the abdominal fascia protrudes but is firm secondary to intraabdominal fat, weight loss through diet and exercise is the best option. Tightening fascia that is tense from intraabdominal fat frequently leads to recurrence of abdominal wall protuberance.

Liposuction

Instrumentation

CANNULAS. Cannulas generally range from 2 to 5 mm in diameter. The aspiration apertures are usually multiple and are positioned several millimeters proximal to a blunt cannula tip. Although larger cannulas permit more rapid fat evacuation, they are also more likely to produce contour irregularities. Small and medium-sized cannulas are used for most applications, particularly for smaller fat deposits and for removing fat closer to the surface of the skin. Vented cannulas permit air to mix with the aspirate as it is flushed down the tubing and increase the speed and efficiency of aspiration.

TUBING. Clear, flexible polymer tubing approximately ½ inch in diameter connects the cannula to the collection device and vacuum pump.

VACUUM PUMPS. A wide variety of aspiration devices composed of collection bottles, filters, and vacuum pumps are available. The pumps should be sufficiently powerful to achieve sufficient negative pressure for vaporization of water. Vaporization of water in the aspirate also increases the efficiency and speed of aspiration by reducing the coefficient of friction of the aspirate.

SYRINGE SUCTION. Small volumes of fat can be efficiently aspirated by using syringes attached to collection cannulas with a Luer-lock fitting. Syringe aspiration is used most frequently when the fat is collected for reinjection into other body areas as autologous fat grafts.

POWER-ASSISTED LIPOSUCTION. Pneumatic and electric-powered handpieces are used to create a reciprocating action of the cannula after it is inserted into subcutaneous fat. Reciprocation occurs at 3000 times per minute over a 2- to 3-mm distance. The reciprocating action facilitates passage of the cannula through fat and avulses small particles of fat as the fat is suctioned into the opening at the distal end of the cannula. Powered devices reduce the surgeon's physical work effort and are particularly useful for large-volume fat removal or for multiple liposuction procedures on the same day. Powered handpieces have the disadvantage of being larger and more bulky than handpieces of conventional cannulas.

ULTRASOUND-ASSISTED LIPOSUCTION. An ultrasonic probe reciprocates at 20,000 to 30,000 cycles per second over a 100-micron amplitude. This creates a powerful energy field at the tip of the probe. The energy field vaporizes the fatty tissue and releases intracellular triglycerides. The mixture of oil and tissue stroma is aspirated with a conventional or powered liposuction cannula. Ultrasound-assisted liposuction reduces the surgeon's work effort and is particularly useful in scarred and fibrous areas. It adds an additional step to the procedure, however, and introduces the potential complication of a thermal burn caused by the high energy at the tip of the probe. Other complications, such as seromas and paresthesias, occur more commonly when ultrasound is used.

Surgical Treatment

The patient is marked preoperatively in the standing position. A series of concentric circles are drawn on the protuberant areas. The innermost circle marks the point of maximum fat deposition. Volumetric estimates of fat extraction can be inscribed on the skin and serve as a useful intraoperative guide. Access incisions for cannulas are also marked (Figs. 1 to 3).

With the patient standing, the skin surface from the upper part of the chest to the toes is prepared with a germicidal agent (povidone-iodine). The patient is placed on a sterile-draped operating table, and the feet are wrapped in sterile towels. A sterile Mayo stand cover under the patient is used as a draw sheet for assistance in positioning during the operation. A sterile barrier sheet separates the anesthesiologist from the operative field. This method of skin preparation and draping keeps the entire body available for surgical manipulation in multiple body positions without having to interrupt the operation for a second sterile preparation and draping.

Most of the body is exposed for the entire operation so that body heat loss is minimized because decreased core body temperature is associated with a variety of complications. For patients under general anesthesia, a countercurrent exchanger in the anesthesia circuit (Bain circuit) reduces heat and vapor loss. Whenever possible, forced-air warming blankets are used on parts of the body outside the sterile field (most frequently, the head, shoulders, and upper extremities). When warming blankets cannot be used or are insufficient to maintain core body temperature, the operating room is heated to 80° F. Fluids for injection are heated to body temperature.

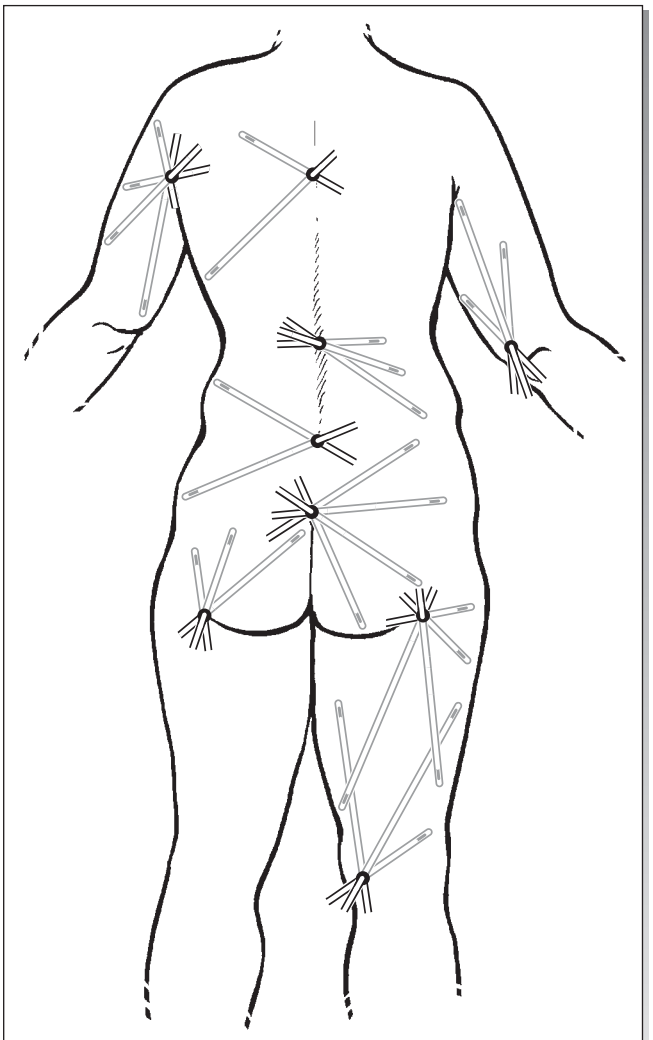

Figure 1 • Access incisions and cannula positions for liposuction of the posterior aspect of the body.

Tumescent Technique

Anatomic areas undergoing liposuction or excisional surgery should be infiltrated with a dilute solution of lidocaine and epinephrine before the start of surgery. Infiltration of large volumes of dilute lidocaine and epinephrine provides the following benefits:

- Decreased blood loss
- Decreased need for systemic anesthetic agents, including postoperative narcotics
- Decreased need for intravenous fluids
- Facilitation of passage of the cannula through tissues
- Facilitation of the use of ultrasound-assisted liposuction

The following recipe for tumescent solution is both safe and effective:

Lidocaine 2%	20 mL
Epinephrine 1:1000	1 mL
Lactated Ringer's solution	1000 mL
Lidocaine 0.04% with epinephrine 1:1,000,000	1026 mL

Each liter of injectate contains lidocaine, 400 mg, at a concentration of 0.04%, the minimum concentration that consistently reduces patients' requests for narcotics in the postoperative period.

The volume of subcutaneous infiltrate varies depending on the volume of aspirate. A 1:1 ratio of injection to aspirate provides total fluid replenishment and adequate hemostasis and analgesia. The total injectate volume frequently exceeds 3 L, which usually delivers lidocaine doses in excess of the manufacturer's recommendations. Nevertheless, extensive clinical experience has established the safety of lidocaine doses up to 50 mg/kg when instilled into subcutaneous fat in a hyperdilute solution mixed with epinephrine. To use lidocaine safely in this off-label manner, however, the surgeon must be thoroughly familiar with the pharmacokinetics of hyperdilute lidocaine-epinephrine solutions delivered to subcutaneous tissues. The anesthesiologist should limit intravenous fluid administration when large volumes of subcutaneous fluid are given.

Use of the tumescent method permits removal of large volumes of fat aspirate with minimal blood loss. If the method is used correctly, liposuction aspirate volumes of more than 5000 mL can be removed without the need for transfusion. It is critical that the surgeon monitor the quality of the aspirate as the operation proceeds. A pure yellow aspirate indicates minimal blood loss. A very red aspirate indicates more than average blood loss, a finding dictating alteration of the operative plan and reduction in the scope of the operation to avoid an unplanned transfusion.

Liposuction begins 20 minutes after completion of preinfiltration with tumescent solution to allow adequate time for optimal vasoconstriction. Whatever anatomic areas are treated, the principles for producing consistently smooth contour reduction remain the same:

- Treat each area through multiple access incisions. It is important to suction each area at different angles to allow more even suction with less noticeable contour irregularities.
- Suction first from deeper tissues. More superficial areas should be addressed after the deeper fat is removed.
- Use narrow-gauge cannulas. Narrow cannulas are less likely to result in depressions, a maneuver that is especially important in superficial suctioning.
- Judge the endpoint of liposuction by the preoperative goals and intraoperative findings.

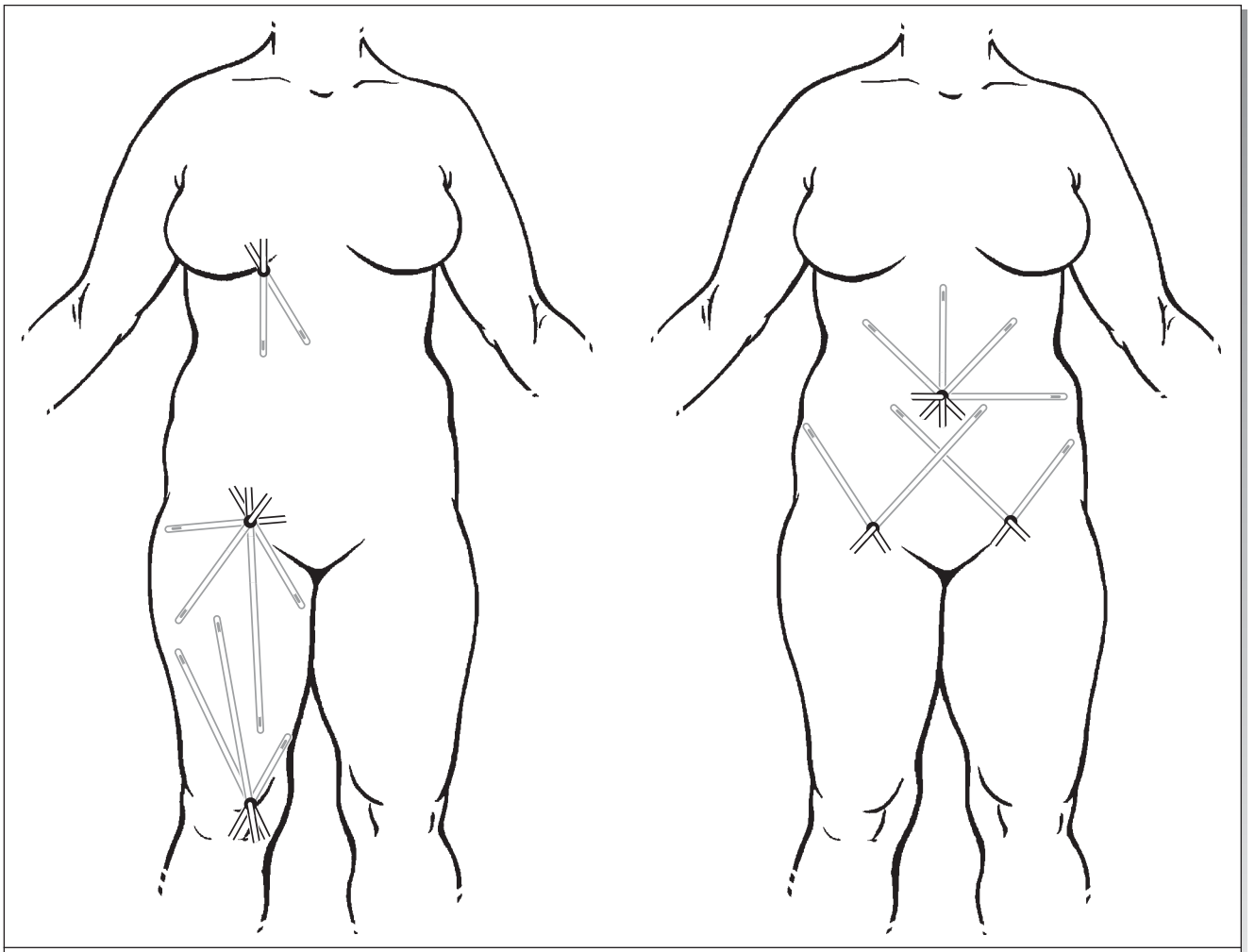

Figure 2 • Access incisions and cannula positions for liposuction of the abdomen and anterior thigh.

- There should be a visible diminution in contour, along with a reduction in pinch thickness. In addition, as preoperative target volumes are reached or changes in the quality of the aspirate are encountered, liposuction should be terminated.

If in doubt, it is better to remove less tissue because overresection is more difficult to correct than underresection.

Aftercare

During the first 2 days after liposuction, large amounts of blood-tinged fluid drain from the incision sites. Patients should prepare their bedding and furniture with protective sheets. Patients undergoing small to average volume removal (<2500 mL) are usually able to return to sedentary work activities within 4 to 5 days. Treatment of multiple operative areas and removal of larger volumes require a more prolonged recovery. Patients undergoing large-volume liposuction (>5000 mL) usually remain overnight in the hospital and usually require 7 to 10 days before return to normal daily activities. Vigorous sports are not resumed until 3 weeks after surgery.

Patients undergoing calf and ankle liposuction benefit from strong support hose for 6 weeks to decrease swelling of the distal end of the lower extremity. Compressive garments in other anatomic areas do not influence the final result but may speed recovery.

Excisional Body Contour Surgery

Excisional body contour surgery consists of various types of abdominoplasty and thigh lift. Abdominoplasty of the anterior abdominal wall is the most frequently performed excisional body contour procedure, but circumferential abdominoplasty and abdominoplasty combined with thigh-hip-buttock lifts have been performed with increasing frequency

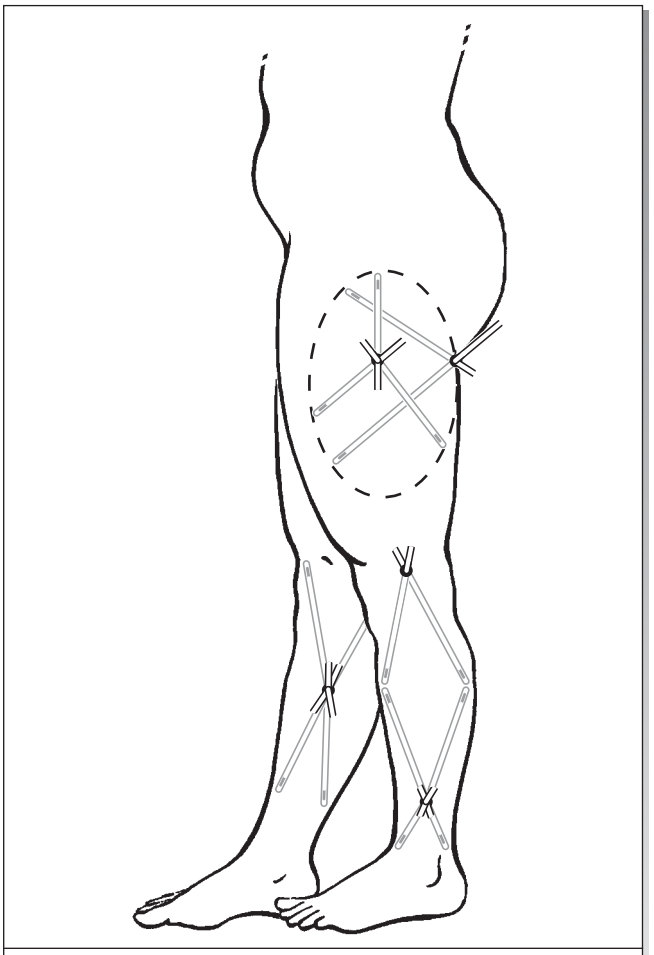

Figure 3 • Access incisions and cannula positions for liposuction of the lower extremity.

in recent years. An ever-increasing overweight population and new techniques and acceptance of obesity surgery are generating large numbers of patients who have undergone massive weight loss. These patients frequently have excess skin that requires excision. Even more commonly, serial pregnancies can result in stretched, excessive skin on the anterior abdominal wall with an underlying abdominal diastasis.

Abdominoplasty

Multiple variations of abdominoplasty can be performed, depending on the precise anatomic location of the skin and fascial laxity.

ANTERIOR ABDOMINOPLASTY. Preoperative marking is performed with the patient standing. The anterior midline is marked from the xiphoid to the anterior labial commissure. A curving, low transverse incision line is marked so that the low point of the planned incision is at the midline, 5 to 7 cm cephalad to the anterior labial commissure with the skin on strong stretch. The incision line curves laterally and cephalad and ends near the anterior superior iliac spines. The umbilicus is outlined as a longitudinal ellipse measuring 2 to 2.3 mm with the long axis vertical (Fig. 4).

A Foley catheter is placed after induction and intubation. After tumescent infiltration, the umbilicus is sharply circumscribed and left on a stalk attached to the anterior abdominal wall. The skin is incised through subcutaneous tissues to the deep fascia. The skin and subjacent fat are elevated from the lower part of the abdomen to the xiphoid, with sparing of the umbilicus. The abdominal musculature is tightened with a permanent midline suture to approximate the left and right rectus muscles and close any midline diastasis. The umbilicus is delivered through a midline incision in the abdominal flap.

Important technical details include the following:

- Wide undermining of the lower (infraumbilical) central and lateral aspects of the abdomen to mobilize maximum skin for excision
- Undermining in the upper part of the abdomen (supraumbilical) confined to a relatively narrow midline area to preserve blood supply to the distal abdominal flap
- Use of adjunctive liposuction in areas that are not undermined to achieve maximum fat removal
- Closure of the superficial fascial system (Scarpa's and Camper's fasciae) under tension with multiple permanent sutures so that the skin is approximated without tension

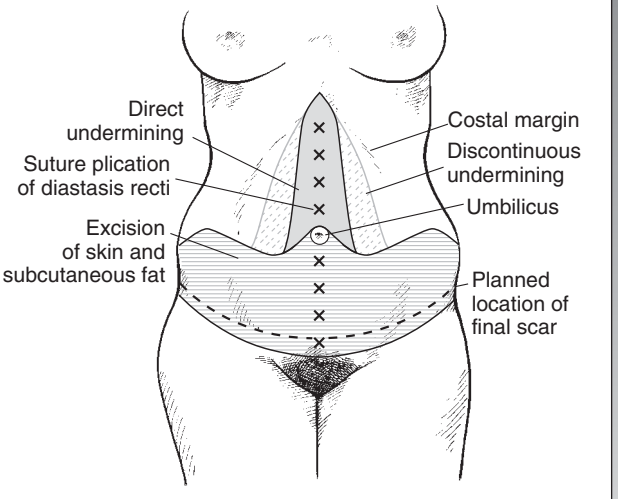

Figure 4 • Anterior abdominoplasty incision markings and extent of dissection. In the figure shown, excision extends beyond the midlateral lines. Excision may also extend to posterior areas to perform circumferential abdominoplasty or anterior abdominoplasty in conjunction with a lower body lift (see Fig. 6).

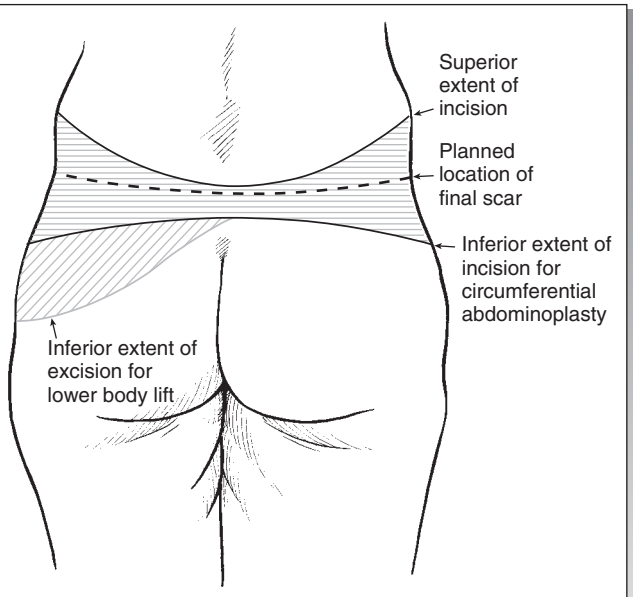

Figure 5 • Posterior incision markings for a lower body lift (patient's left) and circumferential abdominoplasty (patient's right).

- Placement of the neoumbilicus in the midline at an appropriate height (usually 2 to 3 cm cephalad to a horizontal line between the high points of the iliac crests)

CIRCUMFERENTIAL ABDOMINOPLASTY. Patients with skin excess and laxity extending into the flanks and the posterior aspect of the trunk benefit from circumferential abdominoplasty.

Preoperative marking is performed with the patient standing. The anterior midline is marked as for anterior abdominoplasty. The posterior midline is marked from the midthorax to the natal cleft (Fig. 5). Left and right midaxillary lines are also marked. Vertical lines halfway between the posterior midline and the midaxillary lines are outlined bilaterally. On the anterior trunk vertical lines are drawn midway between the anterior midline and the midaxillary lines in the areas of the anterior superior iliac spine (see Fig. 5).

The amount of excess skin is estimated by pinch in the lateral and posterior areas of the trunk above and below the planned incision line. Estimates are also made of the width of excision on the lower anterolateral aspect of the trunk. The pinch estimates determine the cephalad and caudal extent of the skin excision (Fig. 6; see also Fig. 5). Although the actual extent of excision is determined at the time of surgery, the planned excision lines are useful guides in performing the operation and maintaining symmetry.

A Foley catheter is placed and the operative sites infiltrated with tumescent solution. With the patient in the supine position, the anterior abdominal dissection is performed as described for anterior abdominoplasty. With the table slightly flexed, a midline incision is made in the anterior abdominal flap to the estimated point of excision. A temporary suture is used to secure the midline of the incised upper abdominal flap to the midline just above the symphysis pubis.

The table is taken out of the flexed position, the patient is turned into the left lateral decubitus position, and an axillary roll is placed. A padded, sterilely draped Mayo stand is placed beneath the right knee and lower part of the right leg to maintain slight hip abduction.

The previously made incision in the anterior of the abdomen is continued around the trunk to a position approximately 8 cm beyond the posterior midline. Skin-subcutaneous flaps are dissected in a cephalic and caudal direction. The level of dissection is between the superficial and deep layers of the superficial fascial system. Excess skin is excised and closure accomplished in layers with nonabsorbable sutures through the superficial layer of the superficial fascial system. The skin is closed without tension with an absorbable intracuticular suture. Closure extends from the right anterior superior iliac spine to beyond the posterior midline.

The patient is turned to the opposite lateral decubitus position, and the identical procedure is carried out.

The patient is returned to the supine position. The operating room table is flexed slightly, and excess skin from the anterior abdominal region is

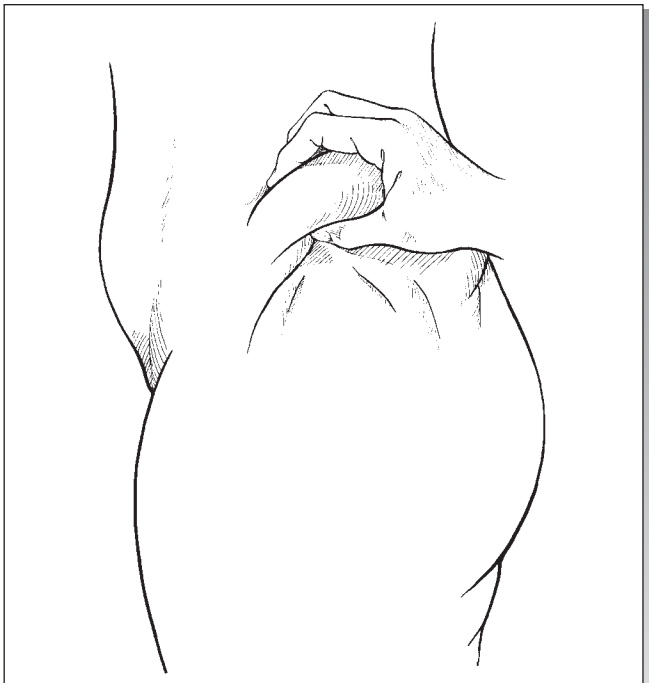

Figure 6 • Pinching and lifting of skin to demonstrate excess skin laxity of the buttocks and thigh. This maneuver simulates the benefit of a lower body lift.

excised. Anterior closure is as described for anterior abdominoplasty.

Thigh Lift

Lateral thigh lifts, also termed lower body lifts, are performed in much the same way as circumferential abdominoplasty. The planned incision lines (and scar) are lower than the circumferential abdominoplasty incision lines in the lateral and posterior aspects of the trunk to facilitate removal of thigh and buttock soft tissue (see Fig. 5). The patient's lower extremities are separated by bolsters to create maximal abduction at the hip, with the feet separated by as much as 3 feet and the angle between the thighs kept as wide as 45 degrees. This type of positioning permits excision of widths of skin in excess of 20 cm over the lateral lower torso–lateral thigh–buttock region.

Medial thigh lifts are rarely necessary because lateral thigh lifts combined with anterior abdominoplasty usually tighten the medial thigh skin as well as the lateral thigh skin.

Postoperative Care

Abdominoplasty patients remain in the hospital for 24 to 72 hours after surgery; thigh lift patients remain 48 to 96 hours. Patients are encouraged to ambulate short distances while in the hospital.

All patients are kept in a semi-Fowler position (about 20 degrees of hip flexion) while in the hospital. Thigh lift patients have pillows placed between their legs to maintain abduction during the postoperative period. Intermittent compression devices applied in the operating room remain on the legs until the patient is fully ambulatory. Foley catheters are removed 1 to 2 days after surgery. For thigh lift patients, Jackson-Pratt suction drains placed deep to the lower abdominal flap are removed on the date of discharge. Voiding and bowel movements are performed from the partially standing position.

Pearls and Pitfalls

- Aesthetic complications are the most common in liposuction and include surface depressions or contour irregularities. They may be prevented by using smaller cannulas and deeper aspiration planes. Overresection should be avoided.
- Excision surgery has a higher rate of complications than does liposuction, including bleeding, infection, and seroma.
- Check frequently for seromas in the first few weeks after surgery. Once the diagnosis is confirmed by aspiration, small seromas (<20 mL) can be left to resorb spontaneously. Larger seromas should be treated aggressively with open, dependent surgical drainage. A suction catheter should be inserted in the seroma cavity and left in place until full absorption of the fluid has occurred and the seroma cavity is obliterated.
- The risk for flap necrosis and wound dehiscence is increased with smoking, extensive undermining, previous surgery, or closure under tension.
- Aggressive tightening of the rectus sheath may compromise the patient's respiratory reserve.
- Pulmonary thromboembolism is the most feared complication of abdominoplasty and excisional body lifts. Risk factors include history of obesity, multiple procedures, and lengthy operations. Lower extremity pneumatic compression boots should be worn for prophylaxis.

SUGGESTED READING

Commons GW, Halperin B, Chang CC: Large-volume liposuction: A review of 631 consecutive cases over 12 years. Plast Reconstr Surg 108:1753-1763, 2001.

Iverson RE, Lynch DJ: Practice advisory on liposuction. Plast Reconstr Surg 113:1478, 2004.

Klein JA: Tumescent technique for local anesthesia improves safety in large-volume liposuction. Plast Reconstr Surg 92:1085-1098, 1993.

Pitman GH: Liposuction and Aesthetic Surgery, 2nd ed. St Louis, Quality Medical, 2006 (in press).

Pitman GH, Aker JS, Tripp ZD: Tumescent liposuction: A surgeon's perspective. Clin Plast Surg 23:633-641, 1996.

Pitman GH, Giese SY: Liposuction. In Mathes SJ (ed): Plastic Surgery, Philadelphia, Elsevier, (in press).

Congenital Deformities

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Congenital urogenital deformities can be broadly categorized into four groups: (1) hypospadias, (2) epispadias and bladder exstrophy, (3) ambiguous genitalia, and (4) cloacal abnormalities. Although common etiologic threads run through all four conditions, the deformities are complex in their origins and treatment. The most common of the anomalies, hypospadias, occurs in approximately 1 in every 300 live male births in Western countries, with a strong familial propensity. Moreover, the incidence of hypospadias in the United States appears to have increased in recent years.

Surgical repair of hypospadias has implications for treatment of all of the aforementioned congenital malformations. This chapter concentrates on hypospadias, identification of its various components, and surgical correction of those components. Mastery of the techniques required to repair hypospadias enables the surgeon to understand the complex nature of all urogenital anomalies and to appreciate the intricate skills necessary to achieve functional and aesthetic success.

Etiopathogenesis

No single etiologic factor has been identified as the cause of all forms of hypospadias. Endocrine, genetic, and environmental influences have been investigated and implicated in the development of hypospadias in the fetus. Endocrine pathways involving the effects of progesterone, luteinizing hormone, and follicle-stimulating hormone on development of the penis have been studied. Defects in testosterone biosynthesis have been identified in association with hypospadias. In addition, absence of the frenular artery has led to speculation of a vascular cause of hypospadias. Maternal exposure to certain drugs, pesticides, and toxins such as polychlorinated biphenyls (PCBs) has been linked to the rise in incidence of hypospadias. In vitro fertilization and maternal vegetarianism have also been implicated.

A familial history of hypospadias leads to a threefold or greater incidence of hypospadias in male offspring. Genetic studies have demonstrated several allele loci associated with hypospadias.

Despite the various possible etiologic factors, hypospadias remains a largely sporadic malformation. Consequently, predisposing factors play only a minor role in the treatment algorithm of this common congenital anomaly.

Pathologic Anatomy

Hypospadias is a defect that occurs from an abnormality in development of the urethral plate. Proximal hypospadias (25%) develops as a result of failure of tubularization of the urethral plate at the 11th week of fetal development, whereas the more common glanular hypospadias (75%) is due to abnormal development of the distal fetal urethra at 16 weeks.

Although hypospadias refers to ectopic ventral placement of the urethral meatus, it is frequently accompanied by penile chordee and a dorsal hood foreskin. Penile chordee is the ventral curvature of the penis resulting from hypoplasia of the ventral urethra and corpus spongiosum. The penis is tethered by attachments of the hypoplastic tissue, which can occur in both the skin and the underlying urethral plate attachments to the corpora cavernosa. Dorsal hood foreskin is a deficiency in the ventral foreskin with a relatively normal dorsal portion that gives the glans a hooded appearance.

The three abnormalities constituting the constellation of anatomic findings often occur together. However, each may occur as a single finding or in association with one of the other abnormalities. It is

not uncommon to identify penile chordee without hypospadias or a dorsal hood foreskin. Similarly, hypospadias and a dorsal hood foreskin may occur without evidence of chordee. Proximal, rather than distal, hypospadias is more commonly associated with the presence of the other two deformities.

There have been many attempts to categorize the severity of hypospadias. An anatomic descriptive terminology is preferred: glanular, subcoronal, midshaft, penoscrotal, scrotal, and perineal. This classification system provides a visual method of describing the various degrees of hypospadias (Fig. 1).

Diagnostic Studies

Diagnostic studies have a limited role in the management of hypospadias. The anatomic issues are visible and external. However, there is an increased incidence of associated urologic anomalies, particularly in the most proximal hypospadias varieties. It is not unreasonable to obtain a screening renal and bladder ultrasound on infants born with proximal hypospadias.

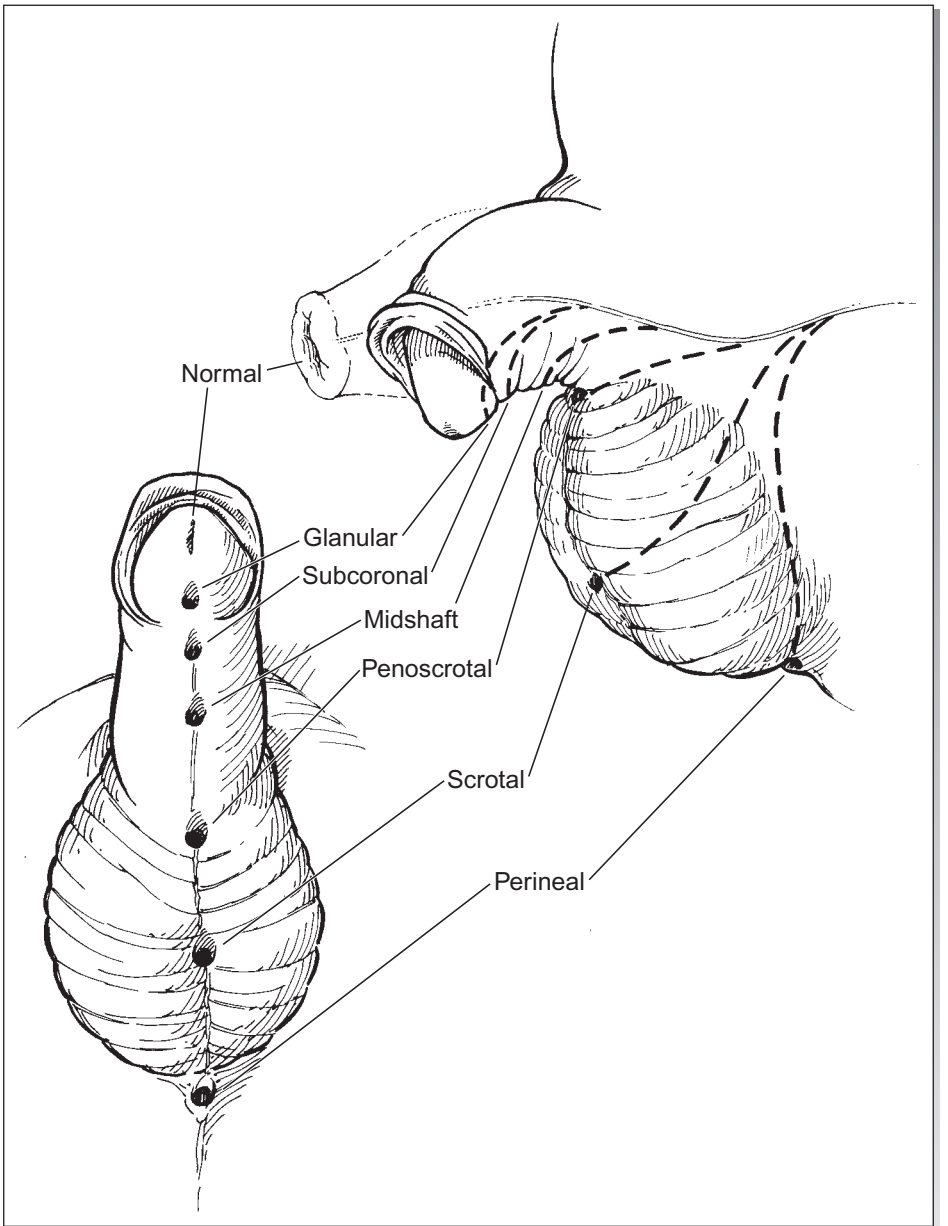

Figure 1 • Various forms of hypospadias.

Reconstructive Goals

The reconstructive goals are:

1. Placement of the meatus at the tip of the glans
2. Correction of ventral angulation of the penis (chordee)
3. Creation of a normal-appearing, circumcised penis

Treatment

Surgical repair of hypospadias has been attempted since the 19th century, with a wide range of outcomes. More than 300 types of repair have been described in the literature, a fact suggesting that no single method has achieved universal acceptance and success without complications. It is also indicative of the anatomic variations that call for different surgical approaches. Successful repair of hypospadias requires imagination, surgical skill, and experience.

During the initial patient evaluation, the parents should be urged to avoid circumcision because the foreskin plays an important role in the eventual repair.

Chordee Repair

Correcting chordee begins with degloving the penile shaft skin to the base of the penis at the level of Buck's fascia. Degloving often adequately corrects all chordee, particularly with distal hypospadias. When chordee persists, the urethral plate should be dissected and elevated from the corpora. A distinct urethral plate may not be identified. In such cases, fibrous bands along the corpora must be resected to straighten the penis. Despite all surgical efforts, chordee may persist and necessitate dorsal corporeal plication.

Urethroplasty

The primary goal of urethroplasty is to advance the urethral meatus to the tip of the glans. Once the penile chordee has been corrected, the distance between the native meatus and the glanular tip increases. Various surgical methods have been developed either to advance the urethral meatus or to create a neourethra that can be added to the native urethra. The procedure of choice depends on the preference of the surgeon and the location of the native urethral meatus.

MEATAL ADVANCEMENT AND GLANULOPLASTY. Meatal advancement with glanuloplasty (Fig. 2) was created to advance the urethra distally without actually performing urethroplasty. It is applicable only for the distal type of hypospadias.

After the penile skin is degloved, a longitudinal incision is made in the urethral plate in the ventral midline from the distal end of the native meatus to the tip of the glans. The incision is closed in a transverse direction with 7–0 Vicryl sutures and the meatus advanced distally. The separated glanular halves are approximated in the ventral midline so that they surround the advanced urethra. The glanular tissue is closed with 6–0 Vicryl suture while avoiding suture placement in the glanular epithelium to prevent obvious scarring. The penile shaft skin is reapproximated to the coronal cuff with fine catgut suture.

Although this repair avoids many of the complications accompanying formal urethroplasty, the aesthetic results are often less than desirable.

MEATAL-BASED URETHROPLASTY. More proximal hypospadias requires the creation of a neourethra that will reach the distal end of the penis. If, after correction of the chordee, the distance to be bridged is moderate and the urethral plate is intact, a meatal-based urethroplasty (Fig. 3) is an excellent option.

A longitudinal skin flap is created on the ventral surface of the penis by incising the skin on both sides of the meatus proximally. The length of the flap should be slightly longer than the distance from the meatus to the tip of the penis. Parallel incisions are carried distally along the urethral plate, extending to the glanular tip. Dissection into glanular tissue on both sides of the urethral plate creates glanular flaps that are used to cover the neourethra. The meatal-based flap is raised off the penile shaft and advanced distally, where it is sutured to both sides of the urethral plate with 7–0 Vicryl suture. A No. 6-French Silastic catheter is placed into the bladder and left as a stent. The glanular flaps are sutured to each other in the ventral midline with 6–0 Vicryl suture to cover the neourethra. The distal flap is sutured to the glanular tip with fine catgut sutures.

PREPUTIAL FLAP URETHROPLASTY. The most proximal hypospadias defects require a urethroplasty longer than can be created by a meatal-based flap. To satisfy this requirement, a vascularized preputial flap can be created and transposed ventrally as an onlay graft (if an adequate urethral plate is present) or as a tubularized neourethra (if there is not an adequate urethral plate) (Fig. 4).

The penile shaft skin is circumferentially incised, leaving a coronal cuff. If an adequate urethral plate is present, it is left intact, dissected off the corpora, and left attached to the urethral meatus proximally. The dissection often corrects the chordee. A preputial graft is created by incising a transverse rectangular flap on the undersurface of the dorsal hood foreskin. Care is taken to avoid damage to the vascular pedicle, which is dissected proximally to

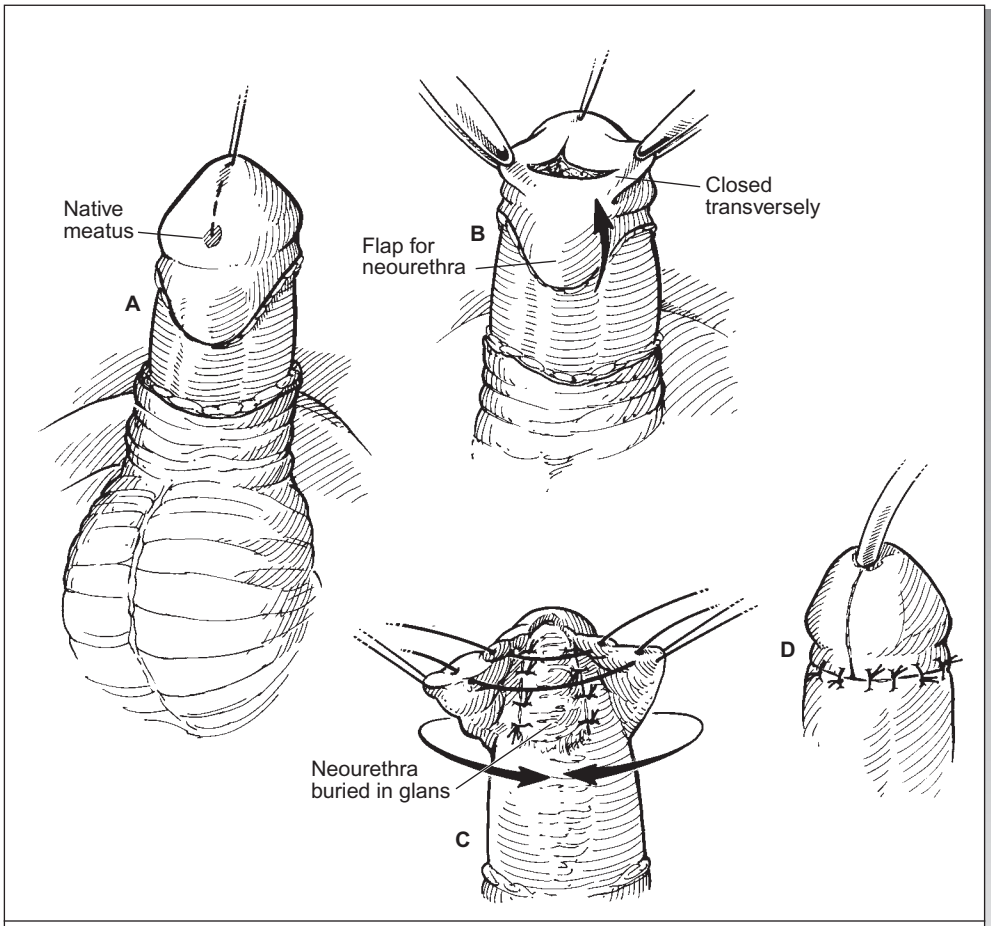

Figure 2 • Meatal advancement and glanuloplasty. **A,** An incision is made from the native meatus to the tip of the glans, which is closed transversely. The penile skin is degloved. **B,** The ventral flap is raised and brought distally. **C,** The glanular flaps are brought around the neourethra. **D,** The incisions are closed.

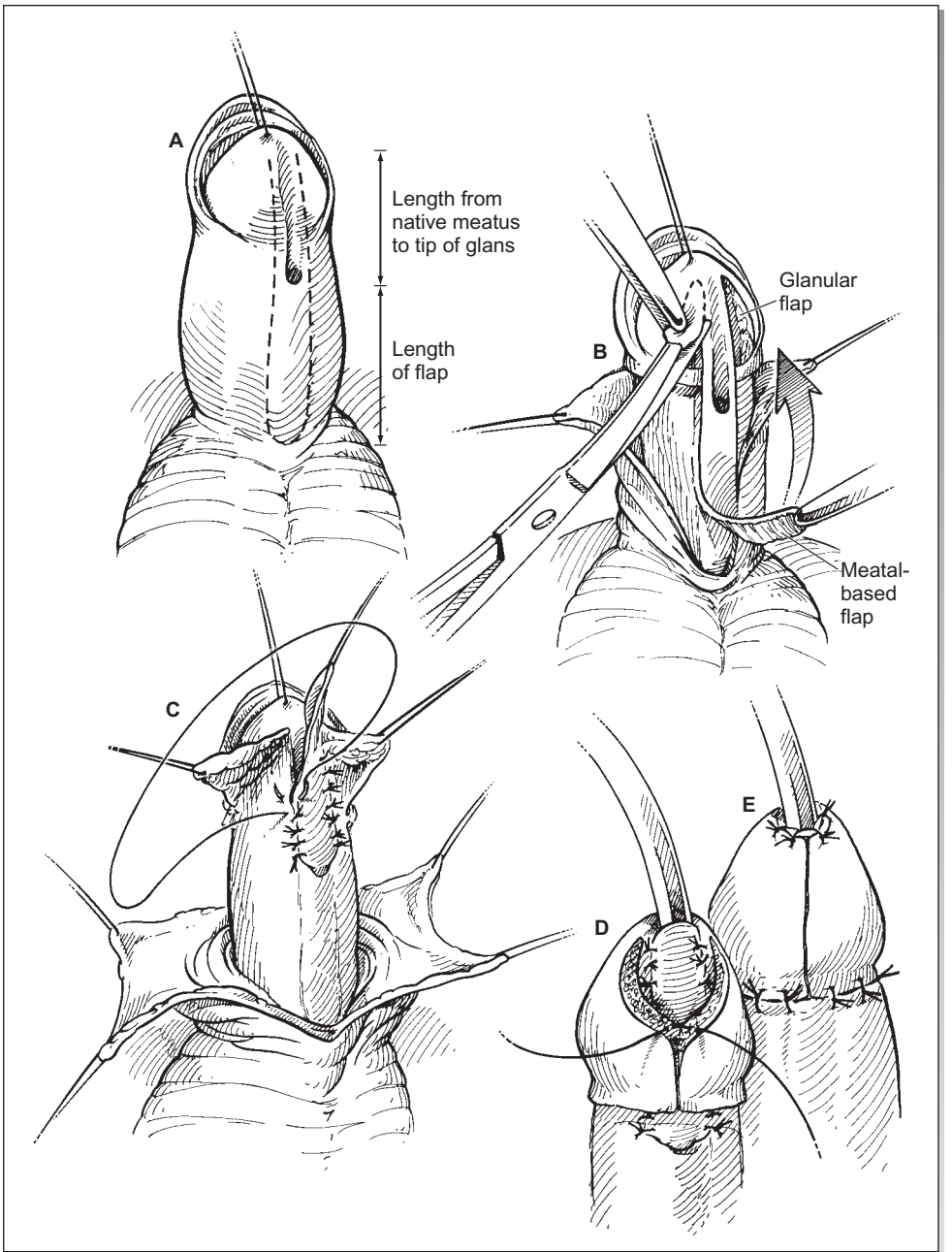

Figure 3 • Meatal-based urethroplasty. **A,** The meatal-based flap must be slightly longer than the distance from the native meatus to the tip of the glans. **B,** The meatal-based flap and glanular flaps are elevated. **C,** The meatal-based flap is advanced distally on the ventral side of the neourethra. **D** and **E,** The glanular flaps are brought around the neourethra.

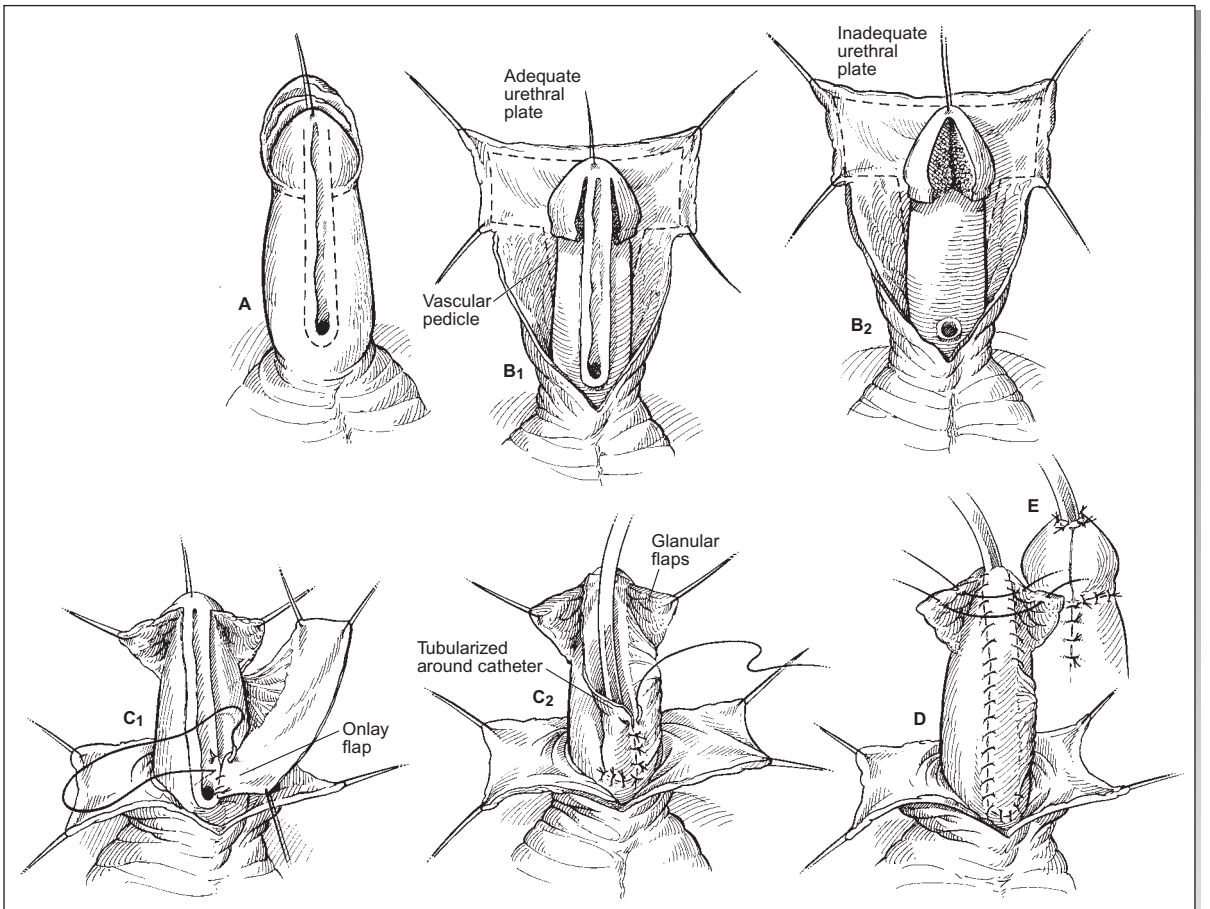

Figure 4 • Preputial flap urethroplasty. **A,** The ventral incisions are outlined. **B_1** and **B_2,** The preputial flap is elevated and based on the dorsal blood supply. **C_1** and **C_2,** The preputial flap is brought ventrally and used to reconstruct either the ventral portion (**C_1**) or the entire urethra (**C_2**). **D** and **E,** The glanular flaps are brought ventrally and the incisions closed.

the base of the penis. The graft is brought to the ventral surface. If a urethral plate is present, the graft is trimmed and used as an onlay flap and sutured to both sides of the plate with 7–0 Vicryl. The graft is tubularized around a No. 6-French Silastic catheter if there is no urethral plate. The graft is then anastomosed to the proximal end of the meatus with 7–0 Vicryl suture. The graft is either tunneled through the glans to the tip or enclosed within glanular flaps. The preputial graft is sutured to the glans with fine catgut sutures. The catheter is left in place as a stent.

TWO-STAGE PROCEDURE. Most hypospadias repairs are completed in one stage. Occasionally, however, a two-stage repair is indicated (Figs. 5 and 6). The first stage consists of correcting the penile chordee and transposing the dorsal foreskin to the ventral surface. After several months are allowed for neovascularization of the transposed foreskin, a urethroplasty is performed with the redundant ventral skin.

HYPOSPADIAS CRIPPLE. When confronted with a failed hypospadias repair, the options for creating a neourethra are limited. Buccal mucosa can be used as a urethral graft. Skin and bladder mucosa have also been described as urethral substitutes, although both have led to significant long-term complications.

Skin Coverage

Adequate skin coverage is often a difficult, but important part of the hypospadias repair. Byar's flaps are created by incising the remaining dorsal foreskin longitudinally (Fig. 7). The flaps can be brought onto the ventral surface to cover the denuded area. An attempt should be made to cover the neourethra before the skin flaps are approximated. Subcutaneous tissue from the dorsum can often be used as coverage. On occasion, the neourethra can be buried between the corpora cavernosa. Optimal coverage helps to decrease the occurrence of urethral fistula formation.

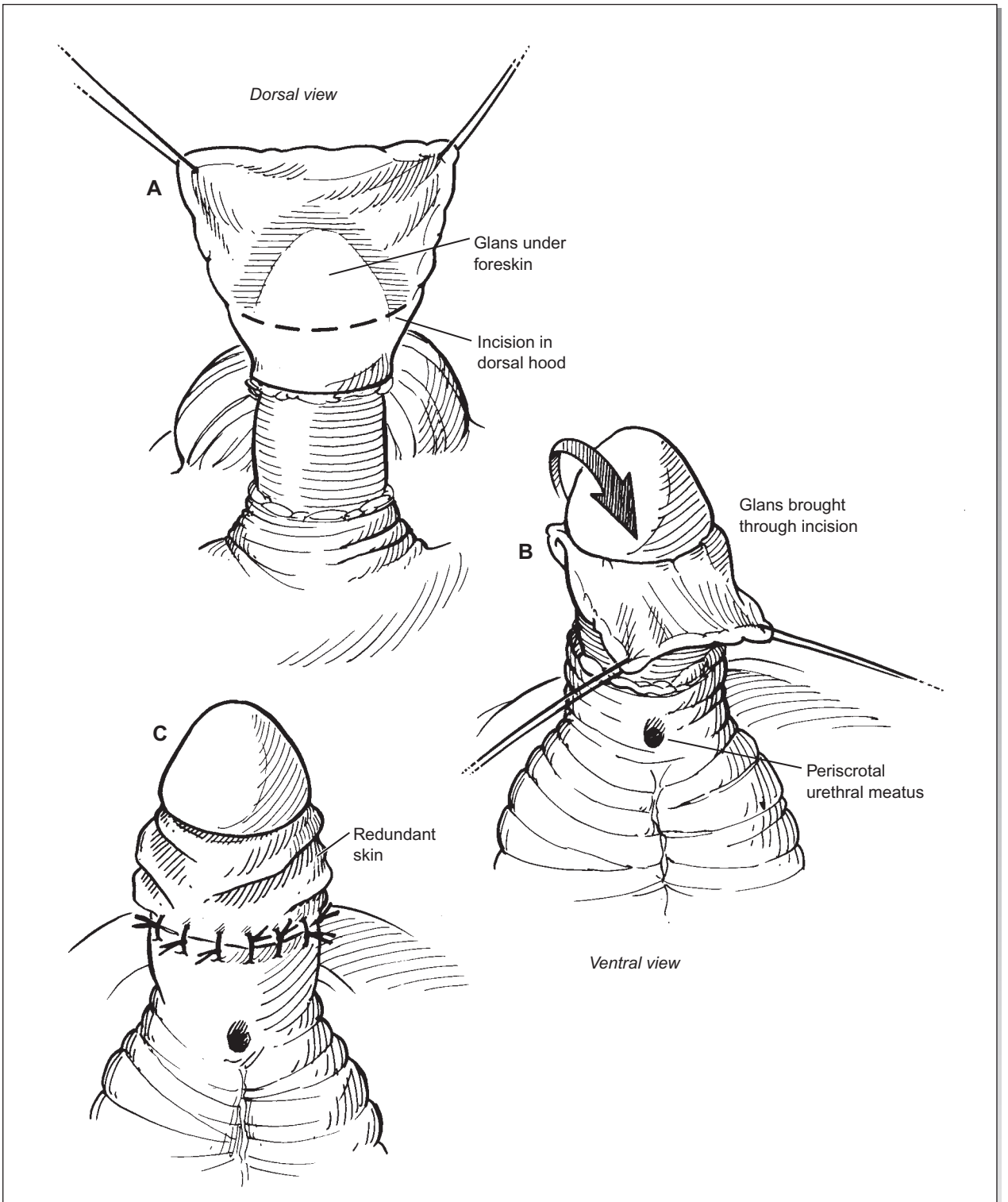

Figure 5 • First stage of a two-stage approach. **A,** Planned dorsal incisions and elevation with correction of the chordee. **B,** Glans delivered through the incision. **C,** Flaps brought ventrally to provide tissue for the second-stage repair.

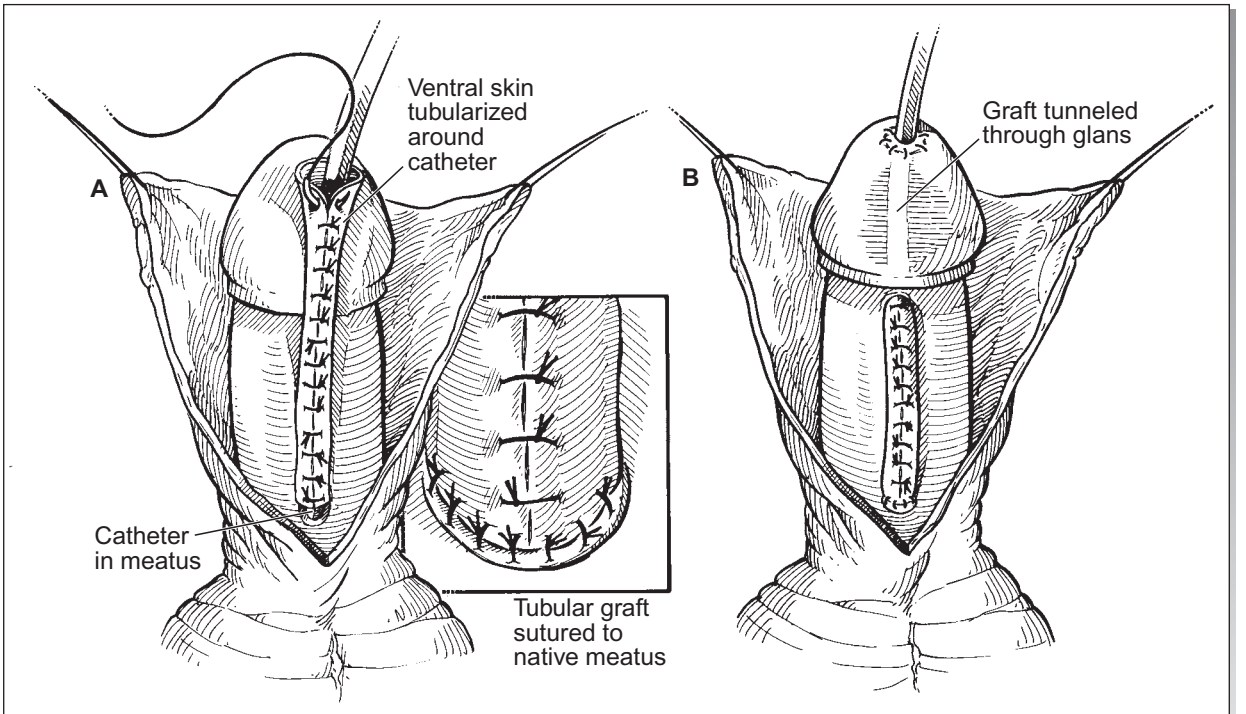

Figure 6 • Second stage of a two-stage approach. **A,** Banked flaps tubularized to form the neourethra. **B,** Neourethra tunneled through the glans.

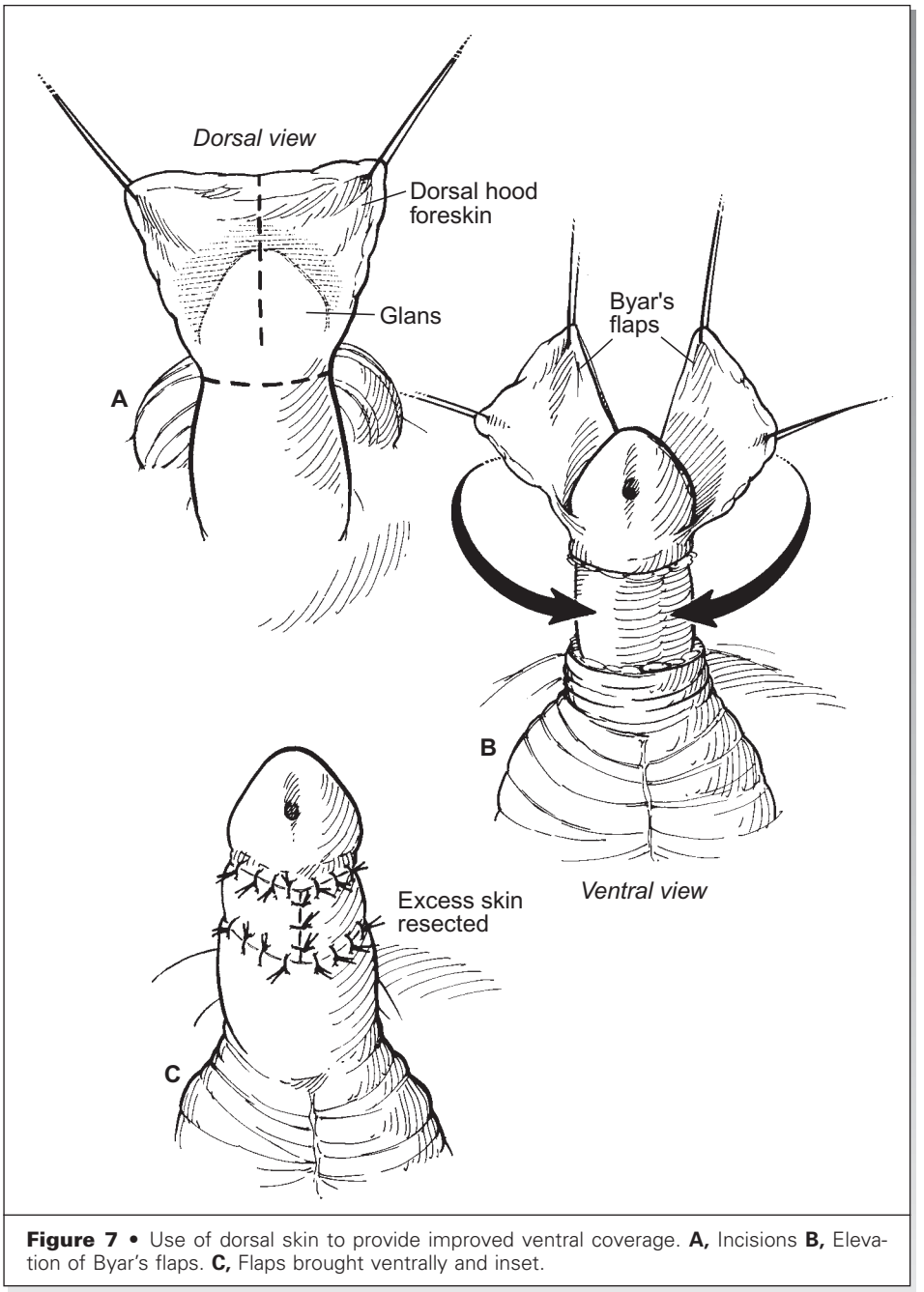

Figure 7 • Use of dorsal skin to provide improved ventral coverage. **A,** Incisions **B,** Elevation of Byar's flaps. **C,** Flaps brought ventrally and inset.

Postoperative Care

Almost all current hypospadias repairs can be performed as ambulatory procedures. However, precise parental instruction and detailed postoperative education are necessary. Urinary catheters are left in place and parents are taught a double-diapering technique in which the catheter is placed through a hole in the first diaper and rests in a second diaper. This maneuver prevents the repair from being bathed in urine until the catheter is removed.

Postoperative medication includes oxybutynin to prevent bladder spasm and prophylactic antibiotics; both are discontinued after the catheter is removed.

A simple bio-occlusive dressing is applied after the procedure. The dressing is usually removed 3 to 4 days postoperatively and the catheter 7 to 10 days postoperatively.

Complications after hypospadias repair are common and occur in up to 20% to 30% of all cases. The most common complications include the following:

1. Urethral fistula
2. Meatal stenosis
3. Urethral stricture
4. Urethrocele
5. Persistent chordee
6. Glanular dehiscence

Pearls and Pitfalls

- Hypospadias repair is an extremely technically challenging procedure. When poorly planned or executed, it can lead to disastrous results.
- Ensure adequate length and width of the urethral graft. Inadequate length produces meatal regression and stenosis. Inadequate width leads to stricture and graft breakdown.
- Multiple layers of coverage over the graft prevent urethral fistulas.
- Glanular flap elevation must be extensive to eliminate the possibility of tension on the suture line and subsequent glanular dehiscence.

SUGGESTED READINGS

Hinman F Jr: Penile plastic operations. In Hinman F Jr (ed): Atlas of Pediatric Urologic Surgery. Philadelphia, WB Saunders, 1994, pp 551–602.

Mouriquand PDE, Mure PY: Hypospadias. In Gearhart JP, Rink R, Mouriquand PDE (eds): Pediatric Urology. Philadelphia, WB Saunders, 2001, pp 713–728.

Duckett JW: Hypospadias. In Frank JD, Gearhart JP, Snyder HM III (eds): Operative Pediatric Urology, 2nd ed. London, Churchill Livingstone, 2002, pp 149–164.

Duckett JW, Baskin LS: Hypospadias. In O'Neill J Jr, Rowe MI, Grosfeld JL, et al (eds): Pediatric Surgery, 5th ed. St. Louis, Mosby, 1998, pp 1761–1781.

Skandalakis JE, Gray SW, Broecker B: The male reproductive tract. In Skandalakis JE, Gray SW (eds): Embryology for Surgeons, 2nd ed. Baltimore, Williams & Wilkins, 1994, pp 773–815.

Posttraumatic Deformities of the Perineum

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The external genitalia and perineum are unique. They are one of the few midline structures of the body, and they have specialized anatomy and function that allow for both urination and sexual activity. In addition, the external genitalia's structure and function are intimately connected to an individual's self-image and sexuality. Physicians treating patients who have sustained genital injury need not only to be well versed in the specialized anatomy and function of these structures but also to be sensitive to the inevitable psychological trauma that most patients experience after such injury.

Treatment goals are best accomplished by having a multidisciplinary team, including plastic surgeons, urologists, and psychologists, involved in each phase of treatment.

Etiopathogenesis

The findings in patients with genitourinary injury are variable. Injuries range from simple lacerations to blunt straddle injuries or severe penile avulsion/amputation injuries (Table 1).

Causes of blunt perineal trauma include assault, falls, bicycle accidents, and motor vehicle accidents. An example of blunt penile trauma is a penile "fracture," which usually requires an erection at the time of injury and is almost always caused by traumatic intercourse. Patients can have either frank fracture of the corpora cavernosum (Fig. 1) or hematoma of the penile shaft without corporal disruption. Blunt trauma to the female perineum is uncommon and may occur secondary to bike accidents and sexual assault.

Penetrating injuries may be caused by gunshot wounds, knives, or other sharp objects. It is important to obtain a complete history and ascertain the direction or trajectory of the penetrating object.

Patient Assessment

After life-threatening and other serious injuries have been addressed on primary and secondary surveys of the traumatized patient, assessment of the perineal injury should be performed. Whether evaluating acutely injured patients or those with a posttraumatic deformity, the surgeon should be able to answer questions such as the following: What structures if any are missing? What structures if any are displaced or damaged? Is it possible to replace normal parts back to their normal position? Do structures have evidence of vascular compromise or insufficiency? Is vascular repair or replantation indicated?

Diagnostic Studies

Preoperative studies may include a retrograde urethrogram for urethral injuries, a cystogram for bladder injuries, and an intravenous pyelogram for renal or ureter injuries. Rigid sigmoidoscopy should be performed if rectal injury is suspected. Computed tomography is an important diagnostic study to identify injuries of the urinary tract and to evaluate concomitant pelvic and abdominal injuries. Plain films of the pelvis may reveal pelvic fractures, which can, in turn, suggest injury to the urethra. In cases of severe pelvic trauma, an arteriogram may be necessary both to identify injury and to delineate potential recipient vessels for reconstruction. Intraoperative ultrasonography and other diagnostic studies may be used to monitor corpora cavernosum blood flow, as well as help to predict the erectile function of the injured penis.

TABLE 1 Common Causes of Genital and Perineal Injuries

Blunt trauma
Contusions
Penile fracture
Penetrating trauma
Avulsion injuries
Compressive trauma
Bite injuries
Iatrogenic trauma
Burn injuries
Scald
Flame
Electrical
Chemical
Frostbite
Self-inflicted injuries (psychiatric)

Reconstructive Goals

Two fundamental concepts should be understood with respect to all reconstructive options: quantitative deformities and qualitative deformities. *Quantitative* deformities are defined by the specific anatomic and functional abnormalities caused by the injury. *Qualitative* deformities are those that may not cause functional impairment, but rather cause emotional and psychological turmoil.

Although the idealized goals of genital reconstruction are to reestablish normal function and appearance, this is not always possible. It is important for the reconstructive surgeon to understand the limitations of surgery and have realistic goals. The surgeon needs to present this information to the patient and together determine a prioritization list of potentially achievable goals. There are some patients whose primary goal is to be able to urinate in a normal fashion. In many male patients, standing to void is an extremely important function, especially in certain work environments, as well as in some social situations. Other patients prioritize sexual function as the most important function to reestablish. Some patients have an overwhelming psychological need to "just have something there." This is true for both genders, whether it be the semblance of a vulva and not necessarily a functional vaginal orifice in a female or a nonfunctioning semblance of a penis and scrotum in a male.

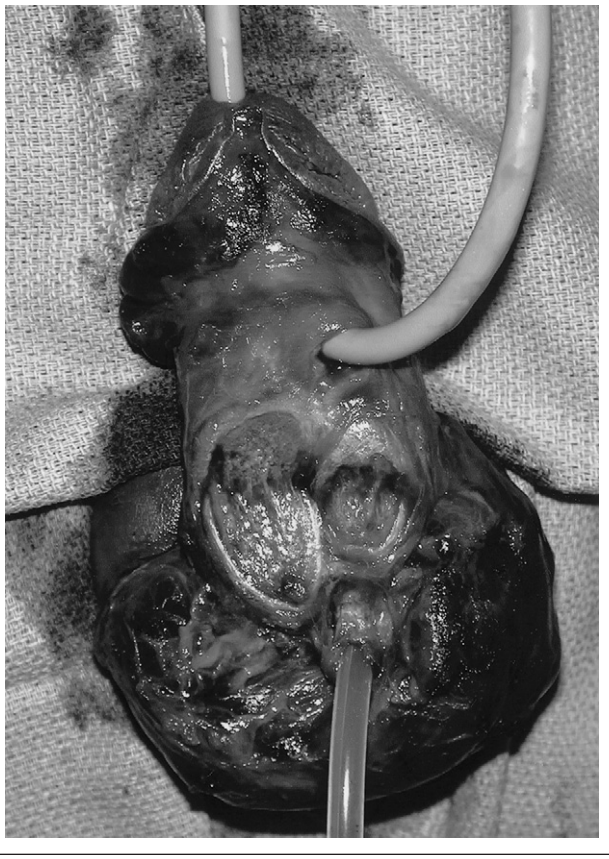

Figure 1 • Penile fracture after blunt trauma.

Treatment

The vital perineal structures likely to be injured in trauma include the penis, urethra, scrotum, and testicles in men and the vulva and urethra in women. The vagina is only rarely injured with trauma, and therefore its reconstruction is not discussed. It should be pointed out, however, that with vaginal injury or posttraumatic vaginal deformity or stricture, standard regional skin flaps cannot generally be used because of their bulkiness, unlike reconstruction after vaginal extirpation for cancer.

General Principles of Reconstruction

Resurfacing extensive areas of skin cicatrix of the perineum and genitalia is a challenge. When the undesirable scar is not extensive and is adjacent to adequate normal skin, tissue expansion is a reasonable technique of obtaining qualitatively normal skin with minimal donor site deformity. However, expanders in the perineal region tend to be associated with a high complication rate. In addition, it is mechanically difficult for patients to tolerate the "bulge" of an expander in the perineum because it interferes with clothing, walking, sitting, and normal daily activities. An alternative to expansion of adjacent flaps is expansion of full-thickness grafts. This technique allows for large full-thickness skin grafts to be harvested with minimal donor site morbidity.

Tightness, webbing, and distortion of the normally mobile male genitalia result from a lack of skin. Vulvar injuries with subsequent webbing may

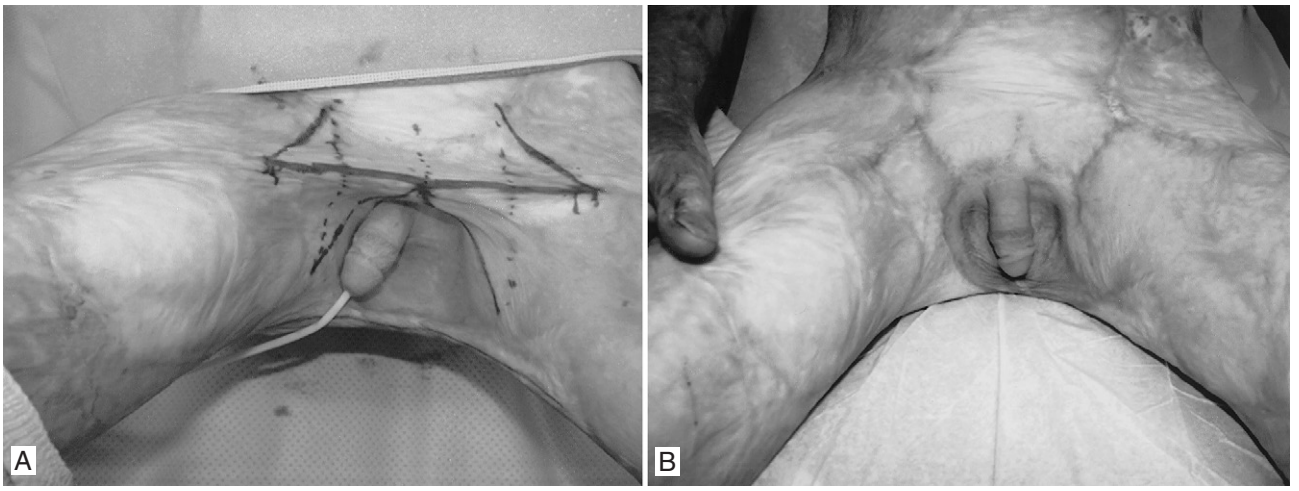

Figure 2 • A and **B,** Double opposing Z-plasty to reduce severe postburn perineal webbing.

compromise normal sexual activity and cause difficulty with urination. If there is pliable, relatively normal adjacent skin, one of the transposition (Z-plasty, W-plasty, etc.) or advancement (V-Y) flap techniques may be used (Fig. 2). This technique works best if normal tissue can be interposed to "break up" the scar contracture. When normal adjacent skin is not available because of extensive scarring, adequate contracture release may not be possible with local flaps alone. Additional skin grafting is frequently necessary to fully correct the contracture or webbing.

The ideal time for contracture release is after active contraction is complete and maximum scar maturation has occurred, usually at least 6 months and up to 2 years after injury. On occasion, early contracture release may need to be performed because of severe functional disability.

Penile Reconstruction

Posttraumatic reconstruction of the penis is generally directed toward correction of deformities caused by scar contracture or tissue loss. The scar contracture may occur via extrinsic contracture; however, more commonly, intrinsic contracture occurs. Simple extrinsic contracture of the penis may lend itself to Z-plasty or surgical release and grafting of the adjacent tethered skin. Intrinsic contracture of the penis may result from injury and subsequent scarring of penile skin, dartos fascia, Buck's fascia, tunica albuginea, or the corpora themselves. The resultant scar causes the penis to deviate from its normal straight vector when it becomes tumescent. Treatment is similar in concept to that of secondary hypospadias repair or Peyronie's disease. The basic principle is to release the scar tissue by surgical incision or, in selected cases, by excision. If the injury is superficial, local flaps or skin grafts are adequate. If the cause is a deficiency or scarring of Buck's fascia or the tunica albuginea, an autogenous dermal graft may be necessary to reconstruct this layer. Plication of the tunica (Nesbit tuck) on the convex side of a penile bend may be helpful in some patients with mild lateral bending. The resultant scarring from burn injury of the penis frequently gives the illusion that it is severely foreshortened or absent. The penis may have retracted into pubic fat or become tethered as a result of scar. Releasing the scar, grafting the skin defect on the penile shaft, and removing some fat from the mons usually improves this deformity.

With complete or nearly complete penile loss, total reconstruction should be considered. For more than 6 decades, many penile reconstructive techniques have been described in which tissue from the torso and extremities was used. Ideally, the reconstructed penis should be aesthetically acceptable with a shaft of adequate length and simulation of a glans. It should have close to normal tactile and erogenous sensitivity. Its girth should be adequate for placement of an erectile prosthesis but not too large to preclude sexual intercourse. The reconstructed penis should include a neourethra that has the potential to be anastomosed to the patient's residual native urethra, thus allowing the patient to stand to void through a meatus in the glans. The reconstructive technique that best fulfills the ideal criteria is the neurosensory radial artery forearm free tissue transfer. The thin flap incorporates a vascularized neourethra by fashioning a "tube in a tube." Coapting the antebrachial cutaneous nerves to the stump of the penile or pudendal nerves allows for the development of protective and erogenous sensation. Its design allows for insertion of an erectile prosthesis, but it is not usually as bulky as other reconstructive options. The forearm donor site is generally covered with a skin graft. Subtotal penile

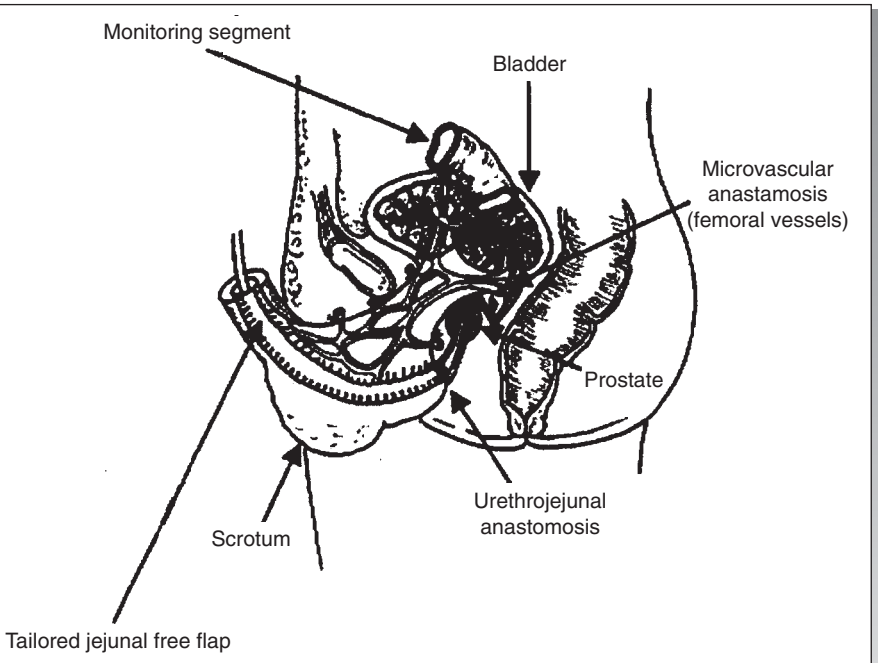

Figure 3 • Jejunal free tissue transfer. The mesenteric vessels are anastomosed to the femoral vessels. The proximal jejunum is tapered longitudinally to allow for end-to-end anastomosis with a proximal urethral remnant. A distant segment of jejunum is exteriorized for postoperative flap monitoring.

reconstruction may also be managed by radial forearm free tissue transfer, with the proximal stump of the native penis serving as the base for the flap. It should be noted, however, that placement of an erectile prosthesis affects functioning of the remaining corpora.

Urethral Reconstruction

Injuries involving the urethral meatus may cause stenosis. Mild stenosis can be treated by repeated dilatation. Stenosis unresponsive to dilation may require Z-plasties or other flap interdigitation of adjacent skin into the constricting band. Distal urethral loss may be repaired by using the principles of hypospadias surgery (see the chapter "Congenital Urogenital Deformities"). Modifications may be necessary, depending on the availability of foreskin and the amount of adjacent scarring.

The techniques of more proximal urethral reconstruction continue to evolve. Many methods of restoring urethral continuity and establishing a normal urethral lumen have been devised. Various autologous grafts and local/regional flaps allow dependable long-term results to be achieved, depending on the quality of the recipient bed. Scarring from previous surgery or trauma may preclude the use of local or regional flaps. In addition, reconstructing the proximal end of the male urethra may push the limits of some of these techniques. When grafts and regional flaps are inappropriate, inadequate, or unavailable, free tissue transfers may be the best option. A tailored intestinal free tissue transfer should be considered for the treatment of extensive urethral abnormalities when the only alternative is chronic supravesical diversion (Fig. 3). The decision regarding which of the various methods available to use for urethral reconstruction must be individualized. Factors used in choosing the appropriate option include the location and dimension of the injured or diseased urethra, the quality of the urethral bed, the availability of local or regional tissue, and the quality and availability of distant tissue.

Scrotum and Vulva Reconstruction

The scrotum and vulva are derived from the same embryologic tissue. The differences are basically that the scrotum descends further than the vulva and fuses to form the median raphae and the vulva forms the vaginal introitus. In addition, the testicles migrate down to fill the scrotum. Reconstruction is guided by what tissue is injured or lost. If only skin is lost, skin grafts can be used successfully. Small to moderately sized skin defects may be treated conservatively by allowing the wounds to heal by secondary intention. The skin of the scrotum is quite forgiving in that large areas of loss frequently heal by wound contraction to produce a satisfactory result. If deeper tissue is destroyed, a semblance of either a scrotum or vulva may be reconstructed with

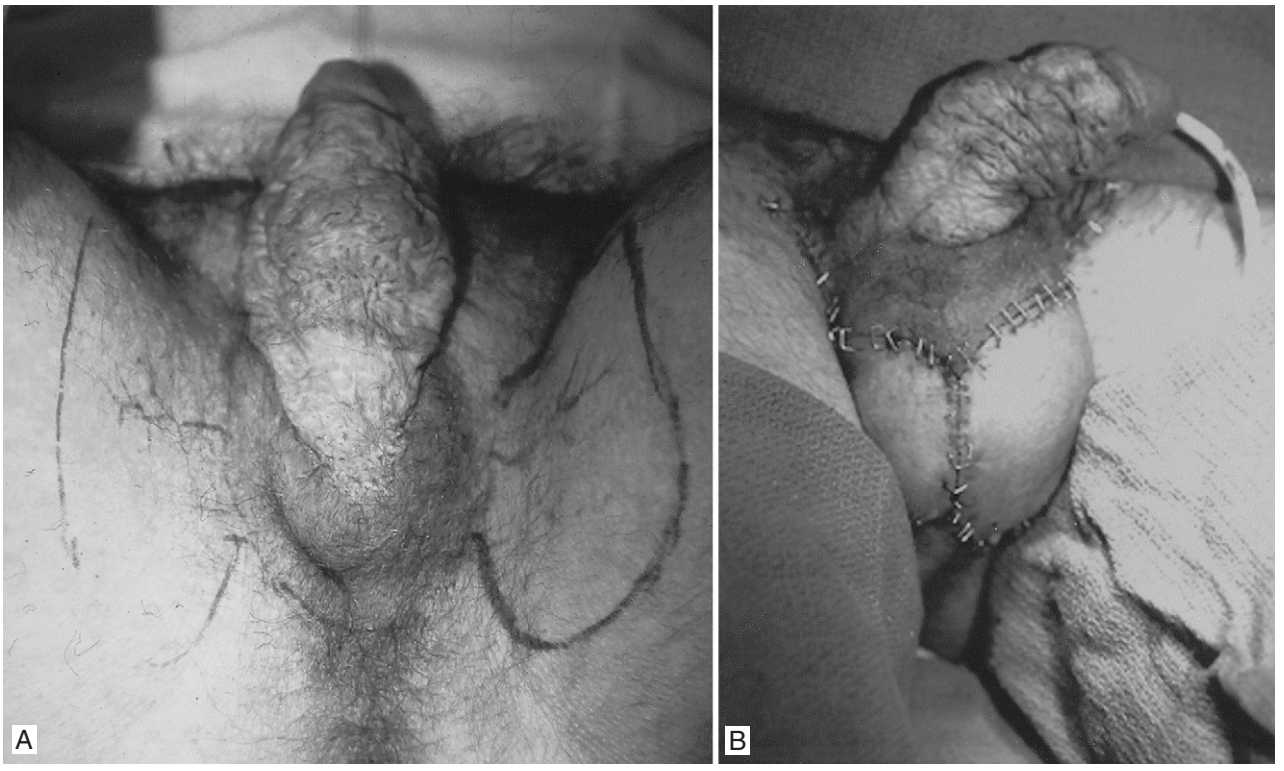

Figure 4 • A and **B,** Bilateral medial thigh flaps based on the pudendal artery system may be used for scrotal reconstruction. The subcutaneous fat of the flaps may give the impression of a normal scrotum with contained testicles.

regional flaps, usually derived from the skin supplied by the pudendal artery system (Fig. 4). A semblance of testicles may be created with the fat of the pudendal artery flaps or with the use of silicone prostheses.

Pearls and Pitfalls

- Reconstruction of the perineum should be guided by using the basic principles and concepts of plastic and reconstructive surgery and by having intimate knowledge of the regional anatomy and recognition of what tissue has been lost or injured.
- Other than revascularization, replantation, urinary tract restoration, and techniques for wound closure, reconstruction should generally wait until the patient has recovered from the initial injury, not infrequently at least 3 to 6 months.
- The main drawback of using the radial forearm free flap for penile reconstruction is the unsightly donor site. This deformity can be improved by using a thick split-thickness skin graft to close the donor wound.
- Virtually all penile prostheses erode through the skin if inserted in an insensate flap. Development of protective sensation is important to minimize the chance of pressure necrosis. Adequate sensation to protect against erosion generally requires 6 to 9 months.
- It is important that the patient and family have realistic expectations. The limitations of reconstruction should be discussed with the patient during the acute phase of care and should be continued throughout the reconstructive process. The dialogue should include discussion of the expected appearance, as well as urinary and sexual function.

SUGGESTED READINGS

Bales GT, Kuznetsov DD, Kim HL, Gottlieb LJ: Urethral substitution using an intestinal free flap: A novel approach. J Urol 168:182, 2002.

Bertini JE Jr, Corriere JN Jr: The etiology and management of genital injuries. J Trauma 28:1278, 1988.

Brandes SB, Buckman RF, Chelsky MJ, Hanno PM: External genitalia gunshot wounds: A ten-year experience with fifty-six cases. J Trauma 39:266, discussion 271, 1995.

Castanheira AC, McAninch JW; Reconstruction for penile trauma. In Webster G (ed): Reconstructive Urology. Boston, Blackwell, 1993, p 967.

Gomes CM, Ribeiro-Filho L, Giron AM, et al: Genital trauma due to animal bites. J Urol 165:80, 2001.

Gottlieb LJ, Levine L: A new design of the radial forearm free flap phallic construction. Plast Reconstr Surg 92:276, 1993.

Gottlieb LJ, Saunders JN: Perineal burns. In Achauer B (ed): Burn Reconstruction. New York, Thieme, 1991, p 165.

Levine LA, Gottlieb LJ: Phallic reconstruction for trauma. In Webster G (ed): Reconstructive Urology. Boston, Blackwell, 1993, p 967.

McAninch JW, Kahn RI, Jeffrey RB, et al: Major traumatic and septic genital injuries. J Trauma 24:291, 1984.

Michielsen D, Van Hee R, Neetens C, et al: Burns to the genitalia and the perineum. J Urol 159:418, 1998.

Vaginal Reconstruction

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NAVIN K. SINGH
NELSON H. GOLDBERG

Reconstruction of a vaginal defect is a difficult challenge, not only because of the complex anatomy involved but also because of the significant emotional, psychological, and sexual implications. In addition to the standard reconstructive goals of obtaining a closed wound, obliterating dead space, and providing healthy vascularized tissue, the surgeon must create a sensate, functional vagina that is capable of sexual intercourse while externally resembling normal female genitalia. These goals require a team approach, including a plastic surgeon, gynecologist, psychiatrist, and sex therapist.

Congenital Vaginal Agenesis

Reconstruction of a congenitally absent vagina requires the creation of a stable, lined space between the bladder and rectum. Split-thickness skin grafts have been used by surgeons as far back as the late 19th century. To minimize the inevitable contracture of the split grafts, Sadove and Horton have described the use of full-thickness skin grafts. Skin grafting procedures should be delayed until the patient has the motivation to comply with a postoperative stenting and dilation regimen. These procedures should be performed at as young an age as possible to allow normal psychosexual development.

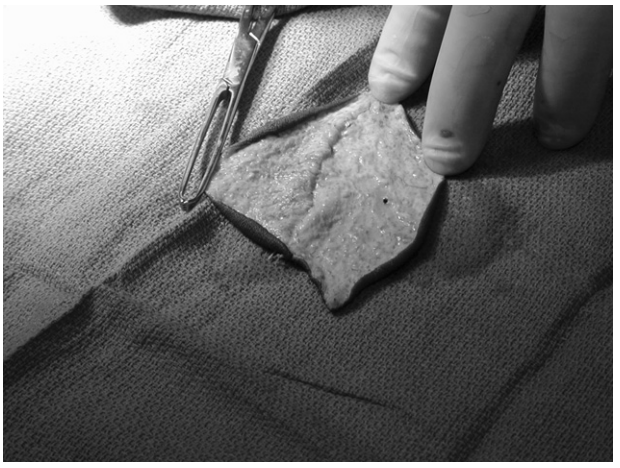
The authors' preferred technique is that of full-thickness skin grafts (Fig. 1). First, a space must be created between the bladder and rectum. Full-thickness skin grafts should be harvested bilaterally either from the lateral (hairless) groin crease or from a lower abdominal crease. The skin grafts should be sutured together over a stent. Many stents can be used for supporting vaginal skin grafts, including condoms stuffed with foam, cotton or gauze sponges over Xeroform, or even commercially available adjustable vaginal stents. An inferiorly based, inverted "V" incision is made in the introitus. The "V" flap is sutured inside the newly created space, followed by insertion of the stent covered with the skin grafts sutured together. The outer edges of the graft are sutured to the "V" flap and the remaining opening of the introitus. A suprapubic tube is recommended to avoid erosion into the urethra as a result of pressure from the stent.

After 5 days, the first stent change is performed under anesthesia. Subsequent dressing changes can be performed without anesthesia. A regimen of daily stent changes, sitz baths, and douching is prescribed for approximately another 3 to 4 weeks. After the first month, a nighttime dilator is used to maintain the space for an additional 2 to 3 months.

Although graft contraction is certainly diminished by using full-thickness grafts, there is still some contraction of the neovagina over time if the space is not maintained by either a dilator or frequent intercourse. In patients not compliant with a dilation regimen, the introitus can contract significantly and make it difficult to introduce a small dilator. For these patients, we have designed a custom-made dilator with a tissue expander. It can be introduced even through a very small opening, followed by gradual inflation of the expander with saline injections. If necessary, labial sutures can be placed to hold the expander in place.

Acquired Vaginal Defects

The vast majority of acquired vaginal defects requiring reconstruction result from surgical resection for malignancy. The defect created is often large, and many of the patients receive postoperative radiation treatment; therefore, flaps are generally recommended over skin grafts. Cordeiro and colleagues



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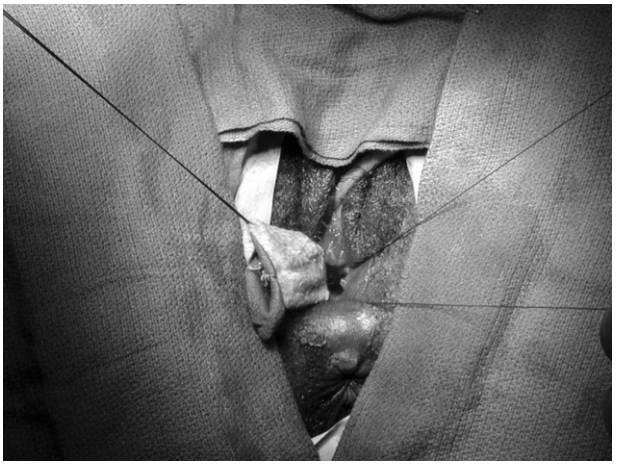

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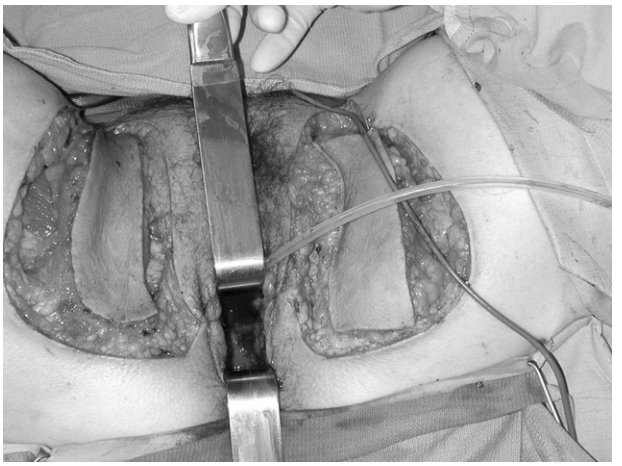
Figure 1 • Full-thickness skin graft technique. **A,** Full-thickness skin grafts from both groins sutured together. **B,** Inserting of the tubed skin grafts.

created a classification system for vaginal defects and generated an algorithm to assist in the selection of an appropriate flap. In general, for small or partial vaginal defects, a fasciocutaneous flap based on the pudendal artery (*Singapore flap*) is recommended, whereas for larger defects, often associated with pelvic exenteration, a myocutaneous flap (either the *rectus abdominis* or *bilateral gracilis*) is preferred.

Singapore Flap

The Singapore flap, first described by Wee and Joseph and later modified by Woods and coworkers, is a fasciocutaneous flap based on the posterior labial arteries. The flap can remain at least partially sensate based on innervation from pudendal nerve branches, as well as branches of the posterior cutaneous nerve of the thigh.

This posteriorly based flap is centered over the medial thigh crease just lateral to the labia majora (Fig. 2). The flap measures 15 × 6 cm with the long



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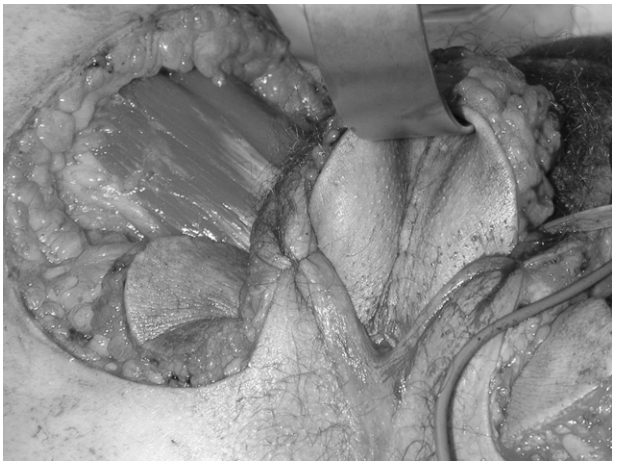

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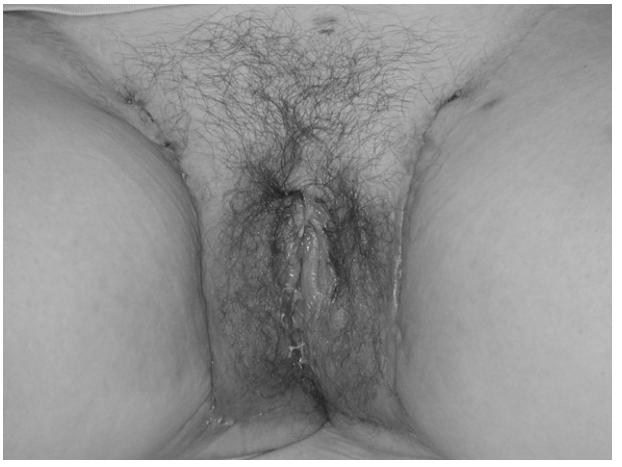


C

Figure 2 • Singapore flap. **A,** Bilateral island flaps centered over the medial thigh crease. **B,** Insetting of the flaps. **C,** Postoperative view of the donor site scars.

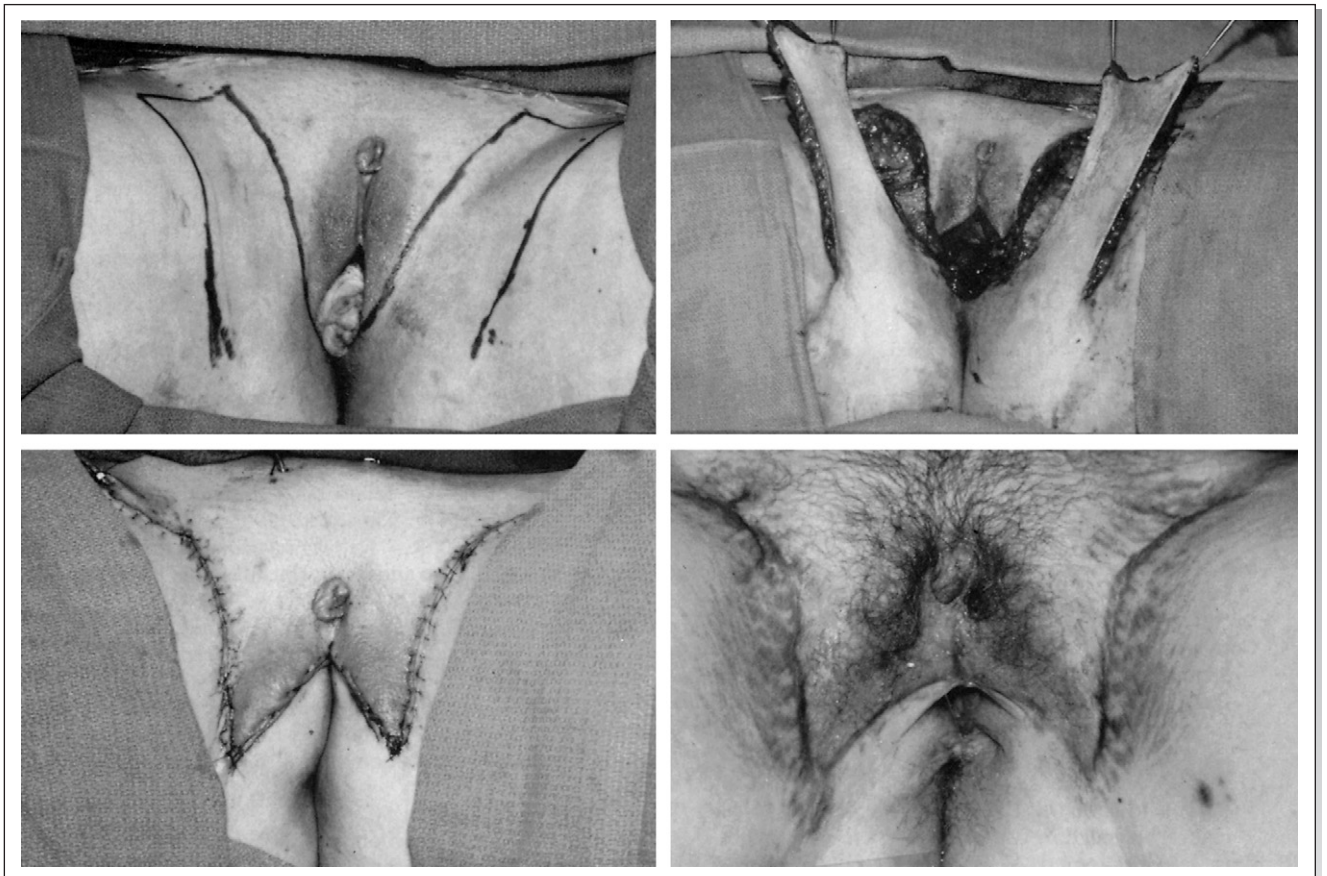


Figure 3 • The Woods modification of the Singapore flap. Note that the inferior edge of the flap is not divided. Instead, the labia are released and allowed to retract anteriorly. (From Woods JE, Alter G, Meland B, Podratz K: Experience with vaginal reconstruction utilizing the modified Singapore flap. Plast Reconstr Surg 90:273, 1992.)

axis parallel to the medial thigh crease. The flaps are elevated in an anterior-to-posterior direction and include the deep fascia of the thigh adductors. The medial, lateral, and anterior incisions are extended through the skin, subcutaneous layers, and deep fascia of the thigh adductors. A posterior incision is extended through skin and subcutaneous tissue to create an island flap. The island flap is rotated approximately 90 degrees and passed through a tunnel deep to the labia majora. In Woods and colleagues' modification, however, the posterior margin of the labia majora is released; there is no posterior skin incision (Fig. 3). The labia are allowed to retract anteriorly and the flaps are rotated into position. In both techniques, the flaps are sutured together and inserted into the defect, with the apex of the flaps sutured to the sacrum (in total exenteration defects) or to any other pelvic structure. The patient is kept in bed postoperatively with the thighs adducted for at least 2 to 3 weeks.

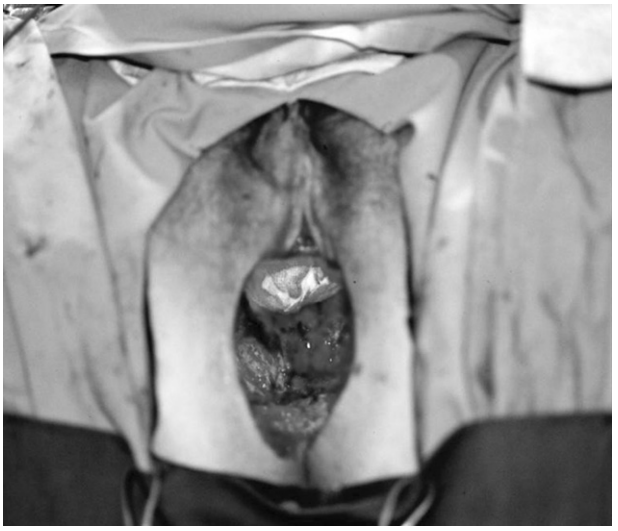
The island flap technique (Wee and Joseph) has the advantage of better-positioned scars and thus more normal-appearing labia. There is a slight risk of flap loss because of the potential for external pressure at the base of the flaps from the tunneling. The Woods modification allows for a more reliable flap, but with a somewhat distorted appearance of the labia. The authors generally prefer the island flap technique; however, the Woods modification should be considered in patients who are at an increased risk for wound complications.

The main advantages of the Singapore flap are that it provides sensation to at least the anterior portion of the flaps and does not require an abdominal incision. The major disadvantage is that it does not provide a large amount of bulk and may therefore not be appropriate for pelvic exenteration patients.

Rectus Abdominis Myocutaneous Flap

For large pelvic exenteration defects, the first choice for reconstruction is the rectus abdominis myocutaneous flap. Based on the inferior epigastric artery, it is extremely reliable and can also provide a large amount of bulk with minimal donor site morbidity (Fig. 4).

A vertically oriented elliptical skin island is designed over the proximal half of the muscle starting approximately 5 cm below the costal margin and extending to a position 3 cm below the umbilicus. The skin island should be 10 to 12 cm wide. The



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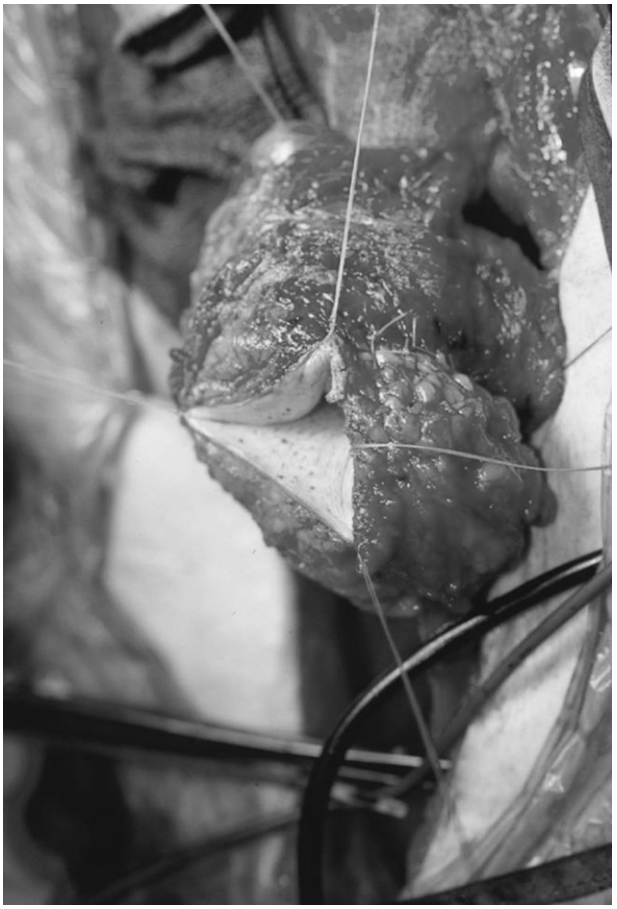

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Figure 4 • Rectus abdominis myocutaneous flap. **A,** The pelvic defect after tumor resection. **B,** The flap tubed to form the neovagina.

remainder of the rectus abdominis can be exposed through a midline incision or paramedian incision. The entire anterior rectus fascia should be taken with the muscle at the level of the skin island, but distally the muscle should be elevated through an incision in the midline of the rectus sheath to spare the fascia. It is usually unnecessary to disengage the muscle from its pubic attachment because such attachment can be helpful in avoiding undue tension on the vascular pedicle.

The anterior rectus donor fascia defect can occasionally be closed primarily in patients with relaxed abdominal walls. Alternatively, the cephalic edge of the anterior rectus fascia, which has been closed primarily caudal to the skin island, can be sutured to the posterior rectus fascia because the skin island is cephalad to the linea semicircularis. This technique allows primary fascial closure, which can be reinforced by an onlay polypropylene mesh. The skin can be closed primarily with undermining.

The skin island is tubed to form the neovagina by suturing the medial, lateral, and caudal skin edges together and leaving the cephalic edge open to be sutured to the remaining perineal skin. The flap can be transposed into position by either tunneling under the skin or connecting the abdominal incision to the pelvic incision. Multiple closed suction drains should be placed in both the donor site and the recipient site to obliterate dead space and prevent seromas. The patient is kept in bed postoperatively (with the thighs adducted) for at least 2 to 3 weeks.

Although the rectus abdominis myocutaneous flap is very reliable and can provide a large amount of healthy tissue for the wound, it has the obvious disadvantages of being insensate and requiring abdominal incisions.

Gracilis Myocutaneous Flap

Because use of the rectus abdominis is not always feasible, an alternative reconstructive option is that of bilateral gracilis myocutaneous flaps based on the ascending branch of the medial circumflex femoral artery, a branch of the profunda femoris (Fig. 5). The flap has the disadvantage of having a somewhat unreliable skin paddle, particularly in overweight or elderly patients with loose and redundant medial thigh skin. Nevertheless, it is still a reasonable option for vaginal reconstruction in patients in whom more bulk is required than can be provided by a fasciocutaneous flap or in whom the rectus is unavailable. Furthermore, this flap should be strongly considered in patients who have had their ablative surgery performed through a perineal incision only because eliminating abdominal incisions decreases the risk for pulmonary complications.

The gracilis is palpated (from the pubis to the medial tibial tubercle) and marked with the patient standing upright to be certain that the skin island is centered over the muscle. The patient is placed in the lithotomy position, and the vascular pedicle is

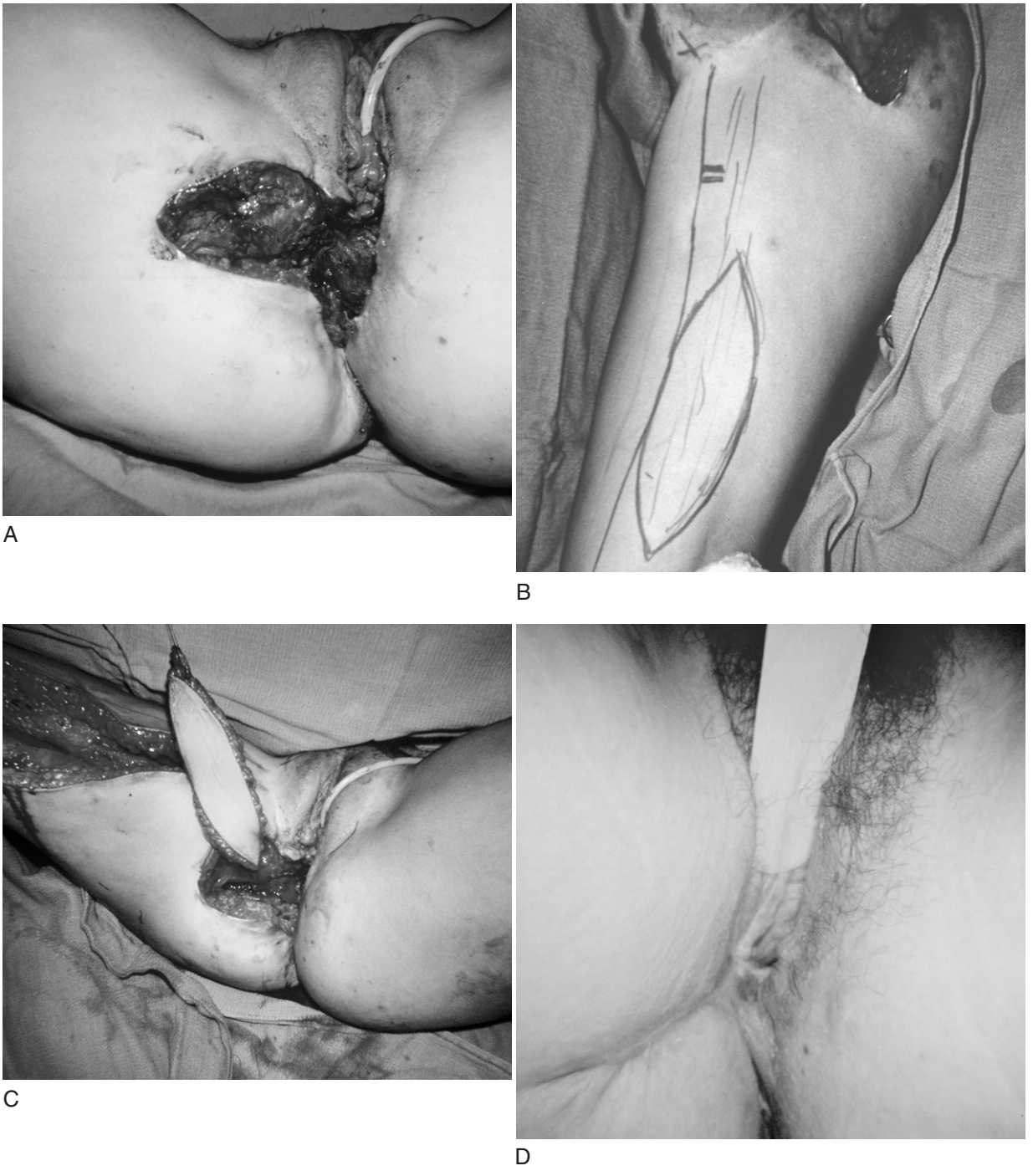

Figure 5 • Unilateral gracilis myocutaneous flaps. **A,** Preoperative view showing a partial vaginal defect. A Singapore flap was not possible because of extension of the defect onto the medial aspect of the thigh. **B,** Markings for the gracilis myocutaneous flap. "X" marks the public tubercle and the double lines mark the vascular pedicle. **C,** The flap being rotated into position. **D,** Postoperative view showing a healed wound and patent neovagina.

identified by Doppler and marked on the skin. The pedicle usually enters the muscle 8 to 10 cm below the pubic tubercle. The skin island, which can be used only over the proximal two thirds of the muscle, measures approximately 6 × 15 cm. The muscle is elevated distally to proximally. Several minor vascular pedicles originating from the superficial femoral artery must be divided. The distal incision is made, and the muscle is identified and divided. The skin island is incised down to the deep fascia of the thigh, and the fascia is elevated until the edge of the gracilis is visualized. The dermis should be sutured to the fascia edge temporarily during elevation to avoid unintentional undermining of the flap. The muscle should be elevated to the level of the dominant vascular pedicle. It is generally not necessary to release the origin of the muscle for insetting of the flap.

The flaps are tunneled beneath the skin of the medial thigh crease and sutured to one another, with the most anterior edge left open. The flaps are inset into the defect. The open edge of the skin island is sutured to the remaining perineal skin. Postoperative care is similar to that for the rectus abdominis flap.

Advantages of this flap include a sufficient amount of bulk for most exenteration defects and avoidance of abdominal incisions. Disadvantages include large medial thigh scars, an insensate flap, and relatively unreliable skin islands.

Pearls and Pitfalls

- Most cases of congenital vaginal agenesis can be managed by full-thickness skin grafts, provided that the patient is able to comply with the prolonged process of dilation and stenting.
- In the case of acquired defects, which are most often a result of tumor ablation, the defect is generally too large for a skin graft, and a flap is required.
- For smaller defects, the Singapore flap is the best option because of its ability to maintain partial sensibility.
- Patients with pelvic malignancy require soft tissue bulk that is best provided by insensate myocutaneous flaps. The rectus flap has a much more reliable skin island and is generally the first choice.
- Many postexenteration patients already have an abdominal incision. In patients without an abdominal incision, the gracilis myocutaneous flap is a satisfactory option.
- With the gracilis flap, the surgeon must keep in mind the large medial thigh scars, as well as the relative unreliability of the attached skin islands.
- It should be noted that the use of bowel to reconstruct the vagina, either as a sigmoid loop or as a free jejunal segment, has been described and should be kept in mind as an alternative method. Bowel is not the preferred choice of the authors because of patient complaints about excessive mucus formation and odor. In addition, it does not provide adequate bulk for large defects.

SUGGESTED READINGS

Abbe R: A new method of creating a vagina in cases of congenital absence. Med Rec NYU 54:836, 1898.

Concannon MJ, Croll GH, Puckett CL: An intraoperative stent for McIndoe vaginal construction. Plast Reconstr Surg 91:367, 1993.

Cordeiro PG, Pusic AL, Disa JJ: A classification system and reconstructive algorithm for acquired vaginal defects. Plast Reconstr Surg 110:1058, 2002.

Emiroglu M, Gulton SM, Adanali G, et al: Vaginal reconstruction with free jejunal flap. Ann Plast Surg 36:316, 1996.

McIndoe A: The treatment of congenital absence and obliterative conditions of the vagina. Br J Plast Surg 2:254, 1950.

Pratt JH, Smith GR: Vaginal reconstruction with a sigmoid loop. Am J Obstet Gynecol 96:31, 1966.

Sadove RC, Horton CE: Utilizing full thickness skin grafts for vaginal reconstruction. Clin Plast Surg 15:443, 1988.

Tobin GR, Day TG: Vaginal and pelvic reconstruction with distally based rectus abdominis myocutaneous flaps. Plast Reconstr Surg 81:62, 1988.

Wee JTK, Joseph VT: A new technique of vaginal reconstruction using neurovascular pudendal-thigh flaps: A preliminary report. Plast Reconstr Surg 83:701, 1989.

Woods JE, Alter G, Meland B, Podratz K: Experience with vaginal reconstruction utilizing the modified Singapore flap. Plast Reconstr Surg 90:270, 1992.

Unilateral Cleft Lip

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This chapter reviews the etiopathogenesis and anatomic pathology of unilateral cleft lip, outlines the goals for reconstruction, and highlights proven treatment methods, both nonsurgical and surgical. Cleft lip patients have varying degrees of deformity ranging from notching of the lip to complete clefting of the lip and primary and secondary palate. As a surgeon evaluates a cleft lip patient, the degree of deformities must be identified and differentiated; only then can a treatment plan be formulated for correction of the individual problem.

Etiopathogenesis

The cause of unilateral cleft lip is multifactorial. Genes, drugs, and environment are contributing factors; yet no clearly defined causal relationship has been identified. There is a strong genetic role with an increased incidence of cleft lip in certain racial groups (from approximately 1 in 500 in Asians to 1 in 2000 in African Americans); however, no specific culprit gene has been detected. In some patients, clefts are seen as part of a syndrome, but these are the minority (approximately 13%) because most represent sporadic cases. When combined with cleft palate, it is more likely (approximately 40%) to be part of a syndrome.

Study of the embryology of cleft lip clearly establishes it as a deficiency state, and the degree of deformities must be individualized so that a treatment plan for correction of individual problems can be devised.

The deficiency in tissue is complicated by malposition of the anatomic components that are present. Interposition of the tongue in the cleft by the fetus widens the cleft and produces many of the deformities that occur.

Pathologic Anatomy

It is mandatory that the surgeon identify the soft tissue and skeletal deformities associated with a cleft so that each of the following deformities may be addressed in the repair:

1. Displacement of Cupid's bow with deficiency of the vermilion (see Fig. 1)
2. Shortened lip on the cleft side
3. Shortened columella on the cleft side
4. Aberrant insertion of the orbicularis in the nasal base
5. Malformed lower lateral cartilage with a depressed dome and flattened lateral segment
6. Lateral and outward rotation of the alar base
7. Webbing of the alar base
8. Deficient vestibular lining
9. Dislocation of the septum out of the vomer
10. Outwardly rotated premaxilla and retropositioned lateral maxilla

Microform or Fusiform Clefts

These deformities are the mildest versions of cleft lip. Frequently, parents will see these clefts as a minor problem and expect full correction with minimal surgery. As a surgeon evaluates a cleft lip patient, the degree of deformities must be identified and differentiated. This type of cleft, which at initial glance seems simple, shares many, if not all of the anatomic features of complete clefts. The exception is that these clefts often have minimal nasal deformity, and thus better aesthetic results can be achieved. The most important point is that the shortened lip must be elongated and the correct

anatomic elements balanced. We prefer to treat microform clefts with greater than 1-mm lip length discrepancy in a manner similar to that of complete clefts. A rotation-advancement flap (to be discussed) provides the best aesthetic outcome.

Incomplete Clefts

Incomplete clefts have at least an intact nasal sill or a Simonart band along the nasal floor. The importance of these clefts is that the maxillary deformity is usually less severe. They are typically treated with a rotation-advancement repair. The nasal deformity is generally less severe than in a wide complete cleft.

Complete Clefts

Complete clefts are those with clefting through the entire lip and nasal floor, as well as clefting of the maxilla up to the level of the incisive foramen. If clefting extends beyond the incisive foramen, a cleft palate is also present. However, complete clefts have a wide variance of severity in the degree of separation and rotation of the alveolar segments. Thus, more severe complete lip clefts, especially those with a cleft palate, will be referred to as a wide complete cleft lip. These clefts require more extensive treatment, with surgical emphasis on realigning not only the soft tissue but also the skeletal deformity.

Diagnostic Studies

Diagnostic studies for evaluation of cleft lip are limited to tests needed for detection of possible syndromic cases. No specific tests are required for additional diagnosis.

Goals of Reconstruction

The goals of repair of a cleft lip are correction of the aberrant anatomy:

1. Balance Cupid's bow.
2. Equalize the vertical height of the lip on the cleft and noncleft sides.
3. Equalize the height of the vermilion on either side of the repair.
4. Maintain an adequate philtral column and dimple.
5. Equalize the length of the columella on the cleft and noncleft sides.
6. Place scars in inconspicuous natural skin lines.
7. Reestablish orbicularis function and orientation.
8. Reestablish a labiobuccal sulcus.
9. Balance and reposition the alar bases for primary nasal reconstruction.
10. Elevate the depressed lower lateral cartilage.
11. Realign the maxillary segments.

Treatment

Nonsurgical

One of the challenges posed by complete clefts is the cleft of the maxilla. As described, the lateral segment of the maxilla is usually rotated outward with interruption of the dental arch. Therefore, one treatment goal is to establish an intact maxillary arch in preparation for future orthodontic work. To this end, it is important, when possible, to convert complete clefts to ones that are more surgically manageable by presurgical orthodontic manipulation. In general, the best cleft repairs are accomplished by teams that include orthodontists committed to cleft care and the fundamentals of presurgical manipulation of clefts. As the skeletal framework of the premaxilla is realigned into the correct orientation and abutment, the orbicularis and other soft tissues likewise fall into alignment.

Presurgical intervention can consist of the following: simple elastic banding to align the maxillary segments; acrylic molding plates, which are adjusted weekly by adding and removing acrylic to move the maxillary segments; screw-retained acrylic devises, which, similar to molding plates, realign the maxillary segments by weekly adjustment; and nasoalveolar molding, which incorporates a nasal strut on the molding plate to reshape the malformed nasal cartilage while simultaneously aligning the maxillary segments.

Surgical

Lip adhesion is a surgical technique that is also used to achieve the aforementioned presurgical goals; however, these procedures increase cost, do not align the alveoli accurately, and produce scarring of the lip tissue. When used in combination with palatal or maxillary stents, lip adhesion can realign malpositioned maxillary segments effectively if orthodontic manipulation is not available.

Presurgical marking of a cleft lip is one of the most important technical aspects and ensures the most precise repair. It is best to use a finely sharpened end of a wood cotton-tipped applicator dipped in ink. Patience with marking is paramount, and no degree of error should be tolerated when marking the following structures (Fig. 1):

1. Midpoint of Cupid's bow
2. High point of Cupid's bow on the noncleft side

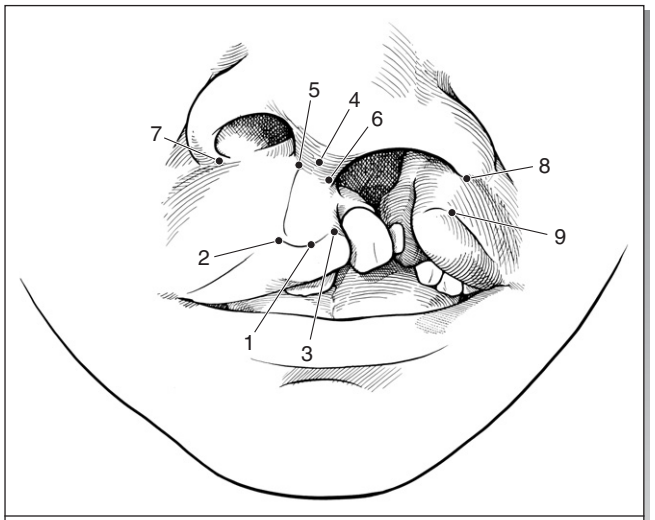

Figure 1 • Markings for unilateral cleft lip repair: 1, midpoint of Cupid's bow; 2, high point of Cupid's bow on the noncleft side; 3, projected high point of Cupid's bow on the cleft side with the distance from 1 to 2 equal to the distance from 1 to the projected high point on the cleft side; 4, center of the columella; 5, columellar base on the noncleft side; 6, columellar base on the cleft side; 7, alar base on the noncleft side; 8, alar base on the cleft side; and 9, projected high point of Cupid's bow on the lateral lip element.

3 Projected high point of Cupid's bow on the cleft side with the distance from 1 to 2 equal to the distance from 1 to the projected high point on the cleft side

4 The center of the columella

5 The columellar base on the noncleft side

6 The columellar base on the cleft side

7 The alar base on the noncleft side

8 The alar base on the cleft side

9 Projected high point of Cupid's bow on the lateral lip element

After the markings are made, the lip is infiltrated with 1% lidocaine with 1:100,000 epinephrine via a 30-gauge needle.

Through the years, many methods have been used for cleft lip repair. In modern cleft lip surgery, the rotation-advancement technique, as described by Millard, and its subsequent modifications serve as the basis for the type of surgical repairs that yield the best results and achieve most of the goals outlined earlier. The technique yields the most satisfactory scars while lengthening the lip and closing the wide nasal floor. Other methods, such as the triangular flap, can produce consistent results and are easier to learn and reproduce; however, they do not yield the optimal results seen with rotation-advancement repairs. The triangular repair has the disadvantage of leaving noticeable scars, a flattened Cupid's bow, insufficient lip length, and a wide nasal base.

The following modification of the extended Mohler rotation-advancement flap is the authors' repair of choice.

TECHNIQUE. An incision is made from the projected apex of Cupid's bow on the medial aspect of the lip along the most lateral border (Fig. 2). The flap is elevated from the muscular lip tissue and eventually used as the inner surface of the lip medially. A second incision is made from that same high point of Cupid's bow along the projected philtral column on the cleft side to a point 2 mm above the base of the columella. The incision is straight in orientation. Under the columella, the incision is extended with an oblique back cut at the columellar base. The back cut allows downward rotation of the medial segment. The C-flap (columellar-based flap) is liberated. The skin on the medial portion of the lip is elevated to the level of the philtral dimple, but not beyond this line.

The first portion of the nasal repair is begun. Through the opening at the columellar base, scissors are used to dissect the nasal cartilage. First, the medial crura are separated. Dissection is extended over the dome on the cleft side, the septal angle, the upper lateral cartilage, and the dome on the noncleft side. The lower lateral cartilage on the cleft side should be completely skeletonized, which is confirmed by freedom of elevation to an anatomic position with a Ragnal retractor.

Attention is directed to the lateral lip segment. An L-flap (laterally based flap) based on the lateral nasal wall and not on the alveolus as initially described is elevated (Fig. 3). A gingivobuccal sulcus–piriform aperture incision is made and extended to the underlying maxilla. Subperiosteal dissection is performed to liberate the alar base for medial movement. The L-flap is folded on itself to fill the piriform aperture defect; the excess of the L-

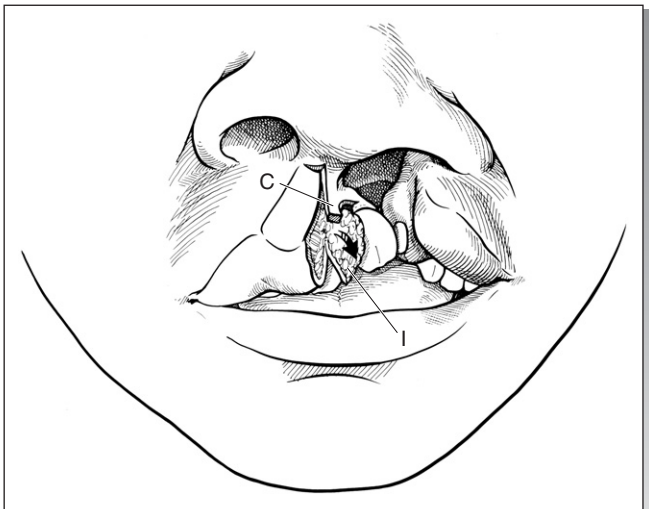

Figure 2 • Elevated C-flap (C) and medial lip segment along with the medial inner lip flap (I).

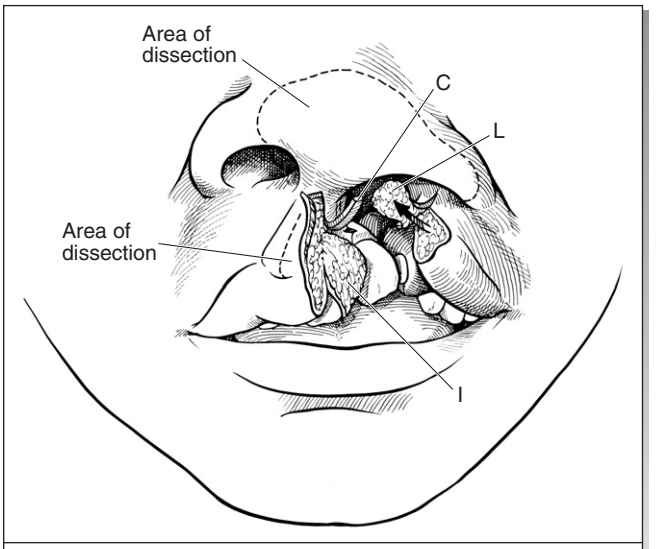

Figure 3 • After elevation of the L-flap (L) with the previously elevated C-flap (C) and the medial inner lip flap (I).

Figure 5 • Repair of the gingiva and labiobuccal sulcus and partial repair of the orbicularis (O).

flap is trimmed. A nasoseptal flap is elevated, and the flap is sutured to the bend of the L-flap to close the floor of the nose (Fig. 4).

The lateral lip element is prepared by elevating the skin and subcutaneous tissue from the underlying musculature. The mucosa is approximated to the previously elevated medial inner lining, gingiva, and sulcus, and the inner aspect of the lip is repaired. The orbicularis is advanced and sutured to the base of the columella (Fig. 5). The remainder of the orbicularis is carefully aligned and repaired. The vermilion is trimmed to achieve symmetry in height of the vermilion on both sides of the repair. The two projected high points of Cupid's bow are approximated, and the vermilion and lower portion of the lip are repaired (Fig. 6). The C-flap is rotated into the defect created by downward rotation of the medial aspect of the lip, and the excess is trimmed (Fig. 7). The nasal floor is repaired.

Definitive nasal repair is performed. The depressed lower lateral cartilage on the cleft side is elevated and fixed with an asymmetrically placed horizontal mattress suture. Placement of the suture is critical because it is the main force that aligns the lower lateral cartilage. The suture enters the cephalic portion of the dome on the cleft side lower lateral cartilage, catches the upper lateral cartilage on the noncleft side, enters the undersurface of the lower lateral cartilage on the noncleft side, and exits

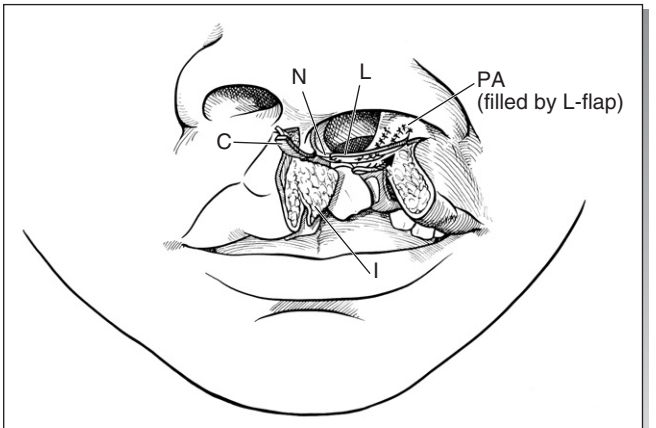

Figure 4 • The L-flap (L) has been folded into the piriform aperture defect (PA) and sutured to the nasoseptal flap (N) for closure of the nasal floor.

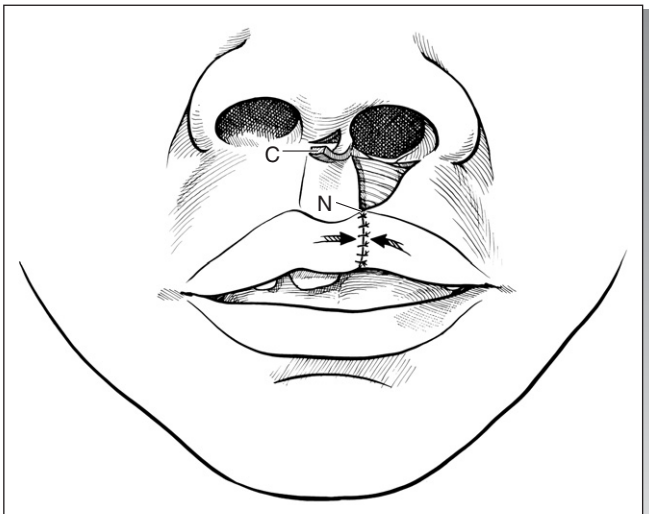

Figure 6 • After repair of the orbicularis and lower lip with restoration of the high point of Cupid's bow on the cleft side (N), the C-flap (C) is been trimmed.

at the proposed apex of the two domes. It passes into the lower lateral cartilage on the cleft side, thereby elevating the lower lateral cartilage and re-creating a sharp nostril apex and nasal tip (Fig. 8). The vestibular web is obliterated by another mattress suture that passes from inside the nose out at the alar/cheek junction and reenters through the same exit hole in the skin. It is tied inside the nose to close the vestibular web. No bolsters or stents are needed.

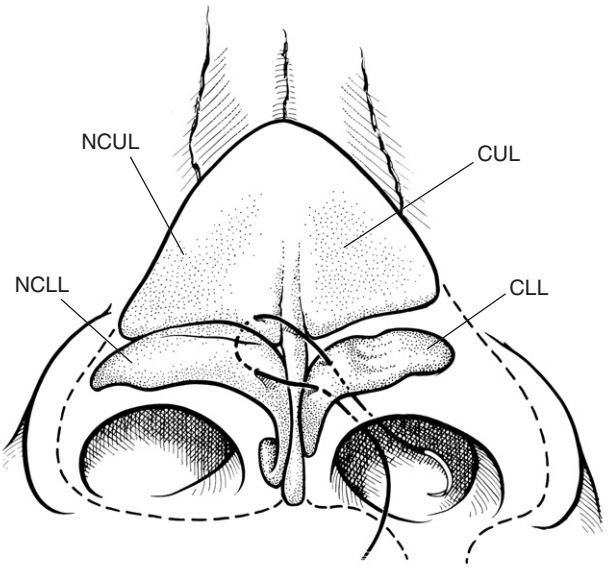

Figure 8 • An asymmetric horizontal mattress suture is used to elevate the dome and the lower lateral cartilage on the cleft side (CLL). CUL, upper lateral cartilage on the cleft side; NCLL, lower lateral cartilage on the noncleft side; NCUL, upper lateral cartilage on the noncleft side.

Postoperative Care

In the postoperative period, children are placed in elbow restraints for several days. A syringe feeder is used for feeding. Half-strength peroxide and saline are used to clean the incision three times a day until the scab is completely removed to facilitate suture removal at 1 week. Sutures are removed under loupe magnification with the aid of a papoose restraint board. After the sutures are removed, the lip is taped in compression for 5 additional weeks.

Pearls and Pitfalls

- The most common mistake in unilateral cleft lip repair is marking Cupid's bow too wide, which makes downward rotation of the medial segment difficult.
- The vermilion under an inaccurately placed Cupid's bow point is usually insufficient and results in a whistle deformity.
- Presurgical nasoalveolar molding is an important component of modern cleft lip repair and allows primary correction of the lip, nose, and alveolus.
- Postoperative taping for 5 weeks has resulted in improved scar appearance.

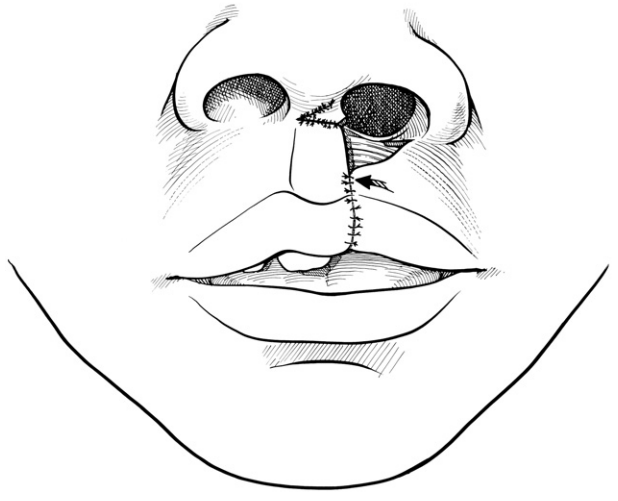

Figure 7 • The trimmed C-flap permits downward movement of the medial rotation flap.

SUGGESTED READINGS

Byrd HS; Cleft lip 1: Primary deformities (overview). In Selected Readings in Plastic Surgery, vol 8. 1997, p 1.
Cutting CB: Cleft surgery virtual surgery videos: Unilateral cleft lip. New York, Smile Train, 2001.
Millard R: Cleft Craft—The Evolution of Its Surgery. Philadelphia, Lippincott, Williams & Wilkinson, 1980.
Millard RD: Unilateral Cleft Lip. Philadelphia, WB Saunders, 1990, p 2627.
Mohler LR: Unilateral cleft lip repair. Plast Reconstr Surg 80:511, 1987.

Bilateral Cleft Lip

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Bilateral cleft lip remains one of the greatest technical challenges for plastic surgeons, and this challenge is evident in the recent evolution of treatment from several stages to a one-stage repair. With recent advances, the results after bilateral cleft lip repair can equal or approach those after unilateral cleft lip.

Etiopathogenesis

The cause of bilateral cleft lip is multifactorial and most likely similar to that of unilateral cleft lip, but it is much less common in incidence and accounts for approximately 10% of clefts. The incidence among racial groups is similar to that for unilateral cleft lip. There is, however, a higher incidence of associated cleft palate (86% versus 68% for unilateral cleft lip).

Pathologic Anatomy

It is useful to divide the anatomic abnormalities into three components: nasal abnormalities, prolabial abnormalities, and premaxillary malposition. The repair should restore as many of the abnormalities as possible to the normal anatomic state.

Nasal abnormalities:

- Wide alar base with a laterally flared nasal valve
- Orbicularis inserted into the nasal base
- Flattened lower lateral cartilage with widely separated domes
- Apparent short columella as a result of malpositioned lower lateral cartilage
- Fibrofatty deposit separating the domes

Prolabial abnormalities:

- Vertically reduced upper lip
- Absent orbicularis muscle
- Absence of normal anatomic landmarks (Cupid's bow, philtral dimple and columns, white roll)
- Absence of a true vermilion (buccal mucosa instead of true vermilion)
- No gingivobuccal sulcus
- Absence of hair follicles

Premaxillary malposition:

- Premaxillary protrusion on a vomerine stem
- Loss of the normal dental arch

Bilateral cleft lips have varying degrees of abnormalities. They may range from partial or incomplete on both sides to wide complete bilateral clefts. Throughout this spectrum, the anatomic derangements remain in varied degrees. Through knowledge of the pathologic anatomy, the surgeon can devise a plan to correct the abnormality.

Diagnostic Studies

Diagnostic studies for evaluation of bilateral cleft lip are limited to tests required for the evaluation of possible syndromic cases. No specific tests are required for additional diagnosis.

Goals of Reconstruction

The goals of repair of a cleft lip are correction of the aberrant anatomy. The ultimate goal should be reconstruction of the lip so that it has normal shape and appearance with the resulting scars resting in

natural lines and at the same time avoidance of disruption in growth of the maxilla and dental eruption. In addition, the goals should ideally be accomplished in a single surgical procedure.

The following criteria further delineate the reconstructive goals:

1. Reconstruction of a symmetric nose with adequate columellar length and a narrow defined tip
2. Closure of the lip with adequate height and restoration of the orbicularis muscle
3. Creation of a normal-size philtrum complete with philtral columns and dimple
4. Creation of a balanced Cupid's bow with a white roll and underlying normal vermilion
5. Establishment of a labiobuccal sulcus
6. Positioning of the premaxilla with reestablishment of the dental arch
7. Placement of the scars in inconspicuous natural skin lines

The paradigm shift in treatment of bilateral cleft lip has been centered on reestablishment of normal positioning of the nasal cartilage rather than the use of skin flaps from the wide prolabium to elongate the columella. Therefore, growth of the lower lateral cartilage in a normal anatomic configuration maintains the surgical correction of nasal form instead of flattening of the nasal tip as seen after skin-based repairs.

Treatment

Nonsurgical

Bilateral clefts have a wide spectrum of manifestations. Simple incomplete bilateral clefts with minimal notching of the lip and without involvement of the nose or maxilla can be accurately repaired in one stage by a variety of techniques. More often, however, significant displacement of the premaxilla precludes simple one-stage repair; usually, something has to be done before definitive lip repair—typically, a lip adhesion procedure. The goal of lip adhesion is to pull back the protruding premaxillary segment and close the gap in the lip to allow for tension-free repair. This procedure places scars in areas that are not critical for the definitive repair and allows the tension across the premaxilla to narrow the gap. The lip adhesion procedure can be extremely effective and, when used with a guiding appliance, can reliably close the wide gap and allow for tension-free lip repair.

More recently, it has been shown that all of these goals can be accomplished without the drawbacks of the additional scarring and inflammation associated with a surgical intervention. With the use of molding plates, the premaxilla can be accurately reduced to reestablish a normal dental arch and close the wide gap. Moreover, with the addition of nasal extensions, these goals can be accomplished at the same time that the nasal correction is nonsurgically initiated. The columella can be elongated and the vestibular lining expanded.

The advantages are obvious, but nasal molding also has its drawbacks. The process is time consuming and requires a motivated family, as well as a trained orthodontist or professional dedicated to cleft management. It should be initiated as soon as the child is born. There is significant time and cost associated with the technique; however, it possibly decreases the need for secondary surgery when combined with primary gingivoperiosteoplasty (which is still experimental at this time) and thus may be cost-effective in the long run.

Surgical

Presurgical marking of bilateral cleft lip is as important as that for unilateral cleft. There is, however, more freedom in that the entire anatomy is to be recreated and liberty can be taken within certain restraints (as in philtral width). Points are tattooed with a 30-gauge needle dipped in ink (Fig. 1):

1. The midpoint of Cupid's bow at the depth of the prolabium
2. The high points of Cupid's bow on the prolabium, two points 2.5 mm on either side of the midpoint of Cupid's bow, with hemicircles connecting the points to mimic a Cupid's bow
3. The alar bases
4. The high point of Cupid's bow on the cleft sides; they must be equidistant from the alar bases and the lip kept as short as possible while still

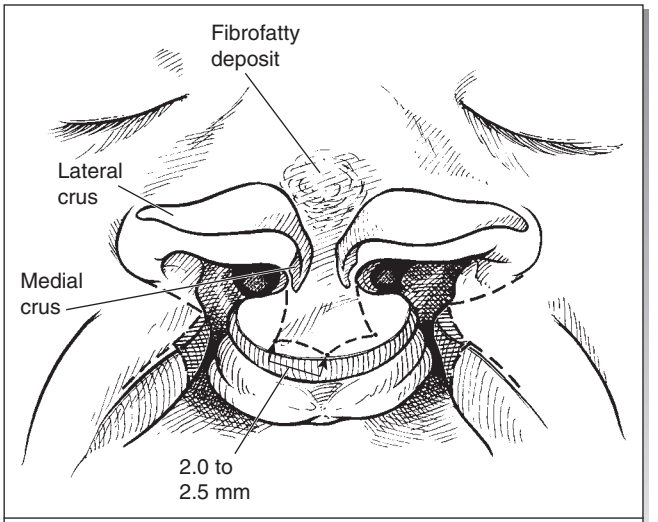

Figure 1 • Markings for bilateral cleft lip repair.

allowing a lateral turndown flap equal in size to the hemicircles

After the markings are made, the lip is infiltrated with 1% lidocaine with 1:100,000 epinephrine.

The new philtrum is outlined. The lines should extend to a point just lateral to each base of the columella. They should be made slightly lateral to the columellar base because the transfixion sutures used for closure will later narrow these points even more as the prolabial skin is recruited to the columella. On the lateral elements, a line is drawn from the high point of Cupid's bow along the border of the cleft toward the nose. This line is connected with a horizontal line to the alar base point. The turndown flap must be designed such that the hemicircles created in the lateral part of the vermilion equal the hemicircles in the new Cupid's bow of the prolabium. All skin incisions are incised. The lateral lip elements are prepared by elevation from the underlying muscle for a short distance. The turndown flaps are fully freed through the substance of the lip. The turndown flaps should be kept bulky to create a slight pout in the midline.

The lateral flap, or "L" flap, is elevated from the medial margin off the underlying muscle of the lateral lip elements (see Figs. 3 and 4 in the chapter "Unilateral Cleft Lip"), as performed for repair of unilateral cleft. The flap is based on the lateral nasal sidewall; it fills the piriform aperture defect and closes the floor of the nose after being sutured to a septal flap.

An incision is made over the piriform aperture, and supraperiosteal dissection is performed to allow the lateral portion of the lip to advance toward the midline. In cases without presurgical orthodontic management, more undermining is needed. The medial edge of the "L" is sutured to the skin at the junction of the alar and piriform aperture defect, and the remainder of the "L" flap is hinged on itself to completely fill the piriform aperture defect. The tip of the "L" flap is trimmed. The medial edge of the "L" flap is brought across the floor of the nose to a small septal flap to complete the closure of the nasal floor.

The buccal mucosa, based on the alveolus, is elevated off the prolabium. This flap should be kept thin and is trimmed and anchored to the periosteum at the new height of the gingivobuccal sulcus. The tip of the mucosa of the lateral lip elements is sutured to the height of the gingivobuccal sulcus. By anchoring these elements to the periosteum, the height of the gingivobuccal sulcus is firmly established. The remainder of the inner lip mucosa is repaired. The orbicularis is also repaired and anchored to the periosteum at this location to further set the height of the sulcus and prevent downward migration. Muscle pennants from the area of the alar base should be repaired in the area of the absent anterior nasal spine to create a smooth, natural nasal sill and give a firm base for the footplates of the medial crura to rest.

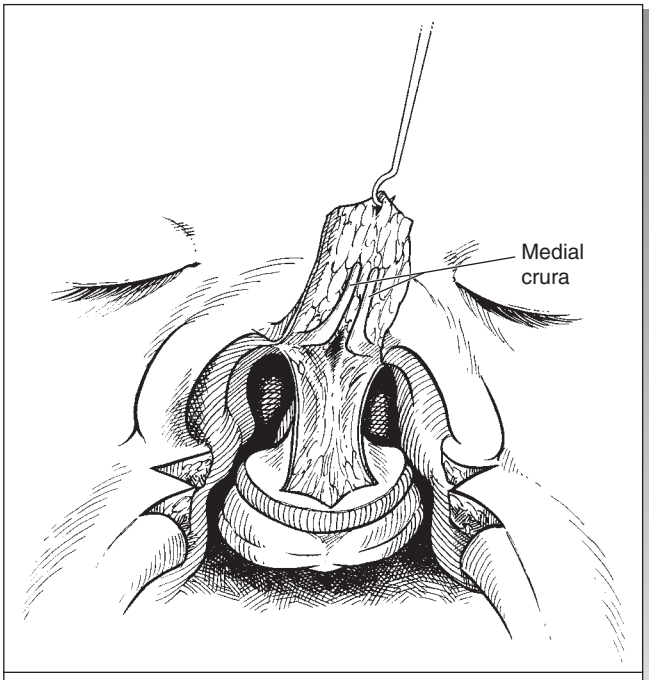

Figure 2 • Elevation of the prolabial flap and dissection of the lower lateral cartilage. The fibrofatty tissue is completely freed from the lower lateral cartilage but is not removed to avoid decreasing the blood supply to the prolabial flap.

The incision in the prolabium is continued up the membranous septum. Elevation of the prolabial flap is continued under the medial crura such that the crura are raised with the prolabium. The superior surface of the domes is fully dissected (Fig. 2). The dissection should be continued fully to the alar rim. Alar rim incisions can be made to facilitate dissection of the domes (and suturing of the domes, which is described later). The alar rim incisions are helpful, but can be avoided as experience with the procedure is gained. The complete dissection serves two important functions. It elevates the fibrofatty deposits from the domes and enables advancement of the lower lateral cartilage to recreate a sharp tip and elongate the columella. It should be noted that the fibrofatty tissue is not removed because of potential injury to the blood supply to the prolabium.

After the nasal cartilage has been fully dissected, the nasal repair can be performed. A horizontal mattress suture advances the lower lateral cartilage in a lateral-to-medial direction and creates sharp dome-to-dome contact (Fig. 3). It also adds nasal tip projection and elongates the columella. Advancement is possible only if the lower lateral cartilages have been liberated, and dome-to-dome approximation is possible only if the fibrofatty tissue has been elevated from between the domes. The horizontal mattress suture is passed directly through the mucosa at the desired new tip point (usually 3 mm lateral to the columellar nasal junction) and back through approximately 2 mm superiorly (see Fig. 3).

Care should be taken to avoid inclusion of fibrofatty deposits in this suture. The transfixion incision is closed with transfixion sutures, and the medial crura are advanced up the septum. The medial footplates are set on top of the repaired muscle pennants to give firm support to the tip and ensure that the gained projection will be permanent.

Often, there is significant excess lateral prolabial skin. Strips of the lateral skin can be deepithelialized to serve as bulk to create philtral columns. A small amount of undermining of the prolabium is needed to create the bulk and give the impression of a philtral dimple. The remainder of the skin is closed (Fig. 4). Occasionally, a small amount of excess skin is present at the nasal sill and should be excised.

The vestibular webs are obliterated by passing a mattress suture from inside the nose out at the alar-cheek junction and reentering through the same exit hole in the skin. The suture is tied inside the nose to close the vestibular web.

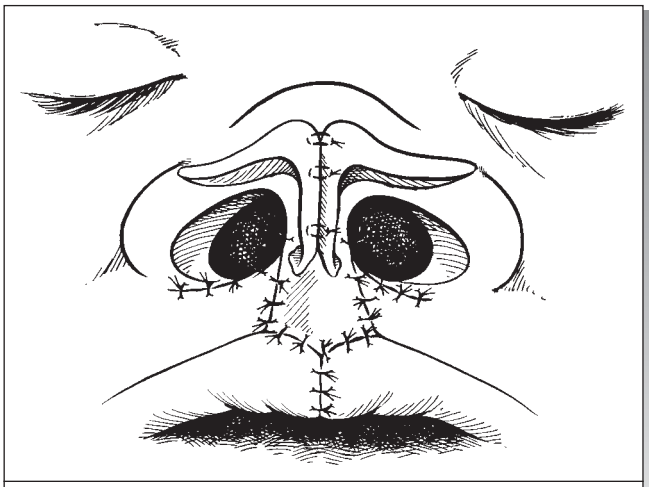

Figure 4 • Final closure. The muscle is repaired under the prolabial flap and anchored to the periosteum of the bone.

Postoperative Care

In the postoperative period, children are placed in elbow restraints for 5 weeks. A syringe feeder is used for nourishment. Half-strength peroxide and saline are used to clean the incision three times a day until the scab is completely removed to facilitate suture removal at 1 week. Sutures are removed under loupe magnification with the aid of a papoose restraint board. After the sutures are removed, the lip is taped in compression for 5 additional weeks.

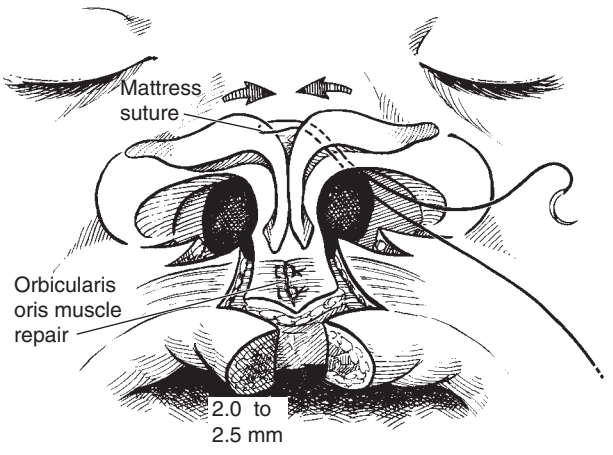

Figure 3 • Horizontal mattress suture used to reapproximate the domes of the lower lateral cartilages.

Pearls and Pitfalls

- The prolabium should be kept narrow because a wide prolabium leaves an unnatural appearance.
- The lower lateral cartilages must be liberated to be advanced, and the fibrofatty tissue must be elevated from between the domes to allow intimate approximation of the domes. This technique allows the cartilage to be repositioned and creates a defined tip with adequate projection.
- The nose often appears short with an overrotated tip at the conclusion of the procedure; however, the appearance of the nose improves as the child grows provided that the lower lateral cartilages have been properly repositioned.

SUGGESTED READINGS

Cleft surgery virtual surgery videos: Bilateral cleft. New York, Smile Train. 2001.

Cutting C, Grayson B, Brecht L, et al: Presurgical columellar elongation and primary retrograde nasal reconstruction in one-stage bilateral cleft lip and nose repair. Plast Reconstr Surg 101:630, 1998.

McComb H: Primary repair of the bilateral cleft lip nose: A 15-year review and a new treatment plan. Plast Reconstr Surg 86:882, 1990.

Millard R: Cleft Craft—The Evolution of Its Surgery. Philadelphia, Lippincott Williams & Wilkins, 1980.

Mulliken JB: Primary repair of bilateral cleft lip and nasal deformity. Plast Reconstr Surg 108:181, 2001.

Repair of Secondary Cleft Lip and Nose Deformities

IAN T. JACKSON

Primary management of cleft lip and palate has improved considerably over the past 30 years. However, a large number of patients have secondary deformities. They may be minor soft tissue problems requiring simple repair or major bony dental and soft tissue deformities requiring major orthodontic and surgical rehabilitation. This chapter builds on the previous two chapters dealing with unilateral and bilateral cleft lip and nose repair. The goals of secondary repair are the same as those of primary repair: normality of form and function in all aspects of the cleft deformity.

Unilateral Clefts

Pathologic Anatomy

In unilateral cleft cases (Fig. 1), the nasal deformity consists of the following:

1. Flattened, asymmetric nose
2. Hypoplastic and retruded bony paranasal area and maxilla on the cleft side
3. Lateral and posterior displacement of the alar base (accentuated by its attachment to the periosteum of the maxilla)
4. Outwardly displaced and everted lower lateral cartilage
5. Notching anterior to the midpoint of the alar rim because of the position of the alar base and the connections of the nasal mucosa to the underlying hypoplastic and laterally displaced lateral edge of the piriform aperture (Note: if a hook is placed anteriorly on the nostril and pulled, a tight band is seen intranasally that extends from the alar rim to the edge of the piriform aperture)
6. Wide, low, and unsupported nasal sill
7. Deviated bony and cartilaginous septum (the vomer on the cleft side is absent)
8. Enlarged inferior turbinate (as a result of the increased intranasal volume on the cleft side caused by deviation of the septum and lack of support for the nasal floor)
9. Constant regurgitation from the oral cavity because of lack of bony support of the nasal floor (or the floor may not have been repaired in the primary procedure)
10. Internal nasal valve collapse, especially with forced inspiration (because of lack of support of the middle vault by the lower lateral cartilage)

In the preoperative assessment, two factors need to be addressed: form and function. It is mandatory to identify the problems, determine which are the most significant, and devise a plan to correct the pathology.

If a nasal deformity is present, there is frequently an associated lip deformity. The lip deformity may result from the severe nature of the original cleft, but it is usually due to a lack of correct muscle reorientation at the primary surgery. In a primary cleft lip, the muscle of the lesser segment is attached to, or close to, the alar base. Pulling on this segment of the muscle everts the alar base and pulls it downward. This phenomenon is seen in all unilateral clefts, and its severity varies directly with the severity of the lip deformity and the hypoplasia of the lesser segment of the maxilla. On the medial side of the lip the muscle is absent, which results in the lip being vertically reduced at the edge of the cleft. There is hypoplasia of the medial edge of the alveolus with absence of half of the nasal spine (Fig. 2).

The anatomic deficiency, if not corrected at the initial repair, results in the following:

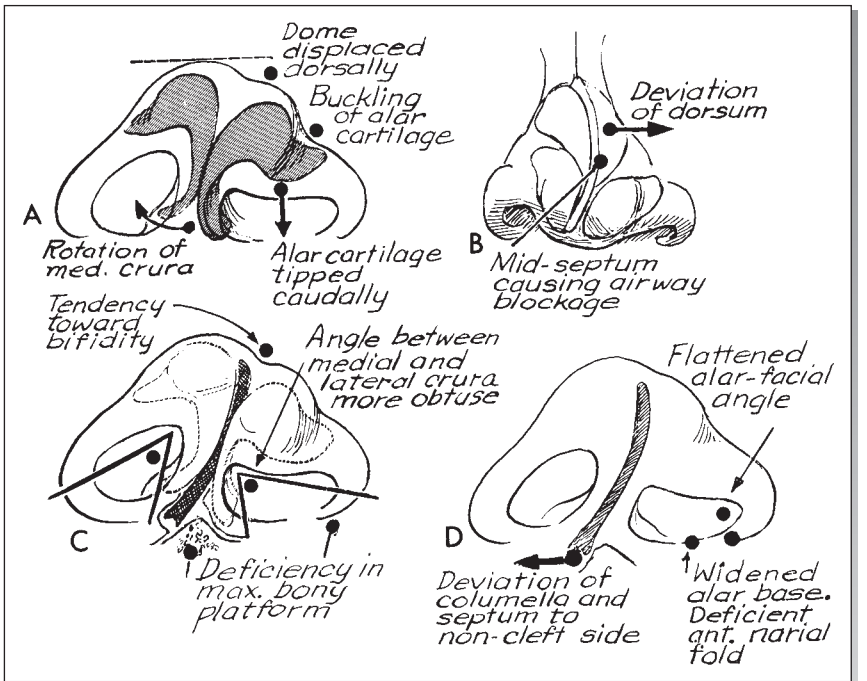

Figure 1 • Deformities of the nasal tip in a patient with cleft lip and nose. **A,** Deviation of the nasal tip. **B,** Deviation of the nasal dorsum. The alar cartilage is tipped caudally. **C,** The angle between the medial and lateral crura is more obtuse, and the dome is displaced dorsally. Note the buckling of the lateral crus and deficiency of the maxillary bone platform. **D,** The columella and caudal septum are deviated to the noncleft side. The septum on the convex side causes varying degrees of obstruction, as well as a tendency toward bifidity. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990.)

1 A short lip
2 Deficiency of tissue directly under the repair
3 A mass of muscle tissue lateral to the lip scar
4 Deficient lip margin
5 Mismatch of the mucocutaneous area

Goals of Repair

PROBLEMS IN THE PRIMARY REPAIR. In the original deformity, the maxilla is hypoplastic on the cleft side with a defect in the dentoalveolus. The anterior aspect of the maxilla is usually displaced laterally by the cleft. On the medial aspect, the septum is deviated to the noncleft side and the nasal spine is absent. The "link" material is the periosteum. If the periosteum is left in its original position, the soft tissue and bone remain displaced. If the soft tissue is elevated from the periosteum, bleeding occurs and scar tissue is formed with a tendency for subsequent displacement and deformity of the soft tissue.

What needs to be appreciated is that in the developing skeleton, subperiosteal dissection *does not* form scar tissue—it forms *bone*. Consequently, rather than deprecating subperiosteal dissection, it should be advocated. In this way, the periosteal envelope can be put into its correct position in association with correct positioning of the overlying soft tissue. The defect that is formed under the alar base can be filled with cancellous bone, thus correcting the underlying deficiency in three dimensions. The primary palate should be closed in two layers at the same time as correction of the nasal defect. In addition, if indicated, a bone graft can be placed in the alveolar gap.

This, of course, is the ideal situation and can be achieved if the advice just given is followed. Furthermore, extensive dissection of the lower lateral cartilage is needed at the primary procedure to place it in a symmetric position relative to the normal side.

If this type of regimen is not followed in the initial procedure, the typical deformity occurs and is related almost directly to the severity of the initial deformity. Asymmetry of the nose results largely from hypoplasia of the cleft maxilla. The alar base is displaced laterally and posteriorly, and the lower lateral cartilage demonstrates its characteristic deformity. Another interesting phenomenon can be demonstrated if a hook is placed on the tip of the nostril on the cleft side and an attempt is made to make it symmetric with the normal side; a tight band develops that leads from the deformed area of the lower lateral cartilage to the anterior part of the cleft maxilla just medial to the alar base. This problem can be corrected at the primary procedure

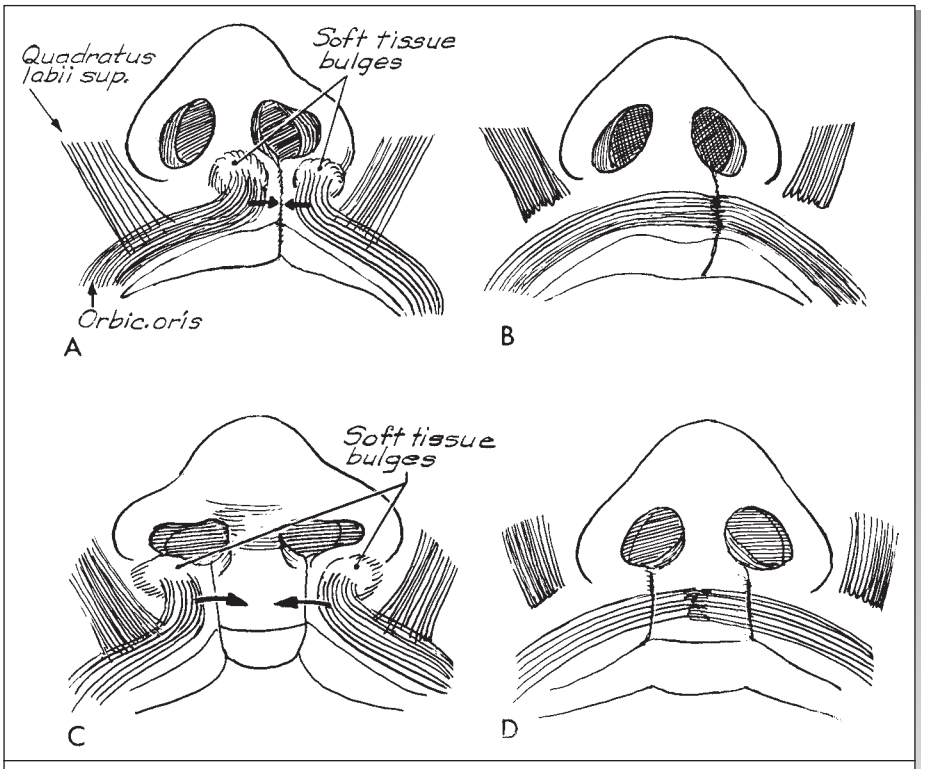

Figure 2 • Orbicularis oris abnormalities. **A,** The orbicularis oris fibers are misdirected in this unilateral cleft lip with the formation of two orbicularis bulges, one on each side of the cleft. **B,** After division of the fibers of the quadratus labii superioris muscle, the orbicularis oris muscle is redirected and sutured. **C,** Similar misdirection is found in a bilateral complete cleft. **D,** The fibers of the orbicularis oris are redirected and introduced into the prolabial segment in which muscle is sparse or absent. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990.)

by extensive subperiosteal dissection and release to allow the base of the lower lateral cartilage to be placed in its correct three-dimensional position.

If possible, a one-stage correction is planned; alternatively, many prefer staged reconstruction. In the majority of cases, orthodontic treatment is necessary beforehand to place the segments of the dentoalveolus in the correct position; such treatment may expose a previously undiagnosed anterior fistula. This is advantageous because wide fistulas are easy to close whereas narrow fistulas are difficult. Although a cleft deformity is occasionally repaired concurrently with performance of a LeFort I osteotomy, it is more common to close and bone graft the fistula before orthognathic surgery. The nose can be repaired at the time of fistula closure or during the osteotomy. If necessary, the lip can be corrected at the same time as the rhinoplasty procedure.

DIAGNOSTIC STUDIES. The external nose must be examined carefully to note asymmetry, deviation, and the sequelae of previous surgery. The patient is asked to inhale through the nose to observe evidence of internal valve collapse. A complete examination with a headlight and speculum gives information about the position of the septum and the size and appearance of the turbinates. The examination can be further enhanced, particularly in terms of the posterior portion of the nose, with the use of a nasoendoscope. The position of the bony septum and the nasal floor can be assessed. Defects in the floor can be confirmed by examination of the hard palate.

If sinus disease is a concern, coronal computed tomography provides all of the necessary information. It also allows accurate assessment of septal form and position, in addition to the size of the turbinate, the shape and size of the nasal cavities, and the position of the nasal floor. In some situations, airflow studies may be used to diagnose airway problems.

Examination should include an estimation of the extent of the deformity, the presence or absence of airway problems, dental occlusion, and the position of the alar base. All of these factors are clues about what needs to be done surgically. It is important to understand that in many patients, the primary deformity of the nose remains but with no further worries about growth. As in primary cases, the maxilla, alveolus, nose, and lip are all involved to a greater or lesser degree. If no additional surgery has

been performed since the original repair, in many ways a successful repair is much easier to achieve because of less scarring and distortion of surgical planes.

Surgery

The nose and anterior aspect of the maxilla on the cleft side are infiltrated with 0.5% lidocaine (Xylocaine) and 1:400,000 epinephrine with or without hyaluronidase.

When vasoconstriction is achieved, an incision is made in the upper labiobuccal sulcus of the lesser segment to examine the extent of the maxillary hypoplasia. The subperiosteal dissection is extended to the nostril rim, the nasal floor, and the nasal spine. The dissection is extensive and should include the anterior aspect of the maxilla, the defect of the dentoalveolus, which may be complete or incomplete, and the nasal spine. This maneuver frees the soft tissue and allows the alar base to move anteriorly and medially. Such dissection not only allows repositioning of the displaced alar base but also provides access for bone grafting of the deficient maxilla.

For patients with septal deviation, a vertical incision is made in the mucoperichondrium over the caudal septum, and the perichondrium and the periosteum of the septum are elevated widely anteriorly and posteriorly. The dissection is carried over the caudal septum to elevate the contralateral mucoperichondrial flap on the noncleft side. The septum is elevated from the septal groove, and the bony septum is completely freed of periosteum. If the latter is deviated, it is removed. In the majority of cases, the cartilaginous septum, whose position is determined by the bony septum, immediately moves into the correct position. A small amount of cartilaginous septum is removed from its lower border to keep it clear of the vomerine ridge. The septum now becomes a "swinging door." If the vomerine ridge is large and displaced, it is removed. The septum, once fully released, centralizes; scoring is almost never required unless there has been severe trauma or previous surgery.

For patients with the typical situation of depressed lower lateral cartilage, a limited intercartilaginous incision is made between the upper and lower lateral cartilage. The lower lateral cartilage and the mucosa on the cleft side are delivered over scissors and the cartilage dissected from the mucosa. The deformed cartilage is trimmed on its cranial edge and scored to normalize its position and shape.

The lower lateral cartilage on the noncleft side is delivered in the same way and trimmed. It is not removed from the mucosa because it will form the template to allow the cleft cartilage to be positioned correctly. The cleft cartilage is sutured to the opposite side to produce a symmetric tip. If necessary, a graft from the normal lower lateral cartilage may be placed and sutured to the anterior aspect of the cleft lower lateral cartilage to enhance overall symmetry. The bony nasal dorsum is exposed and rasped to make it smoothly continuous with the cartilaginous dorsum. In a severe unilateral cleft deformity, conchal cartilage may be used to splint the lower lateral cartilage. In addition, the nasal tip or dorsum can be augmented with conchal cartilage. In only a few situations, through and through sutures may be used to hold the lower lateral cartilage in position and tied over a gauze pledget on the skin overlying the ala.

A Joseph knife is used to create bilateral tunnels on either side of the nasal bones, notched osteotomes are used to make lateral osteotomies, and nasal bone infractures are performed.

It is not unusual to rasp the nasal dorsum to achieve a smooth contour. The septal cartilage may occasionally require dorsal trimming. The upper lateral cartilage is trimmed on its anterior or dorsal aspect; any removal of cartilage from the inferior area, however, must be done with care. Overenthusiastic resection results in weakening of the nasal valve mechanism and secondary airway problems.

Through and through quilting sutures are performed on the septum, and the rim incisions are closed. The nose is stabilized with tape and a splint. The splint is left in position for 1 week. The patient is advised to wear it at night to prevent trauma during sleep.

Bilateral Clefts

It is unusual for a complete bilateral cleft of the lip and the anterior aspect of the palate not to require secondary correction after primary repair. When confronted with this deformity, it is best to place the upper arch in its correct position by orthodontic therapy and then proceed to bone graft the alveolus bilaterally before any soft tissue or nasal correction (see the chapter "Bilateral Cleft Lip"). In addition, the anterior aspect of the maxilla requires augmentation in almost every case, preferably as a separate procedure after the dentoalveolar defect has been treated. The procedure is performed in the same way as in unilateral cases, and the laterally displaced ala is repositioned at the same time.

In a bilateral cleft deformity, secondary revision of the lip for aesthetic and functional deficiencies is frequently required. There is no true vermilion on the prolabial segment in bilateral cleft lips. Consequently, the vermilion may be deficient in the midline. Although this deformity can be improved by V-Y advancement, a relatively simple procedure, it is not ideal because wet mucosa is advanced to a dry area and crusting of this region can occur. A preferred procedure is the Kapetansky vermilion flap (Fig. 3), which consists of triangular island flaps based on the orbicularis muscle that are transferred

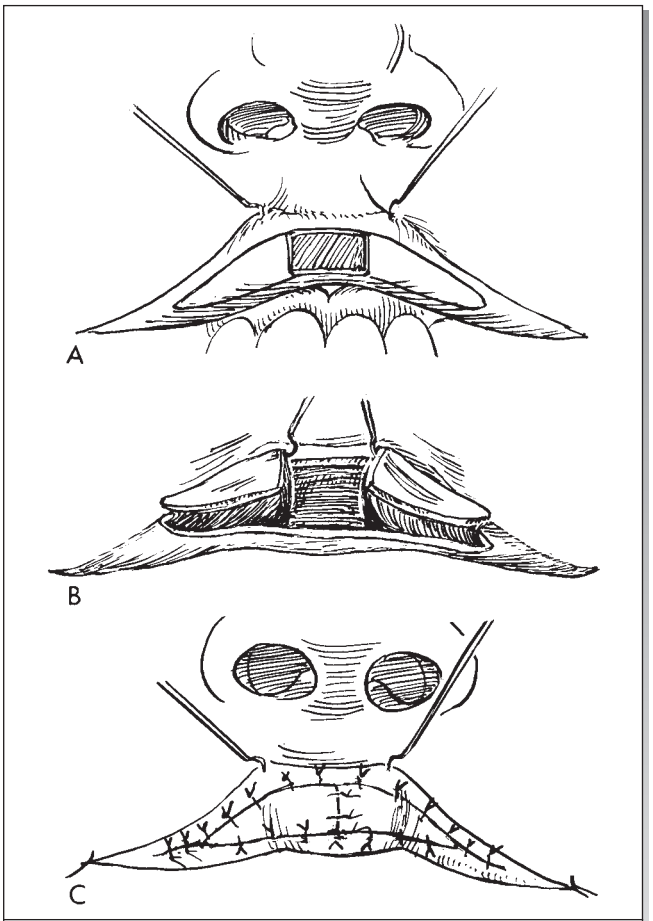

Figure 3 • Double "pendulum" flaps (island flaps with an orbicularis muscle pedicle) to correct notching of the vermilion border. **A,** Two lateral island flaps are designed. **B,** The island flaps before being advanced centrally. **C,** Operation completed. (After Kapetansky; from McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990.)

in a lateral-to-medial direction on a superior subcutaneous/submucosal pedicle. This technique augments the soft tissue medially and decreases bulk laterally—exactly what is required.

In patients with central deficiency, an Abbé flap may be used. This flap is frequently the most useful procedure for correction of the overall deformity because the lower lip is often recumbent in bilateral cleft lip patients. An Abbé flap reduces the excess lower lip and simultaneously augments the deficient upper lip. Although this procedure can produce a satisfactory result, it may not address the muscular deficiency of the upper lip because the muscular activity of an Abbé flap is variable. In most secondary bilateral cleft lips, the muscles are locked into the lateral segments. The muscles, however, can be dissected and reconstructed in the midline to provide a normal muscular sling, which can be covered with an Abbé flap consisting of skin and mucosa—the sandwich Abbé flap (Fig. 4).

Secondary Bilateral Cleft Nasal Correction

A bilateral cleft nose frequently remains flat and unattractive, even if the lip repair has turned out well. This problem is due mainly to the complexity of the nasal deformity but also to hypoplasia of the maxilla around the nasal area. The deformity can be further accentuated by maxillary retrusion.

The sequence of events to correct the most significant deformity is maxillary advancement with bone grafting of the maxilla as required. This procedure gives a satisfactory skeletal platform or foundation for the nose. Judgment has to be exercised

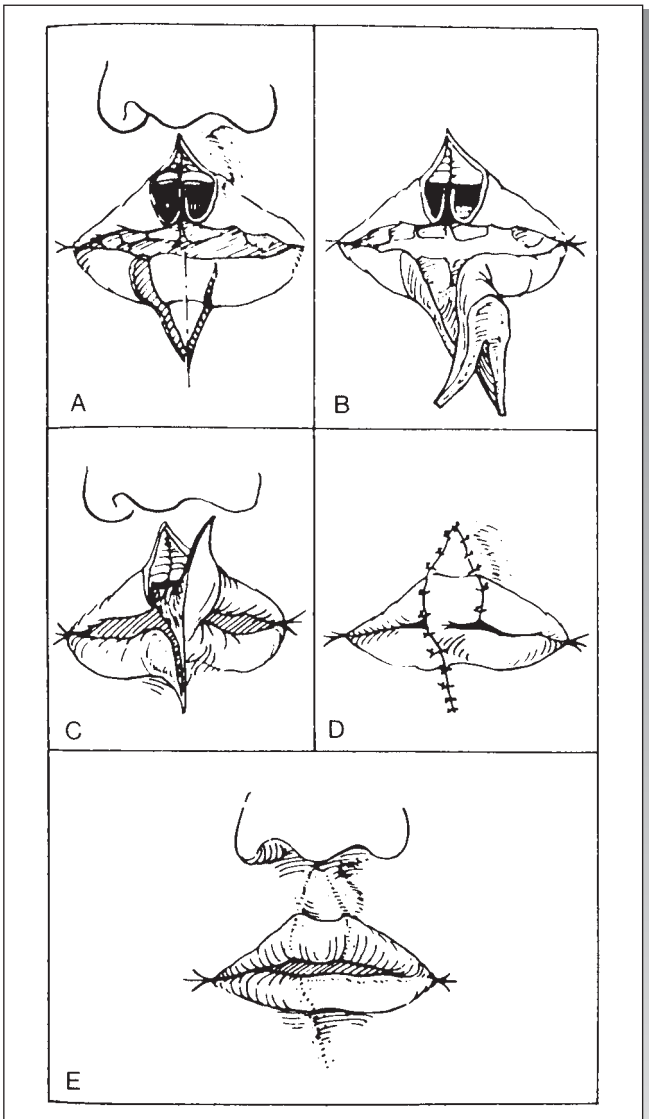

Figure 4 • Operative technique for a sandwich Abbé flap. **A,** Dissection of the orbicularis oris muscle. **B,** Elevation of the Abbé flap. **C,** Transfer of the lower lip flap and insertion into the upper lip. **D,** Sutures hold the flap in position. **E,** The flap is divided after 10 days. (From Jackson IT, Soutar DS: The sandwich Abbé flap in secondary cleft lip deformity. Plast Reconstr Surg 66:38, 1980.)

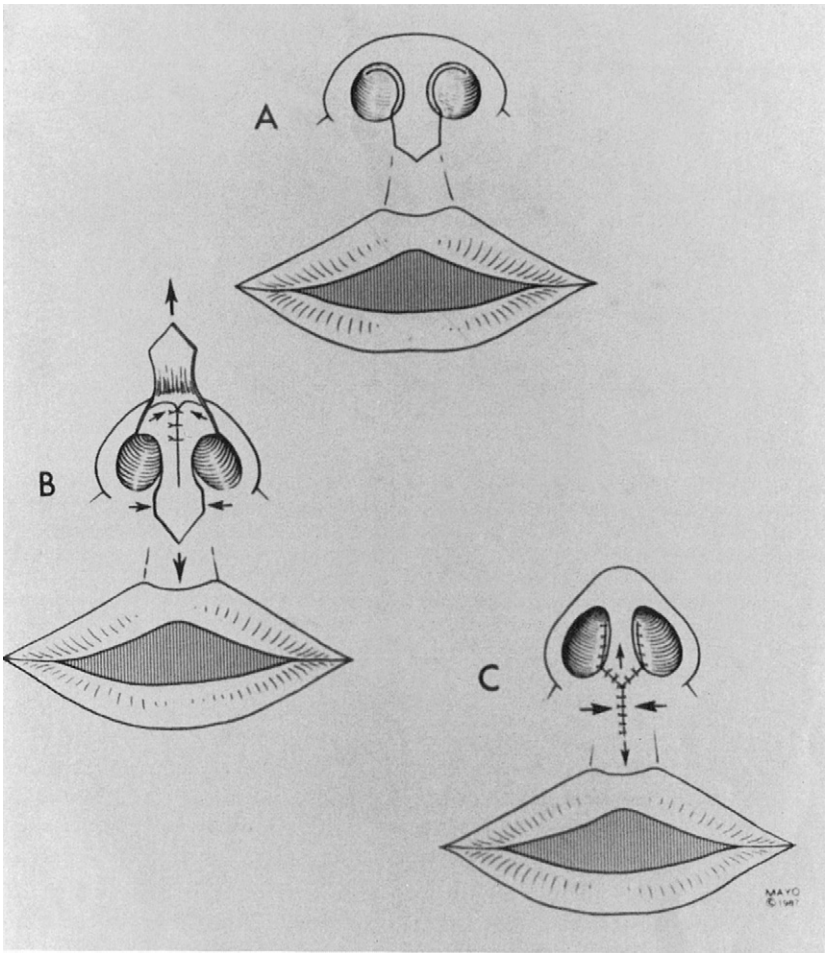

Figure 5 • Central lip flap for columellar lengthening. **A,** Design of the incisions. Note the position of the incision on the columella and inside the alar rims. **B,** Lip-columellar flap elevated to expose the lower lateral cartilages. The latter are dissected and sutured in correct position. **C,** Flap replaced in an advanced position and the lip sutured. (From Jackson IT: Cleft lip and palate. In Mustarde JC, Jackson IT [eds]: Plastic Surgery in Infancy and Childhood, 3rd ed. Edinburgh, Churchill Livingstone, 1988.)

regarding whether the nose should be corrected at the time of maxillary surgery or at a later date; the latter sequencing is preferred.

The columella frequently requires lengthening, which is achieved by making bilateral rim incisions and continuing on both sides of the columella. The incisions are extended on the central area of the lip to form a flap of the required length. The flap is elevated and the incisions are continued into the columella and nasal tip to provide an open approach so that the lower lateral cartilages can be trimmed. The cartilages are sutured together with a stacked graft of septum or ear cartilage to increase tip projection and definition (Fig. 5). Nasal bone infracture may provide a satisfactory dorsal nasal contour, but in more deficient cases, an onlay bone graft is required. The graft is inserted through a small glabellar incision—a slot is cut in the bone and the graft is held in position with a countersunk screw. If a slot of adequate dimensions is cut in the frontal process, the inferior edge acts as a point of leverage, or cantilever, to determine the position of the tip. When this technique is used, there is no need for cartilage grafting of the tip because the dorsal augmentation elevates the tip. Alternatively, cartilage can also be used in cases requiring only minimal dorsal augmentation (e.g., septal, ear, or rib cartilage).

Orthognathic Surgical Procedures

It is not uncommon to have older patients with either unilateral or bilateral repaired clefts who present with jaw disproportion. The maxilla is

usually retruded, but in some cases the mandible may be prognathic; the latter finding may be only relative in relation to the position of the maxilla.

The patient is examined together with the orthodontist; cephalograms and dental models are obtained (see the chapter "Orthognathic Surgery"). A joint decision is made regarding the preferred osteotomy and preoperative orthodontic management. If a fistula is present, closure is deferred until after the orthodontist has achieved the desired occlusion. The bony defect is subsequently grafted. After the bone graft has become incorporated, the orthognathic surgery is undertaken, ideally in a single-stage procedure. If it is a single-jaw case, be it maxilla or mandible, a LeFort I or sagittal split osteotomy is performed to achieve the ideal occlusion. With an intraoperative occlusal splint, the osteotomy is plated and the maxillomandibular fixation is removed. The endotracheal tube is disconnected, grasped from an oral approach, pulled into the mouth, and reconnected. If desirable, it is now possible to carry out safely the nasal correction described earlier.

In two-jaw cases, an intermediate splint is used to position the maxillary segment. A definitive splint is subsequently applied to gain the final occlusion. The maxillomandibular fixation is removed after the sagittal split osteotomy and the tube is again changed to an oral position, and the rhinoplasty or septoplasty is performed.

On occasion, lip revision, with or without bone grafting, is also performed. These procedures are performed before the osteotomies, especially in secondary bilateral cleft procedures.

Performing the secondary surgery in this manner reduces operating time, is more economical, and from the patient's point of view, reduces time off work or school.

Pearls and Pitfalls

- Secondary surgery for cleft lip and palate requires the expertise of an experienced and well-coordinated clinical team. Failures and complications are few when patients are treated by surgeons and clinicians doing this type of work frequently.
- It is important to identify both aesthetic and functional problems associated with the cleft nose deformity.
- Wide subperiosteal undermining allows for repositioning of the ala and bone grafting of the deficient maxilla.
- Iliac crest bone grafts remain the material of choice for alveolar and maxillary bony defects.
- Occasionally, additional orthodontic therapy and surgery may be necessary. Most tertiary procedures are performed on the nose for residual aesthetic or functional problems.

SUGGESTED READINGS

Atlan G, Jackson IT, Topf J, Lash S: Combined composite correction of orthognathic, nasal and cosmetic deformities. Eur J Plast Surg 21:67-71, 1998.

Henderson D, Jackson IT: Combined cleft lip revision, anterior fistula closure and maxillary osteotomy; a one-stage procedure. Br J Oral Surg 13:33-39, 1975.

Jackson IT, Soutar DS: The sandwich Abbé flap in secondary cleft lip deformity. Plast Reconstr Surg 66:38-44, 1980.

Kaplan E: Lip repair: Problems and solutions. In Kernahan DA, Thompson HG (eds): Symposium on Pediatric Plastic Surgery, vol 21. St Louis, Mosby, 1982.

Posnick JC: Cleft orthognathic surgery. In Vander Kolk CA (ed): Plastic Surgery: Indications, Operations and Outcomes, vol 2. St Louis, Mosby, 2000.

Alveolar Bone Grafting

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Children with cleft lip and palate deformity require a specialized interdisciplinary approach to their many complicated problems. Currently accepted protocols call for sequencing of treatment interventions along a timeline extending from birth to adulthood. Such staging balances the needs of the patient with the consequences of the intervention (i.e., maxillary growth). The rehabilitation of dentition is a critical component of this sequence and contributes significantly to oral function, facial aesthetics, and psychosocial development.

Etiopathogenesis

Clefting of the alveolar process is present in the majority of patients with cleft lip and palate. A perialveolar, oronasal fistula, commonly associated with the bony cleft, communicates between the alveolus, anterior hard palate, and floor of the nose. Early correction of the lip and palate fails to close the cleft and oronasal fistula present at the alveolus. The fistula can be a significant source of morbidity by allowing the oral contents to pass into the nasal cavity. Inspissation of food particles contributes to poor oral hygiene, gingivitis, periodontitis, and early dental caries in adjacent teeth. Permanent closure of the cleft with bone grafting and local flaps effectively eliminates these issues.

Goals of Reconstruction

The presence of the cleft destabilizes the maxillary arch and predisposes it to medial collapse. Permanent stabilization of the maxillary segments into a functional dental arch form is achieved by reconstruction of the alveolus and anterior hard palate with bone grafts. Effective alveolar bone grafting also stabilizes the premaxilla of the bilateral cleft with the lateral maxillary segments. In addition, reconstruction of the alveolus is the cornerstone of stabilizing the orthodontic movements required to achieve and maintain normal occlusion throughout the maxilla. Orthodontic expansion and correction of the maxillary arch cannot be maintained unless repair of the alveolar cleft is coordinated with the desired orthodontic movements.

The teeth at the margins of the cleft are susceptible to dental caries and early loss, especially in the presence of oronasal fistulas, because of the tenuous bony and soft tissue support around the margins of the cleft. Grafting of the alveolus allows for orthodontic movement of teeth in and about the cleft and thus eliminates a dental gap and reliance on dental prosthetics.

The roof of the cleft is the base of the nasal ala. Bone grafts to fill the superior defect restore support of the alar base. Lip projection and symmetry and leveling of the slumped ala on the cleft side are also improved by restoring this volume.

Timing

The ideal age at which to reconstruct the alveolus remains controversial. Advocates of early procedures (including primary gingivoperiosteoplasty and primary bone grafting) argue that early cleft correction allows early orthodontic alignment of the teeth, correction of occlusion, and restoration of facial aesthetic balance without interference with subsequent maxillary growth. In contrast, a number

of centers have documented that early intervention causes later restrictions in maxillary growth. At issue is the question of whether elevation of the periosteum off the maxilla significantly retards later appositional growth of the maxilla. Reports comparing the growth characteristics of patients treated by *primary bone grafting* with other patients undergoing *secondary bone grafting* did not demonstrate any significant growth disturbance. These findings may eventually lead to changes in the manner in which the alveolus is approached.

Others have advocated early correction of the cleft alveolus with *primary gingivoperiosteoplasty*. This procedure entails raising small gingivoperiosteal flaps from the margins of the cleft before eruption of the primary dentition. The procedure is highly dependent on presurgical approximation of the cleft margins via orthodontic molding. The composite flaps are reported to induce neoosteogenesis within the cleft, thus obviating the need for grafting. By minimizing the size of the flaps, this procedure may avoid growth disturbances in the developing maxilla.

Growth issues aside, primary grafting has significant limitations. Graft harvest is more complicated in a small child. Operating on the alveolus before the time of primary dentition risks damage to the developing tooth buds. Alveolar bone grafts placed before the eruption of permanent dentition have a tendency to resorb, and secondary grafting procedures may be required. Primary procedures do not seem to result in a significant reduction in the occurrence of oronasal fistulas, and their persistence requires additional operations, thus negating to a large degree the rationale underlying early correction of alveolar clefts. Therefore, until all data concerning more radical approaches have been analyzed, the alveolus should be reconstructed as a secondary procedure at the time of mixed dentition (early secondary grafting).

Maxillary growth and dental age are the predominant considerations in determining the timing of alveolar reconstruction. Maxillary growth is complete near the age of 8 years, whereas the maxillary canine does not usually erupt before the age of 10. Most surgeons agree that grafting should be completed before canine eruption to allow for eruption of the tooth through the graft, thereby promoting stability of the tooth.

The interval between the conclusion of maxillary growth and eruption of the permanent canine allows time for preoperative orthodontic movement of the maxillary segments and complete alveolar reconstruction. The intervention is coordinated with the dental age of the patient and begins after eruption of the first molar. The maxillary arch can be expanded to normal dimensions, a maneuver that often widens the cleft. Alignment and occlusion are maximized during this period, which usually takes 4 to 6 months. Bilateral clefts may require additional time if significant posterior movement of the premaxilla is necessary. Coordination of orthodontic therapy with surgery is required because expansion and alignment are inherently unstable until reconstruction proceeds. After grafting, the reconstructed alveolus needs orthodontic stabilization for at least 6 weeks to allow for consolidation of the graft. Once consolidated, the maxillary arch dimensions can be considered stable, and orthodontic movement of teeth into the grafted alveolus can proceed.

Diagnostic Studies

Early in the orthodontic phase of treatment, the condition of the pericleft dentition should be assessed. Often, the lateral incisor in the margin of the cleft develops roots of insufficient quality to confer long-term stability to the tooth. If extraction is necessary, it should be done early in the orthodontic period and at least 4 weeks before bone grafting.

Survival of the bone graft within the cleft depends to a significant degree on the condition of the soft tissues at the time of grafting. Widening of the cleft as a consequence of orthodontic distraction of the maxillary arch often facilitates proper hygiene within the cleft and associated oronasal fistula. Proper hygiene minimizes soft tissue inflammation, which can be a significant impediment to bone graft survival. In addition, early removal of poor-quality teeth in the region improves the quality of the recipient bed and subsequent bone graft success. The condition of the gingiva is particularly important because coverage of the erupting surface of the cleft with healthy gingiva allows for natural eruption and long-term durability of the tooth.

Radiographic studies, including Panorex, selected periapical films, and occlusal films, are an integral part of the evaluation. They provide a valuable adjunct to clinical examination for assessment of the dentition in the vicinity of the cleft, as well as the dimensions and structure of the cleft itself. Serial films obtained during the period of preoperative orthodontic therapy are helpful in instituting appropriate treatment in a timely fashion.

Surgical Technique

The cleft is visualized in terms of a pyramid with medial and lateral walls (internal margins of the cleft), a roof (nasal floor), and an upward-sloping floor (lingual alveolus and palate) (Fig. 1). To obtain satisfactory soft tissue coverage, which is critical for graft success, all sides of the pyramid should be adequately closed with viable flaps. Ideally all surfaces of the cleft defect should be lined with periosteum or bone to facilitate bone graft take.

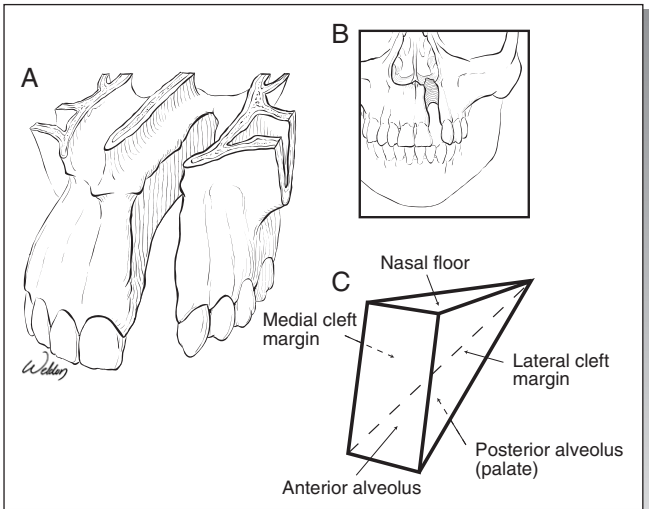

Figure 1 • A and **B,** Alveolar cleft viewed as a pyramid. **C,** Healthy soft tissue coverage on all aspects of the cleft is required for successful reconstruction.

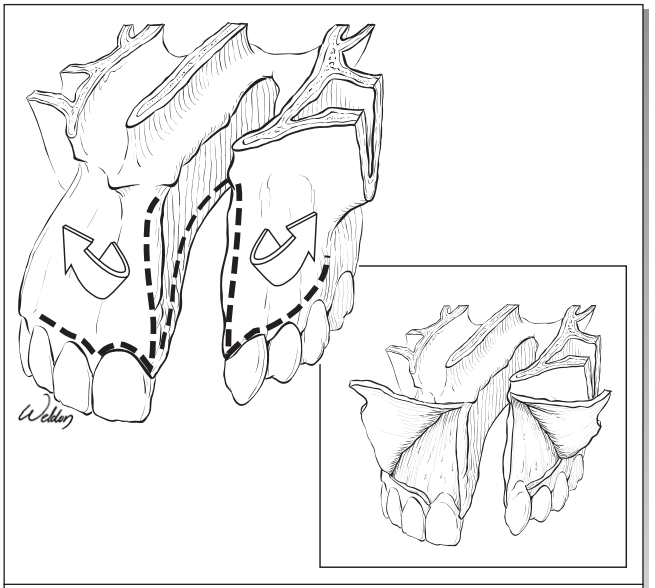

Figure 2 • Planned incisions for flaps used to close the cleft alveolus. Labial and palatal gingivoperiosteal flaps are raised at the gingival margins both medial and lateral to the cleft to provide coverage to the floor of the defect and the anterior alveolus. The incision preserves the gingival margin on teeth that are not adjacent to the cleft. These incisions are adjusted onto the alveolus and palate when the cleft is wide to provide adequate flap length for closure of the nasal floor.

Unilateral Clefts

For unilateral clefts, labial gingivoperiosteal flaps are raised both medial and lateral to the cleft (Fig. 2). The gingival margin of the tooth adjacent to the cleft is raised with flaps to ensure gingival coverage over the tooth-erupting surface. Distal to this tooth the incision is designed to preserve 2 to 3 mm of gingival margin. The incision usually ends near the mesial aspect of the first molar lateral to the cleft. In the case of very wide clefts, the flap is extended distal to the first molar. The incision is extended around the anterior margin of the cleft into the gingivolabial sulcus and medial margin of the cleft and carried to the opposite gingival margin. The medial flap is developed past the opposite central incisor. Subperiosteal dissection is performed to the margins of the piriform aperture.

Similar gingivoperiosteal flaps are developed on the palatal side (the medial and lateral aspects of the cleft). Closure of the palatal and labial gingivoperiosteal flaps together eventually provides coverage for the floor and anterior walls of the theoretical pyramid.

At this stage, the roof of the cleft is still open, and the medial and lateral margins of the cleft are covered by the margins of the fistulous tract. The nasal floor (roof) is closed by forming superiorly based rectangular flaps from the mucoperiosteum lining the medial and lateral margins of the cleft. These flaps are elevated and rotated superiorly in the subperiosteal plane (Fig. 3). Closure is obtained with 4–0 Vicryl sutures. At the posterior junction of the roof and the floor where the four flaps merge (two palatal flaps and the two superiorly based marginal flaps), a single key suture placed through both palatal flaps will hook the most posterior stitch in the nasal floor closure and complete the posterior closure (Fig. 4).

In the case of tenuous soft tissue coverage, a decision to stage the procedure is made, at which time all incisions are closed over the cleft to achieve soft

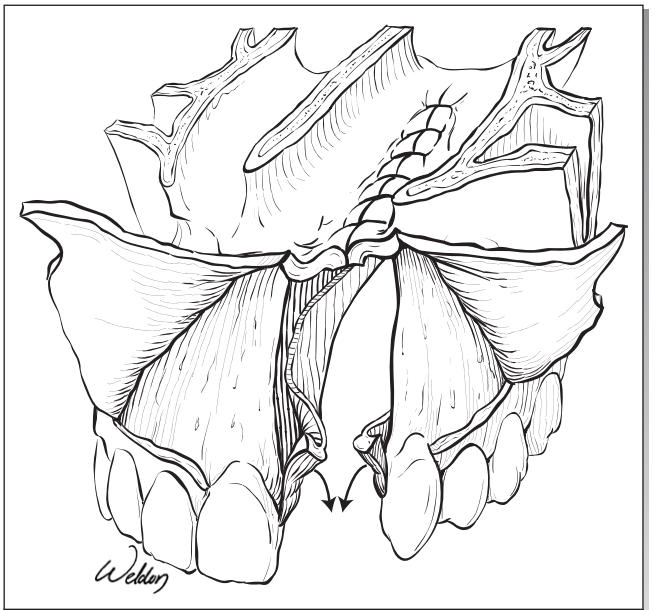

Figure 3 • Soft tissue–periosteal flaps are developed from the medial and lateral margins of the cleft to close the roof of the defect (nasal floor). In the presence of an oronasal fistula, the soft tissue represents the margins of the fistulous tract.

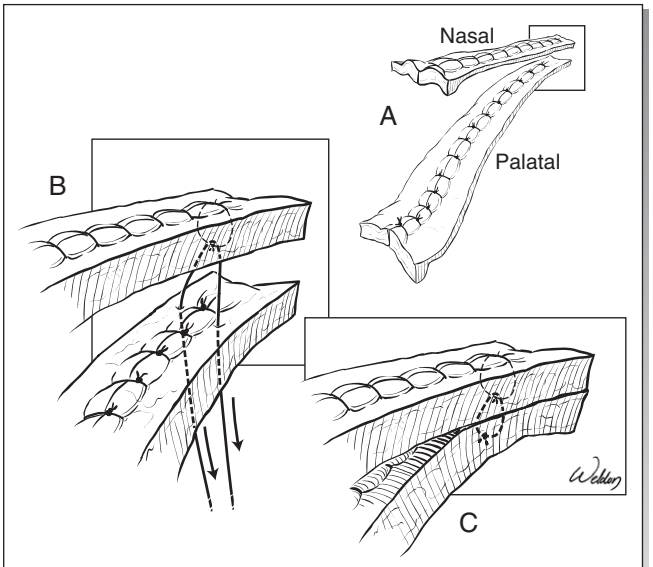

Figure 4 • A and **B,** Diagram illustrating closure in the posterior aspect of the cleft where the floor and the roof merge together. **C,** A single suture is used to close this aspect of the flap and complete the closure of the cleft space.

tissue coverage for future bone grafting. If adequate healthy soft tissue coverage is available, the bone graft is harvested.

A segment of cortical bone can be wedged into the roof of the defect (nasal floor) and the anterior alveolus to provide a durable structure to pack the graft against (Fig. 5). This technique provides additional bulk to the nasal floor and augments the support to the nasal base and upper lip. Alveolar height is slightly overcorrected. The incisions are closed with 4–0 Vicryl (Fig. 6).

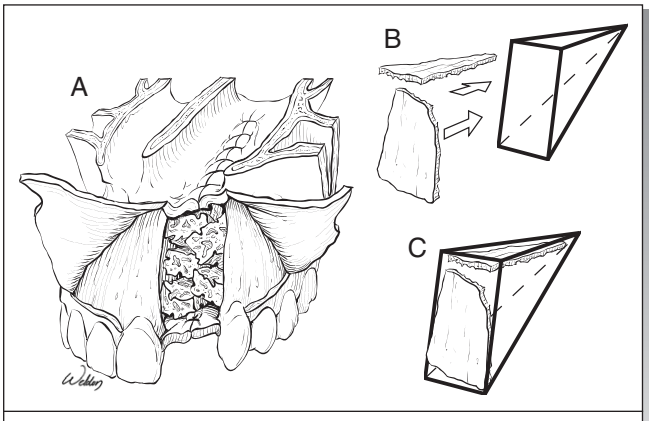

Figure 5 • A, Defect after grafting with autogenous bone. **B** and **C,** Cortical bone is used to augment the nasal floor and anterior alveolus. These bone segments allow the defect to be filled without putting pressure on the flaps.

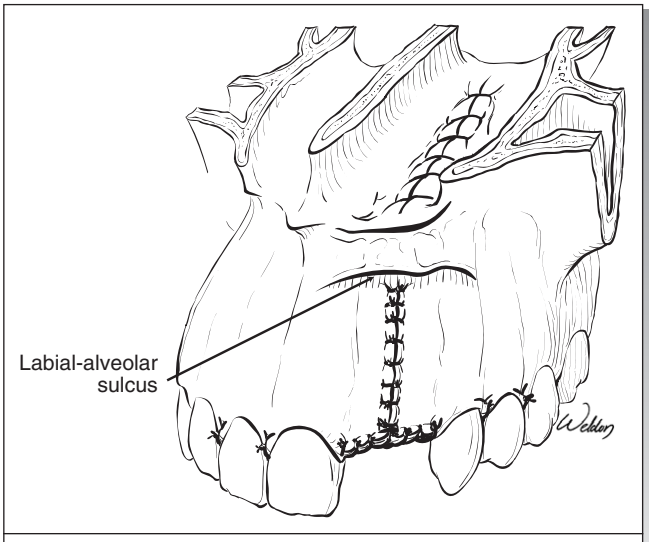

Figure 6 • Final closure of the flaps.

Bilateral Cleft

In the case of a bilateral cleft lip, the limited perfusion of the premaxilla prevents extensive mobilization of the labial mucosa and gingivoperiosteum. The labial soft tissues must be preserved anteriorly to maintain viability of the premaxilla. Without medial labial flaps, the volume of soft tissue available for closure of the cleft is significantly less. Lateral gingivoperiosteal flaps are raised in a fashion similar to that described for a unilateral cleft. Frequently, the lateral flaps must be extended distal to the first molar in an attempt to compensate for the lack of medial flaps.

On the palatal side, gingivoperiosteal flaps can be raised over the premaxilla as long as the labial mucosa is totally preserved. Lateral gingivoperiosteal flaps are developed in a similar fashion. The roof of the clefts is closed with superiorly based nasal mucosa, the margins of which are incised to favor the palatal side so that adequate flap length is developed and the nasal floor (cleft roof) is closed. Bone grafting proceeds in a fashion similar to that in patients with unilateral clefts. If the premaxilla is mobile, it requires immobilization during the postoperative period with a prefabricated acrylic splint placed over the occlusal surfaces of the maxillary teeth and secured to orthodontic brackets.

Because patients with a bilateral cleft and those with a wide unilateral cleft have the lowest success rates, staging significantly improves the overall success of the procedure.

The preference for autogenous bone graft is the iliac crest harvested from a relatively concealed incision. After a small incision is made, subperiosteal dissection proceeds around the crest onto the lateral cortex, where a square-shaped window is removed with a sharp osteotome. Cancellous bone is harvested with a medium-sized curet. The outer

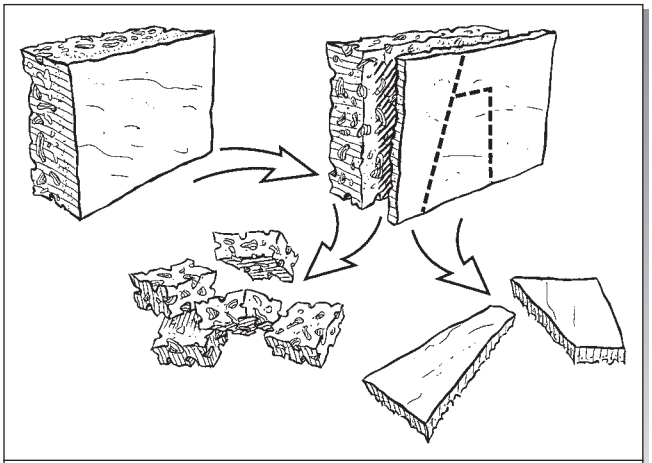

Figure 7 • Cortical segments are designed to fit the nasal floor and anterior alveolus (see Figure 5).

cortical bone is used to augment the nasal floor and anterior alveolus as described earlier (Fig. 7).

Postoperative Care

The iliac crest drain can often be removed the morning of discharge, but it remains if drainage persists.

Beginning the day after surgery, patients are encouraged to brush the operative site with a soft brush after each meal and every morning. A water pick is not used because it disrupts the gingival repair and lateral gingival donor sites, which heal by secondary intention.

Postoperative orthodontic therapy to close the dental gap and guide the erupting canine into position can begin 6 weeks after reconstruction, depending on the condition of the soft tissues. Such a schedule allows adequate time for consolidation of the bone graft and healing of the soft tissues.

Pearls and Pitfalls

- Reconstruction of the alveolus with bone grafts should be delayed beyond the age of 8 years to avoid restriction of maxillary growth.
- If reconstruction is performed before eruption of the canine, a healthy canine can be preserved to allow for later closure of dental gaps.
- Reconstruction of the alveolus also provides the needed volume to support the alar base, which is a significant contribution to the cleft nasal deformity.
- Maximizing the soft tissue, bony, and dental conditions about the cleft in the preoperative period improves outcomes.
- Revascularization of the bone graft is significantly influenced by the quality of soft tissue support provided to the graft. Before attempting reconstruction, every effort should be made to provide for coverage of the graft with healthy tissue, which requires appropriate timing with orthodontic movements, timely removal of teeth in poor condition about the cleft, and proper hygiene, especially in patients with oronasal fistulas.
- Procedures are often staged to ensure adequate soft tissue availability before grafting.

SUGGESTED READINGS

Amanat N, Langdon JP: Secondary alveolar bone grafting in clefts of the lip and palate. J Craniomaxillofac Surg 19:7, 1991.

Freide H, Johanson B: A follow-up study of cleft children treated with primary bone grafting. I. Orthodontic aspects. Scand J Plast Reconstr Surg 8:88, 1974.

Posnick JC, Thompson B: Cleft-orthognathic surgery: Complications and long-term results. Plastic Reconstr Surg 96:255, 1995.

Rosenstein SW, Dado DV, Kernahan D, et al: The case for early bone grafting in cleft lip and palate: A second report. Plast Reconstr Surg 87:644, 1991.

Wood RJ, Grayson BH, Cutting CB: Gingivoperiosteoplasty and midfacial growth. Cleft Palate Craniofac J 34:17, 1997.

Cleft Palate

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A cleft palate precludes the normal functions of speech and swallowing, and the lack of separation of the oral and nasal cavities leads to chronic irritation. The disrupting effects of a cleft palate on eustachian tube function are uniform, and therefore hearing loss can occur. Surgeons treating cleft palate must recognize these important derangements. They must also formulate a treatment plan that restores normal speech function with the least adverse sequelae from the intervention, such as impairment of maxillary development, and at the same time correct the deformity with a low percentage of secondary procedures.

Etiopathogensis

The cause of cleft palate is multifactorial. Genes, drugs, and environment are contributing factors, yet no clearly defined causal relationship has been established. There is a difference in the etiology of isolated cleft palate and cleft palate associated with cleft lip. When associated with cleft lip, the incidence of cleft palate follows that of cleft lip. However, when cleft palate occurs without a concomitant lip cleft, the incidence becomes constant across all racial groups at approximately 1 in 2000. When found in isolation, it is much more likely to be associated with a syndrome. In fact, up to 50% of cleft palates without cleft lip are associated with other abnormalities or are part of a syndrome.

Pathologic Anatomy

The pathologic anatomy of cleft palate can be divided into defects of the hard palate and those of the soft palate. The normal function of the hard palate is to provide a fixed, nonmobile partition between the oral and nasal cavity. It prevents regurgitation of food into the nose and serves as a mechanical block against which the tongue can direct the food bolus to the pharynx. Cleft palate can extend anteriorly as far as the incisive foramen or, in the case of cleft lip and palate, through the alveolus and the anterior maxillae. Reconstruction of the hard palate should reestablish this partition. Although the defect is a combined bony and soft tissue defect, the reconstruction must reestablish only the separating function.

The other pathologic feature is cleft of the soft palate and, most importantly, loss of normal muscular anatomy. In the normal state, the palatoglossus, palatopharyngeus, and levator palati muscles all insert into the contralateral muscle in the midline to form a muscular sling. In cleft palate, they aberrantly insert on the posterior portion of the hard palate. Of these muscles, loss of function of the levator palati muscle is the most crucial because this muscle is responsible for elevation and posterior movement of the soft palate onto the pharyngeal wall. This dynamic obturator allows nasal breathing and escape of nasal consonants when relaxed and permits buildup of pressure in the oral cavity and prevention of nasal regurgitation of air and food when tightened. In the cleft state, the nasal cavity cannot be separated from the oral cavity. It is therefore impossible to build up the oral pressure necessary for normal speech.

In addition, the important function of the tensor tympani muscle is lost because of the aberrant insertion of the musculature. This muscle normally serves, in conjunction with the levator veli palatini, to open the eustachian tube. In adults, the eustachian tube orifice is inferior to the middle ear. As a result, any middle ear fluid passively drains into the pharynx. In children, the eustachian tube orifice is superior to the middle ear cavity. In the normal state, as the tensor tympani contracts in conjunction with the levator palatini, the eustachian

tube orifice opens and the pump-like action of the muscle contraction facilitates drainage of the middle ear. When this function is lost, the middle ear is prone to serous otitis and patients are uniformly left with ear disease and resulting hearing loss if untreated.

It is important to note that cleft palate occurs on a spectrum. One end of the spectrum is a bifid uvula with normal muscular anatomy. This condition obviously does not require any intervention. Cleft palate can also occur as a submucous cleft in which the soft tissue is not separated, but the muscular anatomy is abnormal and consistent with a cleft palate. There may be a zona pellucida (central thin area with no muscle) or a notch in the hard palate. Some patients with a submucous cleft, like cleft palate patients, are unable to obtain nasal competence and therefore require surgical intervention if proper speech is to be expected.

Diagnostic Studies

Diagnostic studies for cleft palate are centered on the middle ear and diagnosis of any associated syndromes. Patients should undergo evaluation for possible placement of middle ear drainage tubes at the time of cleft palate repair because most patients (approximately 95%) have serous otitis. Hearing evaluation may be necessary and should be performed on an individual basis. Because isolated cleft palate is commonly associated with a syndrome, cardiac, renal, and neurologic evaluation should be performed.

Goals of Reconstruction

Cleft palate should be treated such that the patient has no difficulty with feeding, speech quality, or deglutition. Ideally, treatment should have no effect on facial growth. If a cleft palate is left untreated to adulthood, facial growth is unimpaired. However, poor speech patterns and compensatory articulations that cannot be corrected will have developed. Conversely, if the cleft is repaired at the time of lip repair (3 to 4 months), the patient will have no compensatory articulations, no difficulty with feeding, but midface retrusion and poor facial growth may result. Therefore, the surgeon must balance these factors. The inability to perform proper speech rehabilitation leads most surgeons to intervene at the beginning of speech development, or approximately 11 to 13 months of age.

The goal of surgery is to recreate a continuous hard palate with palatal tissue that allows no communication between the oral and nasal cavities. The surgery must also close the soft palate by providing the mobile tissue needed to create a dynamic obstruction separating the oropharynx and nasopharynx. At the same time, it should reconstruct the muscular anatomy to that of the normal state to allow the action of the levator veli palatini muscle to elevate the mobile soft palate and separate the nose. These goals should be accomplished without creating significant scarring of the maxilla. Although all areas of dissection heal with scars, large areas of uncovered bone will have extensive fibrosis and thus increase the incidence of midface retrusion. This should therefore be avoided.

Treatment

Nonsurgical

Nonsurgical treatment (i.e., treatment with obturators) should be mentioned only for historical reasons because it has no place in primary cleft management. Obturators are still occasionally useful for salvage following disastrous complications, but such treatment is unnecessary if proper cleft management is instituted from infancy.

Surgical

A Dingman mouth gag is placed and the patient positioned in a neurosurgical head holder such that the neck is fully extended. The entire hard and soft palate is injected with a solution of 1% lidocaine with 1:100,000 epinephrine. In addition to antibiotics, patients are given 0.5 mg/kg of dexamethasone (Decadron) because it reduces the tongue swelling associated with the procedure and tongue compression from the mouth gag.

Different clinical findings call for different surgical techniques. The von Langenbeck design is useful for posterior clefts, whereas the Bardach design is preferred for unilateral clefts. The Oxford designs can be applied to either bilateral or posterior clefts. Regardless of whether a von Langenbeck, Bardach, or Oxford design is chosen for the hard palate, the principles remain the same: the design should have the fewest number of flaps with the fewest number of tripoints. The Bardach and Oxford designs add technical ease to the procedure by improving visualization, especially with dissection of the pedicle, and in this chapter the Bardach-type design is illustrated. The von Langenbeck technique (anteriorly the flap is not incised free of the palate) yields similar results in experienced hands, uses fewer incisions, and is better suited for repair of an isolated cleft of the soft palate or submucous cleft. The Oxford design is best suited for bilateral cleft lip and palate and is now often performed without the push-back component in order to avoid large areas of raw bone. The push-back technique is not necessary for satisfactory speech results (Fig. 1).

The incision is made to the bone of the hard palate along the margin of the clefts slightly on the

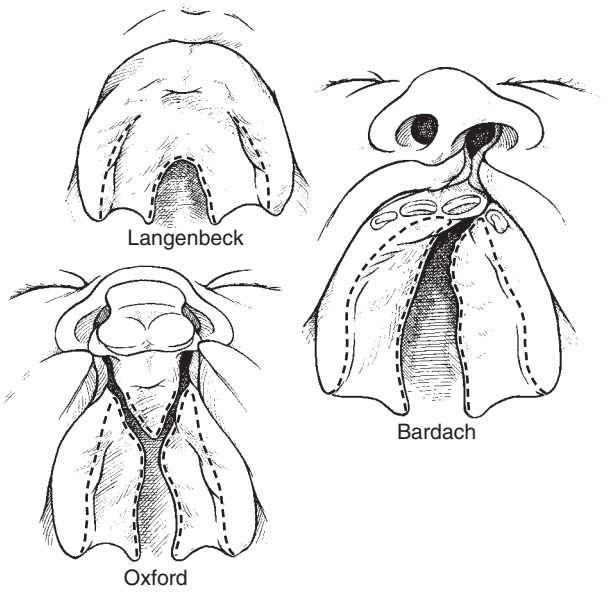

Figure 1 • The three common approaches to the hard palate. The Bardach method is useful for complete unilateral clefts. The Langenbeck method is useful for clefts of the soft palate only. The Oxford method is useful for complete bilateral clefts.

oral side (Fig. 2). Such an incision provides a small amount of extra tissue for nasal closure. The incision is then carried up the soft palate to the tip of the uvula. An incision is made to the underlying bone starting between the maxillary tuberosity and the hamulus and extended anteriorly along the alveolar ridge to meet the medial incision at the anterior limit of the palate. The incision should extend well beyond the area of the incisive foramen because this is the site of potential fistulas. The flap is dissected off the bone with an elevator. After the flap is elevated, the mucosa is raised from the nasal side of the bone. If the cleft palate is associated with a cleft lip, the elevation will probably include elevation of a vomer flap anteriorly.

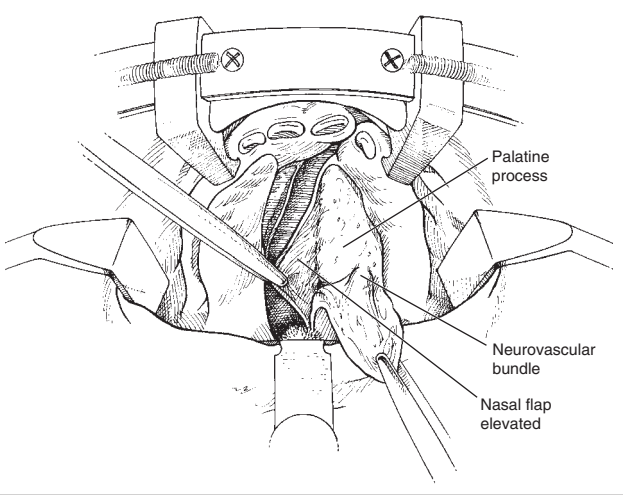

Figure 2 • Dissection of the nasal side of the soft palate.

After the oral and nasal sides of the hard palate are elevated, it is important to use the elevator to dissect the muscle insertion from the posterior aspect of the hard palate. The elevation should proceed cranially, and scissors can often be used to liberate this tissue from the underlying bone. The nasal side of the soft palate is elevated after disinsertion of the muscle of the posterior palate. The thin nasal mucosa is separated from the muscle while taking extreme care to avoid injury of the muscle. The most medial aspect is left slightly thick because the levator is not present in the cleft edge. Two to 3 mm into the dissection, a slight blue hue is encountered when the proper dissection plane is entered. Often, small holes are made in the nasal mucosa as this thin layer is elevated, and they do not need to be repaired.

The mucoperiosteal flap should be completely liberated except for its attachments at the pedicle of the greater palatine artery. It is easily appreciated that the stiff periosteal cone limits movement of the flap and provides excessive tension in the area of the hard-soft palate junction. The periosteal cone is placed on extreme tension and a knife is used to first incise the posterior and lateral aspects (Figs. 3 and 4). Pouching of fat from the incision indicates release. Spreading scissors between the pedicle and the maxillary tuberosity completes the periosteal release, and the flap will move dramatically. The periosteal release must be performed before muscle dissection. In the event that the vascular pedicle is divided, the muscle bundle will provide an alternative blood supply to the mucoperiosteal flap if it is not separated from the flap.

The muscle dissection proceeds in three phases: nasal mucosa dissection (described earlier), oral

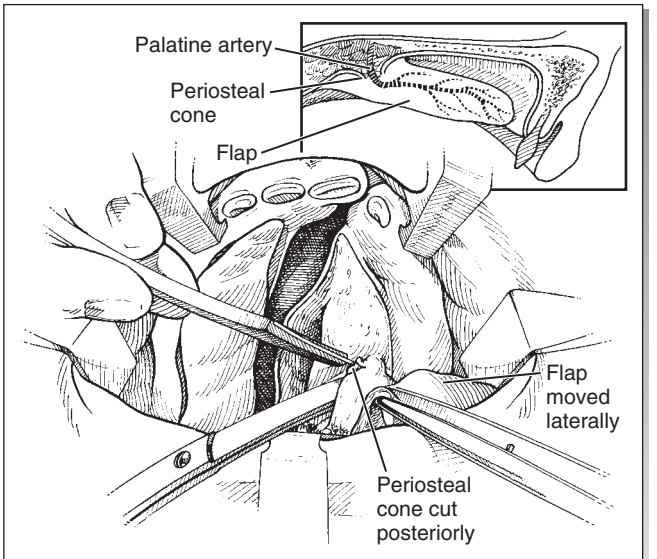

Figure 3 • Release of the periosteal cone. Fat will pouch as the periosteum is lightly incised.

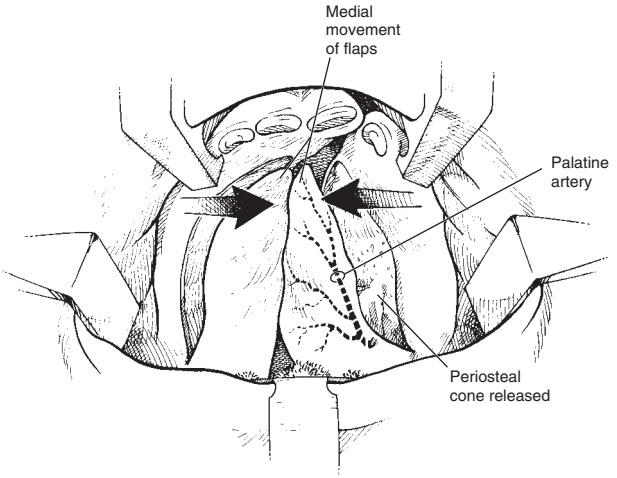

Figure 4 • Mobility of the mucoperiosteal flap after division of the periosteal cone surrounding the vascular pedicle.

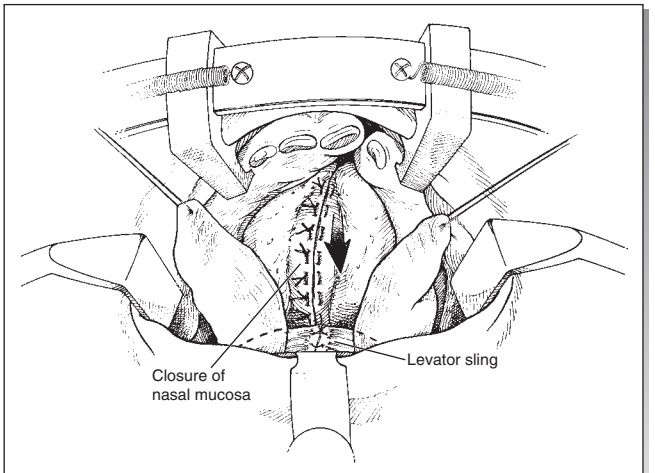

Figure 6 • Repair of the nasal side of the hard and soft palate and repair of the levator muscles.

mucosal dissection, and division of the tensor veli palatini tendon. Again, the first step of the muscle dissection is performed before release of the periosteal cone because the visibility gained facilitates the release and does not risk the secondary blood supply to the mucoperiosteal flap.

The levator muscle is dissected from the palatoglossus and palatopharyngeus muscles. The target for this dissection should be over the hamulus because the levator muscle originates at the skull base. Some surgeons choose to dissect between the mucosa and the palatoglossus and palatopharyngeus muscles. Such dissection yields fine results; however, these muscles are natural antagonists to the important levator muscle, and repair of them is probably not necessary.

Once the levator is completely separated from the nasal and oral layers, it becomes evident how firmly the levator muscle is attached to the hard palate by the tensor veli palatini tendon. This tendon is divided with scissors, and the levator is bluntly dissected free toward the skull base (Fig. 5). The neurovascular pedicle to the levator enters from the lateral side, and care should be taken to avoid injury to the pedicle.

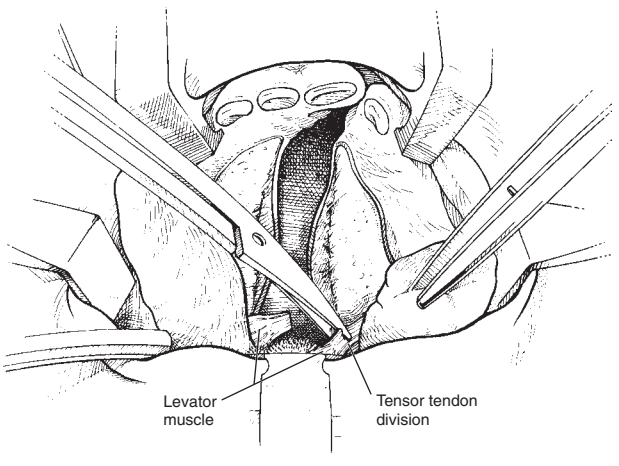

Figure 5 • The most critical part of the muscle repair is division of the tensor tendon.

Closure begins anteriorly and the nasal layer is approximated. The levator muscle is also repaired (Fig. 6). If the muscle repair is made too tight, the patient will have nasal obstruction and even sleep apnea. If made too loose, the patient will have velopharyngeal insufficiency. Judgment and experience must guide the surgeon in placement of proper tension. The oral side is closed, and one or two sutures should include the nasal closure to establish the height of the palatal vault. Loosely tied sutures should be placed to bring the lateral incision together (Fig. 7).

A tongue suture is placed for airway control. The patient should be fully awake before extubation, and emergency airway precautions should be taken.

The Furlow repair is a common technique for cleft palate repair that also yields good results. With the use of a Z-plasty, it avoids a straight line scar of the soft palate and its inevitable contracture. It does not, however, result in an anatomic muscle repair.

Postoperative Care

Elbow restraints are worn continually for 4 to 6 weeks. The airway is observed and the child placed on an oxygen monitor because the cleft patient now has a more obstructed nasal cavity that may cause some difficulty breathing and mild sleep apnea in the early postoperative period. Apneic problems can occur in the immediate postoperative period but can usually be controlled with traction on the tongue

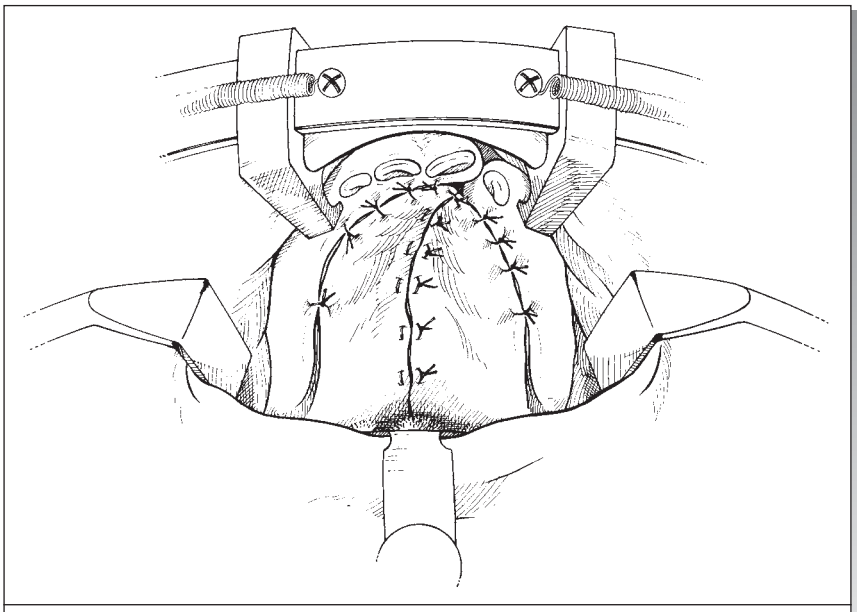

Figure 7 • The final repair. The lateral margins are only loosely reapproximated.

suture. Patients are given a liquid-only diet and are not allowed gelatin. Solids are swallowed by action of the tongue forcing the bolus onto the hard palate and directing it to the pharynx, and this action could disrupt the repair. Pain medication consists of rectal suppositories of acetaminophen and codeine elixir as needed for the first 24 hours. The child is usually discharged the following day after adequate oral intake. The liquid diet is continued for 3 weeks, and the child is transitioned to a soft diet for an additional 3 weeks.

Pearls and Pitfalls

- Interdisciplinary care is essential.
- The periosteal cone around the pedicle of the greater palatine artery must be completely released to allow tension-free medial movement of the mucoperiosteal flaps.
- The periosteal cone should be released before dissection of the levator muscle. The muscle offers an alternative blood supply in the event of injury to the pedicle.
- The tension of the levator sling is critical. Excessive tension results in nasal obstruction or sleep apnea, and a loose repair results in velopharyngeal insufficiency.

SUGGESTED READINGS

Cutting CB: Smile Train Instructional Videos, vol 2. Cleft Palate. Smile Train International.

Millard R: Cleft Craft—The Evolution of Its surgery. Philadelphia, Lippincott Williams & Wilkins, 1980.

Salyer K: Salyer and Bardach's Atlas of Craniofacial and Cleft Surgery. Philadelphia, Lippincott-Raven, 1999.

Sommerlad BC: A technique for cleft palate repair. Plast Reconstr Surg 112:1542-1548, 2003.

Velopharyngeal Insufficiency

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AMY ROGERS

Velopharyngeal insufficiency (VPI) can result from a variety of anatomic and physiologic abnormalities. In this chapter, VPI is discussed mainly in relation to patients with a repaired cleft palate.

Etiopathogenesis/Pathologic Anatomy

There are six possible reasons for escape of excess air from the mouth into the nose:

1. The palate may be anatomically short and thus does not make contact with the posterior pharyngeal wall.
2. The palate is badly scarred, which causes impaired palatal mobility and thereby impedes velar closure.
3. The original operation did not correctly position the palatal musculature sling. This mistake most commonly results from insufficient appreciation of the importance of reestablishing the correct anatomic position of the muscles at the primary palate repair procedure. In this situation, the palate, even if its length is adequate, nevertheless fails to reach the posterior pharyngeal wall.
4. The lateral pharyngeal walls do not move sufficiently medially to effect closure.
5. There is ineffective sphincter closure, perhaps caused by muscular weakness or poor muscle positioning.
6. A large fistula with a poorly fitting obturator is preventing a satisfactory seal.

Speech Pathology

The speech-language pathologist is responsible for monitoring and assessing all aspects of speech and language in patients with cleft lip or palate (or both), including language, articulation, voice, resonance, and oral structure and function. In addition, the speech pathologist plays an important role in assessing velopharyngeal function, determining treatment options in consultation with the surgeon, and evaluating the overall effectiveness of pharyngeal management (Fig. 1).

Children born with a cleft lip and palate are at risk for resonance, articulation, and expressive language problems that may impair their communication for many years. The impact of a palatal cleft may be evident during infant vocalizations and can persist well after an adequate oral-pharyngeal mechanism has been established. The most remarkable speech production problems demonstrated by children with cleft palate are those related to VPI, including hypernasality, audible nasal emission, weak pressure consonants, and compensatory articulation patterns. In considering the speech of individuals with palatal clefts, it is important to remember that many children do not have hypernasality after palatal surgery because valving integrity is frequently achieved in most children. The contemporary clinical expectation is that speech after palatal repair should be normal or nearly so. If this goal has not been realized, explanations should be sought.

Several external variables affect the type and amount of treatment that a child with cleft palate speech receives: cognitive skills, linguistic ability, personality, and motivation. Another factor is the availability of a school-based speech-language

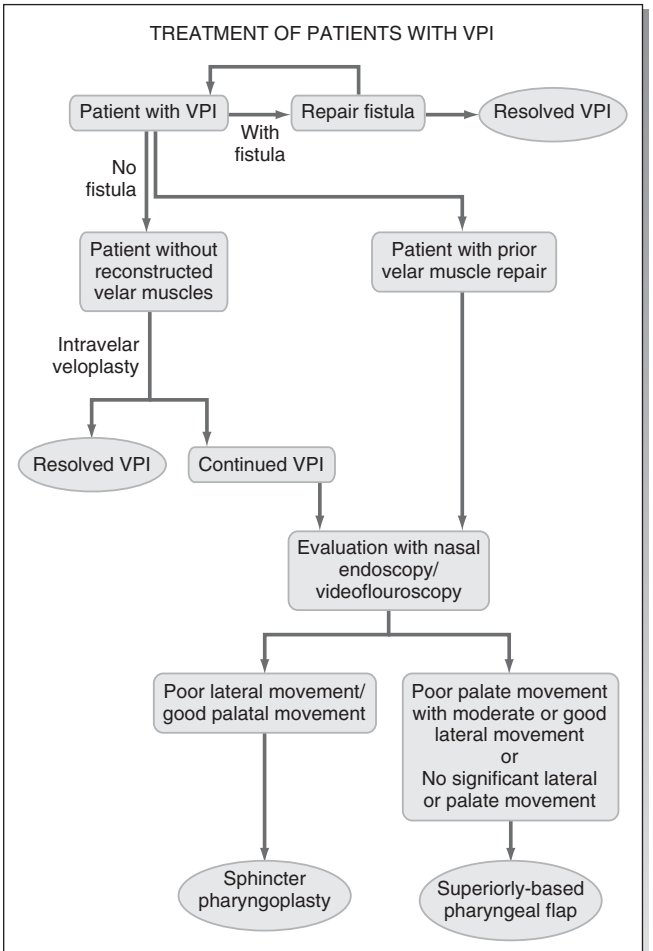

Figure 1 • Algorithm for the treatment of velopharyngeal incompetence (VPI).

pathologist who is knowledgeable about the treatment of speech symptoms related to velopharyngeal dysfunction.

Most children referred for speech therapy generally require intervention to enhance their articulation or phonologic development or their overall expressive language functioning. The speech pathologist must know when it is appropriate to initiate speech therapy, when it is appropriate to refer for physical management, and when it is prudent to defer management of all kind.

The majority of older children with cleft palate who are referred for speech therapy require intervention to establish correct phonetic placement. The initial goal of therapy is to correct articulatory placement. As the child acquires the appropriate phonetic patterns, the focus of therapy may shift to mastery of phonemic patterns. Hypernasal resonance, nasal emission, and weak pressure consonants are frequently reduced or eliminated by surgical or prosthetic management. However, presurgical compensatory articulations often persist postoperatively, even when adequate velopharyngeal valving can be achieved.

Protocol for Assessing Speech Production Problems

The primary purpose of assessing phonologic behavior is to determine whether the individual needs intervention and, if so, the direction of such treatment. The protocol for assessment of speech production problems in children with cleft palate varies, depending on the age of the child and the ultimate goal of therapy.

Several commercial articulation tests are available. For a child with cleft palate and velopharyngeal closure problems, items should be selected to test the child's performance on pressure consonants (i.e., stops, fricatives, and affricates). Two protocols designed specifically to test these consonants are the Bzoch Error Pattern Diagnostic Articulation Test and the Iowa Pressure Articulation Test. Connected speech also provides information about the consistency of the child's errors and the influence of context. Decisions about velopharyngeal function and the need for treatment should be based on how well the individual functions in conversational speech and daily communication. Some persons with marginal VPI sound normal on a single-word test of articulation but have nasalization during connected speech.

Speech analysis also reveals compensatory articulations or articulation patterns that the child has learned in an attempt to compensate for the cleft. Compensatory articulations usually take the form of sound substitutions or abnormal coarticulations, patterns often persisting even after velopharyngeal adequacy has been achieved.

The test battery should include stimulability testing, which consists of imitation of an adult model along with auditory or visual cues to improve a child's articulation of isolated sounds, syllables, words, or phrases. A child who shows potential to improve articulation through imitation may have the capability of imitating the articulatory gesture to produce a sound not typically in the child's phonetic repertoire. When VPI is present, it is important to monitor progress closely to avoid a lengthy program of unproductive therapy. An important part of speech analysis is to categorize the speech errors according to patterns. Such analysis is vital to understand which of the child's speech errors are developmentally age appropriate and which may have a structural or physiologic element as a result of the cleft. The following errors are seen in children with cleft palate: consonant distortions associated with nasal emissions, vowel distortions secondary to hypernasality, compensatory articulations, and selective articulatory backing substitutions.

Nonoperative Treatment of Speech Errors

The clinician should focus instruction on teaching articulatory movement and place of articulation.

Correction of compensatory articulations frequently involves training children to expand or modify their place of articulation repertoire. Cleft palate children may lack the articulatory gestures for correct production; they are taught how to make the target sound. Frequent treatment strategies in the early stages of speech intervention include mirror work, tactile cues, and the use of diagrams to represent articulatory placement. If compensatory coarticulations are evident, it is recommended that correct oral placement be established before attempting to eliminate compensatory coarticulations such as glottal stop.

One should initially select treatment targets that are produced with anterior placement and that are visible and unvoiced. One should also consider how stimulable the child is for correctly producing the sound and how developmentally appropriate that sound is. The goals of placement work should include teaching the child correct placement of the target phoneme, establishment of self-monitoring, and facilitation of generalization and carryover. Therapy should begin with phonemes that are stimulable, have an anterior place of production, are visible, and are developmentally appropriate. The use of whispered speech should be incorporated to facilitate oral valving and to reduce or eliminate glottal valving.

Diagnostic Studies

It is important to understand the methods available for investigation of VPI, as well as the relevance of each (Tables 1 to 4). For accurate diagnosis and formulation of the ideal surgical treatment plan, close cooperation between the speech therapist and surgeon is strongly emphasized.

The palate should first be examined with a speculum to visualize the length of the soft palate, the presence or absence of a fistula, and most important, the type, position, speed, and quality of movement of the soft palate. Limited or absent movement and a shortened soft palate both connote a poor prognosis after surgical rehabilitation. Conversely, documentation of satisfactory lateral wall movement is a favorable indicator for successful surgical repair. The findings on clinical examination dictate the method of repair.

Another finding that increases the possibility of VPI is a gap between the palatal muscles. In an unrepaired palate with VPI, there may be an obvious V-shaped deformity beginning anteriorly and fanning out posteriorly. In this area there is little or no muscular function. The laterally placed muscles, which run anterior to posterior, cause palatal shortening when contracted rather than normal elevation of the posterior third of the soft palate. Another possibility is that the muscles have not been correctly positioned at primary repair.

TABLE 1 Assessment of Palatal Insufficiency (Visualized by Speculum)

Palatal and lateral wall movement
Quality, speed, and point of velopharyngeal closure
Presence, size, and position of fistula
Length of soft palate
Obturator position and effectiveness

Finally, if the palate moves well, as evidenced by upward lifting of the posterior third of the soft palate, but escape is nevertheless present, the lateral pharyngeal walls are probably not contributing sufficiently to closure of the velopharyngeal sphincter.

The next step for the surgeon is to discuss the patient with the speech pathologist. The speech pathologist makes a recording of the patient's speech and often performs more invasive investigations such as nasoendoscopy and cineradiography. For complete evaluation, both procedures must be carried out, preferably by both the therapist and surgeon. If not possible, films of the investigations can be used for joint discussion and for later follow-up purposes.

Nasoendoscopy performed under topical anesthesia is an important three-dimensional investigation that allows assessment of not only completeness of closure but also the level at which closure is taking place. The presence or absence of fistulas is noted; in addition, because the anatomy of the area is completely displaced, the pattern and speed of velopharyngeal closure can be ascertained.

When this investigation has been completed, the following will be determined: position of the palate and the palatal musculature, type of palatal movement, size of the velopharyngeal defect at rest and during function, and in general, the cause of the

TABLE 2 Assessment of Palatal Insufficiency (Visualized by Speculum)

Little or no movement, palate short	Poor prognosis
Satisfactory lateral wall movement	Satisfactory result with correct rehabilitation technique
V-shaped palatal movement, length satisfactory	Repositioning of palatal muscles, buccal myomucosal flaps
Short but satisfactory movement	Sphincter pharyngoplasty
Poor palatal movement but satisfactory lateral wall movement	Superiorly based pharyngeal flap

TABLE 3 Assessment by Nasoendoscopy

Lateral wall movement
Soft palate movement and defects of the soft palate
Length, V-shaped deformity, lateral muscle movement, closure of sphincter, bubbling

VPI. The potential rehabilitation procedure may also be determined at this time. However, it is necessary to carry out additional investigations for confirmation of the findings.

Videofluoroscopy allows accurate representation of palatal movement and demonstrates where movement is deficient.

With nasoendoscopy, videofluoroscopy, and clinical investigation, the surgeon and speech pathologist can establish the cause of the VPI. In the authors' experience, the vast majority of cases of VPI are due to a lack of understanding of the anatomy of the cleft palate by the surgeon; such lack of understanding results in inadequate primary muscle repair and the need for secondary reconstruction of the palatal musculature.

After the diagnosis is established, an appropriate regimen can be formulated. If the cause of the VPI is not known at this time, surgery should not be performed, and the patient should be referred to a more experienced center.

Treatment

Four methods of surgical correction are available, and on occasion two may be combined. The techniques are fistula closure, palatal re-repair with muscle reconstruction, a superiorly based pharyngeal flap, and sphincter pharyngoplasty. Of note, an inferiorly based pharyngeal flap is no longer used in the treatment of VPI.

Fistula Closure

Indications for surgical repair include the presence of a large palatal fistula producing a speech defect not correctable with a simple obturator. The repair is performed by using local turn-in flaps and palatal flaps of the Veau design. Flap closure should be performed without tension. Any anterior defect is closed with a medially based buccal flap consisting of buccal mucosa and, if possible, periosteum from the anterior aspect of the alveolus.

TABLE 4 Assessment by Videofluoroscopy

Speed of palatal movement
Closure of sphincter
Pattern of closure

Large fistulas are addressed by elevating bilateral flaps based on the periphery of the fistula. These flaps are turned in and sutured together. Oral closure is achieved with a pedicled, anteriorly based tongue flap. The flap is divided and inset after 3 weeks. This technique is usually successful and provides tension-free closure.

Extremely large fistulas can be closed without tension by using more of the hard palate. There should be no concern about this maneuver because total nasal closure is the key to successful rehabilitation of VPI.

If significant scarring is present because of breakdown of past repairs, one should not be reluctant to delay the palatal flaps before closure with the tongue flaps (see later).

Palatal Re-repair

Palatal re-repair may be necessary with a totally closed, repaired palate or in the presence of a fistula. It may also be necessary after an anterior fistula has been closed with a tongue flap if attention has not been paid to position of the palatal muscles. If diagnosed, the palate needs to be re-repaired to restore normal muscular anatomy. In the past it was thought that if an associated anterior fistula was present, it might be difficult to close; however, any fistula can be repaired, and reconstructive surgery, in terms of muscular function, can be carried out at the same time. Fistula closure alone in this situation does little to improve speech.

TECHNIQUE. A mouth gag is inserted and the palate is incised to form Veau flaps with a lateral incision and incisions around the fistula, if present. The soft palate is completely divided in the midline in an anteroposterior direction. The palatal flaps are elevated and dissected off the hard palate back to the palatal shelves. At this point the palatal musculature is visualized. In the majority of cases, the musculature was not repaired at the initial procedure, and the muscles are still inserted onto the posterior end of the palatal shelves. They run anteroposteriorly and become narrower until they insert into the medial edge of the hard palate at the end of the cleft via a tendinous insertion.

It is easy to dissect the muscles off the nasal layer after they have been liberated from their insertion on the hard palate. The dissection is continued toward the skull base, almost to the origin of the muscle mass. This process is repeated on the opposite side. The muscles are pennate shaped, and such an appearance is an indication of the completeness of dissection.

When the muscles are completely freed, they should lie in their correct anatomic position—at right angles to the long axis of the cleft in the posterior quarter of the soft palate. The nerve supply of

the musculature can be visualized, dissected, and preserved. The nasal layer is dissected and closed. Lateral dissection in the correct plane prevents midline tightness. After closure of the nasal layer has been achieved, the muscles are approximated in the midline and also onto the nasal layer. This maneuver overcomes any tendency for the muscles to drift back into their previous position.

The nasal layer is transected behind the palatal shelves such that an anterior cuff of nasal layer approximately 0.5 cm wide remains. This technique allows the posterior nasal layer of the palate and the levator muscles to move backward almost onto the posterior pharyngeal wall. Having created this gap, it is necessary to fill it, and the buccal myomucosal flap is excellent for this purpose because of its size, ease of use, and blood supply. The flap is based posteriorly at the level of the upper alveolar tuberosity and outlined on the cheek mucosa. It is approximately 1.0 to 1.5 cm wide and 5 to 6 cm in length. An incision is made around the flap, through the mucosa and the underlying musculature. The flap is dissected back to a level just posterior to the tuberosity. The donor site of the flap is closed primarily. From the posterior aspect of the donor site, a tunnel is made behind the maxillary tuberosity and behind the palatine vessels. The flap is advanced through the tunnel with its mucosal aspect facing into the nasal cavity. The buccal myomucosal flap is sutured into the gap in the nasal layer. The oral layer is closed completely. Each suture in the oral layer should catch the nasal layer as far as the base of the uvula to prevent dead space. Particular attention is paid to suturing the oral layer onto the reconstructed muscular sling. It is usually possible to close all of the lateral defects in the palate, thus leaving it intact with no raw areas.

The described technique always ensures satisfactory muscle dissection, reorientation, and repair without tension. Because of the buccal myomucosal flap, any possibility of forward migration of the muscles is obviated, and therefore shortening of the nasal layer is prevented.

This procedure is the technique of choice for patients with a submucous cleft palate.

Sphincter Pharyngoplasty for a Short Palate with Satisfactory Function and Satisfactory Lateral Pharyngeal Wall Movement

If the primary repair has achieved satisfactory palatal movement in the optimal position but is short and is causing velopharyngeal incompetence, the palate should not be repaired. The goal, however, is to provide a more effective lateral and posterior sphincter to achieve closure.

A short, superiorly based pharyngeal flap is raised; its width encompasses virtually the entire posterior pharyngeal wall, with its base situated above the level of the soft palate. The donor defect is closed primarily. Bilateral, superiorly based flaps are elevated from the posterior pillars of the fauces. They contain the palatopharyngeus muscles with their nerve supply. The donor sites of the flaps are closed. The flaps are sutured onto the superiorly based pharyngeal flap, thus lining its raw aspect. This maneuver ensures that the muscles contained within the flaps form a sphincter in the correct vertical position on the posterior pharyngeal wall with a minimum of fibrosis and scarring. This is indeed a true sphincter because the nerve supply in the flaps should remain functional and the contained muscles should contract as required. The flap donor defects are closed directly (Fig. 2).

Sphincter pharyngoplasty is probably the most frequently performed procedure for correction of velopharyngeal incompetence. With the modern approach to primary palate repair, most patients with velopharyngeal incompetence have satisfactory soft palate movement (i.e., the principal indication for sphincter pharyngoplasty).

Midline Pharyngeal Flap for Poor Palatal Function with Satisfactory Lateral Wall Movement

If examination of a patient with VPI reveals a short palate that cannot be lengthened sufficiently by re-repair of the palate or a poorly functioning palate in the presence of satisfactory lateral wall movement, the procedure of choice is a superiorly based midline pharyngeal flap and shrinkage.

Inferiorly based flaps have been abandoned. The superiorly based flap is positioned in the correct orientation so that it does not drag the palate downward, as can happen with an inferiorly based flap. In addition, all pharyngeal flaps must be lined to prevent fibrosis and shrinkage.

An anteroposterior, full-thickness incision is made in the soft palate. On either side of the midline the mucosa is dissected with a small right-angled scalpel. Flaps based on the posterior margin of the palate are incised on the superior aspect of the palate. The flaps are carefully dissected off the muscle layer; they are made sufficiently wide to provide oral coverage for the superiorly based pharyngeal flap. The flaps are sutured together in the midline.

The pharyngeal flap is formed by incising laterally on the posterior pharyngeal wall. The flap is constructed as wide as possible so that most of the posterior pharyngeal wall is elevated. It should be sufficiently long to cover the raw area on the nasal aspect of the soft palate that remains after flap harvest. Elevation of the flap is continued superiorly to a level approximately 1 to 1.5 cm above the level of the soft palate.

Attention is turned to the palate. The elevated posterior pharyngeal wall flap is sutured to the raw area on the nasal aspect of the palate. The down-turned nasal mucosal flaps of the palate, which have

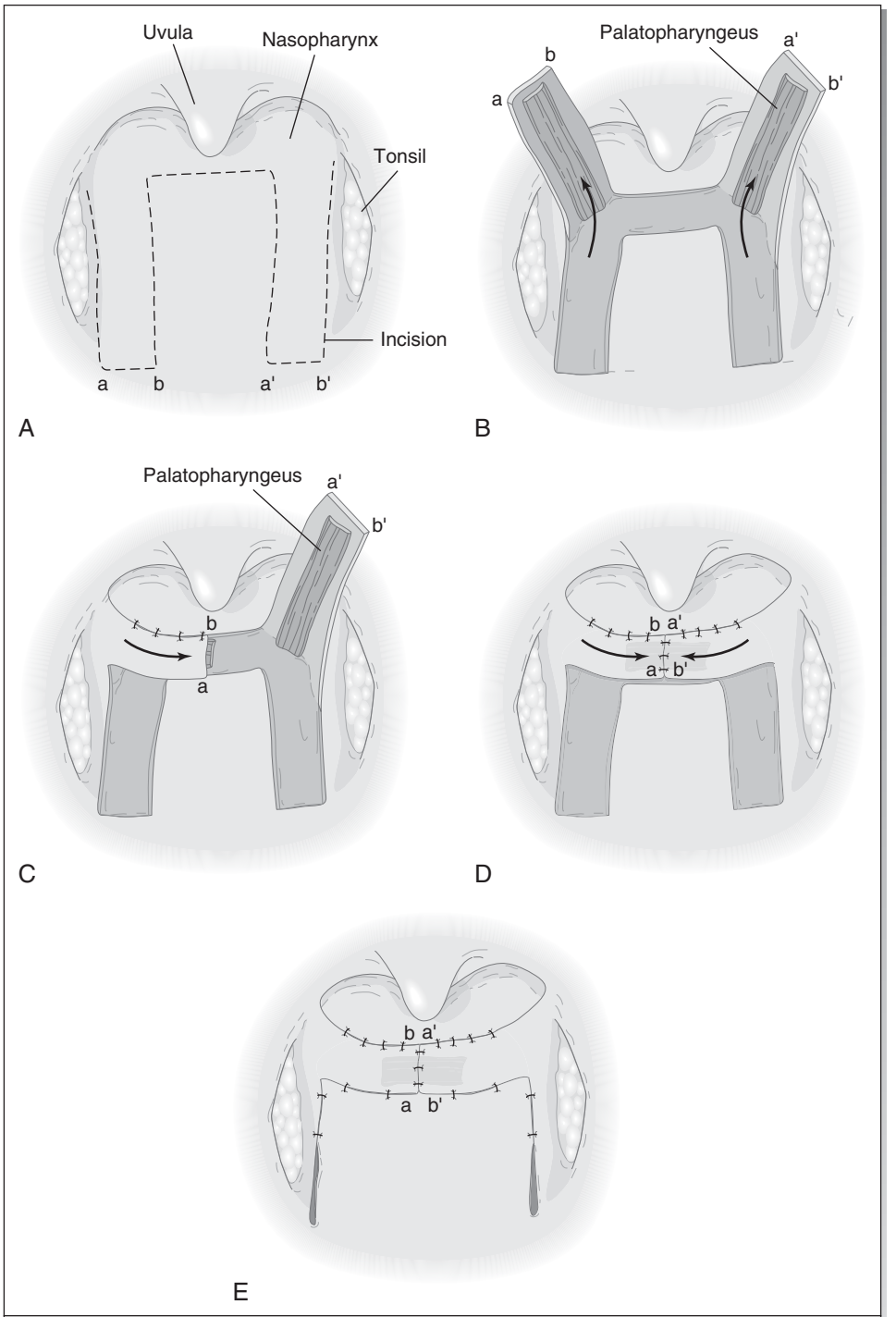

Figure 2 • The modified sphincter pharyngoplasty. **A,** The posterior area tonsillar pillar flaps are outlined, as is the position of the transverse incision in the posterior pharyngeal wall. **B,** The posterior tonsillar pillar flaps are elevated with the palatopharyngeal muscles. **C,** The pillar flap is transferred at 90 degrees, the posterior edge of the flap being sutured to the superior edge of the transverse defect in the posterior pharyngeal wall. **D,** The flaps come to lie in such a position that the distal ends of the flaps abut in the midline. If suturing is continued, a to b′, the anterior edges of the pillar flaps are gradually sutured together; the velopharyngeal connection is decreased in size. **E,** The palatopharyngeal muscles are sutured together. The anterior edges of the pillar flaps are sutured to the inferior edge of the pharyngeal defect of the posterior wall. The lateral defects are closed.

been sutured together in the midline, are sutured to the edge of the pedicle of the pharyngeal flap to eliminate raw areas. The defect in the posterior pharyngeal wall is closed primarily.

Complications

Bleeding during VPI repair needs to be minimal. In all surgical procedures, vessels on the posterior pharyngeal wall need to be visualized to prevent injury. If there is any indication of injury to vessels and it is not possible to control the bleeding, the area must be packed securely and an airway established by tracheostomy. In these situations, the packing remains in place for 10 days and is removed in the operating room.

Sleep apnea has been reported as a complication after sphincter pharyngoplasty. It is best to avoid this procedure in young children (i.e., those younger than 5 years) and children with the Pierre Robin sequence (small, retruded mandible).

Pearls and Pitfalls

- With satisfactory functional repair of the primary cleft of the palate, there is much less need for speech rehabilitation.
- The key points in primary repair of the palate are accurate and stable positioning of the palatal muscles to achieve a functioning palate of adequate length.
- One of the most important goals in primary palate repair is to avoid residual raw areas.
- Attention to these technical points reduces the need for surgical speech rehabilitation to less than 2%.
- Very large defects may not be able to be corrected surgically, as occurs in a badly treated cleft patient or a postcarcinoma patient. The only alternative in such a situation is to use a prosthesis.
- Free tissue transfer should be considered only for the treatment of a hard palate defect after tumor resection or trauma; the flap will, however, be merely a nonfunctional obturator.

SUGGESTED READINGS

Henningson G, Isberg A: Oronasal fistulas and speech production. In Bardach J, Morris HL (eds): Multidisciplinary Management of Cleft Lip and Palate. Philadelphia, WB Saunders, 1990.

Jackson IT: Closure of secondary palatal fistulae with intra-oral tissue and bone grafting. Br J Plast Surg 25:93-105, 1972.

Orticochea M: A review of 236 cleft palate patients treated with dynamic muscle sphincter. Plast Reconstr Surg 71:180, 1983.

Shprintzen RJ, Lewin ML Croft CB, et al: A comprehensive study of pharyngeal flap surgery: Tailor made flaps. Cleft Palate Craniofac J 16:46, 1979.

Trost-Cardamone JE, Bernthil JE: Articulation assessment procedures and treatment decisions. In Moller KT, Starr CD (eds): Cleft palate: Interdisciplinary issues and treatment. Austin, TX, Pro-Ed, 1993.

The Craniosynostoses

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The craniosynostoses represent a wide clinical spectrum ranging from isolated fusion of the metopic suture causing mild trigonocephaly to complex conditions such as Pfeiffer's syndrome with a *kleeblattschädel* cranial deformity, functional midface hypoplasia, and other congenital anomalies. Despite this variety, the requirements for effective clinical management of all craniosynostoses are the same: accurate diagnosis, appropriate timing of surgery, and the use of a reliable surgical technique that maximizes the chance for long-lasting improvement in function and cranial shape and volume.

Etiopathogenesis

Patent cranial sutures are required to maintain normal cranial morphology during the period of rapid brain expansion in the first few years of life. Except for the metopic suture, the cranial sutures do not normally fuse until adulthood. When a cranial suture fuses prematurely, calvarial expansion is locally restricted, and such restriction causes abnormalities in cranial shape and volume.

The exact etiopathogenesis of premature fusion of cranial sutures is an area of active research. A number of genetic mutations have been associated with syndromic craniosynostosis. Fibroblast growth factor receptor (FGFR) 1 and 2 mutations are associated with Crouzon's, Apert's, and Jackson-Weiss syndromes; TWIST mutations with Saethre-Chotzen syndrome; MSX2 mutations with Boston-type craniosynostosis; and FGFR3 mutation with Muenke's craniosynostosis. The majority of patients with craniosynostosis, however, are nonsyndromic. The exact molecular mechanism responsible for in utero or postnatal pathologic fusion of cranial sutures remains unknown. Recent research has identified a number of molecular mediators that may be involved in the signaling pathway responsible for premature fusion, such as members of the transforming growth factor-β (TGF-β) superfamily, fibroblast growth factors, and osteogenic antagonists such as noggin. These factors, however, are probably just "snapshots" of isolated players involved in a complex interconnected communication network encompassing a myriad of molecular messengers.

Although our understanding of the molecular signaling pathways associated with craniosynostosis is increasing, the initial in utero event that activates these pathways remains to be identified. Intrauterine head constraint has been implicated in initiating craniosynostosis and has recently been shown in an animal craniosynostosis model to result in activation of TGF-β isoforms in a pattern similar to normal suture fusion. A number of conditions and teratogens can result in premature fusion of sutures (*secondary craniosynostosis*): hyperthyroidism, rickets, mucopolysaccharidoses and related disorders, hematologic disorders such as thalassemia and sickle cell anemia, and teratogens such as retinoic and valproic acid.

Pathologic Anatomy

Patent cranial sutures allow growth of the skull in a direction perpendicular to the suture. When growth is restricted by early fusion, *Virchow's law* (1851) states that compensatory growth occurs parallel to the fused suture. For example, premature sagittal suture fusion results in constricted biparietal width with compensatory anteroposterior elongation, or *scaphocephaly*; metopic fusion results in constricted anterior transverse growth with a compensatory forehead keel, or *trigonocephaly*; and unicoronal synostosis causes constricted anteroposterior growth of the ipsilateral forehead with compensatory contralateral frontal bossing, or

unilateral frontal plagiocephaly. Even so, Virchow's law fails to explain all of the regions of growth restriction and compensation observed clinically.

The pathologic anatomy is not limited to the calvaria, but also affects the cranial base to varying degrees. In 1959, Moss proposed that the primary pathology is in the cranial base and that the calvarial findings are secondary. If the cranial base pathology becomes normalized after cranial vault remodeling, however, this would suggest that the calvaria and not the endocranium is the location of the initiating event.

Diagnostic Studies

Clinical examination is the cornerstone for diagnosis of the different forms of craniosynostosis. As described earlier, fusion of each suture results in a characteristic change in calvarial morphology. The examination should include observation of the shape of the head, the symmetry and position of the facial bones, the relative ear position, and the alignment of the posterior skull base.

Fused cranial sutures may have a palpable bony ridge, whereas patent sutures can often be demonstrated by ballottement in a young infant. In a calm infant with patent sutures, the anterior fontanelle should be soft and open. A record of previous head circumference measurements plotted on a standardized curve can give an indication of the rate of cranial growth in relation to the child's weight and length. Children with syndromic or familial synostosis should be offered genetic testing.

Children suspected of having craniosynostosis should undergo craniofacial computed tomography (CT) to examine the sutures and rule out an associated intracranial anomaly. A plain radiograph is reassuring if all sutures can be clearly visualized, but it is nonspecific if a particular suture is difficult to visualize. Three-dimensional CT is useful to appreciate the overall shape of the cranium, but it can give false-positive results of suture fusion because of computer averaging; axial cuts should also be examined.

The differential diagnosis of unilateral cranial flattening or plagiocephaly includes unilateral coronal synostosis, unilateral lambdoid synostosis, and nonsynostotic or deformational plagiocephaly. The incidence of deformational plagiocephaly has increased dramatically since institution of the "Back to Sleep" campaign in the early 1990s. This condition can be treated nonoperatively with positional changes or helmet molding in all but a few rare cases. Clinical examination of the shape of the cranium and forehead, the facial midline, the relative ear position, the posterior skull base, and the occiput should result in an accurate diagnosis of unilateral plagiocephaly (Fig. 1).

The metopic suture is the first cranial suture to fuse in a normal infant and does so between 3 and 9 months of age. A controversial subgroup consists of children with the mild form of true metopic synostosis. These children have clinical and radiographic evidence of premature metopic fusion, mild hypotelorism, and variable temporal hollowing and lateral brow recession. Unlike the more severe forms of trigonocephaly, this mild degree of deformity does not always justify an intracranial procedure. Some studies suggest that children with isolated metopic synostosis are at risk for mental retardation or learning disorders, but it is not clear from the data that early surgery in these particular children decreases this risk.

Dolichocephaly of prematurity can be clinically confused with sagittal synostosis; however, on CT examination the former has a patent sagittal suture. As head control improves in the premature infant, the dolichocephaly typically improves but may require molding therapy in some cases. The early surgical procedures that are usually reserved for sagittal synostosis should not be performed on these infants.

Reconstructive Goals

The goal of cranial reconstruction is to create a cranium with a volume and shape that will be appropriate after the anticipated future growth of the child. To accomplish this goal, the areas of restricted growth are expanded and the regions of compensatory bossing are contoured. Except for sagittal synostosis, correction needs to be exaggerated. The retruded brow of coronal synostosis must be overadvanced; the constricted brow of metopic synostosis must be overexpanded. Within months of the operation, soft tissue forces and bone graft remodeling "soften" the reconstruction to achieve a desirable brow contour.

Syndromic craniosynostoses are recognized as having the greatest rate of secondary procedures. This rate can be minimized by operating after 1 year of age when possible (see the section "Timing"), and by consciously overadvancing the bifrontal projection and dropping the turricephaly. The specific reconstructive goals vary depending on the type of craniosynostosis (Table 1).

Treatment

Timing

The optimal timing of craniosynostosis repair depends on a multitude of factors, including the presence of elevated intracranial pressure (ICP), the operative technique used, and surgeon preference.

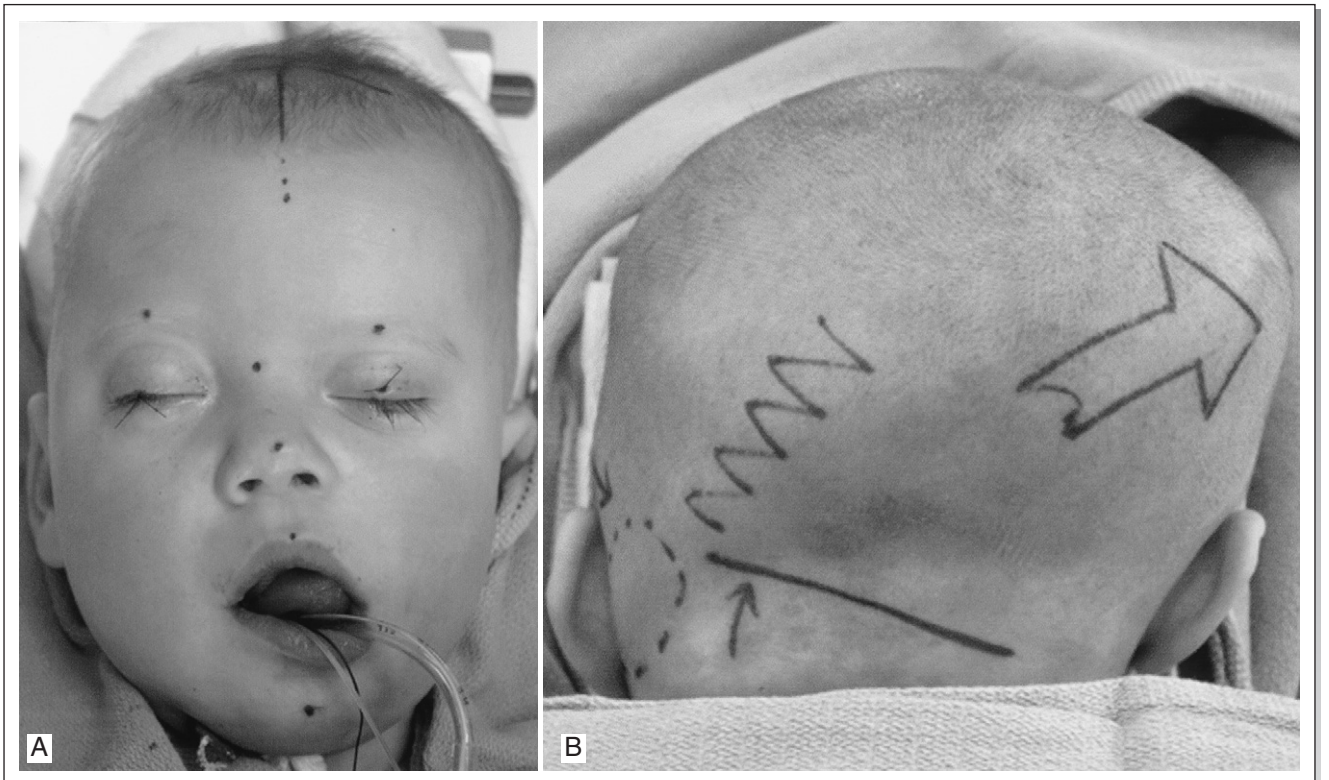

Figure 1 • Clinical findings of unicoronal and unilambdoid synostosis. **A,** The cranial midline, supraorbital rims, radix, nasal tip, Cupid's bow, and chin have been marked on a patient with right unicoronal synostosis. The frontal findings of unicoronal synostosis include ipsilateral frontal flattening, supraorbital elevation and deviation of the nasal root to the affected side, contralateral frontal bossing, and chin deviation. **B,** The posterior findings of left unilateral lambdoid synostosis (fused suture marked with a *zigzag*) include ipsilateral mastoid bossing (*dotted circle*), occipital flattening and upward elevation of the skull base (*small arrow*), and contralateral compensatory parietal bossing (*large arrow*).

Operations performed at a younger age have the potential advantages of prevention of early ICP-related damage, better reossification of cranial defects, and less extensive surgery. In contrast, later surgery is reported to have better long-term results, more substantial bone to remodel, and lower rates of reoperation. The risk for intracranial complications from prolonged elevations in ICP needs to be determined on a case-by-case basis and factored into the timing decision.

For sagittal synostosis, strip craniectomies, including extended strip craniectomies, depend on brain growth to remodel the shape of the skull and therefore need to be performed early (4 to 12 weeks of age) because they are markedly less effective after 6 months of age. More extensive remodeling procedures such as the modified "pi" procedure can be performed at a later age with a predictable result. Total cranial vault remodeling with craniectomy and bone graft repositioning is used to treat sagittal synostosis after 1 year of age.

Performance of cranial vault remodeling from 6 to 10 months of age is preferred to take advantage of a more substantial and stable frontal bandeau construct and to permit reossification of cranial defects.

The results of cranial vault remodeling for syndromic craniosynostosis (e.g., Apert, Crouzon, Pfeiffer) are uniformly worse than those for nonsyndromic cases. Reoperation rates reported in the literature for these patients range from 11% to 27%. At the Consensus Conference on Craniosynostoses in 1995, it was recommended that frontoorbital advancement be performed "as late as possible" for syndromic craniosynostosis to minimize the risk of reoperation. It was recognized that the severity of associated problems, such as increased ICP or exorbitism, might necessitate early surgery. Children with signs of elevated ICP and progressive exorbitism receive an early operation; those who remain stable undergo reconstructive surgery between 12 and 15 months of age.

Surgery

Because of the potential risks of severe morbidity or mortality associated with the surgical correction of craniosynostosis, all precautions must be taken before commencing the procedure. A successful operation requires cooperation between an experienced pediatric anesthesiologist, a craniofacial surgeon,

TABLE 1 Reconstructive Goals of Correction of Craniosynostosis

Unicoronal Synostosis
Advance the affected hemibrow and rotate it anteriorly to increase anterior cranial volume
Rotate the contralateral hemibrow posteriorly to correct compensatory bossing
Flatten the ipsilateral compensatory temporal bossing by flattening the temporal extension of the bandeau
Achieve a normal brow-forehead relationship
Bicoronal Synostosis
Advance the brow and rotate it anteriorly to increase anterior cranial volume
Flatten compensatory temporal bossing as part of the bandeau
Correct compensatory turricephaly
Achieve a normal brow-forehead relationship
Metopic Synostosis
Advance the lateral aspects of the brows to increase anterior cranial volume
Straighten the midline brow angulation
Increase the transverse dimension of the brow and superior orbital rims with bone grafts and contouring
Create a defined lateral brow angulation
Correct the compensatory bitemporal bossing
Achieve a normal brow-forehead relationship
Sagittal Synostosis
Correct occipital and/or frontal bossing
Increase the bitemporal and biparietal distance
Decrease the sagittal length
Unilateral Lambdoid Synostosis
Expand the affected half of the posterior vault
Correct the compensatory ipsilateral occipital bossing
Correct contralateral parietal bossing
Reconstruct the ipsilateral parietal curvature
Create a stable occiput
Bilateral Lambdoid Synostosis
Expand the posterior cranial vault
Create a stable occiput
Correct compensatory turricephaly
Multiple Suture or Syndromic Synostosis
Stage the surgery when necessary
Perform surgery later if possible
Attempt to overcorrect turricephaly and overadvance the bandeau

and a pediatric neurosurgeon to optimize airway and vascular control during the procedure.

ANTERIOR CRANIAL VAULT REMODELING AND FRONTOORBITAL ADVANCEMENT. Anterior cranial vault remodeling with frontoorbital advancement involves removal and remodeling of the abnormal forehead unit as part of a frontal craniotomy and removal and remodeling of the superior lateral orbital rims through mobilization of a supraorbital bandeau with bilateral temporal extensions. Residual parietal bossing posterior to the craniotomy can be corrected with barrel stave osteotomies.

An asymmetric supraorbital bandeau is preferred for the treatment of *unicoronal synostosis* (Fig. 2A). On the side of the fused suture, wide temporal extension with a C-shaped lateral rim osteotomy is performed, and the temporal extension is advanced and rotated forward. On the contralateral (patent suture) side, only a small posterior rotation of the lateral brow region is needed to address the compensatory bossing (see Fig. 2B). To perform this repair, the temporal extension is truncated to approximately 1 cm in length, and the lateral orbital rim is cut at the level of the zygomaticofrontal suture. For *metopic* and *bicoronal synostosis*, the bandeau is raised symmetrically with bilateral temporal extensions and C-shaped lateral rim osteotomies to achieve symmetric advancement (Fig. 3).

On a side table, the supraorbital bandeau is reshaped. Bossing of the temporal flange is straightened with a rib bender. To increase intraorbital width for the treatment of metopic synostosis, a number of techniques can be used. The supraorbital bandeau can be cut in half through the midline and a 1- to 1.5-cm bone graft interposed (see Fig. 3). Another option is to create a sliding step osteotomy to achieve the widening without a bone graft.

The desired position of the supraorbital bar depends on the type of craniosynostosis. For *metopic* synostosis, the cranial expansion mostly comes from advancement and widening of the lateral brow region, with only a small advance at the midline (see Fig. 3). For *unicoronal* synostosis, the affected brow needs to be advanced and rotated anteriorly, whereas the contralateral brow is rotated posteriorly (see Fig. 2). For *bicoronal* synostosis, a large anterior advancement with anterior rotation is required. The altered and advanced bandeau is secured in place with resorbable plates and screws. The tip of the C-shaped lateral rim osteotomy is not returned orthotopically but, instead, is secured to the lateral aspect of the inferior orbital rim to accommodate advancement or expansion of the supraorbital bar. Two wires are used at the nasofrontal junction, except for unicoronal synostosis, for which only the unaffected nasofrontal side is attached because of the twist or torque at the midline of the bar. With large bilateral advancements, such as for bicoronal synostosis, the nasofrontal segment of the bandeau cannot be secured at the nasofrontal junction without the use of interposed bone graft. The temporal extensions of the supraorbital bar are overlapped with the opposing cranial struts and secured with resorbable lag screws or plates.

After the supraorbital bandeau is in the desired position, the bone remaining from the craniotomy is

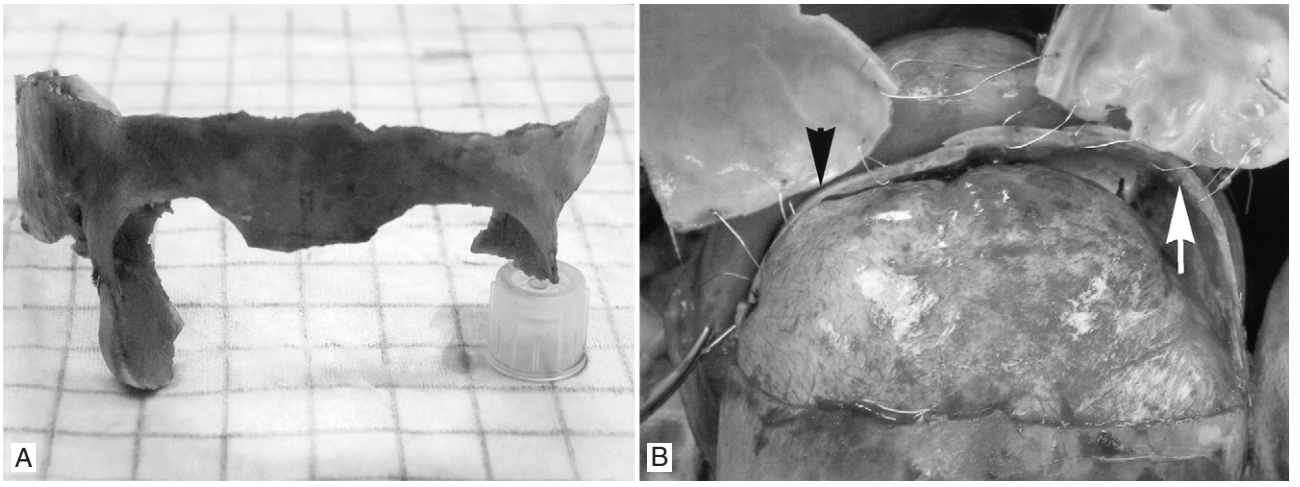

Figure 2 • Supraorbital bandeau alteration for unicoronal synostosis. **A,** Frontal view of a removed bandeau. On the synostosed (patient's right) side, the lateral orbital rim is incorporated into the bandeau with a C-shaped osteotomy. On the contralateral side, the rim is truncated at the zygomaticofrontal suture **B,** Vertex view of an altered supraorbital bandeau before wiring of the frontal bone plates. The synostosed (right) lateral brow has been advanced and rotated anteriorly (*arrowhead*) whereas the contralateral brow (left) has been rotated slightly posteriorly (*arrow*) to achieve the desired overcorrection. The frontal bone flaps have been secured with sutures.

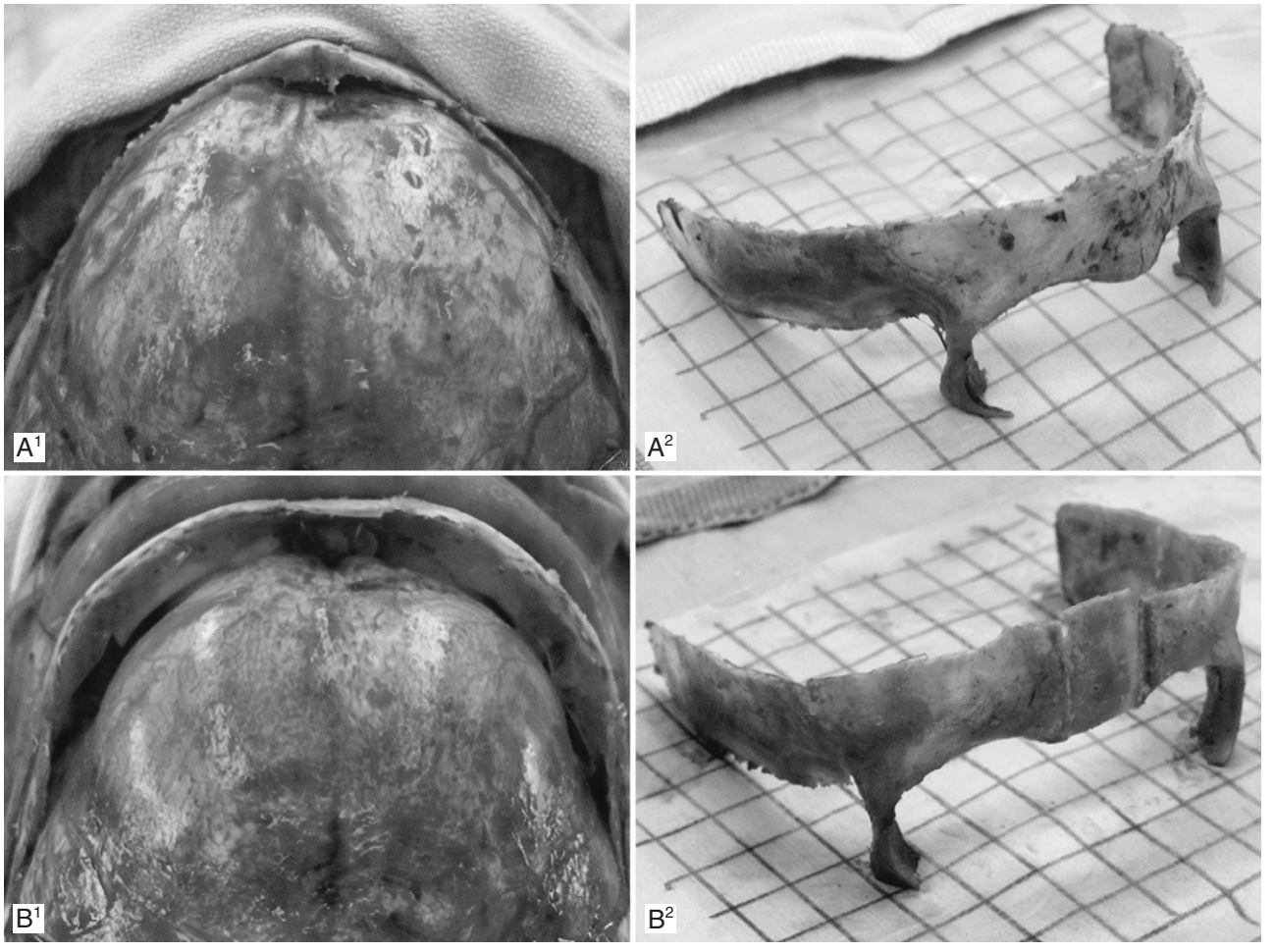

Figure 3 • Supraorbital bandeau alteration for metopic synostosis. **A,** The supraorbital deformity in metopic synostosis includes bilateral temporal compensatory bossing, constriction of the transverse width of the lateral aspect of the brow, and midline angulation. **B,** The bandeau is altered to flatten the temporal bossing and create lateral brow angulation. A 10- to 15-mm-wide bone graft is interposed at the midline osteotomy and held in place with a resorbable plate on the intracranial side. With replacement of the bandeau, there is a small advance at the midline, but the majority of the expansion is at the lateral aspect of the brow.

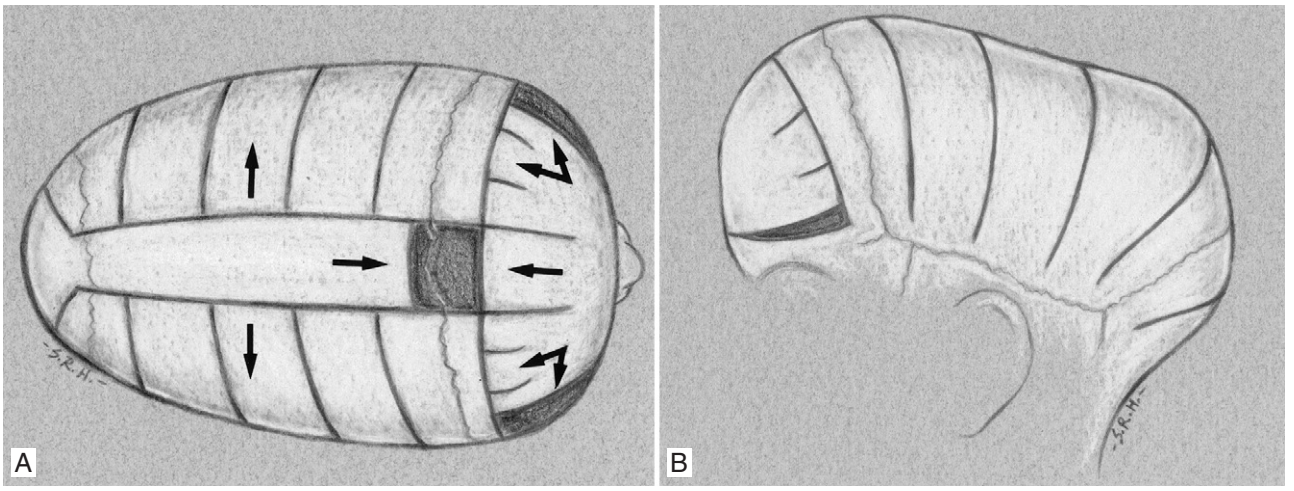

Figure 4 • Vertex **(A)** and lateral **(B)** views of modified pi osteotomies for the correction of sagittal synostosis. The pi pattern is created by osteotomies on either side of the coronal and sagittal sutures. Barrel stave osteotomies down to the cranial base are used to expand the biparietal dimension. Closing wedge osteotomies at the tip of the barrel staves will force an expansion when they are approximated with sutures or wires. The anterior-posterior dimension is decreased by removing a square of bone from the anterior fused sagittal suture and closing the gap under tension with wires. (Courtesy of Dr. Joseph Gruss.)

used to reconstruct the forehead. The segments are rotated and interchanged until the desired brow-forehead relationship is achieved. The frontal bone is secured to the bandeau with resorbable sutures at the points of bone contact.

POSTERIOR CRANIAL VAULT REMODELING. Posterior cranial vault remodeling for *bilateral lambdoid synostosis* or late posterior sagittal synostosis involves removal of the affected cranial bone followed by creation of a stable, symmetric construct with an appropriate shape. With removal of the affected bone, the surgeon must create a stable construct on which the patient can rest postoperatively.

For *unilateral lambdoid synostosis*, the craniotomy should include the majority of the occipital deformity. Any residual contralateral parietal bossing that is not included in the craniotomy can be corrected with barrel stave closing wedge osteotomies. The posterior craniotomy bone is cut in half and the two halves exchanged and contoured until the desired shape has been achieved. The goal is to create the appropriate ipsilateral parietal curvature with calvaria from the contralateral parietal bossing.

CORRECTION OF SAGITTAL SYNOSTOSIS. A variety of techniques ranging from simple strip craniectomies, to calvarial remodeling, to complete calvariectomy can be used to treat sagittal synostosis. Of these techniques, more reliable normalization of cranial proportions can be achieved by cranial vault remodeling than by limited or extended strip craniectomy.

A technique of endoscopic craniectomy for early surgical correction of sagittal synostosis, as well as other types of isolated synostosis, has recently been reported. The advantage of endoscopic craniectomy over conventional open strip craniectomy would be a decrease in the size of incisions, blood loss, operative time, cost, and recovery time. To address previous concerns over the long-term results after simple strip craniectomy, the endoscopic technique is combined with postoperative molding helmet therapy instituted within 2 weeks postoperatively and continued to the age of 8 months.

The authors prefer to treat infants with sagittal synostosis by extended strip craniectomy and biparietal barrel staving at 4 to 12 weeks of age. The cranial vault is effectively remodeled with a variety of other procedures when performed at a very young age. For infants older than 4 months, the authors prefer a modified "pi"-type strip craniectomy with barrel stave osteotomies (Fig. 4). If a child is referred with sagittal synostosis after 1 year of age, complete cranial vault remodeling is considered.

Infants with sagittal synostosis can have one of the following cranial pathologies: (1) predominantly posterior calvarial involvement with an occipital protuberance and biparietal narrowing; (2) scaphocephaly, frontal bossing, and narrowing of the anterior cranial fossa without significant occipital narrowing; or (3) a more severe combination of anterior and posterior deformities. Children with either primarily occipital or frontal narrowing can be treated with one operation in the prone or supine position, respectively. To perform the modified "pi" procedure, osteotomies are performed on either side of the sagittal suture and coronal sutures to create a pattern similar to the Greek letter π (see Fig. 4A). Vertical barrel stave osteotomies are performed on the parietal bones and lambdoid region and

extended as far inferior as possible. A portion of the anterior fused sagittal suture is resected such that when the midline gap is reapproximated with wires, the posterior calvaria is advanced to correct the occipital bossing and prevent further deformity of this region.

The "hung span" or cranial vault remodeling procedure has been reported to stabilize the barrel staves in a separated, expanded position with a long spanning resorbable plate and screws. This technique was described only for use in older children undergoing delayed or late secondary reconstruction when there was concern that the brain and dura would not support the parietal expansion to the same degree as in younger infants. For children older than 12 months, the bone becomes too stiff for modified strip calveriectomies, and the affected bone of the occiput needs to be removed and reshaped as a formal calvarial remodeling procedure.

Surgical Care of Syndromic Craniosynostosis

Patients with syndromic craniosynostosis require multidisciplinary coordinated care through a craniofacial program. Their surgical treatment involves multiple surgical procedures staged over the first 18 years of life (Fig. 5 and Table 2). When compared with isolated craniosynostosis, the craniosynostosis syndromes are associated with higher relapse and reoperation rates after cranial vault remodeling, as well as a greater risk for increased ICP and operative complications. The timing of cranial vault remodeling with frontoorbital advancement is tailored to the individual patient. Infants with severe exorbitism or increased ICP require early aggressive expansion in the first few months of life. In patients without these early functional problems, a more reliable, long-lasting result can be achieved if the surgery is delayed until 12 to 15 months of age.

A majority of the craniosynostosis syndromes are associated with midface hypoplasia, which requires treatment with advancement surgery at the LeFort III level. Timing of this procedure depends on the patient. Functional problems (globe exposure, sleep apnea, nasopharyngeal airway compromise, extreme malocclusion) or severe dysmorphism is treated by early midface advancement after 3 years of age. The earlier the surgery is performed, the higher the risk that repeat advancement will be required. Definitive orthognathic surgery is still required at skeletal maturity, after orthodontic treatment has been completed.

Traditional midface advancement involves advancing the facial skeleton as a LeFort III segment intraoperatively and stabilizing it in the advanced position with autogenous bone grafts. The degree of advancement with this technique is limited to 10 to 15 mm because of resistance from the associated soft tissues. To overcome this limitation and minimize the need for repeat operations, the principle of distraction osteogenesis has been applied to LeFort III or midface advancement. With this technique, the osteotomies are completed in standard fashion; however, after the bone segment has been fully mobilized, the incisions are closed and a postoperative course of distraction or device activation is started at a rate of 1 mm/day. Gradual advancement slowly overcomes the soft tissue resistance and permits advancements of greater than 2 cm. The distraction is followed by a period of consolidation, during which the osseofibrous tissue generated in the osteotomy ossifies and stabilizes the advanced segment.

The vector of midface distraction is planned preoperatively by using cephalogram images, but it is typically parallel to the Frankfort horizontal. The vector should be designed in a manner to optimize the position of the malar prominence, with a secondary consideration being the occlusal relationship. If the LeFort III segment is rotated inferiorly to close the occlusion of syndromic children with large anterior open bites, the vertical height of

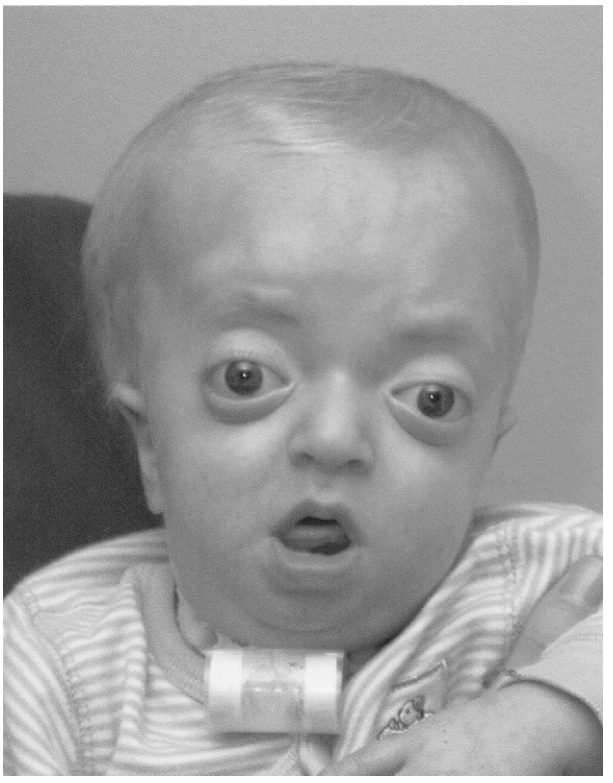

Figure 5 • An infant with a severe Crouzon phenotype. In the first few months of life he has already undergone a tracheostomy for airway protection, as well as early frontoorbital advancement to treat increased intracranial pressure, functional exorbitism, and corneal exposure. A midface distraction will be performed in early childhood.

TABLE 2 Timeline for Surgical Treatment of Syndromic Craniosynostosis

Postnatal
Airway obstruction—tracheostomy
Globe exposure—tarsorrhaphy
Feeding problem—gastric tube placement
Associated congenital anomalies—treat
Early Infancy
Increased intracranial pressure—early cranial vault expansion
Hydrocephalus—ventriculoperitoneal shunt
6 Months
Two-stage cranial expansion needed—posterior vault expansion
9-12 Months
Anterior cranial vault remodeling with frontoorbital advancement
Cleft palate repair
Staged hand surgery commences
12-15 Months
Delayed anterior cranial vault remodeling with frontoorbital advancement (especially Apert's syndrome)
2-3 Years
Large full-thickness cranial defects—autogenous bone graft
Recurrence of deformity—repeat expansion
3-4 Years
Severe obstructive sleep apnea/lack of globe protection/severe dysmorphism—early LeFort III midface advancement
5-7 Years
Symptomatic midface hypoplasia—LeFort III midface advancement
10 Years
Dental/orthodontic preparation for orthognathic surgery
Skeletal Maturity
Appropriate orthognathic surgery
External nasal deformity—rhinoplasty
Forehead contour deformity—onlay bone graft/alloplast

the orbits will be abnormally increased, and an undesirable relationship between the cheek and globe will result. Definitive occlusal surgery can be performed later at skeletal maturity with an osteotomy at the LeFort I level.

Midface distraction devices are either internal or external. The current internal devices *push* the body of the zygoma by using a screw mechanism anchored to the temporal bone. External devices *pull* the maxilla forward with a halo base secured to the cranium (Fig. 6). The disadvantage of internal devices is that the maximum soft tissue resistance during midface distraction is experienced by the maxilla whereas the distraction force is exerted on the zygomas. Between these opposing forces lies the zygomatic-maxillary suture, which is the weakest point of the LeFort III segment and therefore susceptible to fracture or dislocation. This can result in suboptimal advancement of the maxilla with overadvancement of the zygomas. The disadvantage of external devices is the bulky hardware that the child wears for 2 to 3 months; however, the distraction force is exerted on both the maxilla, through a dental splint, and the zygomas, through percutaneous posts, thereby minimizing stress on the zygomaticomaxillary suture.

Postoperative Care

Except in rare cases, infants undergoing surgery for craniosynostosis can be extubated at the end of the procedure. We routinely perform postoperative CT to rule out intracranial bleeding or other complications. Perioperative gram-positive antibiotic coverage is administered until the scalp drains are removed, 2 to 3 days after the operation. The parents should be informed preoperatively about the expected degree of postoperative swelling. After frontoorbital advancement, periorbital swelling typically obscures the patient's vision within 24 to 48 hours. The swelling usually subsides to allow sight around the third or fourth postoperative day. The total hospital stay for craniosynostosis ranges from 2 to 5 days, depending on the procedure performed and any comorbid conditions.

Pearls and Pitfalls

- *Misdiagnosis.* Deformational plagiocephaly, isolated metopic ridging, and dolichocephaly of prematurity must not be misdiagnosed and treated as craniosynostosis.
- *Undercorrection.* It is difficult to overadvance the frontal bandeau when treating unicoronal or metopic synostosis. It is even more difficult with syndromic bicoronal synostosis. At the end of the operation, the brow should be overprojected with smooth, but well-defined lateral angulation. Failure to overcorrect results in a suboptimal outcome within 12 months of surgery.
- *Instability.* The brow and posterior calvaria are particularly susceptible to postoperative deformational forces. The strength of the

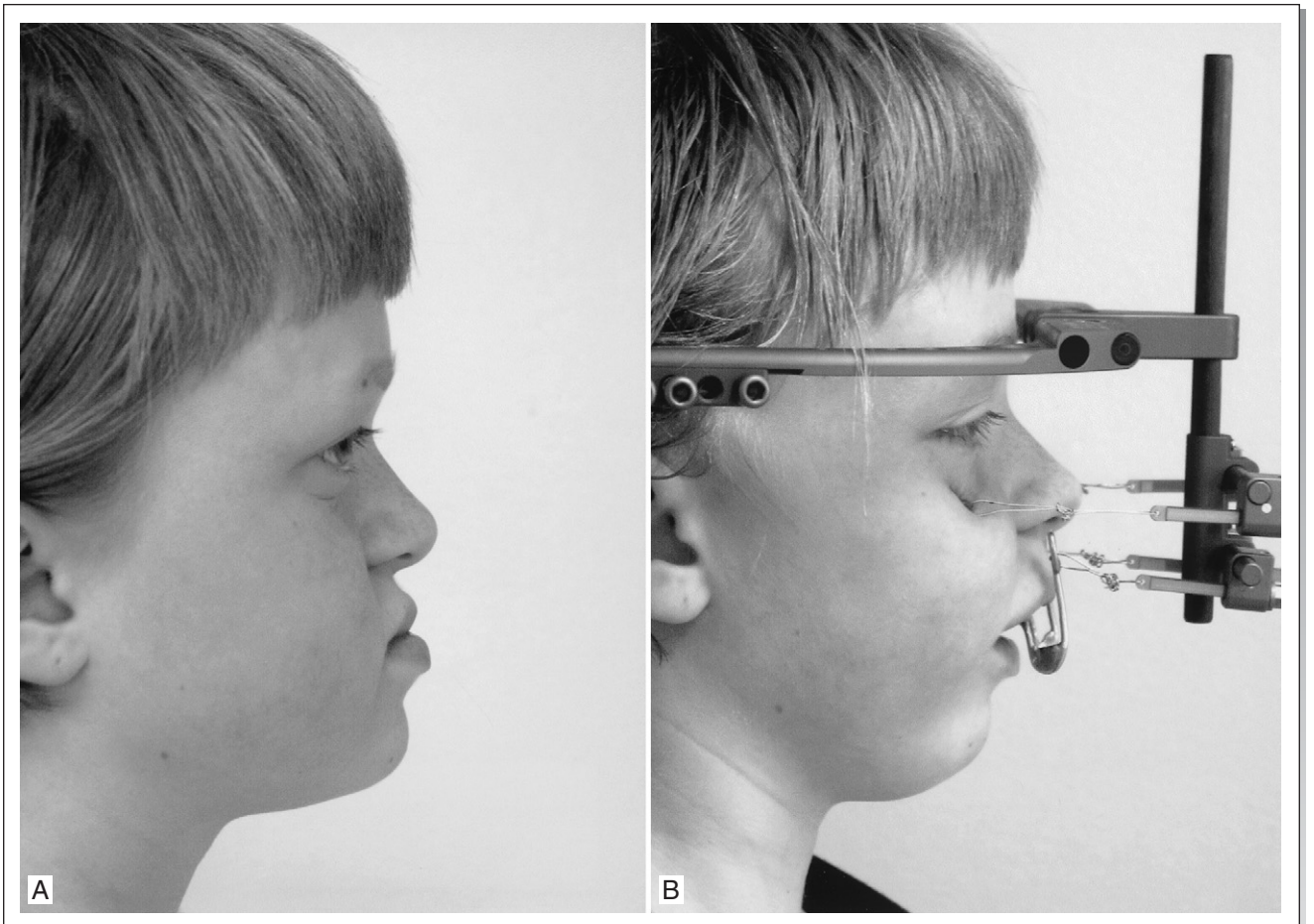

Figure 6 • LeFort III midface distraction osteogenesis performed with an external halo-based distraction device in a patient with Crouzon's syndrome. The patient had previously undergone frontoorbital advancement. **A,** Before distraction, the patient's primary complaint was malocclusion and inadequate globe protection. **B,** After 2 weeks of distraction, the LeFort III segment has been advanced 2 cm to achieve a planned overcorrection in anticipation of future mandible growth. The occlusal relationship will be optimized at skeletal maturity with orthodontic therapy and secondary midface advancement.

reconstruction should not rely solely on the hardware, but also on attention to maximizing bone-to-bone contact at the osteotomy sites.

- *Premature surgery.* Frontal bandeau advancement should be performed as late as possible for the treatment of syndromic brachycephaly.
- *High forehead.* The posterior slope of the forehead unit typically starts around 1 to 1.5 cm above the superior orbital rims. If the slope is designed too high or too steep, a postoperative appearance of a flat, high forehead will result.
- *Palpable hardware.* Care must be taken to ensure that the hardware used to secure the construct is not palpable through the skin. Wire ends can be buried in adjacent bone holes, and resorbable plates can be placed on the intracranial surface. The time frame for complete resorption of current resorbable plates remains unknown, but residual plating material can be found in some patients more than 2 years after the operation.
- *Sphinx position.* Because of the increased risk for venous air embolism, use of the sitting or "sphinx" position for the treatment of craniosynostosis should be limited; a two-stage procedure, prone followed by the supine position, is preferred.

SUGGESTED READINGS

Cohen MM Jr, MacLean RE: Craniosynostosis: Diagnosis, Evaluation and Management. London, Oxford University Press, 2000.

Huang MH, Mouradian WE, Cohen SR, Gruss JS: The differential diagnosis of abnormal head shapes: Separating craniosynostosis from positional deformities and normal variants. Cleft Palate Craniofac J 35:204-211, 1998.

Jimenez DF, Barone CM, Cartwright CC, Baker L: Early management of craniosynostosis using endoscopic-assisted strip craniectomies and cranial orthotic molding therapy. Pediatrics 110:97-104, 2002.

Maugans TA, McComb JG, Levy ML: Surgical management of sagittal synostosis: A comparative analysis of strip craniectomy and calvarial vault remodeling. Pediatr Neurosurg 27:137-148, 1997.

McCarthy JG, Glasberg SB, Cutting CB, et al: Twenty-year experience with early surgery for craniosynostosis: I. Isolated craniofacial synostosis—results and unsolved problems. Plast Reconstr Surg 96:272-283, 1995.

McCarthy JG, Glasberg SB, Cutting CB, et al: Twenty-year experience with early surgery for craniosynostosis: II. The craniofacial synostosis syndromes and pansynostosis—results and unsolved problems. Plast Reconstr Surg 96:284-295, discussion 296-288, 1995.

Posnick JC, Ruiz RL: The craniofacial dysostosis syndromes: Current surgical thinking and future directions. Cleft Palate Craniofac J 37:433, 2000.

Warren SM, Greenwald JA, Spector JA, et al: New developments in cranial suture research. Plast Reconstr Surg 107:523-540, 2001.

Williams JK, Ellenbogen RG, Gruss JS: State of the art in craniofacial surgery: Nonsyndromic craniosynostosis. Cleft Palate Craniofac J 36:471-485, 1999.

Craniofacial Clefts and Orbital Hypertelorism

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HENRY K. KAWAMOTO, JR.

This chapter highlights the etiopathogenesis, clinical manifestations, and treatment options for clefts of the craniofacial skeleton. These clefts are associated with variable involvement of bone and soft tissue. Some display severe deformities of the face and skull, whereas others may be barely noticeable. The surgeon must therefore identify which structures are involved and outline treatment options that adequately address each component of the malformation.

Etiopathogenesis

Two embryologic theories have been proposed to explain the etiology of facial clefting. The classic theory holds that clefting is the result of failure of fusion between the facial processes. An alternative theory suggests that clefting is the result of a failure of neural crest migration.

The true incidence of craniofacial clefting is unknown, with estimates ranging from 1.5 to 6 per 100,000 births. The majority are sporadic, but their occurrence may result from many factors, both environmental and genetic. Suspected environmental factors include infection and prenatal radiation, as well as maternal drug ingestion, dietary deficiency, and metabolic derangement. Drugs with known teratogenic potential include anticonvulsants, chemotherapeutics, corticosteroids, and tranquilizers. Hereditary factors play a stronger role in the Treacher Collins and Goldenhar syndromes.

Classification

In general, facial clefts are described and classified according to both their skeletal and soft tissue involvement.

The most widely used system to classify facial clefts was proposed by Tessier (Fig. 1). This system numbers clefts from 0 to 14, with the center of the orbit used as the reference landmark. A No. 0 cleft is located in the lower portion of the facial midline and continues superiorly as a No. 14 cleft. Clefts 1 through 6 run more laterally in the lower part of the face. Clefts 7 and 8 are located at the lateral corner of the eye, and Nos. 9 through 13 return to the midline in the upper portion of the face. Midline and paramidline clefts often produce hypertelorism as the distance between the orbital cavities is increased.

Diagnostic Studies

Preoperative evaluation of patients with severe craniofacial clefts begins with a complete history and physical examination, including ophthalmologic assessment, to evaluate what tissues are deficient and what tissues may be used in the reconstruction.

Specific radiographic studies include cephalograms and computed tomographic scans of the head and neck.

Goals of Reconstruction

In most craniofacial clefts, reconstruction of both the skeletal and soft tissue defects is required. For mild clefts, there is no urgency, and delay may even facilitate repair. For more severe clefts, the initial repair should be undertaken early. During infancy, procedures are generally limited to the soft tissues, and skeletal reconstruction may best be postponed until childhood.

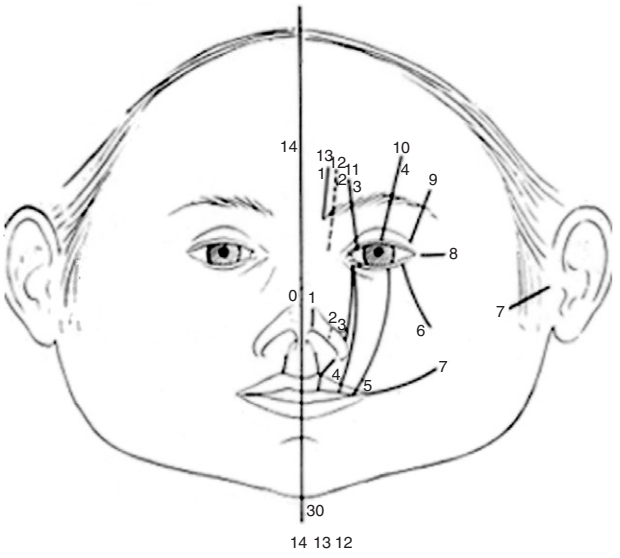


Figure 1 • Facial cleft classification system proposed by Tessier.

In reconstructing the soft tissue defects, local flaps designed along aesthetic lines are preferred. In most instances, multiple procedures over a period of many years are required to achieve optimal aesthetic results. Tissue expanders may be helpful to recruit soft tissue and avoid wound closure under tension.

Skeletal reconstruction may involve osteotomies and bone grafts. When indicated, autogenous bone harvested from the calvaria, rib, or iliac crest (or a combination of these sources) is preferable to alloplastic material. The use of bone grafts in younger children can potentially have adverse affects on growth, and they are prone to resorption. For patients with significant hypoplasia, this problem is less important because their growth potential is already diminished.

Clefts

Each of the following clefts is discussed individually. They have a spectrum of manifestations that may include some or all of the anatomic derangements noted.

No. 0 Cleft

A No. 0 cleft (Fig. 2) involves the midline of the face. It may continue superiorly into the cranium as a No. 14 cleft. The defect may involve hypoplasia or duplications of the affected structures. The abnormalities may be mild, such as minimal notching of the lip vermilion or nose, or severe, such as wide division of all midline craniofacial structures. Midline clefts of the mandible (No. 30) result from failure of fusion of the first branchial arches.

PATHOLOGIC ANATOMY

Osseous Defects

1. Diastema between the central incisors that continues posteriorly in the midline of the premaxilla
2. Keel-shaped alveolus with the teeth directed toward the cleft
3. Anterior open bite
4. Duplicated anterior nasal spine
5. Bifid nose, thickened and often duplicated nasal septum, and broad nasal bones
6. Enlargement of the sphenoid and ethmoid sinuses

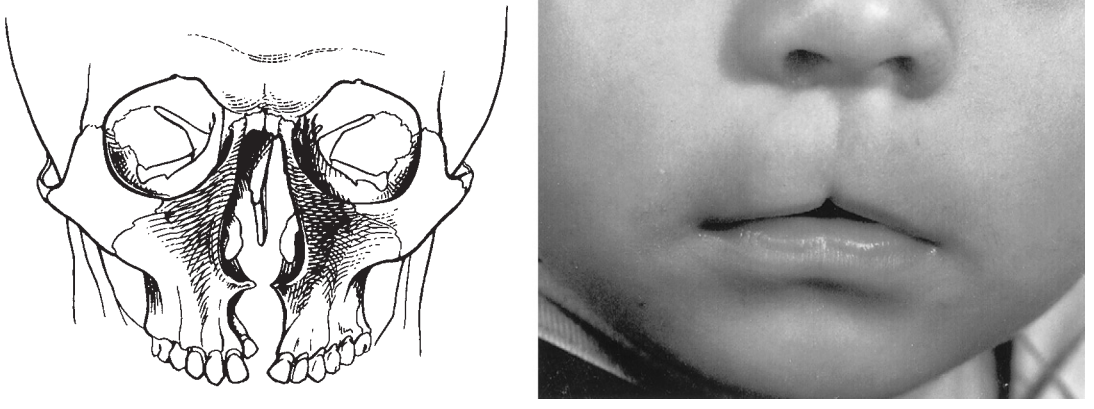


Figure 2 • No. 0 cleft.

7 Reduced midline facial height
8 Associated orbital hypertelorism

Soft Tissue Defects

1 Deficiency in the midline of the upper lip
2 Broad philtral columns
3 Fibrous band extending along the margins of the columella and drawing the upper lip superiorly
4 Duplicated labial frenulum

In hypoplastic cases, any of the following can be partial or absent: philtrum, columella, premaxilla, and nasal septum. There may also be associated deformities of the eye, scalp, and forebrain.

TREATMENT. Reconstruction of the lip should precisely repair the orbicularis muscle and align the vermilion. These repairs should be performed in infancy and, in selected cases, can be done intraorally.

A bifid frenulum does not require repair unless it is associated with a gap in the alveolus between the central incisors. A bifid tongue tethered by a short frenulum can be released with a Z-plasty and reapproximated.

The absence of a premaxilla contributes to collapse of the lateral dental arches. Prevention requires placement of interpositional bone grafts. Established collapse of the maxilla can be corrected with a LeFort I osteotomy and rapid palatal expansion.

No. 1 Cleft

A No. 1 cleft (Fig. 3) is a paramedian cleft of the lower portion of the face that passes through the alar dome of the nose. It may continue superiorly into the cranium as a No. 13 cleft.

PATHOLOGIC ANATOMY

Osseous Defects

1 Diastema between the central and lateral incisors that continues through the piriform aperture lateral to the anterior nasal spine and may traverse the hard and soft palates
2 Keel-shaped alveolus with orientation of the teeth toward the cleft
3 Anterior open bite
4 Cleft at the junction of the nasal bone and frontal process of the maxilla
5 Displaced and flattened nasal bones
6 Associated orbital hypertelorism

Soft Tissue Defects

1 Deficiency within Cupid's bow extending through the dome of the alar cartilage of the nose
2 Short, broad columella

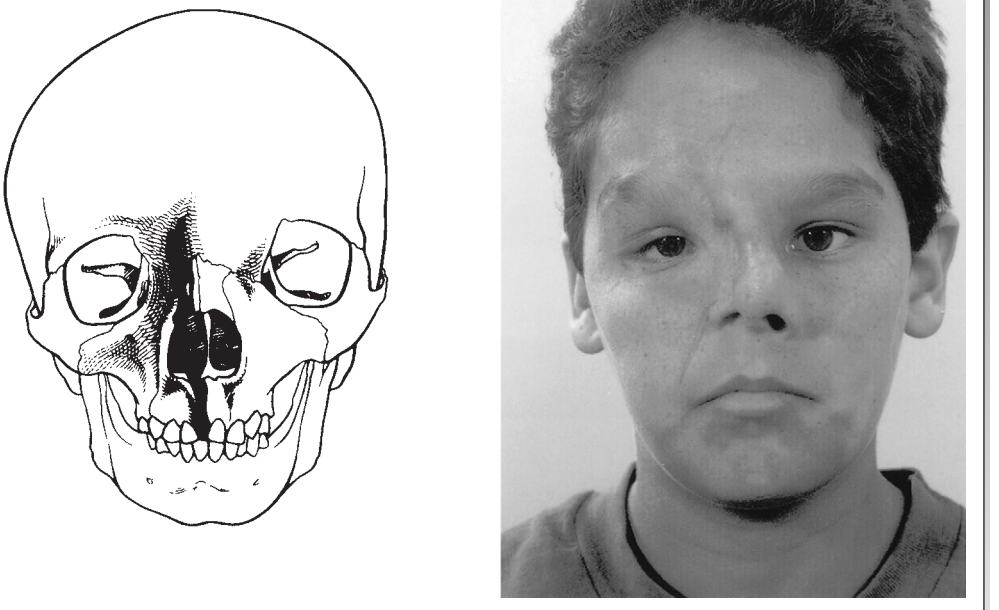


Figure 3 • No. 1 cleft.

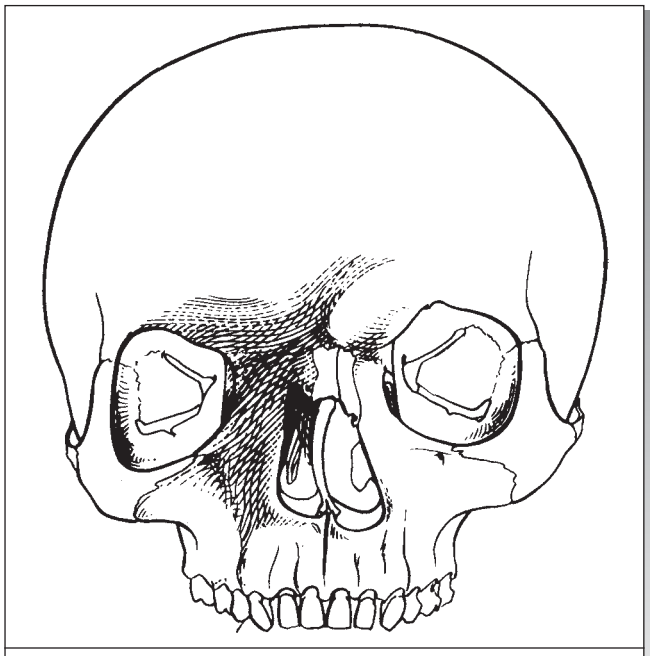


Figure 4 • No. 2 cleft.

3 Deviation of the nasal tip and septum away from the side of the cleft
4 Extension medial to the medial canthus

TREATMENT. Repair of the cleft lip and nasal deformity follows the basic principles of cleft lip repair. Nasal reconstruction requires reapproximation of the alar cartilage and excision of excess skin and soft tissue. Replacement of deficient cartilage can be accomplished with a donor graft from the ear.

No. 2 Cleft

A No. 2 cleft (Fig. 4) is rare. It is also a paramedian facial cleft that lies slightly more lateral than a No. 1 cleft. It may continue superiorly in the cranium as a No. 12 cleft.

PATHOLOGIC ANATOMY

Osseous Defects

1 Diastema between the lateral incisor and canine
2 Intact maxillary sinus
3 Deviation of the nasal septum away from the cleft
4 Continuation between the nasal bones and the frontal process of the maxilla
5 Associated orbital hypertelorism

Soft Tissue Defects

1 In the region of the common cleft lip
2 Hypoplasia of the middle third of the alar rim
3 Broad nasal bridge
4 Flattening of the lateral aspect of the nose
5 Passage medial to the palpebral fissure without involvement of the eyelid
6 Lacrimal system uninvolved
7 Medial canthus laterally displaced
8 Distortion of the medial corner of the eyebrow

TREATMENT. Repair of the lip again follows the principles of standard cleft lip repair. Notching of the lower lateral cartilage can be repaired with an autogenous cartilage graft.

No. 3 Cleft

A No. 3 cleft (Fig. 5) is one of the more common facial clefts and involves the paramidline structures of the face. It has been referred to as an "oblique facial cleft" and "oronasoocular cleft."

Because of its location, communication between the oral cavity, nasal cavity, maxillary sinus, and orbit is created. The inferior punctum of the lacrimal apparatus is displaced inferiorly, thereby causing obstruction of the lacrimal duct. The lower canaliculus is malformed, and the drainage system ends as an opening directly onto the cheek without entrance into the nasal cavity.

A No. 3 cleft tends to have equal sex distribution and an equal distribution of right, left, and bilateral involvement. Bilateral cases are often seen in conjunction with a No. 4 or 5 cleft on the contralateral side.

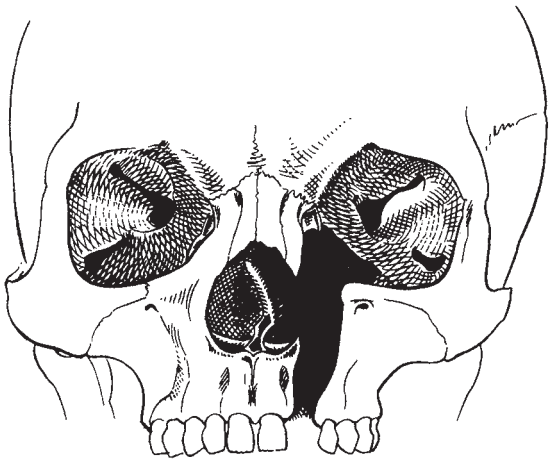
PATHOLOGIC ANATOMY

Osseous Defects

1 Diastema between the lateral incisor and canine that continues superiorly through the lateral piriform rim, medial maxillary wall, and medial third of the infraorbital rim
2 Flattened maxillary arch
3 Location lateral to the nasal bones
4 Involvement of the orbit with inferior displacement of the globe as a result of absence of the orbital floor
5 Frontal process of the maxilla absent with passage of the cleft obliquely through the lacrimal groove
6 Mild narrowing of the sphenoid and ethmoid sinuses

Soft Tissue Defects

1 Cleft extends from Cupid's bow along the philtral column and onto the alar base of the nose
2 A foreshortened nose pulled superiorly at the alar base and flared in the region of the deficient tip



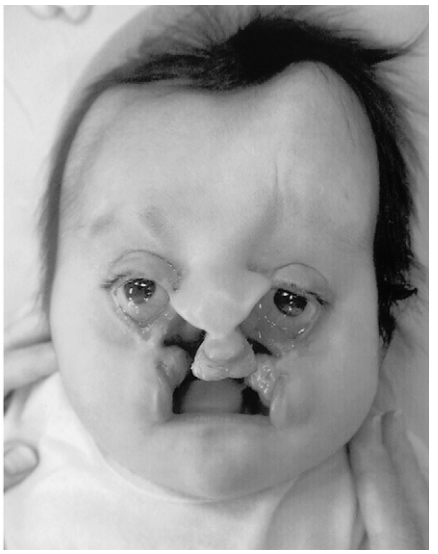


Figure 5 • No. 3 cleft (photograph represents a bilateral example).

3 Deficient perinasal tissues, cheek, and medial third of the lower eyelid
4 Hypoplastic and inferiorly displaced medial canthus
5 Colobomas located medial to the punctum
6 Microphthalmos with inferiorly and laterally displaced eyes
7 Deficiency between the medial canthus and inferior lacrimal punctum

TREATMENT. A No. 3 cleft is one of the most difficult malformations to treat. Early repositioning of the soft tissues of the face is indicated for corneal exposure and the general appearance of the infant.

Soft tissue must be introduced into the medial canthus and the alar base, preferably with a medially based musculocutaneous flap from the upper eyelid. The medial canthal tendon is detached from its inferior position to allow the palpebral fissure to move superiorly to match the contralateral side.

The junction between the skin and mucosa of the lateral cheek elements is incised, and mucosal flaps are elevated into the mouth and nasal cavity. Bilateral gingivobuccal incisions are made to rotate the lip inferiorly and advance it medially. Dissection of the nasal sidewall is performed just above the bone to allow the soft tissues of the nose to move inferiorly. Lining is obtained from the mucosa of the nasal floor or septum.

The medial canthus is reconstructed with transnasal surgical wires. Bone grafts may be used to reconstruct the deficient orbital floor.

Closure of the lip is performed either as a definitive repair or as a lip adhesion. With minimal tension and sufficient length, primary repair of the bilateral lip cleft may be performed. In cases with excess tension and an already lengthy primary procedure, simple lip adhesion may be preferable.

PEARLS AND PITFALLS

- Soft tissue deficiency in the lower eyelids results in corneal exposure.
- Avoid unnecessary procedures on the upper eyelids so that they may be used for reconstruction of the lower eyelids.
- The position of the medial canthus and its relationship to the lateral alar base must be appreciated.
- Secondary attempts to realign the medial canthi in the vertical plane are less successful than a carefully planned primary reconstruction.

No. 4 Cleft

A No. 4 cleft (Fig. 6) passes on the cheek, away from the nose. Unilateral clefts have a 2:1.3 right-to-left ratio and a 2.5:1 male-to-female ratio. Bilateral clefts have an equal frequency in both sexes.

PATHOLOGIC ANATOMY

Osseous Defects

1 Diastema passing between the lateral incisor and canine and continuing superiorly through the maxillary sinus lateral to the piriform aperture and medial to the infraorbital foramen
2 Intact medial wall of the maxillary sinus and separate nasal cavity
3 Termination of the cleft at the medial aspect of the inferior orbital rim

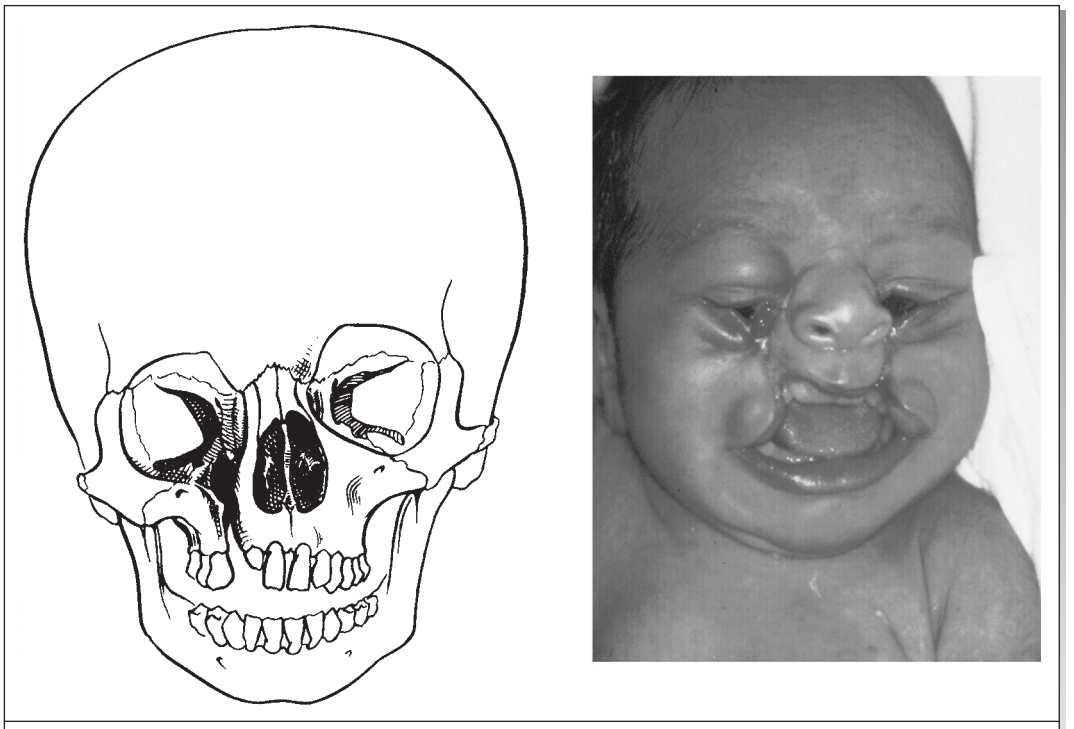


Figure 6 • No. 4 cleft (photograph represents a bilateral example).

4 Associated posterior choanal atresia
5 When bilateral, the premaxilla is often protrusive despite the tendency for midface hypoplasia
6 Orbital floor defect

Soft Tissue Defects

1 Deficiency between the upper lip and lower eyelid that decreases the vertical distance between the mouth and the medial third of the eye
2 Passage lateral to the philtral column and medial to the oral commissure
3 Absent perioral muscles medially
4 Passage lateral to the ala, which is normal in appearance but superiorly positioned
5 Continuation superiorly through the medial aspect of the midface and medial third of the infraorbital rim
6 Sparing of the lacrimal sac but involvement of the lower lacrimal duct and the inferior lacrimal punctum
7 Occasional microphthalmos or anophthalmia
8 Inferiorly displaced globe secondary to orbital floor defects

TREATMENT. Autogenous cranial bone grafts are used to reconstruct the orbital rim and floor.

Soft tissue reconstruction is performed with a superiorly based nasojugal flap transposed and inserted in a subciliary incision after wide soft tissue undermining. The lateral aspect of the cheek is advanced medially toward the medial canthus. The medial canthal tendon has normal structure but is malpositioned, and the inferior tarsal plate is often required for reconstruction of the medial canthal tendon. Despite the ability to repair the lacrimal duct system over Silastic stents, the results have been disappointing. When reconstructing the lip, tissue lateral to the philtral column should be excised and the lateral lip element brought in to reestablish the philtral column.

No. 5 Cleft

A No. 5 cleft (Fig. 7) has also been referred to as an "oblique facial cleft" because of its location just medial to the lateral commissure of the lip. Among the few reported cases, a quarter are unilateral, a quarter are bilateral, and the remainder coexist with other facial clefts.

PATHOLOGIC ANATOMY

Osseous Defects

1 Diastema between the canine and the first bicuspid
2 Continuation through a hypoplastic maxillary sinus, lateral to the infraorbital foramen
3 Termination in the inferolateral portion of the orbital floor

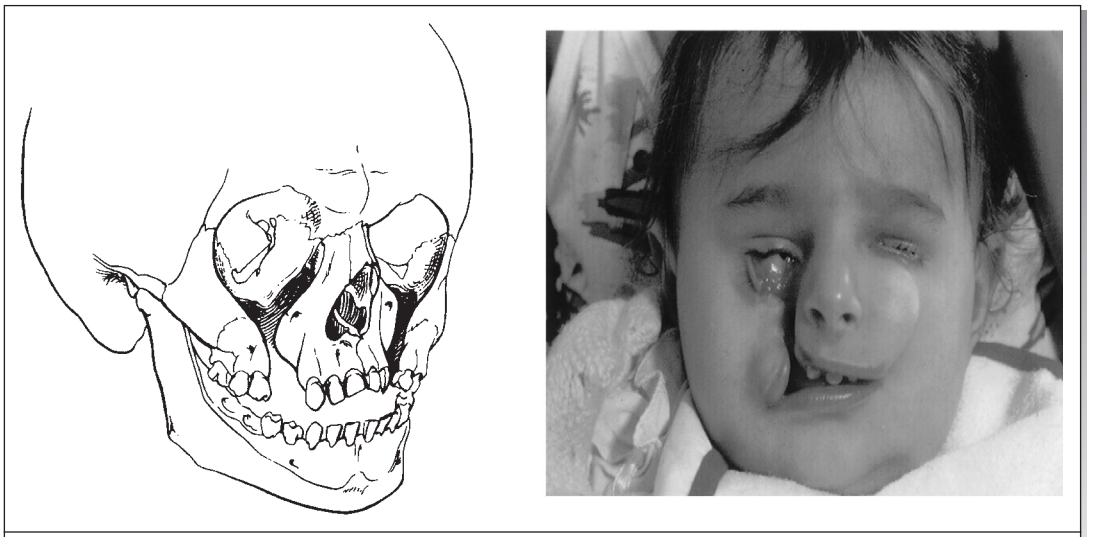


Figure 7 • No. 5 cleft.

Soft Tissue Defects

1. Passage running medial to the oral commissure
2. Continuation superiorly on the cheek
3. Foreshortened nose with elevation of the alar base
4. Termination at the lateral aspect of the lower eyelid (the upper eyelid and eyebrow are spared)
5. Occasional microphthalmos

TREATMENT. Treatment of a No. 5 cleft is similar to that of a No. 4 cleft. Skeletal support can be achieved with cranial bone grafts.

No. 6 Cleft

No. 6 clefts (Fig. 8) are located in the vicinity of the zygomaticomaxillary suture and are similar to those in the incomplete forms of Treacher Collins syndrome.

PATHOLOGIC ANATOMY

Osseous Defects

1. Absence of an alveolar cleft
2. Skeletal cleft between the hypoplastic zygoma and posterior aspect of the maxilla
3. Occlusal cant
4. Posterior choanal atresia
5. Passage into the orbit and communication with the inferior orbital fissure
6. Increased antegonial angle of the mandible
7. Narrow anterior cranial fossa

Soft Tissue Defects

1. A vertical furrow running from the lateral commissure of the mouth to the lateral aspect of the lower eyelid
2. Inferiorly displaced lateral canthus

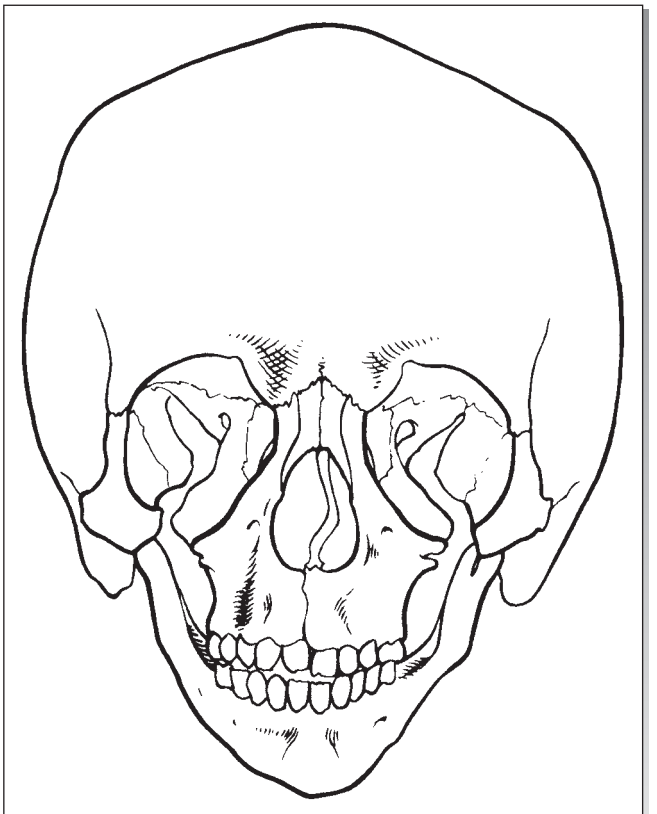
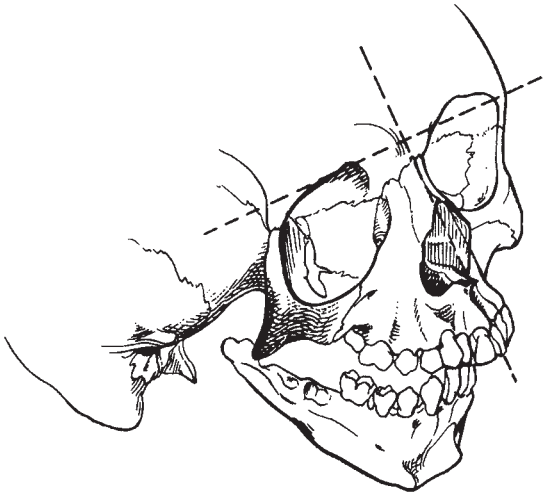


Figure 8 • No. 6 cleft.



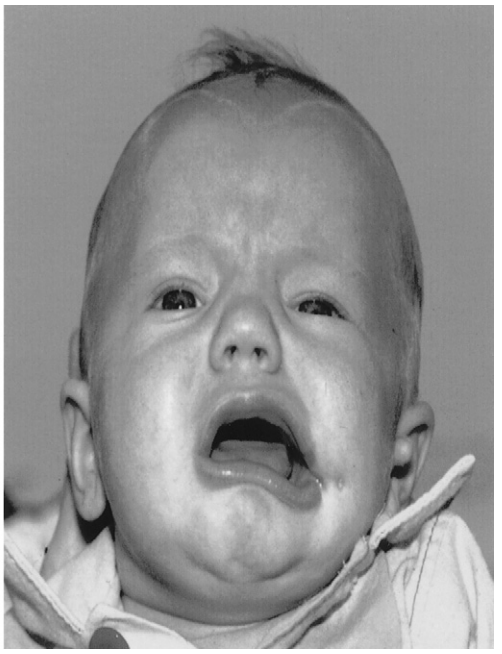


Figure 9 • No. 7 cleft.

3 Colobomas between the middle and lateral thirds of the lower eyelid
4 Microphthalmos
5 Hearing deficits masked by a normal-appearing external ear

TREATMENT. Management of a No. 6 cleft follows the same principles as for a No. 7 cleft. The soft tissue defects should be addressed early. The cleft margins should be completely excised and the defect closed in layers. The skeletal reconstruction must address the zygomatic deficiency. However, because the zygoma is hypoplastic rather than absent, the reconstruction is simpler.

No. 7 Cleft

No. 7 clefts (Fig. 9) are the most common of the atypical facial clefts. They are discussed in the chapter "Craniofacial Microsomia."

No. 8 Cleft

No. 8 clefts (Fig. 10) represent malformations centered around the frontozygomatic suture, usually a temporal continuation of other defined clefts.

PATHOLOGIC ANATOMY

Osseous Defects

1 Deficiency of the lateral orbital rim
2 In the severe form, the greater wing of the sphenoid is the only bony support of the lateral orbit
3 Communication between the orbit and the temporal fossa

Soft Tissue Defects

1 Area of the lateral canthus to the temporal region
2 Antimongoloid slant of the palpebral fissure
3 Associated anomalies of the globe or periorbital structures, such as epibulbar dermoids

TREATMENT. Reconstruction of the lateral canthus is best accomplished by two adjacent Z-plasties lateral to the lateral canthus. A third Z-plasty is placed medially along the lower eyelid to release the skin and allow it to slide laterally.

No. 9 Cleft

A No. 9 cleft (Fig. 11) is rare. It involves the superolateral hemisphere of the orbit and is the extension of a No. 5 cleft.

PATHOLOGIC ANATOMY

Osseous Defects

1 Through the superolateral orbit, upper wing of the sphenoid, and the temporal and parietal bones
2 Hypoplastic greater wing of the sphenoid
3 Foreshortened anterior cranial base

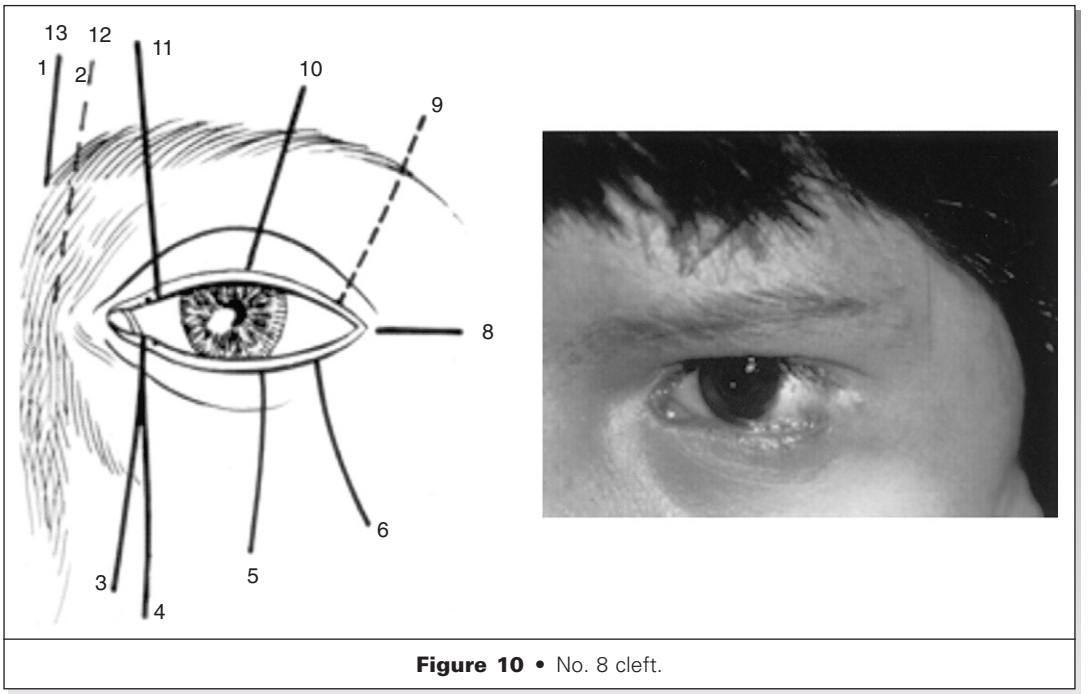


Figure 10 • No. 8 cleft.

Soft Tissue Defects

1 Through the lateral third of the upper eyelid and eyebrow
2 Possible microphthalmos with lateral displacement of the globe
3 Continuation into the temporoparietal region of the scalp with anterior displacement of the hairline
4 Commonly associated with palsy of the frontal branch of the facial nerve

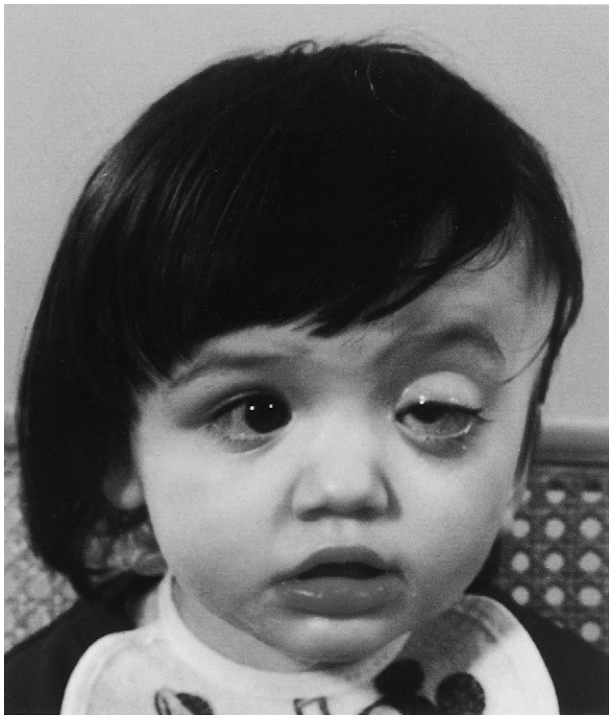


Figure 11 • No. 9 cleft.

TREATMENT. No. 9 and higher clefts involve the cranium, and thus treatment must address the contour of the forehead and cranial vault.

The cleft in the eyelid can be excited and reconstituted with precise alignment of all components.

No. 10 Cleft

A No. 10 cleft (Fig. 12) is the superior extension of a No. 4 cleft and is central in the orbit. It is

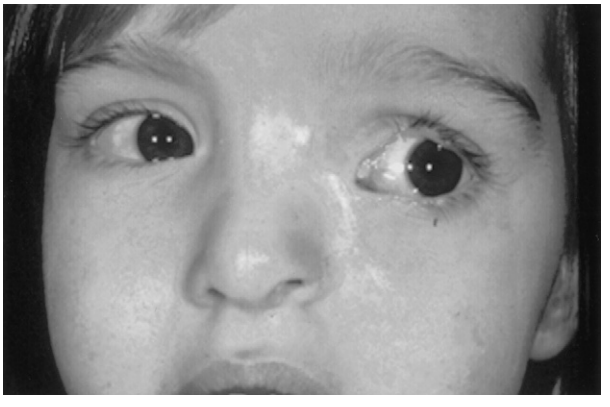


Figure 12 • No. 10 cleft.

sufficiently medial to be placed in the frontal dysplasia group of malformations.

PATHOLOGIC ANATOMY

Osseous Defects

1. Through the middle of the supraorbital rim, frontal bone, and orbital roof lateral to the supraorbital foramen
2. Often associated with a frontal encephalocele
3. Secondary, clockwise rotation of the ipsilateral orbit and counterclockwise rotation of the contralateral orbit
4. Occasional orbital hypertelorism
5. Widening of the anterior cranial base

Soft Tissue Defects

1. Through the middle third of the upper eyelid and eyebrow
2. Coloboma of the upper eyelid
3. Enlargement of the palpebral fissure
4. Globe inferiorly and laterally displaced
5. Frontal hairline projection joining the lateral aspect of the eyebrow

TREATMENT. The cranial vault is reconstructed with autogenous bone grafts. The soft tissue cleft is excised, and the margins of the defect are reapproximated. The orbital hypertelorism is best managed with bipartition of the face.

No. 11 Cleft

A No. 11 cleft (Fig. 13) has not been reported as an isolated entity but rather as the superior continuation of a No. 3 cleft. It runs in the superior and medial aspect of the orbit and is grouped with the frontonasal dysplasias.

PATHOLOGIC ANATOMY

Osseous Defects

1. Either through or lateral to the ethmoid sinus
2. Orbital hypertelorism
3. Normal anterior cranial base, sphenoid wings, and pterygoid plates

Soft Tissue Defects

1. Through the medial third of the upper eyelid and eyebrow
2. Continuation into the medial third of the frontal hairline

TREATMENT. Treatment is similar to that described for a No. 10 cleft.

No. 12 Cleft

A No. 12 cleft (Fig. 14) is also among the frontonasal dysplasias and is the superior extension of a No. 2 cleft. It involves the cranium and medial aspect of the orbit.

PATHOLOGIC ANATOMY

Osseous Defects

1. Through the flattened, frontal process of the maxilla with involvement of the more medial nasal bones and ethmoid labyrinth
2. Enlarged frontal, ethmoidal, and sphenoidal air cells

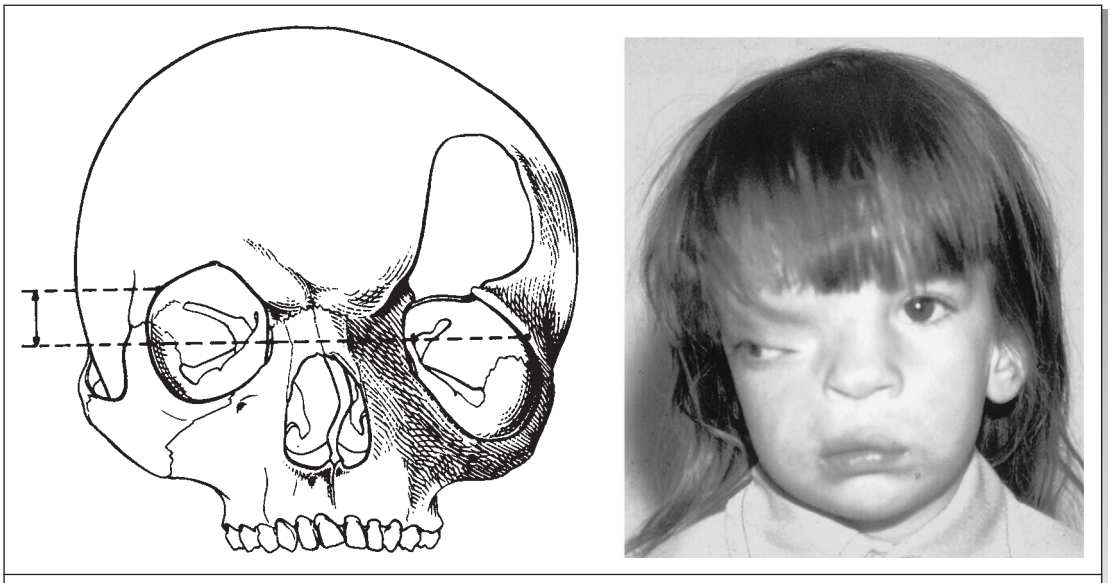


Figure 13 • No. 11 cleft.

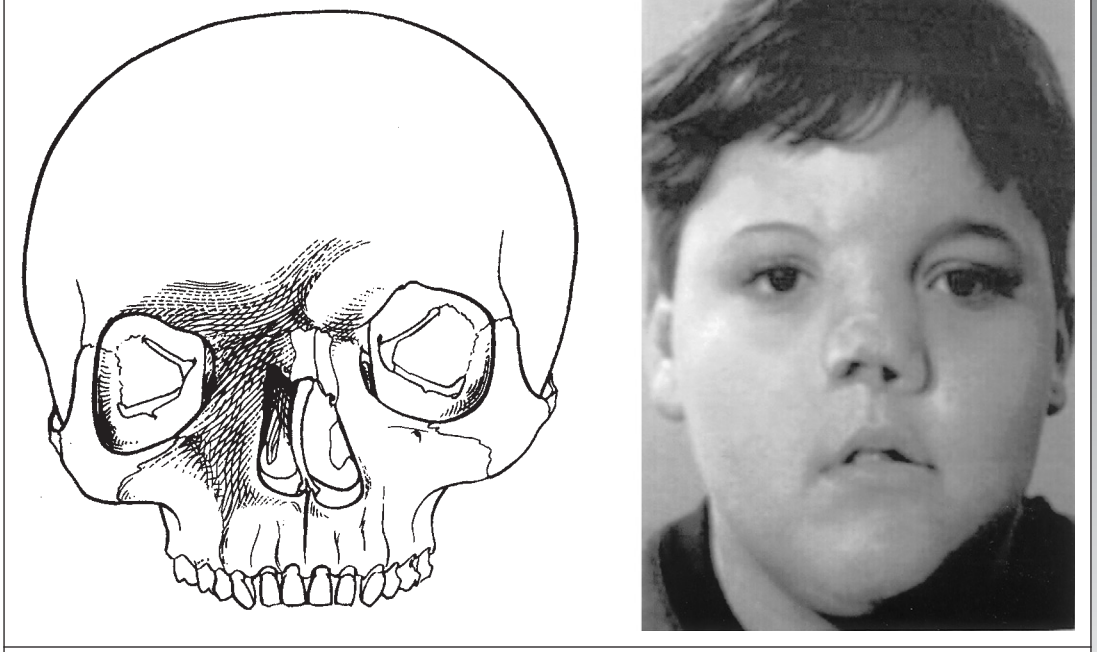


Figure 14 • No. 12 cleft.

3 Hypertelorism, telecanthus, and downward and lateral displacement of the orbit
4 Obtuse frontonasal angle
5 Continuation lateral to the olfactory groove with sparing of the cribriform plate
6 Widened anterior and middle cranial fossae on the cleft side
7 Mild asymmetry of the sphenoid wings and anterior cranial base

Soft Tissue Defects

1 Medial to the medial canthus and continuing through the root, or medial third, of the eyebrow
2 Eyelids not involved
3 Frontal hairline just off the midline of the forehead with a downward projection

TREATMENT. Treatment of the skeletal defect involves reconstitution of the underlying cranial vault and accurate repositioning of the orbit. An osteotomy of the medial orbital wall is required, and reconstruction is performed with autogenous bone.

Because the cleft involves the medial nasal bones and soft tissues of the nose, reconstruction of these structures is indicated.

Length and projection of the depressed and splayed nasal dorsum may be achieved with an autogenous bone graft.

No. 13 Cleft

A No. 13 cleft (Fig. 15) is a paramedian cleft that is the superior extension of a No. 1 cleft. It, too, is grouped with the frontonasal dysplasias. It is the most lateral of the superior clefts to involve the cribriform plate.

PATHOLOGIC ANATOMY

Osseous Defects

1 Between the nasal bones and the frontal process of the maxilla
2 Extension through the frontal bone and along a widened olfactory groove
3 Orbital hypertelorism
4 Cribriform plate displaced inferiorly
5 Displacement of the pterygoid plates, greater and lesser wings of the sphenoid, and anterior cranial fossa.

Soft Tissue Defects

1 Medial to and sparing the upper eyelid and eyebrow
2 Inferiorly displaced tongue of the frontal hairline

TREATMENT. Reconstruction involves the cranial vault and overlying skin and soft tissue with correction of the hypertelorism.

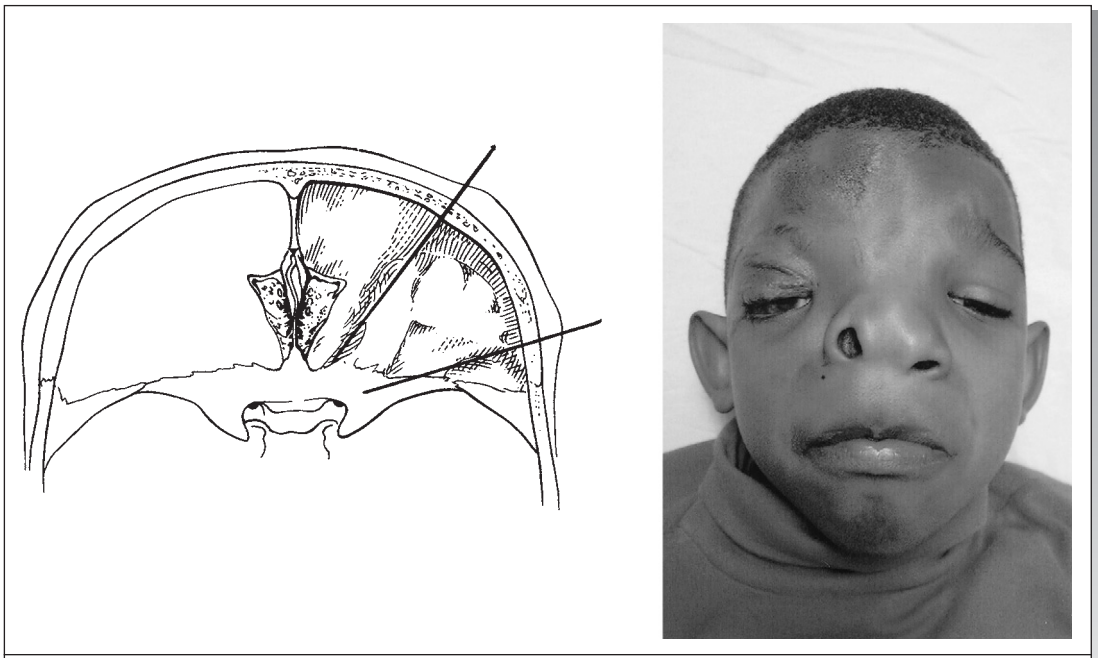


Figure 15 • No. 13 cleft.

No. 14 Cleft

A No. 14 cleft (Fig. 16) lies in the superior midline, often as a continuation of the lower No. 0 cleft. It may be manifested as hypoplasia or hyperplasia of the midline facial structures. Agenesis of the midline structures is seen in holoprosencephalic disorders (cyclopia, ethmocephaly, and cebocephaly). In these cases, microcephaly and orbital hypotelorism are present. The degree of facial malformation usually parallels that of the forebrain.

Widened or duplicated midline structures are observed in patients with frontonasoethmoid dysplasia. In these instances, orbital hypertelorism is

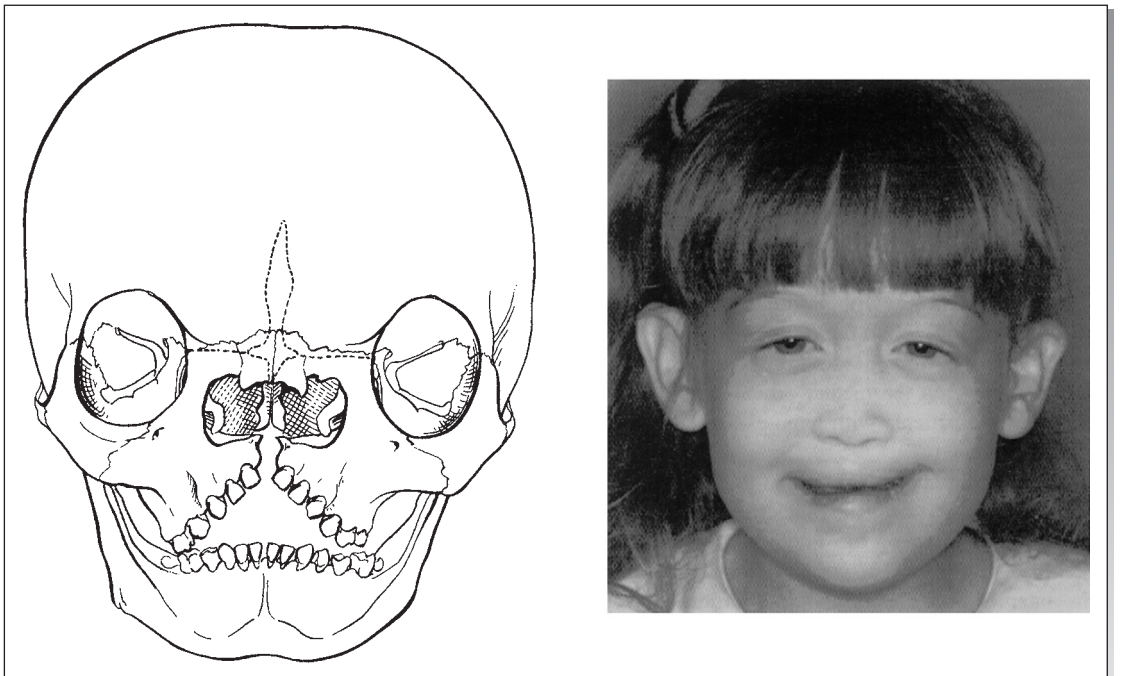


Figure 16 • No. 14 cleft.

produced by midline masses extending from the forebrain. Previously used terms include "median cleft face syndrome," "frontonasal dysplasia," and "holoprosencephaly." In the latter case, the associated cerebral malformations often mirror the external anomalies. The severest forms, which manifest as cyclopia, cebocephaly, or ethmocephaly, are usually fatal.

When examining patients with orbital hypertelorism, the normal anatomic relationships of the eyes should be considered. The palpebral fissure roughly follows the pattern of the eyebrow. The intercanthal distance should be measured between the medial canthi (normal ranges from 28 to 32 mm). In addition, the interorbital distance between the lacrimal crests (as measured on a posteroanterior cephalogram) should be determined (normal ranges from 24 to 32 mm in males and 22 to 28 mm in females). The severity of hypertelorism may be classified into three degrees: first degree refers to an interorbital distance of 30 to 34 mm; second degree, 34 to 40 mm; and third degree, greater than 40 mm.

PATHOLOGIC ANATOMY

Osseous Defects

1. Caudal aspect of the frontal bone displaced
2. Cribriform plate caudally displaced
3. Greater and lesser wings of the sphenoid rotated laterally
4. Broadened sphenoid with displacement of the pterygoid plates away from the midline
5. Orbital hypotelorism or hypertelorism
6. Widened anterior cranial fossa
7. Widened crista galli

Soft Tissue Defects

1. Wide forehead

TREATMENT. Similar to a No. 13 cleft, reconstruction involves the cranial vault and overlying skin and soft tissue. Hypotelorism is usually an indication of underlying brain maldevelopment. In such cases, repair is not generally indicated. Patients with hypertelorism, however, require skeletal reconstruction in the form of facial bipartition, specifically, osteotomies designed to rotate the orbits around a pivot point located between the superior central incisors.

If the patient's occlusion is close to ideal, arch bars may be applied to the maxillary and mandibular teeth. Often, the malocclusion may be too significant to expect an adequate result with facial bipartition alone, and secondary orthognathic surgery is required.

A bifrontal craniotomy is performed for exposure of the anterior cranial fossa. The posterior cranial osteotomy should be placed anterior to the coronal sutures. An osteotomy is made in the midportion of the zygomatic arch. Next, an osteotomy of the lateral orbital wall is performed with the saw inserted from behind the temporalis muscle and angled into the inferior orbital fissure. The bone cut is extended superiorly into the lateral aspect of the orbital roof.

The osteotomy of the roof is performed through the anterior cranial fossa. It is commenced laterally at the pterion, where the lateral orbital wall osteotomy was completed, and continued medially just anterior to the cribriform plate. The medial osteotomies on either side of the face should match up in the midline just anterior to the first olfactory nerve foramina. The medial orbital wall osteotomy is completed with a narrow osteotome posterior to the posterior lacrimal crest and inferior to the inferior orbital fissure.

The septum is sectioned through the anterior cranial base with a straight osteotome anterior to the crista galli. The pterygomaxillary disjunction is performed with a curved osteotome placed in the infratemporal fossa through the mouth. The midface is disimpacted with Rowe nasomaxillary forceps.

Bipartition of the face is performed with a midnasal osteotomy from above and oriented between the central incisors of the maxilla. A sagittal split of the maxilla is completed through a small gingivobuccal sulcus incision. When doing so, the palatal mucosa should be left undisturbed. The facial halves are repositioned medially, and a rotary bur may be used to remove any bony interference. If occlusion is to be set for maxillomandibular fixation, the orotracheal tube needs to be repositioned as a nasotracheal tube. Midface advancement may be included with the maxillomandibular fixation, if indicated. Zygomatic advancement is achieved first and stabilized with miniplates, as is advancement at the upper part of the orbits. The opening between the anterior cranial fossa and the nasal cavity is occluded with Gelfoam, and fibrin glue is placed in the gap.

The anterior cranial vault is recontoured and replaced. The lateral canthi are resuspended through separate drill holes in the lateral orbital rims. The temporalis muscle is used to fill the areas of advancement and is resuspended off the temporal bones.

In all cases, any associated nasal deformity must be evaluated and addressed. Nasal projection will be lost if bone is resected centrally and not replaced. Nasal reconstruction can be accomplished with a cantilevered bone graft.

Reconstruction of a mandibular cleft requires placement of an interpositional bone graft. Absence of bone in the region of the symphysis produces narrowing of the dental arches and malocclusion. Reconstruction is best delayed until after eruption of the permanent incisors. Rigid fixation in the soft bones of a child is difficult and unwarranted.

Postoperative Care

Patients who undergo solely soft tissue rearrangement may be discharged home immediately. In such cases, the typical complications include wound infection and dehiscence.

Reconstruction of the bony skeleton is a larger insult and requires several days of observation. The greatest concern after facial bipartition is the development of intracranial infection as a result of seeding of the central nervous system through the anterior cranial fossa from its communication with the nasal and oral cavities.

The more common complaint after orbital repositioning is diplopia, which may result from setting the orbits at different vertical heights or releasing the anchoring periosteum of the extraocular muscles. In either instance, most cases resolve spontaneously. In those that do not, resetting of muscle lengths by an ophthalmologic surgeon may be required.

SUGGESTED READINGS

Gorlin RJ, Pindborg JJ: Syndromes of the Head and Neck. New York, McGraw-Hill, 1964.

Kawamoto HK Jr: The kaleidoscopic world of rare craniofacial clefts: Order out of chaos (Tessier classification). Clin Plast Surg 3:529, 1976.

Tessier P: Colobomas. Vertical and oblique complete facial clefts. Panminerva Med 11:95, 1969.

Tessier P: Orbital hypertelorism. Plast Reconstr Surg 53:1-18, 1974.

Tessier P: Anatomical classification of facial, cranio-facial, and latero-facial clefts. J Maxillofac Surg 4:69, 1976.

Craniofacial Microsomia

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Among congenital anomalies of the head and neck, craniofacial microsomia is second in incidence only to cleft lip and palate. Because the clinical manifestations can be varied (Fig. 1), many terms have been used to designate the anomaly, but *craniofacial microsomia* is preferred since the cranial bones can be involved. Other commonly used names include hemifacial microsomia, first and second branchial arch syndrome, and otomandibular dysostosis. Goldenhar's syndrome, a variant of craniofacial microsomia, is also known as oculovertebral sequence and is characterized by lipomas and dermoids of the globe, coloboma of the eyelid, and cervical vertebral anomalies.

The incidence of bilateral involvement (Fig. 2) ranges from 5% to 30%, with a slight male preponderance. In addition to the obvious problems in craniofacial form, there can also be functional deficits characterized by respiratory insufficiency, swallowing disorders, and hearing deficits.

Etiopathogenesis

Two major theories have been proposed to explain the etiology of craniofacial microsomia.

Teratogenic Theory

The teratogenic theory is based on animal studies and clinical observations. For example, triazine maternally administered to mouse embryos results in craniofacial microsomia–like phenotypes. The traditional explanation is that the drug had a direct teratogenic effect. Hemorrhage of the stapedial artery resulting in the development of a hematoma that injures the developing structures of the first and second branchial arches is now regarded as a side effect. Administration of the retinoic acid derivative etretinate to rats early in development results in offspring with a phenotype similar to that of craniofacial microsomia. This effect has been explained by the fact that neural crest cells express large amounts of retinoic acid binding protein and, as a result, cell migration is impeded by the drug. Finally, thalidomide and ethanol ingestion in pregnant women, as well as gestational diabetes, can result in human offspring with craniofacial microsomia.

Genetic Theory

In a mutant mouse model, an insertional deletion on chromosome 10 demonstrates an autosomal mode of transmission with a 25% rate of penetration for the development of craniofacial microsomia. Moreover, human genetics studies have also demonstrated positive family histories in 9% to 44% of probands, a finding suggestive of autosomal dominant inheritance with high variability and low penetrance.

A study of twin pairs with craniofacial microsomia has shown that most affected subjects display a heterogeneous pattern of inheritance.

Overall, however, a multifactorial etiology (e.g., abnormal genes, teratogens, vascular events) is most likely responsible for most cases.

Pathologic Anatomy

Skeletal

The mandibular pathology is the pathognomonic finding of the syndrome. The classic deformity consists of underdevelopment of the ramus and condyle. The associated dentoalveolus and dental structures are likewise affected. Pruzansky proposed a classification of the mandibular deformity that was subsequently modified by Kaban and Mulliken (Fig. 3).

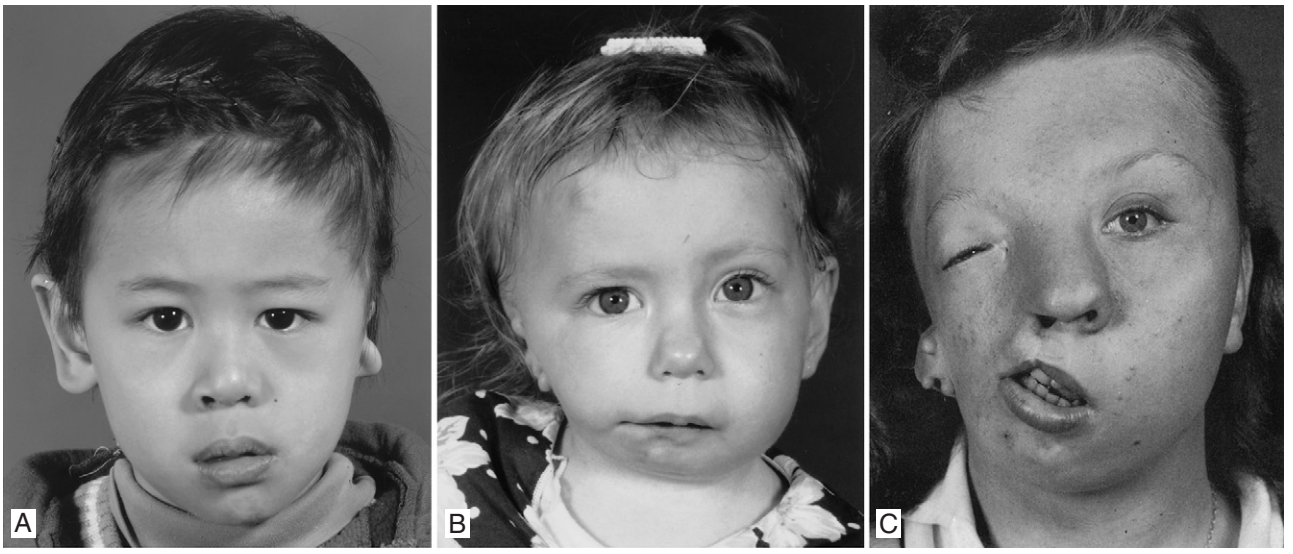

Figure 1 • Variable clinical manifestations of unilateral craniofacial microsomia. **A,** Mild example characterized by microtia, canting of the oral commissure and alar base plane, deficiency of the affected cheek soft tissues, and deviation of the chin to the ipsilateral side. **B,** Moderate example. Note the microtia, deficient cheek soft tissue, macrostomia, canting of the oral commissure, and retruded and deviated chin. **C,** Severe example with microtia, microphthalmos, retrusion of the brow, occlusal cant, elevation of the oral commissure, and deviation of the chin. (From Mathes S, Hentz, R [eds]: Plastic Surgery. Philadelphia, Elsevier, in press.)

Figure 2 • Bilateral craniofacial microsomia. Note the microtia, micrognathia, and underdevelopment of the lower third of the face. (From Mathes S, Hentz R [eds]: Plastic Surgery. Philadelphia, Elsevier, in press.)

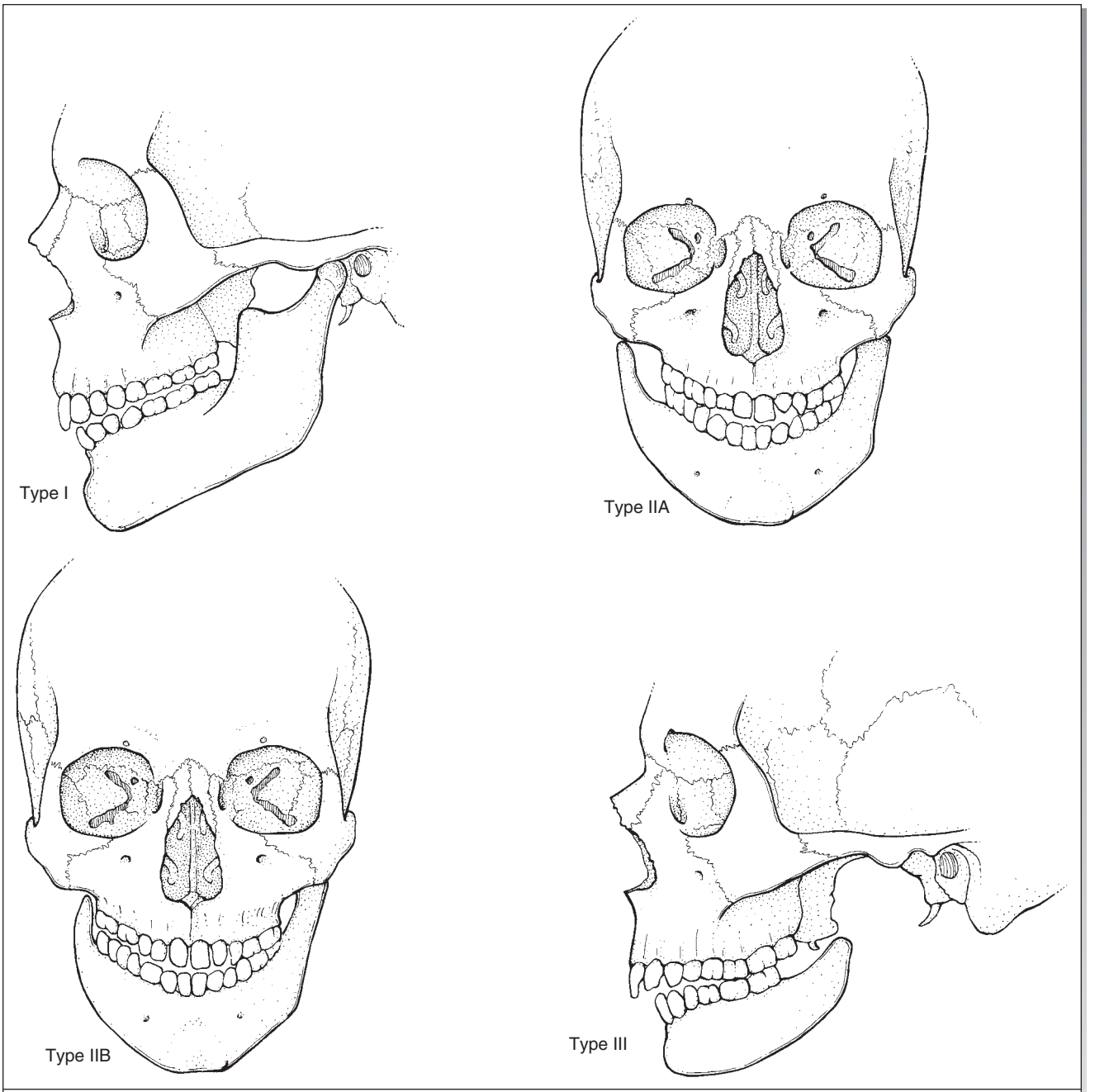

Figure 3 • Pruzansky (1969) classification of the mandibular deformity, as modified by Kaban and colleagues (1981). Type 1: the mandibular deficiency is only mild. Type IIA: the condyle and ramus are small, but the condyle and glenoid fossa are anatomically oriented. However, a flattened condyle can be hinged on a flat, often hypoplastic infratemporal surface. The coronoid process may be absent. Type IIB: similar to type IIA except that the vertical or superoinferior plane of the condyle-ramus is medially displaced. No functioning glenoid fossa is present. Type III: absence of the ramus, condyle, and coronoid process. (From Mathes S, Hentz, R [eds]: Plastic Surgery. Philadelphia, Elsevier, in press.)

Type I. The body, ramus, and condyle are abnormal in shape with reduced volume.

Type IIA. The body, ramus, and condyle are significantly reduced in volume, but the vertical orientation of the ramus is normal.

Type IIB. The same as IIA except that there is a medial or midline inclination of the ramus in the vertical dimension.

Type III. Absence of the ramus and condyle.

Other components of the craniofacial skeleton may also be involved. The maxilla is deficient in all dimensions on the affected side, and there is an upward occlusal cant of the maxilla on the ipsilateral side. The upward cant is likewise reflected in the ipsilateral piriform aperture. The zygoma, orbit, and frontotemporal bones can also be affected in shape and volume. The affected zygoma can be hypoplastic and retruded. Orbital dystopia and anterior (frontal) plagiocephaly are observed in more severe cases.

Soft Tissue

The skin over the affected skeleton is often thin with a bluish hue, and the underlying fat is reduced in volume. The distance between the oral commissure and the ear remnant is reduced; the oral commissure is elevated on the affected side (Fig. 4).

Meurman has designated a classification of the auricular deformity (Fig. 5):

Grade I. Normal shape of the auricle with all components present but deficient

Grade II. Only a vertical remnant of cartilage and skin

Grade III. Only a residual lobule

Neuromuscular deficits can likewise be present. Branches of the facial (VII) nerve, in particular the marginal mandibular branch, may be nonfunctional. The muscles of mastication, especially the masseter, pterygoids, and temporalis, are reduced in volume.

The principal and associated anomalies of craniofacial microsomia are outlined in Table 1.

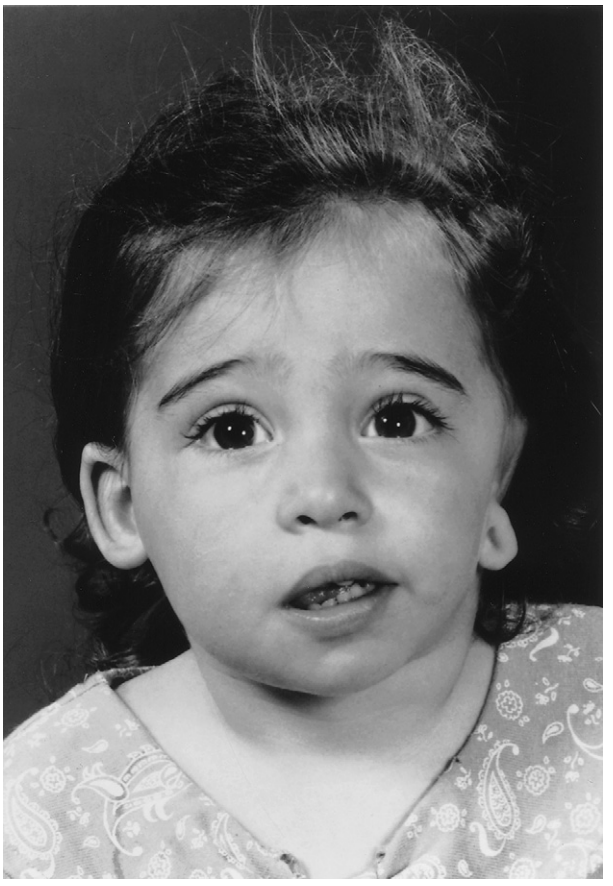

Figure 4 • Patient with left-sided craniofacial microsomia demonstrating the characteristic upward occlusal cant on the affected side with associated cheek hypoplasia and ear anomaly. (From Mathes S, Hentz, R [eds]: Plastic Surgery. Philadelphia, Elsevier, in press.)

Classification

The modified Pruzansky classification of mandibular pathology has been universally accepted (see Fig. 3). The OMENS clinical classification (Table 2) was designed to integrate the **o**rbital, **m**andibular, **e**ar, **n**erve, and **s**oft tissue defects and to detail a more global classification of the deformity.

Goals of Reconstruction

A patient with craniofacial microsomia poses numerous challenges to a plastic surgeon. A multidisciplinary approach using a wide variety of treatment modalities is essential to address the functional and aesthetic problems. Reconstruction requires the surgeon to use the full panoply of plastic surgery techniques with the collaboration of other members of the craniofacial team.

The goals of reconstruction can be segregated into those addressing function and those addressing form.

Function

- Restore airway and alimentary function
- Maximize hearing for proper speech development
- Restore corneal protection
- Correct malocclusion

Form

- Correct deficiency and asymmetry of the mandible and maxilla; level the occlusal plane and centralize the chin point to the midline
- Restore frontotemporozygomatic contour
- Restore cheek soft tissue deficits

TABLE 1 Primary and Associated Anomalies of Craniofacial Microsomia

PRINCIPAL ANOMALIES	ASSOCIATED ANOMALIES	OTHER ANOMALIES
Mandibular	Craniofacial	
Mandibular hypoplasia (89%-100%)	Velopharyngeal insufficiency (35%-55%)	Vertebral/rib defects (16%-60%)
Malformed glenoid fossa (24%-27%)	Palatal deviation (39%-50%)	Cervical spine anomalies (24%-42%)
Ear	Orbital dystopia (15%-43%)	Scoliosis (11%-26%)
Microtia (66%-99%)	Ocular motility disorders (19%-22%)	Cardiac anomalies (4%-33%)
Preauricular tags (34%-61%)	Epibulbar dermoids (4%-35%)	Pigmentation changes (13%-14%)
Conductive hearing loss (55%-66%)	Cranial base anomalies (9%-30%)	Extremity defects (3%-21%)
Middle ear (ossicle) defects	Cleft lip and/or palate (15%-22%)	Central nervous system defects (5%-18%)
Midfacial	Eyelid defects (12%-25%)	Genitourinary defects (4%-15%)
Maxillary hypoplasia	Hypodontia/dental hypoplasia (8%-25%)	Pulmonary anomalies (1%-15%)
Zygomatic hypoplasia	Lacrimal drainage abnormalities (11%-14%)	Gastrointestinal defects (2%-12%)
Occlusal canting	Frontal plagiocephaly (10%-12%)	
Soft tissue	Sensorineural hearing loss (6%-16%)	
Masticatory muscle hypoplasia (85%-95%)	Preauricular sinus (6%-9%)	
Macrostomia (17%-62%)	Parotid gland hypoplasia	
Nerve VII palsy (10%-45%)	Other cranial nerve defects (e.g., V, IX, XII)	

The prevalence rates were summarized from 19 reports in the literature from 1983 to 1996. Studies based on selected samples were omitted to minimize selection bias. It was recognized by the authors that the prevalence rate may be falsely elevated because the reporting tertiary centers may have a referral bias of more severely affected patients.

Adapted from Cousley RR, Calvert ML: Current concepts in the understanding and management of hemifacial microsomia. Br J Plast Surg 50:536-551, 1997.

TABLE 2 OMENS Classification

ORBIT

O_0	Normal orbit size and position
O_1	Abnormal size
O_2	Abnormal position
O_3	Abnormal size and position

MANDIBLE

M_0	Normal mandible
M_1	Mandible and glenoid fossa are small ("minimandible")
M_2	Mandibular ramus short and abnormally shaped
M_{2A}	Glenoid in acceptable position
M_{2B}	Temporomandibular joint medially displaced
M_3	Complete absence of ramus, glenoid fossa, and temporomandibular joint

EAR

E_0	Normal
E_1	Mild hypoplasia and cupping
E_2	Absence of external auditory canal
E_3	Malpositioned lobule with absent auricle

NERVE

N_0^7	No facial nerve involvement
N_1^7	Upper facial nerve involvement
N_2^7	Lower facial nerve involvement
N_3^7	All branches affected

SOFT TISSUE

S_0	No soft tissue deformity
S_1	Minimal (mild) tissue deformity
S_2	Moderate tissue deformity (between the two extremes)
S_3	Major (severe) subcutaneous and muscular deficiency

Adapted from Vento AR, LaBrie RA, Mulliken JB: The O.M.E.N.S. classification of hemifacial microsomia. Cleft Palate Craniofac J 28:68-76, discussion 77, 1991.

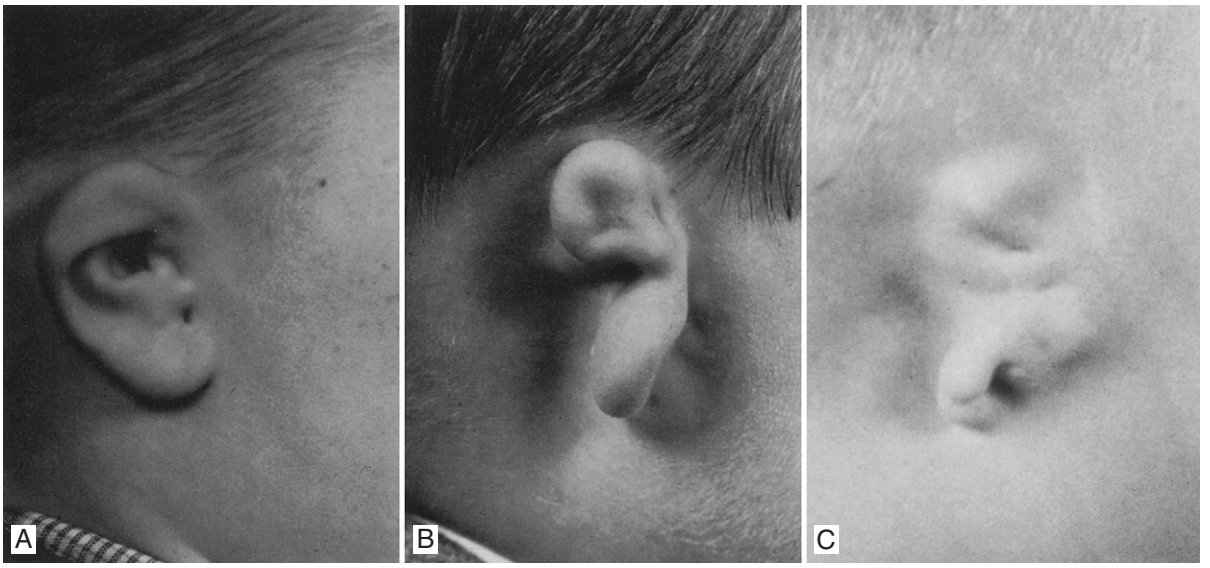

Figure 5 • Example of the three grades of auricular anomalies in the Meurman classification. **A,** Grade I: small malformed ear with most components present. **B,** Grade II: vertical remnant of skin and cartilage. There is atresia of the external auditory meatus. **C,** Grade III: the auricle is almost entirely absent except for a misplaced lobule and diminutive skin and cartilage remnants. (From Mathes S, Hentz, R [eds]: Plastic Surgery. Philadelphia, Elsevier, in press.)

Address facial nerve deficits
Reconstruct the external ear

Preoperative Evaluation

Physical examination documented by photographs is critical to establish a baseline record. Symmetry/asymmetry in the horizontal plane at the level of orbits, external nares, and oral commissures must be documented, as well as the distance between the oral commissure and ear remnant. The auricle must be graded according to the classification of Meurman (see Fig. 5). There may be ear tags or branchial sinuses associated with the auricular deformities. The soft tissues of the cheek must also be evaluated for thickness and contour. In addition, it is important to assess neuromuscular function, including the individual facial nerve branches.

Intraoral examination includes evaluation of the status of the dentition, the occlusion, and the inclination of the occlusal plane. The temporomandibular joint is assessed by recording the maximum distance on full opening, as well as mandibular protrusion and lateral deviation. The eyes should be examined for the presence of ocular dermoids and colobomas of the eyelids.

Diagnostic Studies

Photography is important to document baseline anatomy and to monitor progress during treatment. Frontal, lateral, three-quarter, submental vertex, bird's eye, and occlusal views should be included. Cephalograms also document the initial skeletal deformities and aid in surgical planning and goals. Other studies include Panorex, computed tomography (CT) scans (Fig. 6), and cervical spine films. Patients should also have renal sonograms and additional studies, including genetic analysis, as needed.

Treatment

Orthodontic Therapy

Orthodontic therapy is critical during the entire period of treatment and in preparation for mandibular distraction osteogenesis. The orthodontist often has to set abnormally inclined teeth upright, establish optimal intermaxillary arch width relationships, level the occlusal planes, and guide eruption so that the active phase of distraction results in optimal mandibular position. The rationale for preorthognathic surgical and predistraction orthodontic therapy is similar.

In preparation for mandibular distraction, the orthodontist must establish that a sufficient number of nonmobile deciduous or permanent teeth (or both) are present to serve in the construction of a distraction stabilization appliance.

PREDISTRACTION ORTHODONTIC THERAPY. Predistraction orthodontic removal of dental compensations (i.e., bringing teeth over the basal bone) is

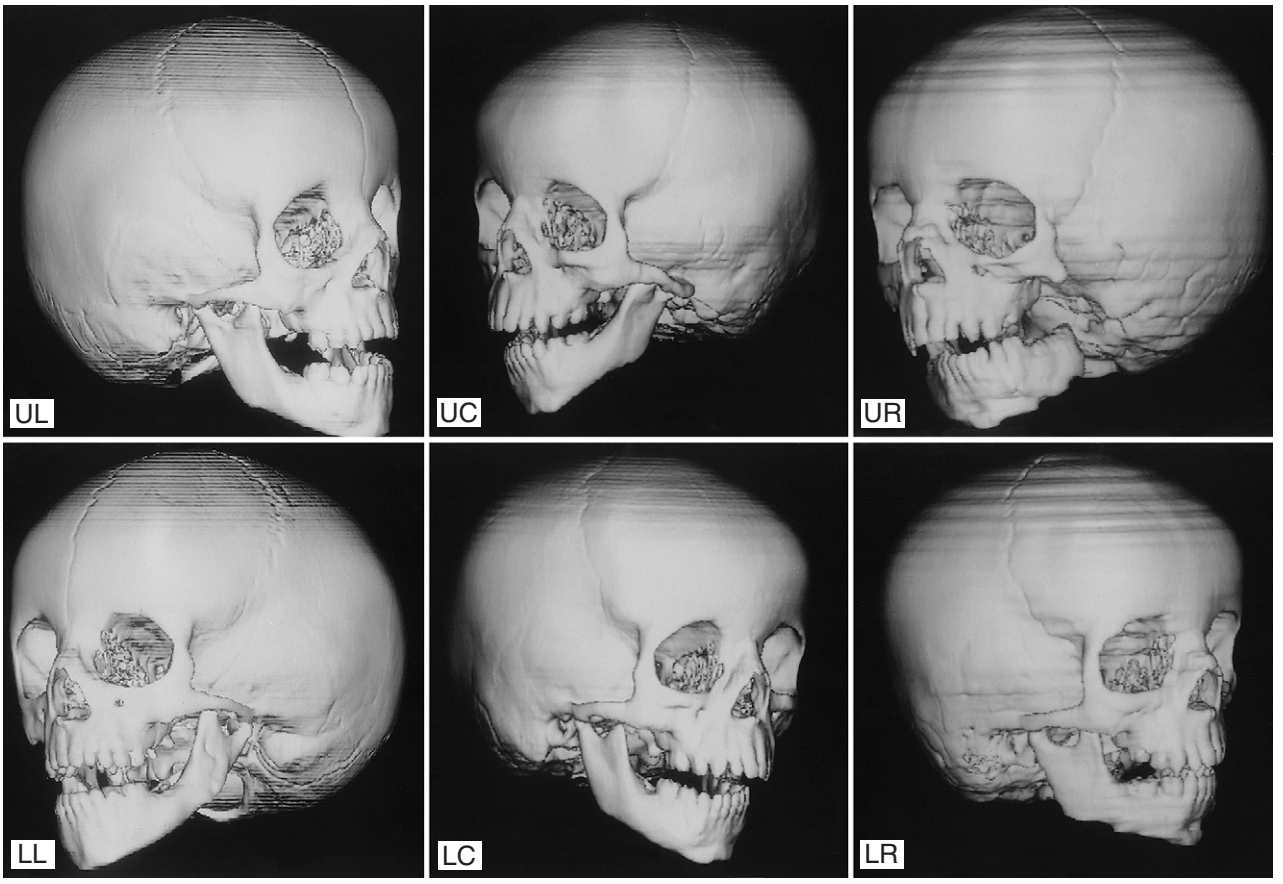

Figure 6 • Three-dimensional computed tomographic scans of three patients with unilateral craniofacial microsomia. Severity increases from left to right. The affected side of each patient is on the *top panel*, with the corresponding normal contralateral side on the *lower panel*. The three patients correspond to the Pruzansky classification of mandibular deformity described in the chapter. Type I, *left*; type II, *center*; Type III, *right*. (From Mathes S, Hentz, R [eds]: Plastic Surgery. Philadelphia, Elsevier, in press.)

carried out so that the final position of the mandible is not compromised by abnormal position of the teeth.

Orthodontic leveling of the occlusal planes is performed so that premature contact of individual teeth does not prevent achievement of optimal jaw relationships during distraction.

Orthodontic coordination of the maxillary and mandibular arch width before or during distraction is necessary to establish correct mandibular position.

A "distraction stabilization appliance" that will be used to engage heavy guiding elastics is constructed before surgery. The elastics are used to aid in control of vectors during the active phase of distraction. The appliance, which consists of a maxillary expansion device and a mandibular lingual arch, is designed to buttress the dental arches against intermaxillary, oblique elastics that would otherwise move individual teeth and distort dental arch symmetry.

INTRADISTRACTION ORTHODONTIC THERAPY. Elastic traction is applied during distraction to modify the device vectors and thereby optimize both the dental and skeletal correction. Transverse (cross tongue) elastics prevent severe lateral shifting of the dental arches. By maintaining the mandibular dental arch centered on the midsagittal plane, an increased amount of vertical ramus lengthening, mandibular advancement, and bite opening is produced on the distracted side.

POSTDISTRACTION ORTHODONTIC THERAPY. Closure of a posterior open bite is achieved by guiding the maxillary teeth down to the mandibular occlusal plane. This can be accomplished in deciduous and mixed dentition through the use of bite blocks and intermaxillary elastic therapy. In permanent dentition, the potential for vertical elongation of the dentoalveolar process is diminished, and therefore a concurrent maxillomandibular distraction (see Fig. 10) or subsequent LeFort I osteotomy is sometimes required.

Mandibular Surgery

In recent years, distraction techniques have revolutionized lengthening and augmentation of a deficient mandible (Fig. 7). The treatment protocol is as

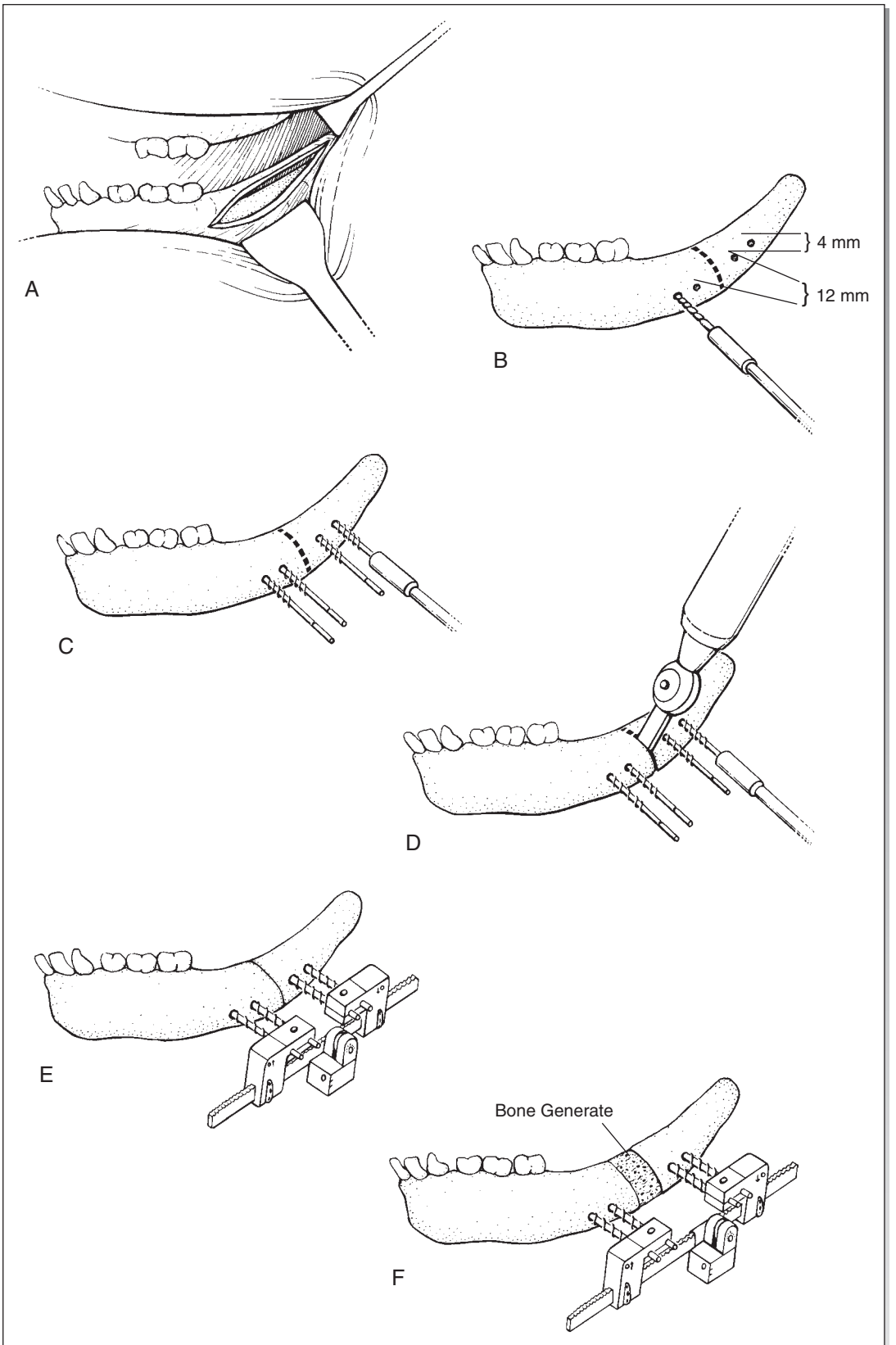

Figure 7 • Technique of extraoral distraction. **A,** Intraoral incision. **B,** Line of osteotomy and sites of insertion of self-drilling pins. **C,** Partial osteotomy and insertion of a fourth pin. **D,** Completion of the osteotomy. **E,** Application of device. **F,** Bone generation after activation of the device. (From Mathes S, Hentz, R [eds]: Plastic Surgery. Philadelphia, Elsevier, in press.)

follows. An osteotomy is made in a non–tooth-bearing segment of the deficient mandible and a distraction device is applied. The orientation of the distraction device establishes the vector of distraction: *vertical* (perpendicular to the maxillary occlusal plane), *horizontal* (horizontal to the maxillary plane), and *oblique* (intermediate orientation). After a *latency period* of 5 days, the device is activated at the rate of a millimeter a day *(activation period)*.

In unilateral cases, activation is continued until the chin point is returned to the craniofacial midline or slightly to the contralateral side, the ipsilateral oral commissure is lowered, and the mandibular occlusal cant is corrected. A posterior open bite usually results and is managed orthodontically through the use of bite blocks and guide elastics.

In bilateral cases, the endpoint is a symmetric edge-to-edge anterior or class III occlusion in a growing child.

After completion of the activation phase, the mandible is maintained in fixation for an additional 2 months *(consolidation period)*. During the activation and early consolidation periods, the newly generated bone can be "molded" with intermaxillary elastic and device forces to optimize skeletal form and occlusal relationships.

In a patient with a Pruzansky III mandible (absent ramus and condyle), the ramus is reconstructed with several costochondral rib grafts (the cartilaginous portion serving as the condyle). Rigid fixation is established at the junction with the mandibular body, and the patient is placed in intermaxillary fixation (Fig. 8). If necessary, distraction can be subsequently performed on the consolidated rib grafts.

In a skeletally mature patient (female approximately older than 16 years, male older than 18 years), bilateral sagittal split osteotomy of the ramus allows appropriate advancement and three-dimensional rotation of the tooth-bearing segment (Fig. 9). However, a fairly well developed ramus and body are required for successful completion of this osteotomy. It is usually complemented with a LeFort I osteotomy and genioplasty.

Maxillary Surgery

The maxilla can be distracted in a patient during the phase of mixed dentition by establishing intermaxillary fixation with an osteotomized mandible undergoing mandibular distraction. A LeFort I osteotomy/corticotomy allows concomitant distraction of the ipsilateral maxilla (maxillomandibular distraction) (Fig. 10).

In a skeletally mature patient, the LeFort I osteotomy can be performed and combined with bilateral sagittal split osteotomy of the mandible to correct the occlusal cant, move the chin point to the midline, and lower the ipsilateral oral commissure (see Fig. 9).

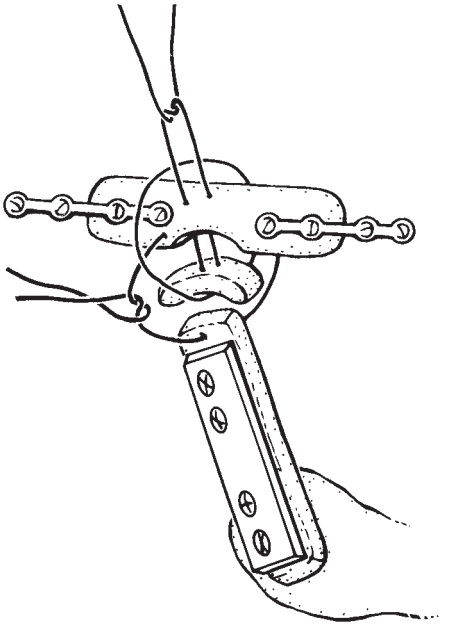

Figure 8 • Lateral view illustrating the details of the ramus/condyle and glenoid fossa reconstruction. Note that a cartilage cap is interposed between the bone grafts reconstructing the ramus/condyle and glenoid fossa (undersurface of the bone graft—reconstructed zygomatic arch). A resorbable suture approximates the latter. Note the rigid fixation to the mandibular remnant. (From Mathes S, Hentz, R [eds]: Plastic Surgery. Philadelphia, Elsevier, in press.)

Chin Surgery

Genioplasty or horizontal osteotomy of the anteroinferior border of the mandible allows three-dimensional rotation/advancement of the chin (see Fig. 9); bone grafts can be placed in the resultant defects.

Surgery on the Frontotemporozygomatic Region

Occasionally, fronto-orbital advancement with cranial vault remodeling is indicated in patients with associated retrusion of the supraorbital rim and frontal bone (see the chapter "The Craniosynostoses").

Soft Tissue Surgery

A microvascular free flap is the preferred method of augmenting the hypoplastic soft tissue of the cheek and temporal region. Although dermis-fat grafts (nonvascularized) have been advocated, their long-term survival is unpredictable, and there can be resulting contour irregularities with resorption and contraction of the grafts.

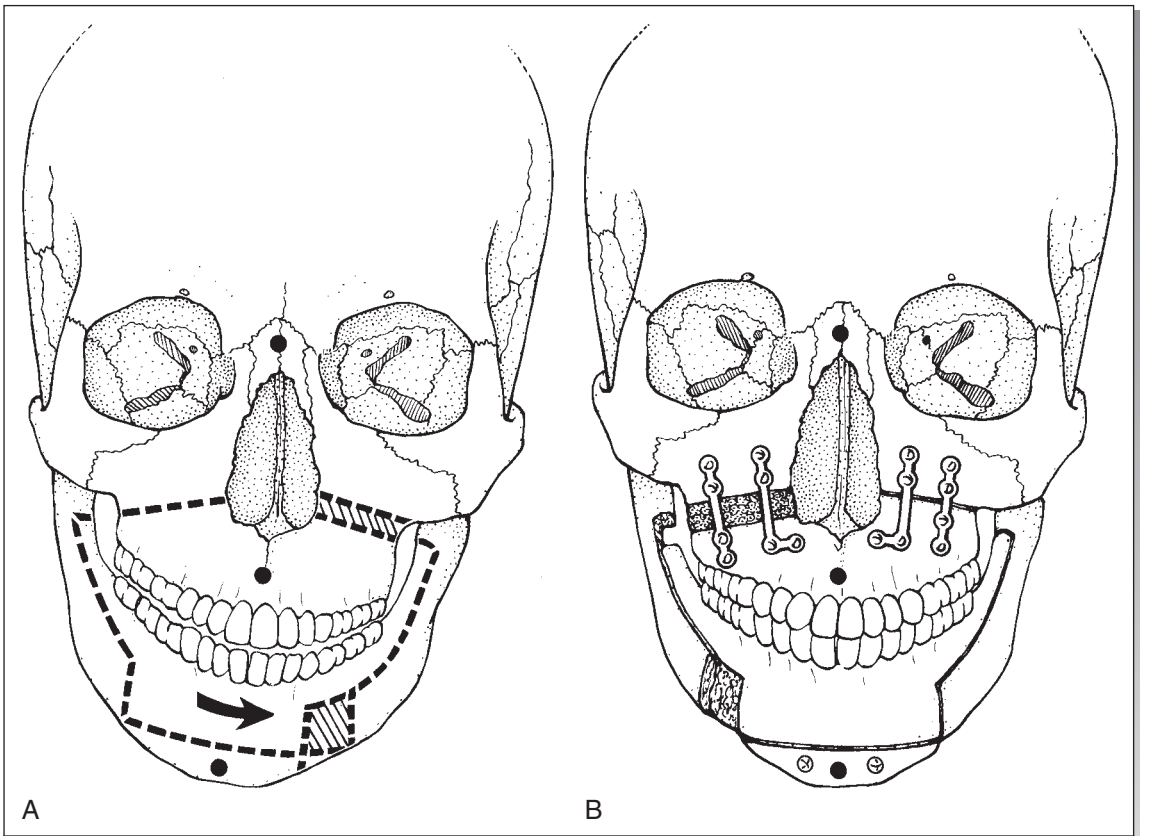

Figure 9 • Combined bilateral sagittal split osteotomy of the mandible, LeFort I osteotomy, and genioplasty. **A,** Lines of osteotomy. The *dark dots* illustrate the deviation of the craniofacial midline, and the *arrow* designates the direction of movement of the mandible (and maxilla). The *shaded areas* represent the portions of the maxilla and the buccal cortex of the mandible to be resected. Note the area of resection and impaction of the maxilla on the same side. **B,** After bone grafting of the maxillary defect and rigid fixation of the LeFort I segment. Lag screws are used on the mandibular rami and genioplasty segment. Note the change in the *dark dashes*. (From Mathes S, Hentz, R [eds]: Plastic Surgery. Philadelphia, Elsevier, in press.)

Treatment Algorithm

Neonatal Period/Infancy

In an infant with craniofacial microsomia (birth through 18 months), the respiratory function of the child must be assessed. With severe mandibular deficiency and associated glossoptosis, the nasal and oral pharyngeal spaces are severely constricted. A pediatric otolaryngologist is invaluable in documenting the respiratory status of the patient by physical and endoscopic examination; CT scans and sleep studies complement this evaluation.

In infants with respiratory insufficiency, the airway problem must be addressed first, traditionally by tracheostomy. More recently, mandibular distraction has obviated the need for tracheostomy in many patients, but endotracheal intubation must be maintained during a portion of the treatment period. In a patient with a previous tracheostomy, distraction can permit earlier decannulation than in the past.

Infants with sleep apnea often have associated feeding problems. Gavage feeding can be provided initially, but if the feeding problem continues, gastrostomy should be considered. Feeding problems, including gastroesophageal reflux, are usually corrected only with mandibular advancement or growth of the mandible.

Fronto-orbital advancement/cranial vault remodeling is occasionally indicated in a small percentage of infants with fronto-orbital retrusion. Such surgery should be delayed until the child is at least 12 months of age.

Repair of an associated cleft of the hard or soft palate (or both) should be performed at approximately 12 months to facilitate speech development. This is also the optimal time to repair macrostomia or a lateral facial cleft.

Ear tags or cartilaginous remnants in the cheeks can be removed within the first year.

Early Childhood

During the period of early childhood (18 months through 3 years), reconstruction can be undertaken in a child with respiratory insufficiency or moderate to severe skeletal deficiency.

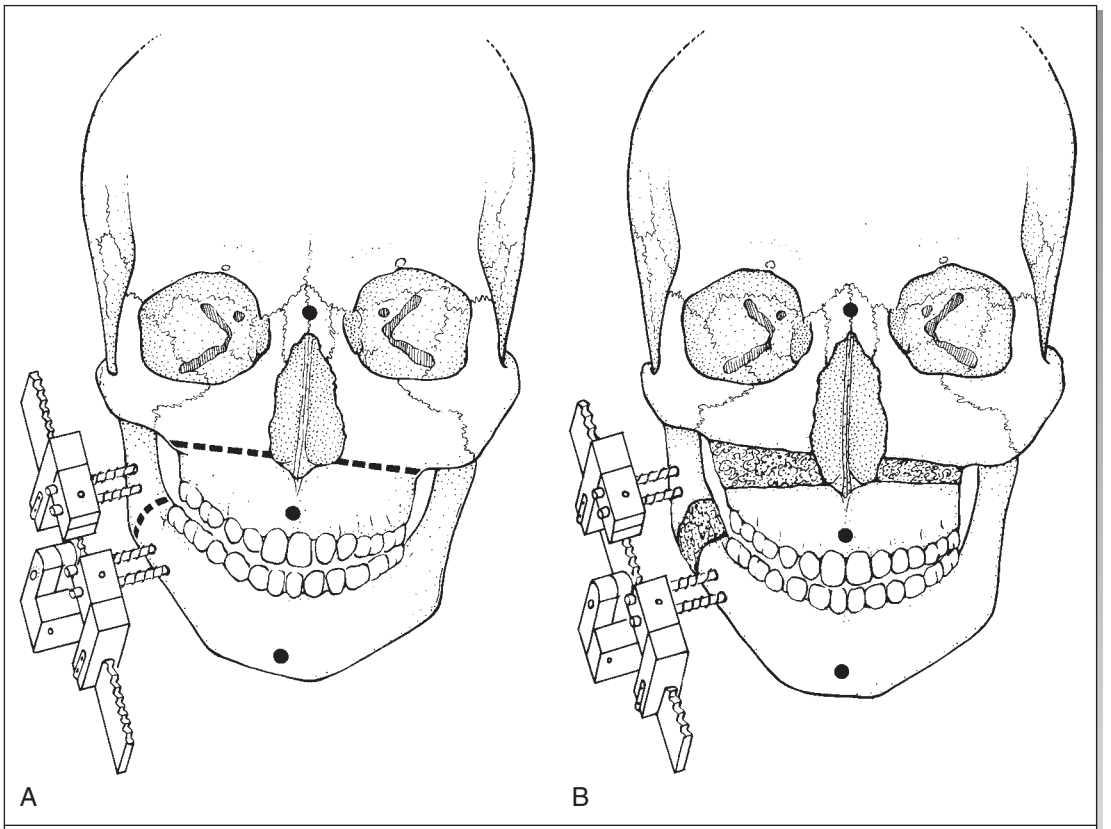

Figure 10 • Technique of maxillomandibular distraction. **A,** The *dots* designate the asymmetry of the craniofacial midline. Note the LeFort I and mandibular lines of osteotomy. A mandibular distraction device (four pins) has been applied. **B,** After activation of the device. The intermaxillary wires (or elastics) are not illustrated. Note the restoration of the craniofacial midline (*dashes*), improvement in the occlusal plane and chin position, and rotation/augmentation of the maxilla and mandible. (From Mathes S, Hentz, R [eds]: Plastic Surgery. Philadelphia, Elsevier, in press.)

In a child with a Pruzansky III mandible (absent ramus and condyle), autogenous rib or iliac bone grafting of the hypoplastic mandible should be undertaken. Once revascularized, the bone can be distracted for further lengthening. This technique results in both an improved airway and facial appearance. It also facilitates orthodontic work by establishing a level occlusal plane and optimal masticatory function.

In a patient with a class IIA or class IIB mandible, distraction could be undertaken. In a unilateral case, activation of the distraction device should continue until the occlusal plane has been leveled (or overcorrected), the oral commissure has been lowered, and sufficient anterior thrust of the mandible and bilateral symmetry of the mandibular angles have been achieved.

Soft tissue augmentation, definitive orthodontic therapy, and auricular reconstruction should be deferred to a later date.

Childhood

Childhood (4 through 13 years) is the age of active growth and, for much of the time, is defined as the period of mixed dentition.

Primary mandibular distraction could be undertaken during this period if there is evidence of a significant occlusal cant, disparity in the oral commissures, and chin asymmetry or retrusion (or both).

Experience with mandibular distraction in patients younger than 3 years shows that there is usually spontaneous descent of the associated maxillary dentoalveolus with vertical elongation of the ramus, provided that the mandibular plane is maintained in its corrected portion through the use of a bite plate. In older patients, the maxilla can be managed in two ways. In the first program, mandibular distraction can be accomplished and an orthodontic bite block constructed at the time of device removal to fill the resulting posterior void between the maxillary and mandibular dentition. Subsequently, the bite block is gradually reduced to allow serial descent of the maxillary dentoalveolus. Alternatively, combined maxillomandibular distraction can be performed. A LeFort I corticotomy is performed in conjunction with the application of a mandibular osteotomy/distraction device and maxillomandibular distraction. With activation of the device, there is associated distraction of the maxilla with leveling of the occlusal plane (see Fig. 10).

It is during the childhood years that auricular reconstruction is undertaken and microvascular free flap augmentation of the cheek soft tissues can be considered.

Adolescence/Adulthood

Adolescence/adulthood is the period when craniofacial growth and development are completed, usually at a minimum of 16 years in females and 18 years in males. Treatment planning at this stage must also take into consideration the variable of a minor amount of posttreatment craniofacial growth and development.

Traditional orthognathic surgery at this point allows reconstruction of the craniofacial skeleton and occlusion in a single surgical procedure. The combined surgical procedure of LeFort I osteotomy, bilateral sagittal split osteotomy of the mandible, and genioplasty (see Fig. 9) has proved to be the therapeutic workhorse in this situation.

In recent years, with the refinement of distraction devices and techniques, the role of maxillomandibular distraction in a mature patient has increased. For example, a LeFort I osteotomy can be performed with three-dimensional repositioning of the maxilla and rigid skeletal fixation. Intraoral mandibular distraction can then be performed and the mandible and its occlusion "docked" into the repositioned maxilla with optimal occlusion. The distraction technique also improves soft tissue contour and is associated with a lower rate of relapse. The development of the concept of *molding of the regenerate* by a combination of multiplanar distraction devices and skeletally and dentally anchored intermaxillary elastics has ensured the achievement of optimal occlusion and skeletal morphology.

Genioplasty alone is the only skeletal reconstruction required in patients with a mild deformity characterized by functional occlusion and only chin retrusion and asymmetry. Three-dimensional genioplasty can provide anterior advancement and three-dimensional repositioning to correct chin asymmetry.

Pearls and Pitfalls

- Multidisciplinary team care is essential.
- In neonates and infants, address respiratory/sleep apnea and nutritional/feeding problems.
- Longitudinal follow-up is critical, especially until craniofacial growth is complete.
- Avoid bone carpentry alone; soft tissue and dental considerations cannot be ignored.
- Distraction has provided a more predictable reconstructive alternative when severe maxillomandibular asymmetry is associated with marked deficiency of soft tissue and bone.
- Soft tissue augmentation of the deficient cheek is an important treatment component.

SUGGESTED READINGS

McCarthy JG, Grayson BH: Craniofacial microsomia: Reconstruction. In Mathes S, Hentz, R (eds): Plastic Surgery. Philadelphia, Elsevier, 2005.

McCarthy JG, Schreiber JS, Karp NS, et al: Lengthening of the human mandible by gradual distraction. Plast Reconstr Surg 89:1, 1992.

Obwegeser HL: Correction of the skeletal anomalies of otomandibular dysostosis. J Maxillofac Surg 2:73, 1974.

Pruzansky S: Not all dwarfed mandibles are alike. Birth Defects 1:120, 1969.

Siebert JW, Longaker MT: Microsurgical correction of facial asymmetry in hemifacial microsomia. Op Tech Plast Reconstr Surg 1:93, 1994.

Giant Congenital Nevi

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The term "giant congenital nevus" generally refers to a melanocytic cutaneous lesion present at birth, with an anticipated diameter of 20 cm or larger in a mature adult. The malignant potential of the lesions justifies their removal. The challenge of the lesions lies not in their removal but in the functional and aesthetic outcome of their reconstruction.

This chapter reviews the incidence, natural history, and pathology of giant melanocytic nevi, discusses the benefits and drawbacks of various reconstruction methods, and presents a rational approach to the management of these patients that involves balancing the need for excision against aesthetic and functional outcomes.

Etiopathogenesis

Congenital melanocytic nevi (CMNs) are ectopic rests of nevus cells. Melanoblasts, the precursor cells of melanocytes, migrate from the neural crest to the skin, mucosa, eyes, mesentery, meninges, and chromaffin system, where they differentiate into dendritic melanocytes. Wherever a disturbance in this migration occurs, the ectopic population of nevus cells results in a hamartoma, or a melanocytic nevus. The histologic finding of nevus cells along the skin appendages or blood vessels is specific for CMNs, but not all lesions demonstrate this finding. Because the nevus cells extend beyond the dermis into the depths of the subcutaneous layer and even into the fascia, adequate margins for resection include the soft tissues beneath the lesion as far as the fascial level.

CMNs are divided into three groups: small, intermediate, and large/giant. *Small* lesions measure less than 1.5 cm in diameter, are present in 1 in 100 births, and can generally be removed in a single excision with direct closure. The *intermediate* group measures between 1.5 and 20 cm, occurs in 6 in 1000 births, and often requires the same management techniques as for giant nevi. *Giant* nevi are larger than 20 cm or cover greater than 2% of the body surface area and are present in 1 in 20,000 births.

The risk for malignant degeneration is uncertain and rates are reported to range from 2% to 31%, thus providing little help to the surgeon in managing these cases. Quaba and Wallace reviewed a large series of patients with lesions covering greater than 2% of body surface area and found the risk for malignancy to be 8.5% during the first 15 years of life. Approximately 50% of malignancies that occur are observed by 3 years of age, 60% by childhood, and 70% by puberty.

CMNs positioned over the axial skeleton can also be a marker for neurocutaneous melanosis, a syndrome that manifests as developmental delay, seizures, and hydrocephalus. Asymptomatic leptomeningeal and central nervous system involvement can be identified by T1 shortening on magnetic resonance imaging (MRI). Asymptomatic leptomeningeal melanosis may portend a risk for the later development of either benign or malignant lesions, but long-term documentation of these findings is not yet available.

Diagnostic Studies

Raised and ulcerated areas within a giant nevus merit biopsy to exclude malignant changes. Children with large nevi over the midline scalp and back should undergo MRI to document the presence or absence of leptomeningeal melanosis. Once documented as either positive or negative, serial studies are not indicated in the absence of associated symptoms.

Reconstructive Goals

The treatment goals for patients with giant congenital nevi are seemingly straightforward—excise as much of the nevus as possible without sacrificing vital structures, and resurface the areas with tissue of like color, texture, and composition with the least amount of donor site morbidity. These goals, however, can be exceedingly difficult to achieve. Giant CMNs are not amenable to serial excision, although some of the intermediate lesions may be. Many children who have undergone extensive resections with split-thickness skin autograft resurfacing have poor aesthetic results and functional difficulties related to hypertrophic scarring and contracture.

The suboptimal results have led physicians to try other methods of treatment. Curettage, especially in the neonatal period, initially produces impressive results, but the coloration and texture changes recur and confound the ability to observe the lesion for malignant changes. Although this technique has been advocated for tumor load reduction, it also eventually requires further reconstruction. Selective photolysis with lasers is likewise unsuccessful. Lasers lighten the color of the lesion temporarily until the melanin content returns, but they do not remove the underlying premalignant nevus cells.

The preferred procedures available for reconstruction remain skin grafting, local flaps, pedicle flaps, tissue expansion, and free tissue transfer. Application of these techniques needs to be adjusted to the pediatric patient and to the anatomic area being resurfaced. Reconstruction of the defects after resection of giant congenital nevi can be addressed with an algorithm for each anatomic area: head and neck, trunk, buttocks and perineum, and extremities. The following suggestions are made after reviewing the senior author's (B.S.B.) series of 245 patients treated from 1979 to 2001.

The most useful modality has proved to be tissue expansion with flaps of similar tissue to provide excellent color and texture match and nearly normal contour match. Even when regional flaps alone are not available, expansion of full-thickness skin graft donor sites, expansion of flaps for free tissue transfer, and expanded pedicled flaps provide new alternative methods for treatment of these complex problems.

Treatment by Anatomic Area

Scalp

Tissue expansion is the mainstay of treatment of giant nevi of the scalp. Increasing experience has demonstrated that improved flap design significantly limits the need for or number of serial expansions. In particular, the use of large expanded transposition flaps allows for better restoration of natural hair patterns and hairline reconstruction. Transposition of the expanded posterior parietal/occipital region of the scalp is particularly effective in coverage of the hemiscalp and temporal hairline.

The incisions for expander placement are made within the borders of the nevus to be excised, and dissection is extended in the subgaleal plane. Expansion commences 1 to 2 weeks postoperatively and continues until adequate tissue is recruited, usually 10 to 12 weeks. Expansion in the scalp, as at other sites, begins at a frequency of once per week, but it may be increased to every fourth to fifth day to safely complete expansion within the projected 10 to 12 weeks. When additional time is required, the date for expander removal is modified. Scalp expansion can begin at 6 months of age without permanent skull remodeling. When a flap requires serial expansion, the second expander should be placed after a delay of 3 to 4 months.

Face

Facial excision and reconstruction follow the aesthetic facial units. Whenever possible, expansion is preferred for resurfacing the forehead, cheeks, nose, and neck. The periorbital and nasal areas are grafted as a unit with a full-thickness graft harvested from an expanded supraclavicular donor site. A single full-thickness graft can be harvested to cover the entire upper and lower eyelids, as well as the periorbital and nasal defect, thereby minimizing the number of scars resulting from multiple grafts patched together from different donor sites. When planning expansion and reconstruction of the forehead, consideration must be given to minimizing distortion of the brow-hairline relationship. Nevi extending into the temporal area require expansion of both facial skin and hair-bearing scalp to establish normal position and hair direction in the temporal region. Expansion of a large rotation or transposition flap from the neck and postauricular region provides optimal coverage of the aesthetic unit of the cheek, with a significantly lower risk for postoperative ectropion and canthal distortion, in contrast to an expanded advancement flap from the same region. Prefabricated flaps based on the superficial temporal artery can also be used in the facial area when the neck and postauricular skin is likewise involved with nevus.

Trunk

Significant advances have been made in the application of tissue expansion to giant nevi of the trunk. Improved expanded flap design and the use of free tissue transfer have virtually eliminated trunk grafting and at the same time improved the functional and aesthetic outcome.

The use of large expanded transposition flaps from the mid/upper back and upper thigh/lower buttock region, with subsequent reexpansion of these flaps and further advancement or transposition, allows coverage of the entire lower back and buttock area without the need for skin grafts. In the shoulder and neck region, transverse rectus abdominis myocutaneous (TRAM) flaps can be placed by microvascular transfer, like a shawl, with excellent match of color, thickness, adnexal structures, and contour. To allow primary closure of the donor site, the skin below the planned TRAM flap is expanded for 10 to 12 weeks.

Anterior trunk lesions are treated with abdominoplasty-like operations, with the planned flap and groin tissue expanded if necessary to obtain primary closure. Large and giant nevi of the breast (in females) are not removed until breast development has progressed to the point that excision of the nevus will not risk injury to the breast bud. Close examination at prescribed intervals with biopsy of suspect areas should be performed.

Extremities

Management of large nevi of the extremities, especially circumferential lesions, remains challenging because of the difficulties inherent in using expansion in these areas. Although previously recommended, expanded full-thickness skin grafts for the extremities result in late contour defects and skin graft pigmentation abnormalities. In the proximal end of the upper limbs, expanded flaps from the shoulder and upper part of the back are recommended to provide the extra skin to avoid constriction of the circumference of the extremity. Likewise, expanded groin and lower abdominal and lower back flaps can be used for the proximal end of the thigh. Free tissue transfer of the expanded TRAM flap can provide coverage for the combined upper arm and shoulder region, whereas expanded pedicled flaps may provide excellent contour and color match for the upper extremity in the area extending from the elbow to the wrist. Expanded full-thickness grafts provide excellent coverage of the hand and fingers.

Large, but not giant, nevi of the lower extremity may be excised with serial expansion and advancement. However, as in the upper extremity, movement of expanded regional flaps (and consequently the overall tissue gain) is limited by the geometry of the extremity. The principles outlined earlier, including the use of free tissue transfer of expanded flaps for coverage after excision of extensive circumferential nevi of the lower extremity from the knee to the ankle, have yielded encouraging early results, thus indicating once again that expansion may play a role in providing better contour, color, and durable skin in situations in which split-thickness skin grafts have historically produced disappointing results.

Postoperative Care

Preparing families for tissue expansion is critical to the success of the procedure. Before surgery, families attend a teaching session at which photographs of patients undergoing expansion are demonstrated. They also have a chance to see the expanders, drains, and bandaging materials and to ask questions. Patients are generally kept in the hospital overnight for pain management and for teaching suture line care and drain management. Perioperative antibiotics, usually cefazolin, are administered. Drains are left in place until scant drainage is noted after both expander placement and flap advancement. Expansion is commenced 1 to 2 weeks after insertion. Families are given the option to perform expansion at home or to return to the office weekly. EMLA cream is used to diminish the pain associated with expansion. Preoperative teaching and careful case-by-case planning are essential for parents and patients to work through the postoperative period with the greatest ease.

Complications associated with skin expansion include infection, device failure/exposure, and inadequate flap movement. If early signs of infection are treated aggressively with resumption of antibiotics and if broad-spectrum antibiotic coverage is resumed when a child shows obvious signs of another infectious source (upper respiratory infection, ear infection, etc.), expander removal may be avoided or at least delayed. If control is not possible, the expander is removed and any available expanded skin is used to complete part of the excision. When expanders are infected but not exposed, expansion can be continued judiciously and the patient electively taken to surgery for flap advancement, rotation, or transposition in the recognition that once the expander is removed, the infection will resolve. Although at times and in special cases flaps are pushed to their limits, with acceptance of the possibility of a small area of tip necrosis, effort should be made to not “overreach” in flap movement and acknowledge the possible need for repeat expansion. These possibilities should be discussed when outlining the initial treatment plan. If flap complications occur after inset, conservative dressing changes and office débridement are usually all that is required.

Pearls and Pitfalls

- Risk of malignancy is uncertain but decreases with age.
- Nonsurgical treatment with curettage and lasers does not address the underlying malignant potential and makes screening for malignancy difficult.

- Expansion of local tissue with similar color and texture match gives the best results.
- Distal extremity lesions are well suited to expanded free tissue transfers.

SUGGESTED READINGS

Bauer BS, Few JW, Chavez CD, Galiano RD: The role of tissue expansion in the management of large congenital pigmented nevi of the forehead in the pediatric patient. Plast Reconstr Surg 107:668, 2001.

Elder D, Elenitsas R: Benign pigmented lesions and malignant melanoma. In Elder D (ed): Lever's Histopathology of the Skin, 8th ed. Philadelphia, Lippincott-Raven, 1997.

Foster RD, Williams ML, Barcovich AJ, et al: Giant congenital melanocytic nevi: The significance of neurocutaneous melanosis in neurologically asymptomatic children. Plast Reconstr Surg 107:933, 2001.

Magalon G, Casanova D, Bardo J, Andrac-Meyer L: Early curettage of giant congenital nevi in children. Br J Dermatol 138: 341, 1998.

Quaba AA, Wallace AF: The incidence of malignant melanoma (0-15 years of age) arising in large congenital nevocellular nevi. Plast Reconstr Surg 78:174, 1986.

Flexor Tendon Repair

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The hands and upper extremities are often subject to trauma, and flexor tendons are frequently injured. Because reconstruction of flexor tendons and restoration of function of the involved fingers can be a challenge to hand surgeons, meticulous care and attention to detail during the repair are mandatory to ensure recovery.

Anatomy

The flexor tendons originate in the forearm with the muscle bellies arranged in three distinct layers: superficial, intermediate, and deep. The superficial layer consists of the pronator teres, flexor carpi radialis, palmaris longus, and flexor carpi ulnaris, which proceed in a radial-to-ulnar direction, respectively. The pronator teres and flexor carpi radialis longus are innervated by the median nerve, whereas the flexor carpi ulnaris is innervated by the ulnar nerve. The flexor digitorum superficialis (FDS) musculotendinous unit constitutes the intermediate layer. The deep layer consists of the flexor digitorum profundus (FDP), flexor pollicis longus, and pronator quadratus. The flexor pollicis longus, pronator quadratus, and FDP to the index and long fingers are innervated by the anterior interosseous nerve, whereas the FDP to the ring and small fingers is innervated by the ulnar nerve.

Nine tendons pass through the carpal tunnel before traveling to their respective fingers. The flexor pollicis longus is the most radial structure in the carpal tunnel. All of the FDP tendons are aligned in order in the base of the carpal tunnel. The FDS is aligned such that the tendons to the index and little fingers are deep to the tendons to the long and ring fingers. The flexor pollicis longus inserts into the distal phalanx of the thumb ray, the FDP tendons insert into the base of the distal phalanx, and the FDS tendons insert into the base and shaft of the middle phalanx. In the palm, the FDS tendon is initially volar to the FDP. The FDS tendon decussates at the level of Camper's chiasm, thus allowing the FDP to become more volar in the finger. The FDS and FDP tendons travel in the finger in a fibroosseous canal that is lined by synovium and reinforced by a pulley system.

The reinforcing pulleys of the fibroosseous canal have different designs for different functions (Fig. 1A). The A1, A3, and A5 pulleys prevent bowstringing of the flexor tendon across the metacarpophalangeal, proximal interphalangeal (PIP), and distal interphalangeal joints, respectively. The A2 and A4 pulleys prevent bowstringing of the flexor tendon at the level of the proximal phalanx and middle phalanx, respectively. The cruciate ligaments, which are interspersed among the annular ligaments, constitute the C0, C1, C2, and C3 pulleys. Biomechanically, the A2 and A4 pulleys are considered the most important for prevention of bowstringing and for minimizing the moment arm.

Five zones have been described for flexor tendons of the hand. *Zone I* consists of the FDP tendon alone and encompasses the insertion of the FDP tendon. *Zone II*, once called "no man's land," is defined as the area of insertion of the FDP tendon to the proximal end of the A1 pulley. *Zone III* is the zone of lumbrical origin of the palm, *zone IV* is within the carpal tunnel, and *zone V* is proximal to the carpal tunnel within the forearm.

Each tendon receives nutrition via an intrinsic and extrinsic source. Extrinsic nutrition is provided by synovial fluid, which is pumped into tendon fibers during flexion and extension of the fingers. Intrinsic nutrition is provided by vascular perfusion through three separate sources: (1) longitudinal vessels that enter the palm and travel in the endotendinous channels, (2) vessels that enter at the level of the osseous insertion, and (3) vincula (two short and two long vincula). Most of the internal vascularity within the tendon is present on the dorsal side of the tendons and is primarily incorporated within the septa of the endotenon separating the tendon fascicles.

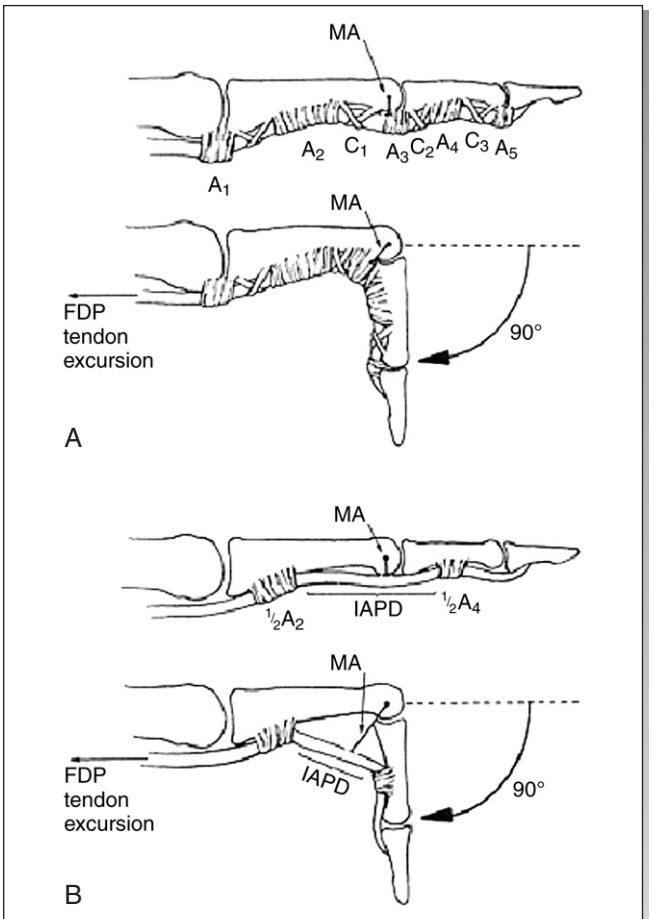

Figure 1 • The pulley system of the fibroosseous canal keeps the tendons in close approximation to the bony skeleton. An intact pulley system fosters optimal muscle amplitude for tendon excursion during flexion. Loss of the pulley system increases the moment arm (MA) and decreases flexor tendon excursion because of the notable bowstringing. IAPD, interannular pulley distance. (From Green DP, Hotchkiss RN, Pederson WC [eds]: Green's Operative Hand Surgery, vol 2, 4th ed. Philadelphia, Churchill Livingstone.)

Appropriate function of the flexor tendons depends on a number of factors, including supple joints, tendon excursion, intact pulley system, and the presence of lubricating synovial fluid. Adhesions between tendons and bone or the synovial sheath, as well as scarring around the joint, prevent tendon excursion. Loss of the pulley system results in bowstringing of the tendons and an increase in the moment arm. A greater amplitude of muscle contraction is required to achieve an equivalent amount of flexion if bowstringing is present (see Fig. 1B). A weakened grip and incomplete flexion result.

Pathologic Anatomy

The zone of tendon injury is important because the location of the injury has a significant impact on the outcome. The overall prognosis and indications for repair may be altered by identifying the mechanism of injury and the composite tissue involved. Zone I injuries typically include avulsion and laceration injuries. Avulsion injuries are usually the result of closed trauma in which the FDP is torn from its insertion in the distal phalanx. The injury is generally associated with forced extension during active flexion of the digit, the so-called Jersey finger, and the ring finger is the most commonly affected digit. Most injuries to the flexor tendons in zone II are the result of open trauma. The hand surgeon must have a high level of suspicion for other vital structures involved, such as the neurovascular bundles on either side of the flexor tendon sheath, and repair these structures as needed. Injuries in zones III, IV, and V are often associated with neurovascular compromise because of the intimate anatomic relationship of the nerves, vessels, and tendons.

Laceration of the flexor tendons results in local bleeding, inflammation, loss of continuity of the flexor sheath, and possible disruption of other local structures such as nerves and arteries. Healing of these structures with diffuse scarring may be detrimental to the functional outcome of the tendon repair. An anatomic repair is required for the tendons, especially in zone II. Zone II is unique because of its tight fibroosseous canal with limited volume for the FDS and FDP tendons to glide. Excess scarring and adhesion formation result in firm adherence of the tendon to the phalanges or the flexor sheath.

Diagnostic Studies

Clinical examination remains the mainstay for identifying FDS and FDP injuries. Lacerations to the tendons are identified clinically by observing loss of the normal cascade. Extending and flexing the wrist may also identify disruption of the flexor tendons through the tenodesis effect. By compression of the forearm, the fingers flex down in tandem. If a breech in continuity of the flexor tendon has occurred, one or multiple fingers may remain extended after this compression maneuver.

Diagnostic studies are of limited value in acute flexor tendon injuries. Magnetic resonance imaging or ultrasound may be of assistance in locating a retracted FDP tendon after a Jersey finger injury. The FDP tendon may retract into the palm (type 1) or the PIP joint (type 2) or may remain caught at the A4 pulley (type 3). Clinical examination, however, will frequently pinpoint the site of the tendon. An inability to flex the PIP joint is often evidence of the FDP tendon trapped at this joint. Fullness in the palm may be an indication of a type 1 avulsion injury.

Reconstructive Goals

The goals of primary repair of flexor tendon injuries are as follows:

- Coaptation of the FDS and FDP tendons
- Anatomic repairs with a limited accordion effect at the repair site
- Multistrand repair to permit active range-of-motion rehabilitation
- Pulley reconstruction to minimize bowstringing
- Atraumatic surgical technique to minimize adhesion formation
- Early active motion to minimize adhesion formation
- Strict adherence to rehabilitation protocols

Treatment

The ideal flexor tendon repair should be reliable, simple, and strong. Most tendon repairs should be performed within the first 24 to 72 hours because delay increases the likelihood of adhesions and scar tissue and decreases the final function. Exposure of the tendons is achieved through either a Brunner (zigzag) or a midaxial incision. If the finger was in the flexed position at the time of injury, the distal end of the flexor tendon is drawn distally as the finger assumes an extended position. If in extension at the time of injury, the distal flexor tendon can usually be identified at the site of the laceration. The proximal tendon end generally retracts into the palm, although intact vincula may limit proximal tendon retraction.

After exposure of the fibroosseous canal, care is taken to avoid disruption of the tendon sheath. Calculated incisions in the sheath prevent the need for pulley reconstruction. Occasionally, a lateral incision in the sheath is required to retrieve the proximal and distal tendon ends. Blind tendon retrieval may be complicated by injury to the distal or proximal segment of the sheath and result in adhesion formation. After the edges are retrieved into the wound, the tendon is secured out to length with a 25-gauge needle or a Keith needle, which pierces through the skin into the tendons at the palm. This maneuver should keep the tendons at the appropriate length for repair without tension. If the laceration is at the A2 or A4 pulley or if repair is hindered because of the close proximity of the pulleys to the severed ends, Z-plasties in the pulleys or partial releases may be required. The pulleys are repaired after the tendon is coapted. Completely disrupted pulleys should be reconstructed with either a slip of the FDS tendon based distally, free tendon grafts, or an extensor retinaculum free graft.

Minimal tendon manipulation is mandated to prevent further injury to the tendons and decrease the likelihood of adhesion formation. Forceps should be used to pick up the tendon only at its severed end and not along the sides of the tendon. Repairs must coapt the edges without bunching or gap formation and provide sufficient strength to prevent rupture. Aggressive bunching impedes tendon excursion and hinders gliding. A gap at the tendon coaptation site results in a weak repair, adhesion formation, and subsequent rupture.

The technique of flexor tendon repair depends on the zone of injury. Avulsions or open lacerations of the FDP in zone I more often than not require repair over a dorsal button because primary repair of the short distal end is difficult. The proximal end of the FDP tendon is drawn through the wound and maintained in position with a Keith needle. Any remaining distal tendon and a sleeve of periosteum are elevated, and the volar cortex of the distal phalanx is abraded. A Bunnell or modified Becker type of suture technique is used to grasp the proximal end of the tendon. The suture is passed through or around the distal phalanx. If it is passed through the distal phalanx, two Keith needles are mounted onto the K-wire driver and passed partway through the distal phalanx and the nail plate. The suture is passed through the eye of the needle and the needle is passed through the dorsal distal wound. The two sutured ends are tied over a button. The proximal tendon end must be cinched into the distal phalanx to allow adequate contact of tendon to bone. The button is maintained in place for 4 to 5 weeks, and the modified Kleinert or Duran protocol is followed for rehabilitation.

For lacerations in zones III, IV, and V, wide exposure is usually required to visualize the nerves, vessels, other tendons, and any bone that may be involved. Although the tendons do not lie within the tight fibroosseous canal, meticulous technique with use of the suture noted in Table 1 is required. It is often necessary to tag and align appropriate tendons to ensure proper coaptation with the respective tendons when multiple tendon injuries have occurred.

For partial tendon lacerations (less than 25% severance), no repair is required. If a 25% to 50% laceration has occurred, a peripheral epitendinous type

TABLE 1 Tendon Repair Strength—Factors

CORE SUTURE—*CONSIDER:*	EPITENDINOUS SUTURE—*CONSIDER:*
Material used	6–0 Monofilament
Caliber	Depth
Strands crossing repair	Locking
Knot location	Crosshatched
Dorsal versus ventral	Simple running

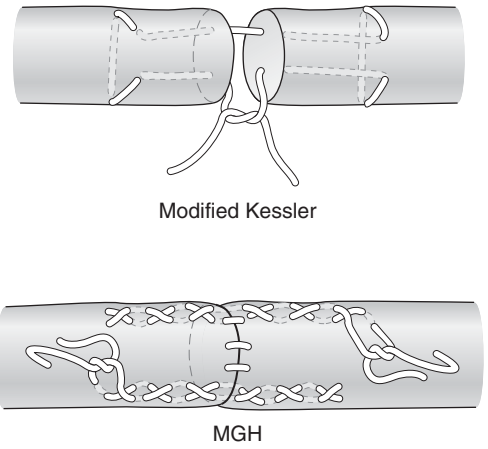

Figure 2 • The modified and MGH Kessler repairs. (Adapted from Lee WPA, Gan BS, Harris S: Flexor tendons. In Achauer BM, Eriksson E, Vander Kolk C, et al [eds]: Plastic Surgery: Indications, Operations, and Outcomes. St. Louis, CV Mosby, 2000, p 1970.)

of suture may be needed to realign the severed edge. Core sutures, as well as a peripheral suture, are required for lacerations greater than 50%.

Flexor tendon injuries require meticulous care and strict adherence to rehabilitation protocols. Early diagnosis and adherence to the principles of wound irrigation, débridement, and atraumatic technique promote excellent results.

Types of Repair

Numerous types of repair have been described for flexor tendon coaptation, the most common of which is a modified Kessler technique with 3–0 Prolene suture. This technique does not reliably permit an active motion rehabilitation protocol because of the risk of tendon rupture. Tendon repair strength is proportional to the number of strands placed across the repair site. There are also many different types of suture material that have been used for flexor tendon repair: Tycron, nylon, Prolene, Ethilon, Mersilene, and stainless steel wire. The suture material should be nonreactive, of relatively small caliber, and strong, with excellent knot-holding characteristics. A commonly used caliber for suture material is 3–0 or 4–0 in either a braided or monofilament design. Commonly used techniques for end-to-end flexor tendon repair are illustrated in Figure 2. The suture techniques, including cruciate, mattress, and cross-stitch techniques, have different grasping qualities. Knots tied within the repair site may occupy a significant amount of space and decrease healing across the repair. Knots located outside the repair along the edge of the tendon may result in friction and adhesion formation.

A volar location for the core sutures has traditionally been advocated because the main blood supply within the tendon is located on the dorsal surface. However, some reports have found a biomechanical advantage with the dorsal core sutures. The modified Becker/MGH repair is an extremely strong suture technique with greater tensile strength than many of the other repairs. The epitendinous suture itself can permit an increase in the strength of the repair and can tuck in any frayed edges that may have been present as a result of the core suture.

No matter what type of repair is performed, adhesions can decrease function after a flexor tendon laceration. Adhesions are promoted in injuries associated with composite tendon-tissue damage, loss of the tendon sheath, gap formation, ischemia, immobilization, persistent inflammation, and secondary trauma. Reconstruction of the flexor sheath after tendon repair is controversial. It may aid in providing extrinsic nutrition to the tendon and in promoting glide-through synovial fluid formation, but it is often very difficult to establish continuity of the flexor sheath. In general, however, only the appropriate pulleys and not the entire flexor sheath need to be reconstructed.

To optimize repair strength, both gapping and bunching should be avoided. Dissection should be minimized to prevent long segments of tendon ischemia. Stress loading should be added to promote more rapid and stronger healing. Controlled active motion during the rehabilitation decreases adhesion formation. The strength of the repair decreases up to 50% between the first and third weeks after repair if the tendon is not stressed. Less of a decrease in strength is noted after early stress to the repaired tendon. Failure of the repair is often due to suture or knot rupture.

Treatment of Late Flexor Tendon Injuries

Indications for flexor tendon grafting include late rupture of the flexor tendon repair, rupture or a gap at the tenolysis site, and delay in treatment after flexor tendon injury. Flexor tendon grafting can be performed in a single stage or as two stages in which the first stage involves placement of a silicone rod, pulley reconstruction, and joint contracture release. Single-stage tendon grafting can be considered for tendon deficits in zones III, IV, and V. For coaptation of the tendon graft to the proximal and distal ends of the tendon, the interweave technique is preferred because it has been shown to be the strongest in biomechanical studies. Two-stage tendon grafting is recommended when patients require tendon grafting in conjunction with pulley reconstruction or joint contracture release. Two-stage grafting is also recommended if collapse of the sheath or excessive scarring is encountered at the time of tendon grafting or if soft tissue reconstruction is required over the graft.

Several donor tendon site options are available: the palmaris, plantaris, extensor digitorum longus,

extensor indicis proprius, and extensor digiti minimi. Selection of a tendon graft is based on the length requirement and the number of tendons requiring graft reconstruction. The palmaris longus provides 16 cm, or sufficient length to graft from the palm to the fingertip. If more graft is needed, lower extremity donors can provide 30 to 35 cm. The palmaris is generally the preferred graft because it is in the same operative field and provides sufficient length. The technique of harvesting a tendon involves dividing the tendon distally and pulling the tendon through a tendon stripper, which releases the tendon from the muscle substance. The palmaris longus is absent in 15% to 25% of patients. Presence of the palmaris longus can be determined by asking the patient to oppose and flex the wrist against resistance. Another potential upper extremity donor is the injured FDS. When multiple tendon grafts or one long tendon graft from the wrist to the fingertip is required, the longer lower extremity donors are useful. The plantaris provides 35 cm of tendon but is absent in 7% to 20% of patients. The plantaris can be located anterior and medial to the Achilles tendon. If the plantaris and palmaris are absent, the extensor digitorum longus from the second, third, and fourth toes can provide multiple segments of tendon 30 cm in length. The extensor digitorum longus is harvested through an incision over the metatarsophalangeal joint. These tendons, however, can be fused at the ankle level, thus necessitating a second incision.

At the first stage of two-stage tendon grafting for zone II reconstruction, the sheath is exposed with midlongitudinal incisions to minimize the risk of silicone rod exposure. The FDP can be used as the motor unit and is generally identified just proximal to the A1 pulley because it is held there by the origin of the lumbrical. The distal part of the FDP is preserved to suture to the silicone rod and the tendon graft in the first and second stages, respectively. Alternatively, the FDS can be used as the motor unit to avoid the potential risk of tendon imbalance with quadriga or a lumbrical-plus posture. The distal end of the FDS is preserved to adhere to the flexor canal for prevention of PIP hyperextension. If the PIP joint already hyperextends, the FDS tail can be tenodesed to the flexor canal to treat the hyperextension. Joint contractures should be released at the first stage. The distal portion of the silicone rod should be secured distally and left free proximally to prevent the rod from being pulled or migrating from the appropriate position. Pulley reconstruction should be performed at the first stage over the silicone rod. Of the various techniques described, the encircling repair technique with a tendon graft has been shown to be the strongest. In this technique, the tendon graft is passed circumferentially around the phalan and silicone rod at the level of desired pulley construction. When reconstructing the A2 and A4 pulleys, the graft should be passed dorsal to the extensor tendon. For reconstruction of the pulley system of the thumb, the oblique pulley has been shown to be the most critical.

The second stage should be performed at 3 months to allow for the development of a pseudosheath. Radiographs can be obtained to confirm proper position of the rod. After limited proximal and distal incisions, the tendon graft is sutured to the rod proximally and pulled distally, and the graft is left in the pseudosheath. Of the various distal juncture techniques, repairing the tendon to tendon has been shown to heal most reliably. The distal juncture can also be repaired with a pullthrough suture through the sterile matrix of the nail bed and nail plate over a button. The proximal end of the tendon graft is repaired to the motor unit with the interweave technique, which allows for size discrepancy. This technique also allows for setting the appropriate length of the tendon graft. If the tendon graft is too short, quadriga and a weakened grip can result. If the tendon graft is too long, a lumbrical-plus posture results with paradoxical hyperextension of the PIP joint on attempted finger flexion. These potential complications can be avoided by using the proximal FDS as the motor unit to avoid quadriga and a lumbrical-plus posture. Immediately after the second-stage operation, early active motion can be initiated with passive flexion and active hold exercises. Patients can begin active flexion exercises at 4 weeks postoperatively. At 6 weeks, the dorsal blocking splint can be discontinued. The patient is allowed to participate in regular activities at 12 weeks.

Possible complications of tendon grafting include adhesions, infection, rod exposure, synovitis, rupture, and tendon imbalance.

Postoperative Care/Rehabilitation

One of the most important factors for success of flexor tendon repairs involves close adherence to a regimented hand therapy rehabilitation program. Various rehabilitation protocols may be used after flexor tendon repair. Because each protocol places different tensile stress demands on the tendons and their repair sites, the choice of rehabilitation protocol depends on the type of repair performed. Other variables include edema and scar tissue, both of which increase drag on the tendons and, in turn, increase the force needed to produce a given flexion task, a situation which may result in tendon rupture. It is therefore important to limit scarring through meticulous surgical technique and to minimize edema with positioning and therapy. Controlled stress is supplied in proportion to the increase in tensile strength. Stressed tendons heal faster, gain strength more rapidly, have fewer adhesions, and result in better excursion.

Passive range-of-motion regimens, including the modified Kleinert and Duran protocols, involve the

use of a dorsal blocking splint with the wrist at 20 to 30 degrees of flexion, the metacarpophalangeal joints at 70 to 80 degrees of flexion, and the interphalangeal joints straight. All digits are placed within the splint and active extension is permitted. Passive proximal and distal interphalangeal motion within the restraints of the dorsal blocking splint is permitted four times a day. At 4 weeks, active composite flexion and extension out of the splint are permitted, and dorsal blocking is continued between exercises. At the fifth week, the dorsal blocking splint is discontinued. Blocking exercises may be initiated at 6 weeks. Gentle passive extension is initiated and a static extension splint used if extrinsic flexor tightness is noted. At 8 weeks light strengthening is initiated, and at 10 weeks resisted exercises are begun. By 12 weeks, normal activities are permitted. The Kleinert protocol uses nail plate hooks with elastic bands attached to the palm and wrist to draw the fingers passively into flexion. The Duran protocol allows the therapist or patient to passively move the fingers into flexion. In both cases, patients can actively extend their fingers.

In active range-of-motion protocols, for the first 3 weeks the patient is placed in a splint similar to the modified Kleinert and Duran protocols. However, in addition, place-and-hold exercises are instituted in which the patient passively flexes the digits and is allowed to contract the muscles to hold the digits in the fist position. At 3 weeks, gentle tenodesis exercises are initiated out of the splint. No active composite flexion is allowed until 4 weeks, at which time active composite flexion exercises out of the splint are initiated. Differential gliding exercises are also begun; the splint is discontinued at 6 weeks and passive extension is initiated. At 7 weeks light strengthening is permitted, and by 12 weeks, normal activities ensue.

Complete immobilization is often recommended for children younger than 10 years and for patients unable or unwilling to follow a controlled motion (passive or active) protocol.

Pearls and Pitfalls

- The strength of tendon repairs is directly related to the number of strands of suture material crossing the repair site and the use of epitendinous suture.
- Strength decreases up to 50% between 1 and 3 weeks if the repair is unstressed.
- Avoid tendon bunching.
- Avoid exposure of knots.
- Use atraumatic technique to decrease adhesion formation in uninjured tendon.
- Gap formation increases the risk for rupture.
- Avoid trapping the repair behind a pulley.
- For best results, initiate rehabilitation protocols immediately.

SUGGESTED READINGS

Boyer MI, Strickland JW, Engles D, et al: Flexor tendon repair and rehabilitation: State of the art in 2002. Instr Course Lect 52:137-161, 2003.

Elliot D, Harris SB: The assessment of flexor tendon function after primary tendon repair. Hand Clin 19:495-503, 2003.

Ingari JV, Pederson WC: Update on tendon repair. Clin Plast Surg 24:161-173, 1997.

Neumeister MW, Brown RE: Flexor tendon repair. In Evans GRD (ed): Operative Plastic Surgery. New York, McGraw-Hill, 2000, pp 890-898.

Strickland JW: Flexor tendon injuries. Part 1. Anatomy, physiology, biomechanics, healing, and adhesion formation around a repaired tendon. Orthop Rev 15:632-645, 1986.

Wilson S, Sammut D: Flexor tendon graft attachment: A review of methods and a newly modified tendon graft attachment. J Hand Surg [Br] 28:116-120, 2003.

Extensor Tendon Injuries

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MICHAEL W. NEUMEISTER

Because the extensor tendon and mechanism are located superficially under the skin on the dorsum of the hand, injuries are relatively common. Over the years, extensor tendon injuries have not attracted the same degree of attention as flexor tendon injuries. However, diagnosis and treatment of extensor injuries require comparable knowledge and skill on the part of the surgeon. The purpose of this chapter is to review extensor tendon injuries and time-proven treatment regimens.

Etiopathogenesis

Extensor tendon injuries can result from sharp or blunt mechanisms. Injury to the extensor tendon at the more distal zones, such as zones 1 and 3, is more commonly a result of blunt trauma. Proximal extensor tendon injuries usually occur from sharp trauma. Extensor tendon injuries can also result from the chronic inflammatory milieu associated with arthritis. The most common extensor tendons to rupture in patients with rheumatoid arthritis include the extensor pollicis longus and extensor digiti minimi tendons.

Pathologic Anatomy

The substance of the extensor tendon changes distally to proximally. It is thinner and more superficial distally, whereas at the wrist the extensor tendon is thicker and lies deeper. Therefore, treatment of extensor tendon injuries is influenced by the site of injury. Moreover, outcomes after repair of extensor tendons vary by level of injury. Thus, extensor tendon injuries have been classified into different zones to assist and direct management (Fig. 1 and Table 1).

There are 11 extensor tendons. The extensor digitorum communis (EDC) muscle has four tendons passing to the index, middle, ring, and small fingers. It is not uncommon for EDC muscle bellies to be independent of each other. The index finger has a second extensor, the extensor indicis proprius (EIP) tendon; the small finger also has another extensor tendon, the extensor digiti minimi (EDM). This anatomic arrangement provides the index and small finger with independent hyperextension at the metacarpophalangeal (MP) joint. Another anomaly is the extensor indicis et medii communis, which splits to the index and middle fingers. The extensor digitorum brevis manus is another aberrant muscle infrequently found on the dorsum of the hand. The thumb is extended at the interphalangeal joint by the extensor pollicis longus (EPL), which is longer and therefore inserts more distally onto the distal phalanx. The extensor pollicis brevis (EPB) extends the thumb at the MP joint. The wrist is extended by the extensor carpi radialis longus and brevis (ECRL, ECRB), which insert at the second and third metacarpal bases, respectively. The extensor carpi ulnaris (ECU) also extends the wrist and inserts onto the fifth metacarpal base.

At the level of the wrist, the wrist and digit extensor tendons pass under the extensor retinaculum through six tunnels, or dorsal compartments, that are numbered in the radial-to-ulnar direction (Fig. 2). The EPB and abductor pollicis longus are contained in the first dorsal compartment. The ECRL and ECRB pass through the second compartment. The EPL courses ulnar to Lister's tubercle in the third tunnel. The fourth dorsal compartment contains the EDC and EIP tendons. The EDM and ECU course through the fifth and sixth compartments, respectively.

Other pertinent anatomy includes the sagittal bands and transverse retinacular ligaments that centralize the extensor tendon over the MP and proximal interphalangeal (PIP) joints, respectively

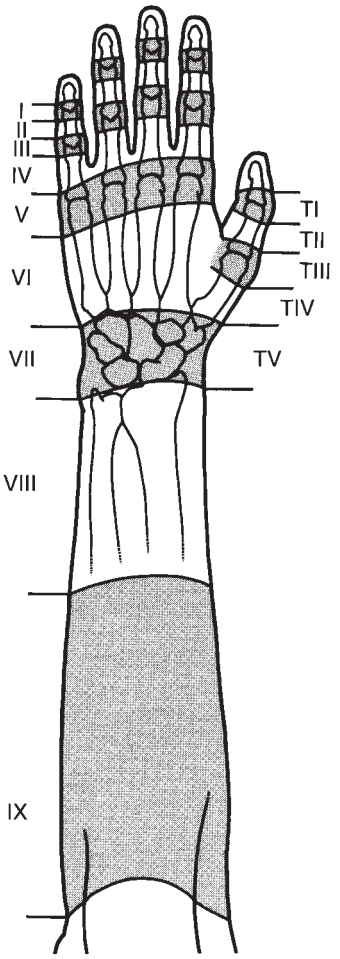


Figure 1 • The nine zones of extensor tendon injuries. (From Doyle JR: Extensor tendons: Acute injuries. In Green DP, Hotchkiss RN, Pederson WC [eds]: Green's Operative Hand Surgery, vol 2, 4th ed. Philadelphia, Churchill Livingstone, 1999, pp 1950-1987.)

(Fig. 3). The sagittal band arises from the volar plate and the intermetacarpal ligaments and neck of the metacarpals. Lateral bands are extensions of the intrinsic hand muscles that are required for extension of the interphalangeal joints; they contribute to flexion of the MP joints. The lateral band structures extend along both sides of the PIP joint to attach to the base of the distal phalanx as the terminal extension of the extensor.

Preoperative Evaluation

The extensor tendon inserts into the distal phalanx as the terminal extension of the joining lateral bands (zone 1). Zone 1 extensor injuries can result from an open or closed mechanism. The most common cause of a zone 1 extensor tendon injury is forceful hyperflexion while the digit is extended (i.e., a mallet finger deformity). When the tendon is injured at this level, the patient is unable to extend the distal phalanx, and the finger assumes a flexed posture at the distal interphalangeal (DIP) joint. The severity of the deformity varies from a few

TABLE 1 Extensor Tendon Zones of Injury

ZONE	FINGER	THUMB
1	DIP joint	Interphalangeal joint
2	Middle phalanx	Proximal phalanx
3	PIP joint	MP joint
4	Proximal phalanx	Metacarpal
5	MP joint	Carpometacarpal joint/radial styloid
6	Metacarpal	
7	Dorsal retinaculum	
8	Distal forearm	
9	Mid to proximal forearm	

DIP, distal interphalangeal; MP, metacarpophalangeal; PIP, proximal interphalangeal.

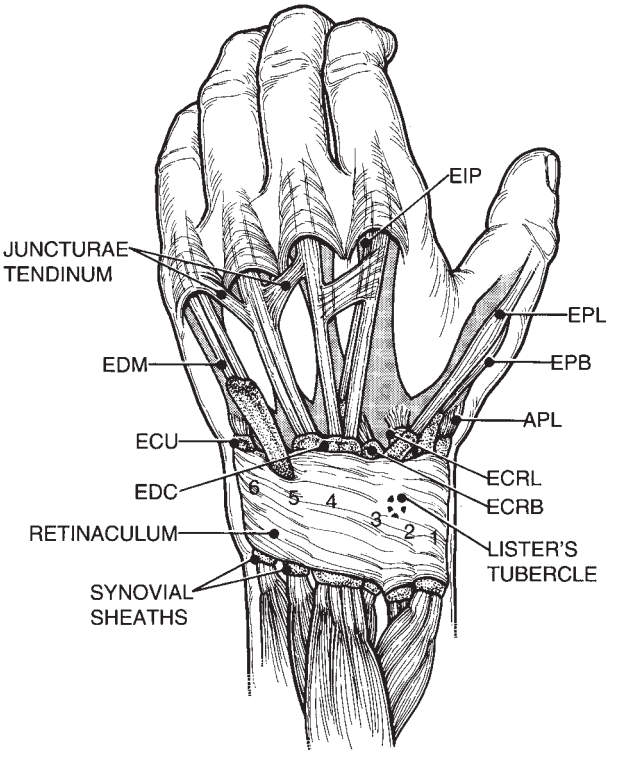


Figure 2 • At the level of the wrist, the wrist and digit extensor tendons pass under the extensor retinaculum through six tunnels known as dorsal compartments. The dorsal compartments are numbered in the radial to ulnar direction. (From Doyle JR: Extensor tendons: Acute injuries. In Green DP, Hotchkiss RN, Pederson WC [eds]: Green's Operative Hand Surgery, vol 2, 4th ed. Philadelphia, Churchill Livingstone, 1999, pp 1950-1987.)

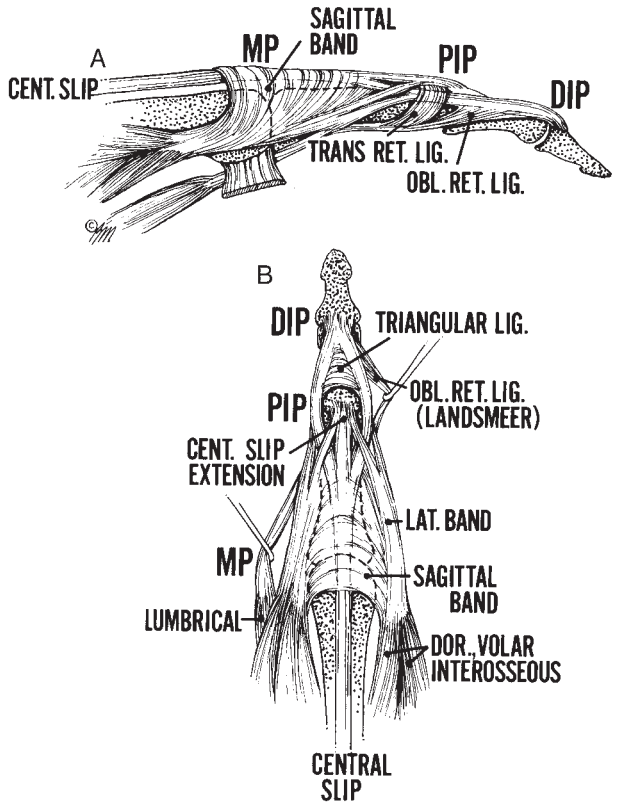


Figure 3 • The sagittal bands and transverse retinacular ligaments that centralize the extensor tendon over the metacarpophalangeal (MP) and proximal interphalangeal (PIP) joint. (From Doyle JR: Extensor tendons: Acute injuries. In Green DP, Hotchkiss RN, Pederson WC [eds]: Green's Operative Hand Surgery, vol 2, 4th ed. Philadelphia, Churchill Livingstone, 1999, pp 1950-1987.)

degrees to 70 degrees. Closed injuries should be evaluated with a radiograph to assess for a possible fracture. Mallet finger deformities have been classified. Type 1 mallet finger deformities, the most common, result from closed or blunt trauma with loss of tendon continuity, with or without a small avulsion fracture. Type 2 injuries occur from a laceration at or proximal to the DIP joint with loss of tendon continuity. Type 3 injuries are caused by deep abrasions and result in loss of skin, subcutaneous cover, and tendon substance. Type 4A injuries occur with transepiphyseal plate fractures in children. Type 4B injuries result from hyperflexion injury, with fractures encompassing 20% to 50% of the articular surface. Type 4C mallet injuries occur with fractures of the articular surface (greater than 50%) and with early or late volar subluxation of the distal phalanx.

Loss of extension at the PIP joint can result from both open and closed injuries. An injury to the central slip can occur as a result of acute forced flexion of the PIP joint. When the central slip of the EDC has been cut or it ruptures from the base of the middle phalanx, loss of PIP joint extension can occur. Over time (usually 10 to 21 days), a central slip injury can progress to a boutonnière deformity in which the proximal phalanx protrudes dorsally through the lateral bands like a button. The proximal and volar vector or lateral band pull causes flexion of the PIP joint and hyperextension of the DIP joint. Because the deformity is not initially present, early diagnosis of the injury is often missed; however, provocative maneuvers have been described for earlier diagnosis of central slip injuries. Loss of extension greater than 15 degrees at the PIP joint with wrist and MP flexion, weak PIP extension against resistance, and some loss of passive flexion of the DIP joint while passively extending the PIP joint are signs that suggest central slip injury. It is important to differentiate a boutonnière deformity from a pseudoboutonnière flexion contracture of the PIP joint because their treatment is much different.

Loss of extension at the metacarpal joint can occur from an injury to the extensor tendon or sagittal band or it can be caused by trigger finger syndrome. Sagittal band injury is suggested if the finger can be held in extension after it is passively extended; this maneuver centralizes the EDC. Trigger finger syndrome is usually suggested by previous popping with extension and tenderness over the A1 pulley. An injury to, or long-standing compression of, the radial nerve (posterior interosseus nerve syndrome) proximal to the innervation of the EDC and EDM muscles can cause loss of extension to the MP joints.

Reconstructive Goals

The results of extensor repairs can be judged by recovery of total active motion, loss of extension and flexion, and grip strength. A widely accepted system for assessing the outcome of extensor repairs involves documentation of loss of extension and flexion. A patient with full flexion and extension is considered to have an *excellent* result. Loss of 10 degrees or less of extension plus loss of 20 degrees or less of flexion represents a *good* result. A *fair* outcome results when 11 to 45 degrees of extension is lost and 21 to 45 degrees of flexion is lost. A patient is considered to have a *poor* outcome if more than 45 degrees of flexion and extension is lost.

Treatment

Suture Techniques

Because of the small caliber of the extensor tendons in the fingers, the suture techniques commonly used for flexor tendon injuries have to be modified when

applied to the extensors. The thickness of the extensor tendon in zones 1, 2, and 4 ranges between 0.55 and 0.65 mm, whereas its thickness in zones 3, 5, and 6 is between 1.0 and 1.7 mm. Flexor tendon suture techniques can be applied to extensor repairs in zones 3, 5, and 6, where the tendon more closely resembles the thickness of a flexor tendon, thereby providing better strength and lower risk for rupture with early motion postoperative protocols. Early motion protocols are not required for zones 1 and 2 extensor repairs.

The authors prefer to use the Massachusetts General Hospital (MGH) technique for zones 3 to 7 extensor repairs to minimize the risk of rupture with early motion postoperative protocols (see Fig. 2 in the chapter "Flexor Tendon Repair"). When the MGH technique is used for a wider tendon such as in zone 4 injuries, six strands instead of four are placed across the tendon, four on each side and two in the center. Zones 1 and 2 extensor injuries can be repaired with a continuous suture augmented with a cross stitch epitenon technique and with a static splint postoperative regimen.

Treatment of Zone 1 Injury

Treatment of zone 1 injuries depends on the type of deformity. Closed type 1 injuries are treated with a DIP splint. The DIP joint is splinted at 0 degrees and the PIP joint is left unrestrained for active movement. Both the DIP and PIP joints were previously maintained in slight hyperextension for the treatment of closed mallet deformity; however, this technique has been shown to be unnecessary. A padded aluminum splint can be used dorsally or volarly until the swelling has subsided to allow for active PIP movement. The patient is instructed to wear the splint at all times for 8 weeks. If the patient removes the splint and bends the finger even momentarily, the 8 weeks of splint immobilization begins again. Alternatively, a longitudinal K-wire can be placed across the DIP joint. Eighty percent of patients treated with a proper splint for zone 1 injuries experience good to excellent outcomes.

If the zone 1 extensor injury is open, the tendon is repaired with a continuous suture augmented with a cross stitch epitenon technique and splinted in extension for 6 weeks. If an extensor tendon deficit exists, it can be grafted primarily or later if soft tissue reconstruction is required. If a fracture involves more than a third of the articular surface of the distal phalanx, it is treated by open reduction and internal fixation. A pullout wire loop over a volar button and a longitudinal transarticular DIP joint K-wire are used for fixation of the fracture (Fig. 4). The finger is splinted for 6 weeks, and the transarticular DIP joint K-wire is removed at 6 weeks. Because the articular surface of the distal phalanx remodels well, some surgeons reserve open fixation of a mallet fracture for only volarly subluxed injuries.

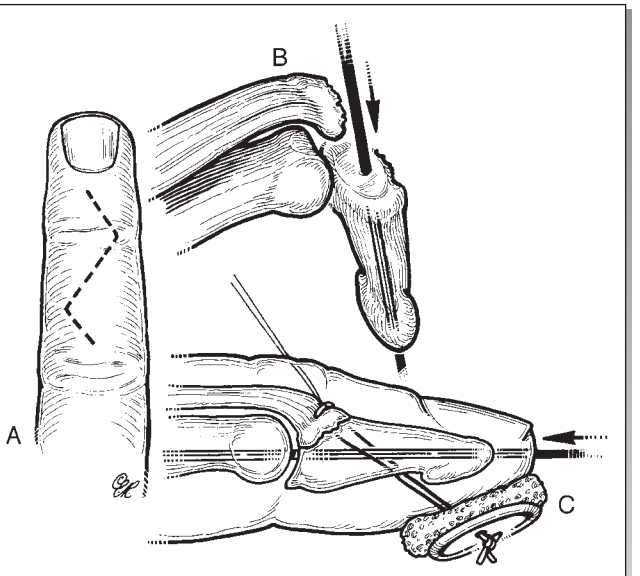


Figure 4 • Treatment of zone 1 extensor injuries that involve a fracture with involvement of more than a third of the articular surface. A pullout wire loop over a volar button and a longitudinal transarticular distal interphalangeal joint K-wire are used for fixation of the fracture. (From Doyle JR: Extensor tendons: Acute injuries. In Green DP, Hotchkiss RN, Pederson WC [eds]: Green's Operative Hand Surgery, vol 2, 4th ed. Philadelphia, Churchill Livingstone, 1999, pp 1950-1987.)

Treatment of Zone 2 Injury

Injuries to the extensor tendon in zone 2 usually occur secondary to a laceration. Lacerations of the extensor tendon within this zone are treated in a manner similar to open zone 1 injuries: a continuous suture augmented with a cross stitch epitenon technique, followed by a static splint regimen for 6 weeks (Fig. 5). Partial tendon injuries involving less than 50% of the tendon substance can be treated by skin wound care and splinting for 10 days.

Treatment of Zone 3 Injury

When a closed central slip injury is diagnosed within 30 days, immobilization of the PIP joint in extension for 6 weeks can provide good to excellent results. Other reports have demonstrated success with immobilization of closed injuries beginning up to 12 weeks after injury or as long as the PIP joint can be passively extended. The preferred technique for PIP immobilization is with a Bunnell-type safety pin or a volar thermoplastic static splint. The splint immobilizes the PIP joint in extension and allows active flexion and extension of the DIP joint. The wrist and MP joints do not require immobilization. Alternatively, a cast or oblique transarticular K-wire can be used. Mobilization of the PIP joint can begin when full flexion of the DIP joint and passive extension at the PIP joint are achieved and full passive extension at the PIP joint is documented. During the first week of mobilization, the PIP is actively extended with a

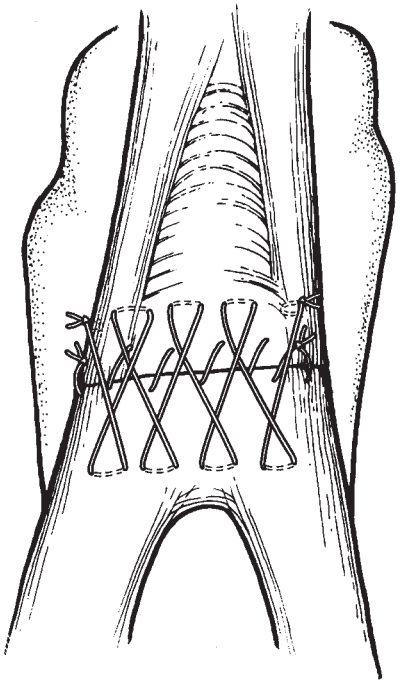


Figure 5 • Treatment of a laceration of the extensor tendon within zones 1 and 2 with a continuous suture augmented with a cross stitch epitenon technique, followed by a static splint postoperative regimen for 6 weeks. (From Doyle JR: Extensor tendons: Acute injuries. In Green DP, Hotchkiss RN, Pederson WC [eds]: Green's Operative Hand Surgery, vol 2, 4th ed. Philadelphia, Churchill Livingstone, 1999, pp 1950-1987.)

neutral MP joint and limitation of PIP active flexion to no more than 30 degrees. If no extensor lag develops by the second week, motion can progress to 50 degrees. Motion can be increased 20 degrees per week. Forced flexion should be avoided for 12 weeks. If an extensor lag develops during rehabilitation, it is treated by extension static splinting until the aforementioned criteria are observed. If the patient has an avulsion fracture at the base of the middle phalanx that involves more than a third of the articular surface, open reduction with internal fixation is required. The fracture can be secured with an intraosseous loop wire or a K-wire and is splinted for 4 weeks.

Extensor lacerations in zone 3 are likely to enter the PIP joint. If so, it is important to repair the tendon primarily and irrigate the joint thoroughly. Either the Kessler or the MGH technique can be used for extensor tendon repairs in this zone. Alternatively, zone 3 tendon injuries can be repaired with a continuous suture and cross stitch technique. The PIP joint is immobilized postoperatively with a volar splint while the DIP joint is free for active flexion and extension for 6 weeks. If the central slip and lateral bands were repaired, the finger is placed in a static splint to hold both the PIP and DIP joints in extension. The remaining postoperative regimen follows as discussed earlier for closed zone 3 injuries.

A deficit of the extensor tendon can be repaired by one of two described techniques. A distally based retrograde flap from the central slip can be turned over the tendon to reinforce the central slip. The retrograde central slip flap should be as wide as possible while allowing primary closure of the central slip donor area. Alternatively, the central edges of the lateral band can be split for 2 cm and approximated longitudinally over the center of the PIP joint. These techniques can also be used if the central slip is deficient distally.

A chronic boutonnière deformity can be corrected by approximating the lateral bands dorsally, wrapping them with a tendon graft, and creating a Fowler extensor tenotomy distally over the DIP joint. When performing the tenotomy, the tendon should be fractured with a celery stick technique until hyperextension is corrected. The DIP and PIP joints are then internally splinted with a longitudinally placed intraarticular K-wire for 6 weeks.

Treatment of Zone 4 Injury

Extensor tendon injuries in zone 4 are usually partial lacerations. If the laceration involves only the lateral band, it should be repaired with a figure-of-eight suture and early initiation of protected motion. Because these injuries involve direct inspection for diagnosis, even partial injuries can be repaired with figure-of-eight sutures and early initiation of protected motion. A complete laceration can be repaired with the MGH technique by using six core sutures because of the width of the tendon at this level. Alternatively, a complete tear can be repaired with a continuous suture and cross stitch technique. An early relative motion protocol is begun on the first postoperative day as described later. In general, the outcomes of complete zone 4 injuries are inferior to those of zone 5.

Treatment of Zone 5 Injury

Zone 5 extensor tendon injuries over the MP joint most commonly occur from a clenched fist punch to the mouth region. A radiograph should be obtained to evaluate for a fracture or foreign body. The wounds and the MP joint should be thoroughly explored, irrigated, and left open. Antibiotics should be administered. Most of the associated extensor tendon injuries are only partial. Complete tendon lacerations can be repaired at 5 to 7 days because the extensor tendon ends do not significantly retract at this level.

Treatment of Sagittal Band Injuries

Closed sagittal band injuries are more common than open sagittal band injuries because of the relatively

protected position of this structure. The mechanism for closed injury to the radial sagittal band involves ulnarward subluxation or dislocation of the extensor tendon with forceful flexion or extension of the finger. The typical injury includes ulnar subluxation associated with painful snapping of the tendon when making a fist, followed by an inability to extend the ulnar-deviated finger. The most commonly affected finger is the middle one. If the diagnosis of a closed sagittal band rupture is made within 2 weeks, the injury can be successfully treated by 6 weeks of continuous immobilization of the MP joint in neutral to slight flexion (20 degrees) while allowing active motion of the interphalangeal joints. When the injury is diagnosed after 2 weeks or after an open injury, repair is indicated. If loss of sagittal band substance precludes primary repair, a proximally based portion of the EDC can be passed around the lateral band to centralize the extensor tendon. The tension on the repair should allow normal flexion of the MP joint while maintaining centralization of the tendon with flexion and extension. The MP joint of the involved digit is immobilized postoperatively in neutral position for 4 weeks.

Treatment of Zone 6 Injury

Extensor tendon repairs in zone 6 achieve better results than more distal injuries because they are less likely to have associated injuries and they usually have less subcutaneous tissue and less tendon excursion. Flexor tendon repair techniques are more easily applied to extensor injuries in this zone because of the thicker tendon substance at this level. Postoperative management involves the early relative motion protocol and a splint that maintains the wrist at *0 degrees* extension and the MP joints at 0 to 15 degrees for 4 weeks postoperatively.

Treatment of Zone 7 Injury

Zone 7 injuries can be challenging to treat because of retraction of the extensor retinaculum and tendon. However, it is usually possible to preserve portions of the retinaculum to prevent bowstringing. Complete resection of the retinaculum is seldom necessary because limiting adhesions can be avoided by early protected motion. Primary repair of the tendon can be performed with a standard core-type suture of nonabsorbable material. Postoperative therapy and splinting are identical to that for zone 6 injuries: early relative motion and a prefabricated splint with the wrist at *0 degrees* extension.

Tendon ruptures in this zone, such as rupture of the EPL from rheumatoid arthritis or a Colles' fracture, are best treated by transfer of the EIP rather than a tendon graft because of the risk for attenuation and rerupture of the tendon. ECU subluxation is treated by repair of the fibro-osseous sheath or a tendon graft or with a portion of the extensor retinaculum turned over the ECU, which is then sutured to itself. Subluxation of first dorsal compartment tendons can be treated by reconstructing the first dorsal compartment with a portion of the brachioradialis turned over and sutured to itself.

Treatment of Zone 8 Injury

Although lacerations distal to the musculotendinous junction can be directly repaired, injuries at the musculotendinous junction do not usually hold sutures well. Such repairs at the musculotendinous junction are commonly tenuous, and side-to-side transfers are preferred if feasible. When side-to-side transfers cannot be performed, such as when multiple tendons have been severed at the musculotendinous junction, they should be repaired with multiple figure-of-eight nonabsorbable sutures and immobilized for 5 weeks with the wrist extended at 45 degrees and the MP joints at 20 degrees.

Treatment of Zone 9 Injury

Penetrating wounds in the proximal third of the forearm with functional loss should be explored to determine whether the cause is neurologic or results from loss of muscle integrity. If the wound extends only through the muscle belly, careful repair of the muscle belly is performed by using multiple figure-of-eight sutures. Properly repaired muscle lacerations restore some element of useful function. Because the muscle planes can be difficult to identify, complete hematoma evacuation and identification of the intramuscular fibrous septa and fascia can be helpful in repairing the muscle and preventing suture pulling through the muscle. Tension-free approximation of the muscle and avoidance of suture pull through can be aided by wrist extension. Patients are immobilized in a static splint or cast for 4 to 6 weeks postoperatively. If a suitable repair cannot be performed, tendon transfers may be indicated.

Postoperative Care and Complications

The early relative motion protocol (Merritt) involves the use of two static-type splints that protect the tendon repair during active motion. The distal splint is fabricated out of thermoplastic material in an elongated figure-of-eight configuration. The volar aspect of the injured finger rests on support between two uninjured fingers, which fit through the openings in the figure-of-eight splint. The design limits MP flexion to 0 to 15 degrees but allows for active PIP flexion and extension. The repaired finger must be kept a half to one finger width higher than the noninvolved fingers. The distal splint can be widened to incorporate both the small and index fingers if the ring and middle finger extensors were

repaired. If the index or small finger was repaired, the distal splint can be modified to block index and small finger MP flexion by extension of thermoplastic material in a figure-of-eight pattern around the middle and ring fingers. The proximal splint blocks the wrist at *0 degrees* of extension. The splints are worn continuously for 4 weeks. The early relative motion protocol was initially described for zone 5 to 7 extensor tendon repairs, but it can also be used for zone 4 injuries with a modification of the distal splint to limit PIP joint flexion to 30 degrees.

Ruptures of extensor tendon repairs can occur, although they are less common than flexor tendon ruptures. Ruptures occur most frequently in noncompliant patients, but they can also result from improper technique. Ruptures should be repaired and placed in a static splint or cast if noncompliance is suspected. Adhesion formation can complicate extensor tendon repairs. The risk for adhesions can be minimized by early protected motion. Combined extensor tendon and bone injuries that require immobilization are at greatest risk for adhesion formation. Adhesions are suspected by loss of passive and active flexion and extension. When performing extensor tenolysis procedures, it is beneficial to use local anesthesia to allow for intraoperative assessment of lysis of adhesions. If the repair site is not attenuated, patients should begin early active motion immediately after tenolysis. If the extensor tendon injury is associated with a soft tissue deficit, soft tissue reconstruction should be completed before tendon grafting.

Imbalance deformities of the extensor mechanism result in loss of the physiologic biomechanics of the extensor mechanism. Although the extensor digitorum tendon stays solely on the dorsum of the finger and inserts onto the central slip at the base of the middle phalanx, the lateral bands arise from the volar aspect of the digit and come to lie dorsal to the pivot axis of the PIP joint and terminate in a conjoint tendon on the dorsal aspect of the base of the distal phalanx. Any alterations in the direction of pull of the intrinsic muscles relative to the pivot axis of the PIP joint result in significant changes in the resting posture of each digit. Two such imbalance deformities of the extensor mechanism are the *swan neck* and the *boutonnière* deformity.

The *swan neck deformity* is characterized by hyperextension of the PIP joint and flexion of the DIP joint. The lateral bands migrate dorsally as the transverse retinaculum ligament attenuates. There is relative lengthening of the extensor mechanism with resultant flexion of the distal phalanx. Primary swan neck deformities result from laxity of the volar plate at the PIP joint, as seen in rheumatoid arthritis. Secondary swan neck deformities, however, are the result of disruption of the terminal tendon, foreshortening of the extensor mechanism, dorsal migration of the lateral bands, and attenuation of the transverse retinaculum ligament. With time, attenuation of the volar plate also occurs and results in PIP joint hyperextension. The loss of continuity of the terminal tendon results in the distal phalanx being drawn into flexion because of the unopposed action of the flexor digitorum profundus tendon. Treatment of swan neck deformities is usually surgical, although early splinting should be attempted. The splint is manufactured to maintain the PIP joint at neutral or approximately 10 degrees of flexion. The patient is allowed to actively flex and extend the finger while in the splint. A trial of at least 6 weeks of splinting is recommended, but it should be noted that splinting has a limited role in correcting swan neck deformities. Causes of swan neck deformity may involve pathology such as imbalance secondary to a mallet deformity, fracture of the middle phalanx that has healed in hyperextension, or laxity of the volar plate at the PIP joint. Tendon reconstruction of a swan neck deformity should not be undertaken in patients with significant joint disruption or arthritis; arthroplasty or arthrodesis may be more appropriate in these patients. There are three techniques of surgical reconstruction of a swan neck deformity. The first procedure involves using the oblique retinaculum ligament. Based distally, the oblique retinaculum ligament is transected proximally. The ligament is wrapped volar to Cleland's ligament across the finger at the PIP joint and inserted into the proximal phalanx of the contralateral side. The PIP joint is kept in approximately 10 to 20 degrees of flexion. The oblique retinaculum ligament is fixed over a button with pull through sutures or with a Mitek anchor.

As an alternative to the oblique retinaculum ligament repair, a tendon graft can be used to perform a spiral oblique retinaculum ligament reconstruction. The graft is attached distally at the base of the distal phalanx and is passed volar to the PIP joint to the contralateral proximal phalanx. The graft is levered around Cleland's ligament to maintain its position. The graft can be fixed over a button or with a Mitek anchor. The palmaris longus tendon is the most commonly used donor tendon. The graft is adjusted to keep the PIP joint flexed at 20 degrees with the distal phalanx kept at neutral.

The third technique for reconstruction of a swan neck deformity includes tenodesis of the superficialis tendon. One slip of the flexor digitorum superficialis (FDS) tendon is transected proximally while still based distally. With the PIP joint in flexion, the FDS tendon slip is passed through the proximal phalanx and secured with a Mitek anchor or over a button.

For each technique of swan neck reconstruction, a K-wire is used to maintain the PIP joint in approximately 20 degrees of flexion and the DIP joint in neutral. The K-wires are left in place for 4 weeks, and the patient is started on active range of motion with a blocking splint in place. Passive range of motion is initiated at 6 weeks.

The *boutonnière deformity* is a second type of imbalance deformity of the extensor mechanism and

is characterized by DIP joint hyperextension and PIP joint flexion. The boutonnière deformity is often associated with compensatory MP joint hyperextension. In this deformity, attrition or disruption of the central slip and transverse retinaculum ligament results in volar migration of the lateral bands at the PIP joint. Because the lateral bands fall volar to the pivot axis of the PIP joint, the bands become flexor tendons instead of extensors of the PIP joint. The PIP joint is drawn into flexion, but because of the attachments of the lateral bands to the terminal tendon, the distal phalanx is drawn into extension as the lateral bands contract. In the early stages of a boutonnière deformity, it is reducible and splinting may be corrective. A three-point splint is used to extend the PIP joint and allow flexion of the DIP joint. Unlike swan neck deformities, boutonnière deformities respond well to splinting. At least 8 to 12 weeks of conservative splinting therapy should be completed before surgery is contemplated. Many surgical procedures are fraught with subsequent stiff joints, and therefore all attempts should be made to conservatively treat these patients initially. Procedures performed for correction of a boutonnière deformity have included direct excision and tightening of the central slip transversely with an end-to-end repair, as well as tenotomy of the terminal tendon to restore flexion of the DIP joint. Tendon graft procedures have also been described for stabilization of the dorsal PIP joint to prevent a resting flexion posture. Tendon grafts are attached to the proximal phalanx but passed through a dorsal hole in the base of the middle phalanx to reconstruct the central slip. The lateral band transfer procedure has been described to add support to the dorsal aspect of the PIP joint. The ulnar lateral band is transected distally at the level of the DIP joint, and the radial lateral band is transected more proximally at the level of the middle phalanx. The proximal ends are crisscrossed and sutured to the contralateral distal ends of the lateral bands. Tendon transfers and graft procedures require 4 weeks of immobilization followed by 2 weeks of passive range-of-motion and subsequent active range-of-motion and gradual strengthening exercises. Tenotomy of the terminal tendon is often considered a more reliable procedure to correct boutonnière deformities. The extensor mechanism is divided near the terminal tendon through the triangular ligament and the lateral band. The oblique retinaculum ligament is left intact to avoid complete loss of distal phalanx extension.

Complications of secondary reconstruction procedures include infection, bleeding, wound dehiscence, scarring of the extensor tendon, joint stiffness, and rupture of the tendon grafts or repairs. When tendon grafts or tendon transfers are used, there is the potential to create an excessively tight or loose repair. Strict adherence to the rehabilitation protocol is required to achieve optimal results. Because of the intricate finely tuned nature of the extensor mechanism, reconstructive efforts are often fraught with complications such as stiff fingers.

Pearls and Pitfalls

- It is critical to diagnose and treat extensor tendon injuries as early as possible.
- The most optimal results can be achieved by using tendon repair techniques that allow for early protected motion.
- Complications can be minimized by frequent follow-up with the surgeon and hand therapist and by placing casts on potential noncompliant patients.

SUGGESTED READINGS

Doyle JR: Extensor tendons: Acute injuries. In Green DP, Hotchkiss RN, Pederson WC (eds): Green's Operative Hand Surgery, vol 2, 4th ed. Philadelphia, Churchill Livingstone, 1999, pp 1950-1987.

Evans RB, Burkhalter WE: A study of the dynamic anatomy of extensor tendons and implications for treatment. J Hand Surg [Am] 11:774-779, 1986.

Harris C, Rutledge GL Jr: The functional anatomy of the extensor mechanism of the finger. J Bone Joint Surg Am 54:713-726, 1972.

Mowlavi A, Schall J, Wilhelmi BJ: Extensor hallucis longus tenorrhaphy by using the Massachusetts General Hospital repair. J Foot Ankle Surg 43:412-418, 2004.

Newport ML, Blair WF, Steyers CM Jr: Long-term results of extensor tendon repair. J Hand Surg [Am] 15:961-966, 1990.

Wasserman RJ, Howard R, Markee B, et al: Optimization of the MGH repair using an algorithm for tenorrhaphy evaluation. Plast Reconstr Surg 99:1688-1694, 1997.

Fractures and Dislocations of the Wrist and Fingers

GREGORY A. DUMANIAN

Innumerable injury patterns can occur in the hand. Rather than provide a laundry list of hand injuries and their treatment, this chapter illustrates common hand fractures and dislocations along with their treatment to demonstrate principles of management. By understanding the fundamentals of hand fractures and knowing the anatomy, one can formulate treatment plans for both familiar and rare injuries.

Etiopathogenesis

The hand is the means by which we interact with our environment and protect ourselves. It is therefore susceptible to injury. Hand injuries are the most common orthopedic injuries seen in emergency rooms. The small bones of the hand are comparatively fragile and can fracture or dislocate with relatively small force. In older patients, the bones can be affected by osteoporosis, thus making them more likely to fracture. In younger patients with stronger bones, stressful activities can lead to both fractures and dislocations.

Diagnostic Studies

The most important diagnostic tool in hand fractures is a detailed history and physical examination, which will elucidate most injuries and direct further testing. In general, standard radiographs are the most important study. This generally includes 3 views (anterior-posterior, lateral, and oblique). Each suspected injury (discussed in the individual sections) may prompt specialized radiographic techniques that best demonstrate fractures or dislocations. Occasionally, computed tomography (CT) and magnetic resonance imaging (MRI) are used, although this is the exception rather than the rule.

Reconstructive Goals

Even though each injury has specific considerations, the overall goals of management of hand fractures and dislocations are as follows:

1. Pain-free hand
2. Normal (or functional) range of motion
3. Adequate strength
4. Normal sensation
5. Minimal scarring or cosmetic deformity

These goals should be accomplished with minimal cost and risk to the patient. Patient age, hand dominance, and hand use patterns affect decision making. Elderly patients with “low-use” hands do not require the same function as a young laborer or athlete.

Dorsally Comminuted Distal Radius Fractures

Patients with distal radius fractures have typically fallen onto an outstretched hand. The force is delivered at 40 to 90 degrees off the long axis of the radius; it travels through the volar cortex and causes dorsal compaction of cancellous bone and comminution of cortical bone. The greater the energy absorbed by the hand, the greater the initial displacement, comminution of the articular surface, and soft tissue swelling. Patient age and bone density have an effect on the ability of the extremity to tolerate and absorb the delivered energy.

Examination should begin with evaluation of swelling, ecchymosis, and any dysfunction of the median nerve. Swelling within the carpal canal is a common cause of nerve dysfunction. Other causes include direct nerve contusion, nerve traction injuries with significantly displaced fractures, and compression from tight-fitting splints. Light touch is a quick test, although two-point discrimination or monofilament testing is a more objective measure of nerve function. Acute carpal tunnel syndrome requiring treatment is uncommon and not generally seen in ambulatory patients. Patients with reduced distal radius fractures and median nerve symptoms that worsen over days of observation should undergo carpal tunnel release.

Multiple classification systems have been devised for distal radius fractures. The "universal" classification system stratifies fractures into displaced versus nondisplaced, extraarticular versus intraarticular, and stable versus unstable fractures. It is critical to examine both the initial emergency room films and any postreduction films to classify the fracture. The worse the initial film, the greater the chance that the fracture is unstable and will need further treatment. Assessment of fracture severity includes evaluation of dorsal tilt (normal, 11 degrees palmar), radial inclination (normal, 22 degrees), radial length (normal, 1 cm), and ulnar variance (normal, neutral, and/or similar to the contralateral radius) on the posteroanterior view. A distal radius fracture with significant dorsal angulation will cause a visible deformity of the upper extremity so that the hand no longer seems to be located at the distal end of the forearm. Additionally, significant dorsal tilt left untreated limits wrist flexion and forearm rotation. The combination of dorsal angulation and radius shortening shifts the normal load-bearing pattern of the wrist toward the dorsum of the radial articular surface and the ulna. These changes in load bearing of the wrist correlate with decreased grip strength, decreased wrist motion, and poor functional outcome. Articular incongruity greater than 2 mm is associated with the development of carpal instability, arthritis, and poor long-term function. At the initial office visit, if there is any question regarding the degree of comminution, dorsal tilt, radius shortening, or articular step off, preoperative CT with coronal and sagittal reconstruction is helpful.

Treatment

The majority of distal radius fractures heal with immobilization and follow-up. Patients typically come to the office within 1 week of the injury. A follow-up visit at 2 weeks allows a second set of radiographs to be taken to confirm fragment position and conversion to a short arm cast with the metacarpophalangeal (MP) joints free. Shoulder, elbow, forearm, and digit range of motion should be encouraged to minimize stiffness. In patients with extraarticular fractures, the cast can be changed to a Velcro carpal tunnel splint at 4 weeks, which can be removed for bathing, arm hygiene, and early active protected motion. At 6 weeks, films are repeated and therapy begun, if needed. A final visit occurs 8 to 10 weeks after injury, and the patient is discharged.

Patients with high functional demands on their hands and initial films showing fractures with greater than 20 degrees of dorsal tilt, 10 mm of shortening, 2 mm or more of intraarticular step off, and dorsal comminution should be considered for surgery (Fig. 1A and B). Even when the postreduction films are improved, the fragments will often gradually slide back to their initial position and leave the patient and the surgeon to deal with the more difficult situation of a several-week-old fracture with associated swelling and malposition. In a patient with films showing acceptable reduction, the risk of surgery is compared with the risk of redisplacement. Younger patients with a few large fragments and adequate bone stock are less likely to suffer redisplacement. It is helpful to look at the initial film as it represents the worst case of redisplacement and decide whether this result would be associated with adequate wrist function. If not, a stronger case is made for surgery. If so, the risks and benefits are described as clearly as possible to the patient for input. For a patient with a persistently displaced fracture, the risks of surgery are compared with the risk of wrist deformity and pain.

A combination of external fixation, internal fixation, bone grafting, and supplemental pins is used for surgical treatment. Placement of external fixators has become simplified with standardization of devices. Two pins are inserted into the second metacarpal and two pins into the bare spot of the radius. The external fixator spans the fracture site. The superficial branch of the radial nerve should be protected while placing the pins. By the process of ligamentotaxis, stretching of the soft tissues and periosteum around the fracture pulls the fragments into place. It also unloads the wrist joint from the pull of the forearm muscles and thus decreases the tendency for redisplacement. The wrist should not be "overstretched," and overstretching can be prevented by closely watching the distraction at the midcarpal joint and assessing for extrinsic tightness of the extensor tendons. Overdistraction results in stiffness and an increased risk for reflex sympathetic dystrophy. Because simple ligamentotaxis may not restore alignment, open reduction may be necessary. Reduction is assessed by portable fluoroscopy. A small 1-cm incision is made dorsally just proximal to Lister's tubercle between the third and fourth extensor compartments, the fracture site entered, and the fracture tilt improved with a periosteal elevator. To hold and buttress the articular surface, iliac crest bone graft or bone substitutes can be placed through this incision. If palmar tilt has been reestablished but there is a risk for loss of

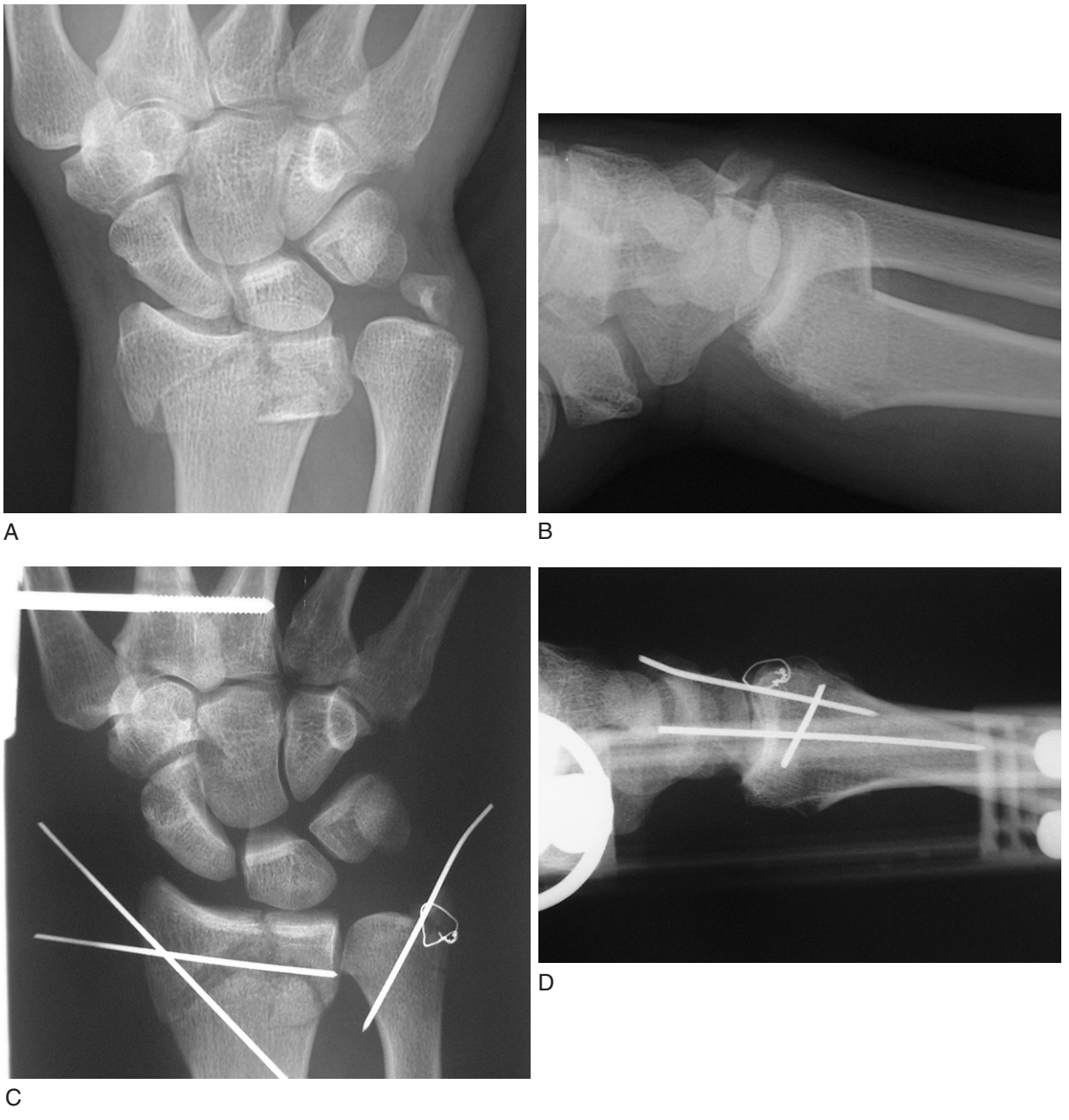


Figure 1 • Anteroposterior **(A)** and lateral **(B)** views of an intraarticular distal radius fracture with significant shortening and dorsal tilt. There is an associated rotated ulnar styloid fracture. Anteroposterior **(C)** and lateral **(D)** views after external fixation and supplemental K-wire fixation. The distal end of the ulna was thought to be unstable and was therefore opened and fixed with a K-wire and an intraosseous wire.

correction, 0.045-inch pins can be introduced percutaneously through the radial styloid across the fracture into the more proximal end of the radius (Fig. 1C and D). Percutaneous pins are not generally combined with bone grafting to preserve palmar tilt because of fear of infection of the bone graft.

Internal fixation is possible when the fragments are large and other pathology is present that needs attention, such as repair of a torn scapholunate ligament. Open reduction may also be necessary for displaced intraarticular fractures to ensure adequate alignment of the articular surface. The amount of soft tissue dissection required for placement of a dorsal plate is much greater than it is for a "mini-open" procedure to place a dorsal bone graft. Dorsal plates can irritate the overlying tendons, and despite newer low-profile plate designs, secondary surgery for plate removal is often necessary. In com-

parison, volar plates are tolerated quite well. For these reasons, many surgeons believe that it is best to avoid dorsal plates and rely on a dorsally placed bone graft through mini-incisions to control articular step off and tilt after placement of the external fixator. Others are more aggressive in open reduction and internal fixation of these fractures. Overall hand and wrist function is similar between groups of patients undergoing either external fixation or internal fixation of intraarticular distal radius fractures with displacement. Both groups do significantly better than those treated by cast fixation.

The newest technique is treatment of dorsally displaced radius fractures through a volar approach. New locking plate designs and extensive exposure are required for accurate reduction of the fracture fragments. Volar placement of plates obviates the need for external fixation. Volar hardware under the pronator quadratus is typically well tolerated, and delayed hardware removal is generally not necessary.

After treatment of the distal radius fracture, attention is diverted to the distal end of the ulna and the distal radioulnar joint (DRUJ). Large distal ulnar fractures are treated with either wires or pins; smaller ulnar styloid fractures with a stable DRUJ (no excessive anteroposterior mobility) can be ignored. The fixator is kept in place for 6 to 8 weeks, depending on the assessment of union, and therapy is begun. Motion of the shoulder, elbow, and fingers is maintained while the fracture heals.

Acute Scaphoid Fractures

The scaphoid bone links the proximal and distal carpal rows, and integrity of this bone is critical for maintenance of the normal mechanics of the wrist. Compressive force along the volar axis of the scaphoid causes the bone to assume an apex dorsal angulation when fractured across its waist. Over time (weeks to months), the volar aspect of the fragments is reabsorbed as a result of micromotion. If left to heal in this position, the scaphoid would have an apex dorsal angulated shape or "humpback" deformity. Scaphoid humpbacks cause carpal malalignment, carpal instability, and progressive radiocarpal degenerative changes over time. Apex dorsal angulation of unstable scaphoid fractures is a related, but distinct injury pattern after a ruptured scapholunate ligament. When the scaphoid is intact in these injuries, the longitudinal compressive force across the scaphoid causes the entire scaphoid to flex (or, in another way of thinking, for it to assume an apex dorsal angulation with the radius). As the scaphoid flexes, the lunate extends or points dorsally or assumes a DISI (dorsal intercalated segment instability) injury pattern.

Another critical concept in the treatment of scaphoid fractures is the relatively poor blood supply to the proximal fragment after a fracture; the majority of the bone surface is articular cartilage, and there is little area for vessels to enter. Most of the blood supply enters the scaphoid along the dorsal ridge, distally on the bone, and flows in a retrograde manner. Because of the retrograde supply, transverse fractures have a high incidence of interrupted blood flow to the proximal fragment, thereby resulting in avascular necrosis of the proximal fragment. Poor blood flow to the proximal fragment is one reason for the slow healing time of scaphoid fractures.

Scaphoid fractures may initially be seen in one of three ways: acute injuries with snuffbox tenderness after a fall onto the outstretched wrist, subacute injuries weeks to months after a self-treated "wrist sprain" in which the pain never quite resolved, and chronic injuries with old fractures and changes in carpal bone architecture along with changes in the articular surface of the radius and adjacent carpal bones. This chapter focuses on patients with acute injuries. Such patients can be of any age group, but in general, these fractures occur in active young adults, with fractures occurring in men more frequently than in women.

Examination reveals the area of maximal tenderness to be over the snuffbox and at the tubercle of the scaphoid. A scaphoid series with a minimum of four views is obtained. A posteroanterior view with the wrist ulnarly deviated is helpful to align the scaphoid for viewing along its long axis. Particular emphasis is placed on looking for signs of other injuries, especially disruption of the scapholunate ligament. Views of the opposite wrist are helpful for comparison. If snuffbox tenderness is still present despite normal films, the patient should be placed in a well-molded short arm thumb spica cast and asked to return in 2 weeks for repeat films. Alternatively, CT or MRI of the wrist is an option for immediate diagnosis. If the patient is still tender at the repeat 2-week visit, CT or MRI is performed and the splinting continued. Wrist immobilization is continued until the patient is pain free or additional imaging studies are negative.

Treatment Options and Surgical Decision Making

Scaphoid fractures should be categorized as stable or nonstable. Stable fractures include nondisplaced fractures of the waist and distal pole, but treatment of such fractures is controversial. Satisfactory results can be obtained in nondisplaced or minimally displaced (less than 1 mm) fractures with long arm thumb spica immobilization for 3 weeks, followed by short arm casting until healing is confirmed by plain films or CT. Prevention of pronation and supination of the wrist with elbow immobilization decreases micromotion at the fracture site, and several studies have implied more rapid healing with above-elbow casts. Distal pole fractures heal

faster than middle third or proximal pole fractures, and the above-elbow component may be omitted. In general, patients should be educated about the slow healing of scaphoid fractures. Over the first several weeks, radiographs are taken frequently and casts are changed often to ensure immobilization and prevent fracture displacement. Afterward, patients are seen monthly until healing is complete. Nondisplaced fractures treated promptly have a 90% to 95% healing rate. More recent literature suggests that internal fixation, even for stable fractures, has certain advantages over cast immobilization because it avoids prolonged immobilization.

Unstable fractures are those with more than 1 mm of displacement, fractures of the proximal pole, and fractures initially seen more than 4 months after injury. They require operative intervention. Although the surgical technique depends on the location of the fracture and surgical expertise, the goal is to anatomically align the scaphoid and obtain secure fixation. A volar approach is useful for apex dorsal fractures because it ensures adequate fracture visualization and reduction and placement of a volar bone graft. A dorsal approach may be more useful for proximal pole fractures that do not require bone grafting. Fixation options include differential pitch compression screws or K-wires. In cases without volar bone loss or resorption, compressive screws placed across the fracture are effective in obtaining fracture union. Placement of the screw requires experience, and it is especially difficult in patients with volar bone loss. K-wires are an effective and simpler method of maintaining reduction. They are useful for chronic fractures when a large corticocancellous graft is placed volarly between the fracture fragments. Jigs can be difficult to place in this instance because of the relative instability of the construct, as opposed to K-wires, which can efficiently spear the fracture fragments and the graft.

Dislocations of Carpal Bones

Carpal bone dislocations are high-energy injuries that occur during motor vehicle accidents or following falls from a significant height. Patients with these injuries seek medical attention for wrist pain and swelling. Because of the frequency of associated injuries, attention is often shifted to life-threatening issues, and in the absence of a fracture, the dislocation is often missed. The injuries are severe, and acute compression of the median nerve is a possibility.

A unifying pathomechanic description of the spectrum of carpal bone dislocations has been termed "progressive perilunate instability." In the cadaver laboratory, an array of carpal bone dislocations was created by forced wrist extension, ulnar deviation, and intercarpal supination. With wrist extension and ulnar deviation, the scaphoid extends whereas the lunate is locked into position by the tighter radiolunate ligament. Simultaneous supination of the hand tears the connections between the scaphoid and lunate and therefore ruptures the scapholunate ligament (stage I). Further hyperextension plus supination tears the ligaments between the lunate, scaphoid, and capitate (stage II). The next ligaments to tear are those between the lunate and triquetrum (stage III). If the lunate remains in the lunate fossa of the distal end of the radius and the midcarpal joint is dorsally displaced, the result is a perilunate dislocation. When the midcarpal bones return to be collinear with the distal part of the radius, the unstable lunate is pushed out of its facet volarly through the torn midcarpal ligaments (the space of Poirier), an injury termed a lunate dislocation (stage IV).

Fractures can be added to the concept of "progressive perilunate instability." In pure dislocations, energy is dissipated by tearing of the intercarpal and radiocarpal ligaments around the lunate, a condition termed a "lesser arc" injury. Rather than going around the lunate, the energy can travel through the carpal bones and cause fractures of the radial styloid, scaphoid, capitate, and triquetrum in so-called greater arc injuries.

Accurate diagnosis requires careful evaluation of a true lateral wrist radiograph because a posteroanterior radiograph can look relatively normal. With all wrist injuries, the capitate must remain normally aligned in the distal facet of the lunate on all views. Displacement of the lunate on the lateral radiograph is known as the "empty teacup" sign and is indicative of carpal dislocation. Early reduction is performed to relieve compression on the median nerve and because reduction is easier in the acute setting. To reduce the dislocation, anesthesia is induced and traction applied for at least 10 minutes. The wrist is extended, and volar force is applied to the palm to reduce the lunate and keep it in proper position. The wrist is gently flexed to seat the capitate on the distal articular surface of the lunate. Closed reduction is usually possible, and postreduction films are obtained to identify any missed fractures and plan for later operative repair.

Treatment Options and Surgical Decision Making

In most cases, because of the unstable nature of these injuries, they require surgical intervention after reduction. The only situation in which closed reduction alone is used is in a low-demand patient with a scapholunate interval less than 2 to 3 mm after reduction and without flexion of the scaphoid or dorsal tilt of the lunate on lateral view. These cases can be managed by casting for 8 to 10 weeks with upward volar pressure on the distal pole of the scaphoid and dorsal downward pressure on the capitate. Close radiographic follow-up of the scapholunate interval is essential.

Most surgeons favor open reduction and restoration of carpal bone alignment with K-wire fixation and direct ligamentous repair (Fig. 2). Fractures are treated by stable internal fixation, which can be performed as a primary repair at the time of the initial reduction or as a delayed repair 4 to 7 days after injury when the swelling has begun to subside. Acute median nerve compression and an inability to achieve closed reduction are indications for immediate surgery.

Restoration of carpal alignment begins with the lunate. The lunate is exposed through a dorsal incision and a longitudinal capsular approach. It is fixed in its facet in neutral position (without dorsal or volar tilt) with a 0.045-inch K-wire emerging from the distal end of the radius. At each step of the procedure, bone position is confirmed by fluoroscopy. The remainder of the carpal bones are reduced around the neutral lunate with the aid of fluoroscopy to ensure adequate alignment. Torn ligaments between the proximal carpal row are identified and repaired if possible. Bone anchors can be used to facilitate repair. For the scapholunate ligament, the most important area is dorsal and distal. The sutures are left long to be tied without tension after K-wires are placed between the scaphoid and lunate. The scaphoid is extended from its flexed position by using K-wire joysticks. A towel clamp placed with its jaws between the lunate and scaphoid is used to decrease the bony interval. K-wires are passed between the scaphoid and lunate, and a supplemental pin is inserted between the scaphoid and capitate to prevent scaphoid flexion. When proper reduction is achieved, the proximal articular surface of the capitate should not be visible because it is covered by the lunate. K-wires are also placed between the lunate and the triquetrum. The pin from the radius to the lunate can usually be removed at this stage to allow wrist flexion for exposure. After reduction is achieved, the scapholunate ligament sutures or bone anchors are tied. Despite being more controversial, an extended carpal tunnel incision can also be made to inspect and repair the torn volar ligaments. The latter maneuver is performed to repair the important volar radiocarpal ligaments, to add support to the carpal bones so that they retain their surgically reestablished alignment, and to release the median nerve. Patients are kept is a short arm splint for 8 to 10 weeks, at which time the K-wires are removed and hand therapy begun.

Metacarpal Fractures and Dislocations

Numerous metacarpal fracture patterns exist, with the largest number of fractures occurring in the border digits. The majority of fractures heal with 3 to 4 weeks of splinting and early active motion. The surgeon must identify fractures with expected slow healing times or with the potential for healing with a problematic deformity. These fractures should undergo surgical treatment, which allows for early active motion and therefore fewer secondary problems of joint stiffness and tendon adhesion.

Metacarpal Base Fractures

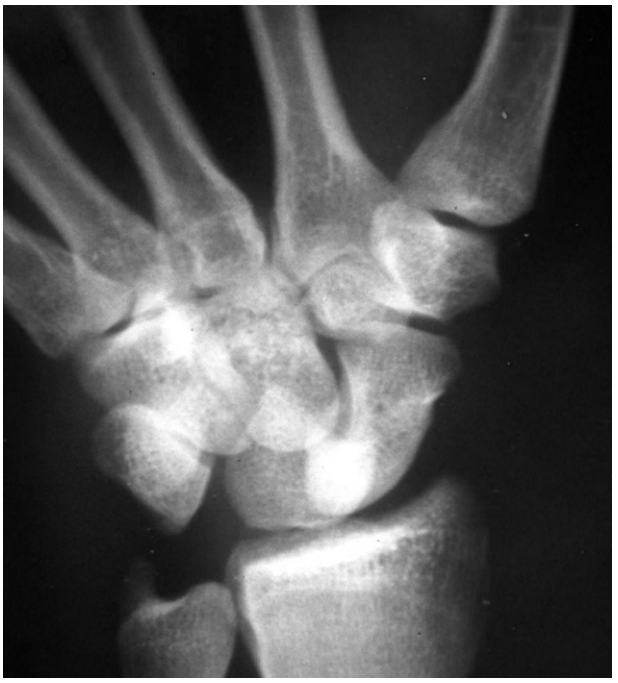
Intraarticular metacarpal base fractures of the thumb (Bennett's fracture) and the fifth finger (reversed Bennett's fracture) are common fractures requiring intervention, as opposed to extraarticular metacarpal base fractures, which are typically seen in the central digits and do well with splinting. Because of pull on the major fragment by the abductor pollicis longus (APL) and adductor for the thumb and by the extensor carpi ulnaris (ECU) for the fifth finger, these fractures often displace despite adequate immobilization. The initial emergency room films are important because the amount of displacement before reduction is the "worst-case" scenario for the fracture if splinting is not effective.

Significant displacement of the thumb metacarpal away from the trapezium should be treated by reduction and K-wire fixation. When possible, the treatment should be done in closed fashion. It is not necessary to pass the K-wire between the major and minor fragments when the minor fragment is small, although the reduction should be as close to anatomic as possible. K-wires passed through the metacarpal base into the trapezium neutralize the deforming force of the APL and allow the fracture to heal. Supplemental fixation between the thumb and index finger can also be used. For a large minor fragment, anatomic open reduction through a volar approach becomes more feasible and beneficial. For minor fragments greater than 30% of the articular surface, screws can be used to fix the fragments.

Two different approaches are used for reversed Bennett's fractures of the fifth metacarpal. The first method treats the fractures in a manner similar to Bennett's fractures; specifically, K-wires are used to neutralize the pull of the ECU by passing from the fifth metacarpal base into the fourth metacarpal base or into the hamate (or into both). The second approach entails a 3-week course of splinting followed by active motion. If symptoms develop, fusion of the fifth carpometacarpal (CMC) joint is performed. In general, the fifth finger is treated less aggressively than the thumb because of the comparatively lower demand on this digit and the relatively easier salvage procedure than a thumb CMC fusion.

Metacarpal Shaft Fractures

Patients with metacarpal fractures should be examined for positional deformities. The examination must be performed by actively (not passively) forming a fist of all four digits to give an accurate



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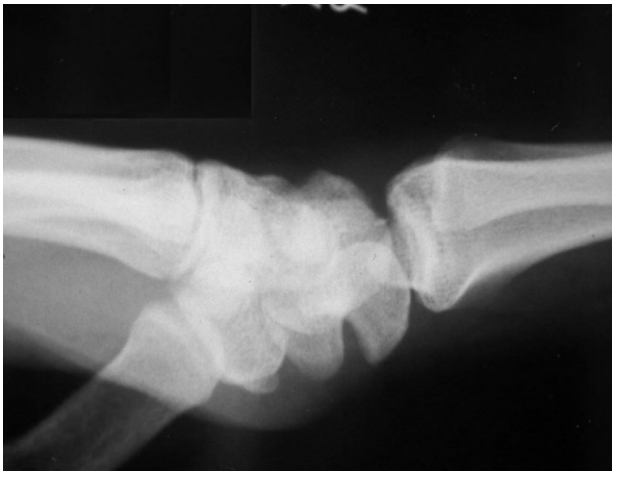

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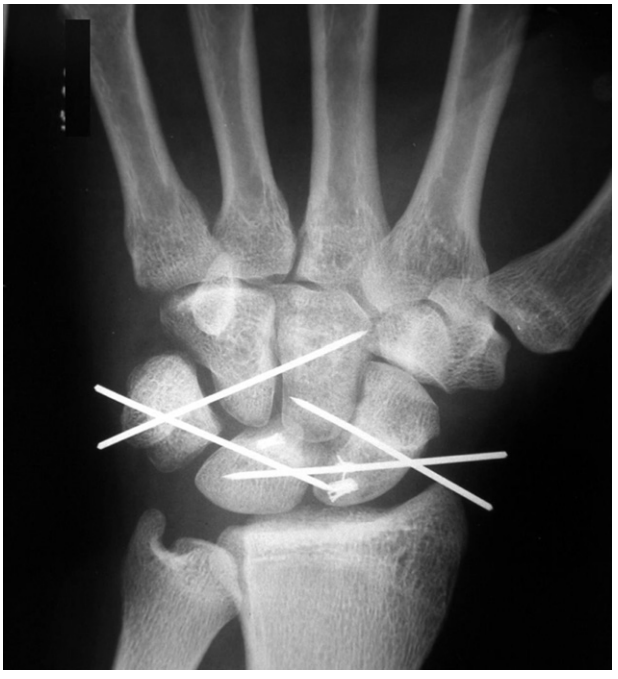

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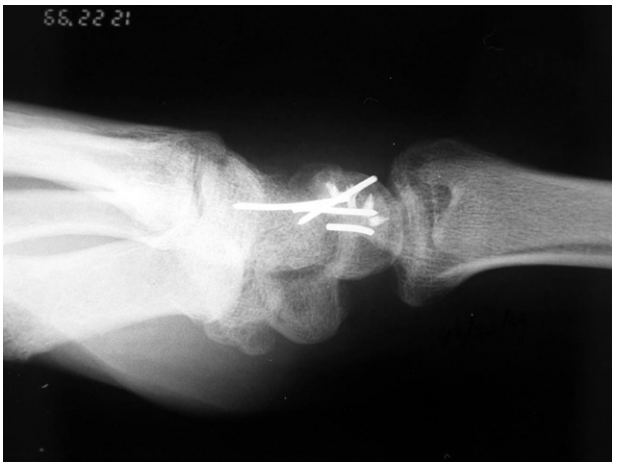

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Figure 2 • Anteroposterior (**A**) and lateral (**B**) views of a perilunate dislocation. On the anteroposterior view, the lunate has a triangular rather than a trapezoidal shape, and there is no clear interval between the scaphoid and lunate. Because no fractures are seen, this injury is often missed, and the unusual appearance of the torn scapholunate ligament is passed off as a "rotated film." On the lateral view, the lunate is seen out of its articular facet and has a "empty teacup" appearance. Anteroposterior (**C**) and lateral (**D**) posttreatment views of the same patient as in (**A**) and (**B**). A bone anchor with K-wire fixation was used to repair the avulsed scapholunate ligament.

assessment of fracture rotation. Rotation of the fracture fragments causing scissoring of the digits is one of the clearest indications for operative reduction and fixation. Other operative indications become more difficult to delineate. Gross displacement is an indication for surgery because the fracture fragments should overlap cortical surfaces by 50%. Metacarpal fractures tend to angulate apex dorsally. The more proximal the fracture and the greater the apex dorsal angulation, the further the metacarpal head will be directed into the palm. The index and middle fingers tolerate less angulation than the ring and fifth fingers because of the increased mobility of the ulnar CMC joints. Reduction is indicated for greater than 10- to 20-degree angulation of the index and middle fingers and for 30- to 40-degree angulation of the ulnar two digits. If the reductions cannot be held in a splint, fixation is indicated. Multiple metacarpal fractures are another indication for operative intervention because the adjacent digit is no longer sufficiently stable to provide stabilization of the distal fracture fragment via the deep transverse metacarpal ligament. Fractures with butterfly fragments are inherently more unstable than two-part fractures and tend to have greater motion between fragments. The greater motion leads to slower healing time, a longer period before clinical union, and therefore delayed resumption of active motion. Oblique fractures tend to be more unstable than transverse fractures and have a greater tendency for fracture rotation. Shortening of the metacarpal more than 4 mm may lead to extensor lag in the digit because of relative laxity of the extensor mechanism. Despite all of the relative indications for operative reduction and fixation, most metacarpal fractures heal with splinting for 3 weeks and resumption of early active motion when pain-free palpation at the fracture site (clinical union) has been achieved.

Patients often make their first office visit by 1 week after the initial injury. Radiographs are repeated, and if acceptable, a new plaster forearm-based splint is fashioned with the MP joints at 70 degrees to stretch the MP collateral ligaments and take tension off the intrinsics. No special effort is made to immobilize the interphalangeal (IP) joints with the splint. The following week (week 2), the splint is redone and finger rotation rechecked both clinically and radiographically. At week 3 or 4, depending on the inherent stability of the fracture, the plaster splint is switched to a removable aluminum splint held on the hand with an elastic bandage, and the patient can remove the splint as often as desired to actively open and close the fingers without resistance. By 6 to 8 weeks after injury, all restrictions are removed from the patient, and therapy is instituted if required. Alternatively, for nondisplaced fractures, patients can move directly to an aluminum splint (or a custom-fashioned Orthoplast forearm-based splint) held in place with an elastic bandage, and this splint can be removed several times a day to perform gentle active unresisted motion.

TREATMENT OPTIONS AND SURGICAL DECISION MAKING. Options for fixation of metacarpal shaft fractures include K-wires, intraosseous wiring, lag screw fixation, and plate and screw fixation. Although all of these options can provide adequate fixation, they also create scar with an increased chance for tendon adhesion, infection, and possible need for secondary surgery to remove the hardware. K-wires are a quick means to reduce and stabilize otherwise unacceptable fracture patterns. When placed percutaneously, there is minimal additional soft tissue trauma or scarring. The pins are removable as an office procedure, and no hardware remains after fracture treatment. However, K-wires do not provide as adequate stabilization for early active motion as other fixation techniques such as lag screws or plates. The bone fixation methods allow early active motion because of their stability against torsion and bending. The improved fracture stability is at the price of greater soft tissue dissection and scar formation.

In general, unstable transverse fractures do well with closed reduction and the introduction of K-wires just lateral to the metacarpal head in the area of the collateral ligament to cross the fracture site. Two 0.045-inch K-wires stabilize the fracture either by cortical purchase or by an intramedullary spanning device. One should continually check for rotational deformity by flexing the fingers into the palm. When it is difficult to place a K-wire in this manner, a salvage technique is to introduce the K-wire through the extensor mechanism at the level of the knuckle prominence into the metacarpal head and directly up the shaft of the metacarpal into the proximal fragment. However, this technique precludes early motion because of entrapment of the extensor mechanism.

Unstable oblique midshaft fractures with a fracture length at least two times the bone diameter can be treated with lag screws (Fig. 3), and greater fracture obliquity allows more room for screw placement. Four steps are necessary for this procedure. The fracture must be found (step 1), it must be reduced (step 2), and, most important, the fracture must be temporarily held in its new reduced position (step 3). Special bone clamps of varying shapes and sizes must be available to hold the fracture fragments in position while reduction of the fracture is checked radiographically and clinically. The fingers must be flexed into the palm to assess for rotational deformity. When satisfactory reduction is ascertained, the fracture can be fixed (step 4). In placement of a lag screw, the near cortex is overdrilled so that only the far cortex screw threads have purchase. The fracture line is compressed between the screw threads in the far cortex and the screw head against the near cortex. To prevent bone fracturing, the screw holes should be placed two screw diame-

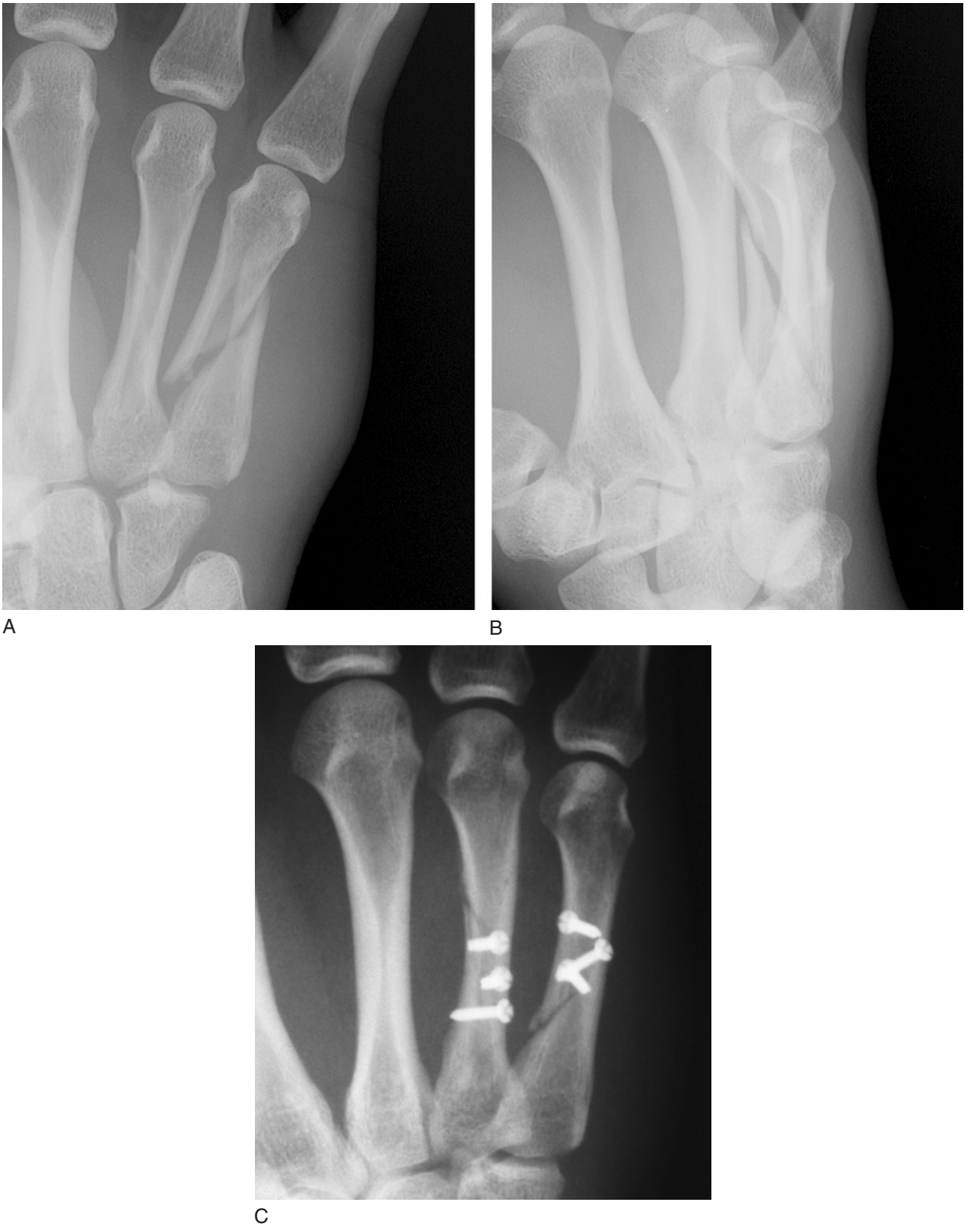


Figure 3 • Anteroposterior (**A**) and oblique (**B**) views of fourth and fifth finger oblique fractures of the metacarpal shafts. Apex dorsal angulation and significant instability are apparent on examination. **C**, Lag screw fixation of each fracture with three separate 2.0-mm screws.

ters from the fracture line. Two to three 2.0- or 2.7-mm screws are sufficient for holding the fracture. Lag screw fixation requires less soft tissue dissection than plate and screw fixation, and less hardware is exposed. As a result, there are fewer problems than with plate and screw fixation, including decreased tendon adhesion and hardware irritation. Plate and screw fixation is more appropriate when there is bone loss from severe comminution or when the fracture geometry is more transverse and does not allow placement of lag screws (Fig. 4). Soft tissue coverage of the plate is also a requirement, and strategies for local or distant flaps must accompany the plan for bony fixation in patients with associated soft tissue trauma.

Metacarpal Neck Fractures

The most common metacarpal neck fracture is the extraarticular "boxer's" fracture of the fifth finger. The fracture is apex dorsal, and the amount of angulation is assessed on a lateral film. Fractures angulated more than 40 degrees and seen within a few days of injury should have an attempt made at closed reduction with the Jahss maneuver. After a metacarpal or wrist block, the MP joint is flexed to 90 degrees to tighten the collateral ligaments around the metacarpal head. A reduction force applied to the proximal phalanx is effectively transmitted to the metacarpal head to reduce the metacarpal neck fracture. A forearm-based ulnar gutter splint is applied with the MP joint flexed and the IP joints straight for 3 weeks, at which time active motion can begin (Fig. 5).

Persistent apex dorsal angulation greater than 40 degrees can result in secondary deformities and functional problems. Loss of prominence of the metacarpal head along with hyperextension at the CMC joint brings the metacarpal head out of the palm. The intrinsics are functionally lengthened by the shortened distance to the proximal interphalangeal (PIP) joint, and therefore PIP extension is limited. The problems with an angulated metacarpal neck fracture must be balanced against the tissue injury and stiffness from operative intervention. Some surgeons, including the author, tolerate 60 to 70 degrees of apex dorsal angulation for the fifth finger, although much less for digits 2 to 4. Most patients do not complain of metacarpal "head-in-palm" or pseudoclaw deformity, but some are bothered by stiffness after operative intervention.

Metacarpophalangeal Joint Dislocation

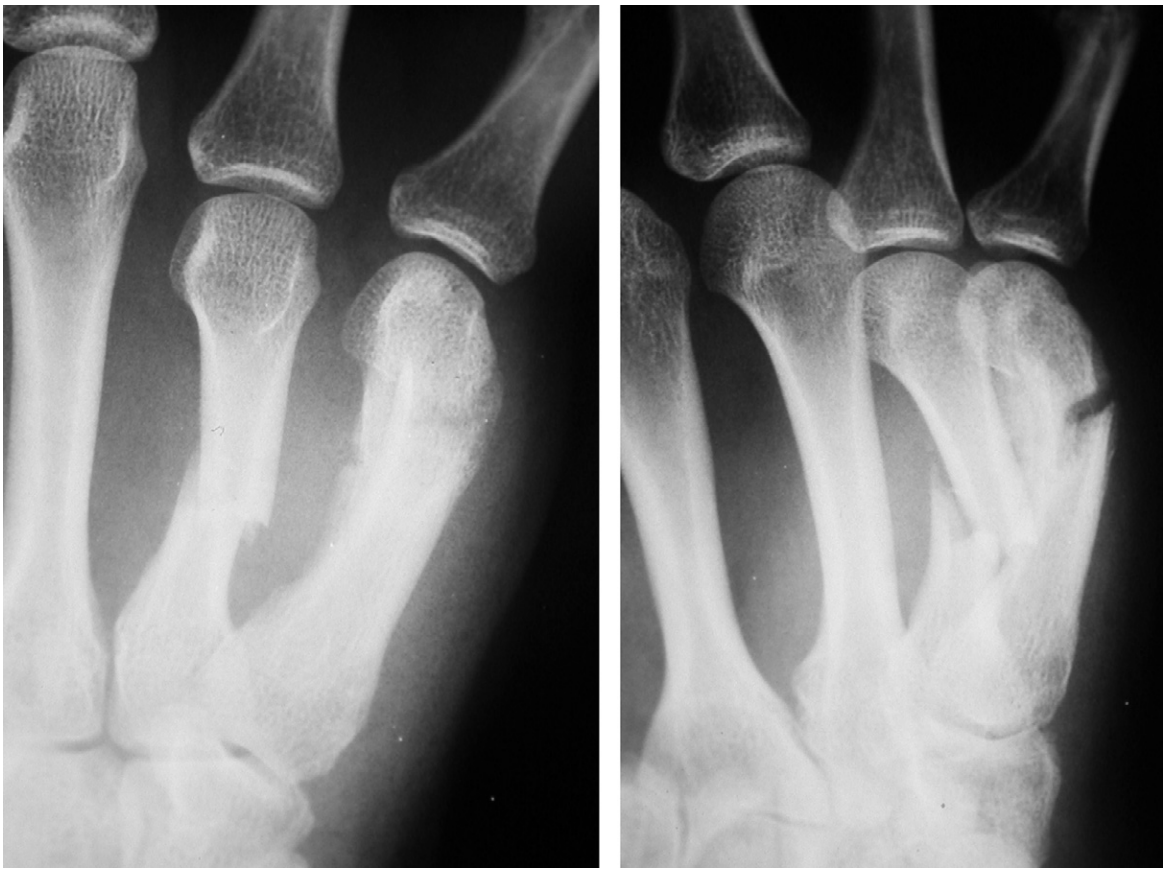
The majority of MP dislocations occur in a dorsal direction with the metacarpal head volarly displaced. The injuries occur as a result of hyperextension of the proximal phalanx. The index finger is the digit most frequently affected, followed by the fifth finger. Unlike the more common PIP dislocation, the volar plate remains attached to the distal bone. It is torn from its proximal loose membranous attachments to the metacarpal neck and remains attached to its distal insertion on the volar lip of the proximal phalanx.

MP joint dislocations can be of two types, depending on the position of this volar plate. In incomplete dislocations, the proximal phalanx is extended but is still in contact with the most dorsal portion of the articular surface of the metacarpal head. The joint space is not widened, and the proximal aspect of the volar plate is still oriented toward the palm. Incomplete dislocations can be reduced after obtaining adequate analgesia, extending the MP joint to 90 degrees, relaxing the flexor tendons with wrist flexion, and applying gentle dorsal pressure onto the base of the proximal phalanx. The idea is to slide the volar plate (which is oriented in the proper direction) back into position. This maneuver runs the risk of converting an incomplete to a complete dislocation, and the patient and surgeon must be prepared for open reduction if such conversion occurs.

Complete or complex MP dislocations have a widened joint space as a result of interposition of the volar plate between the articular surfaces. The metacarpal head travels through the area of the torn volar plate, and the head is resting volarly under the skin. For the index finger, the metacarpal neck is tightly flanked on its radial side by the lumbrical muscle and on its ulnar side by the flexor tendons. For the fifth finger, the tendons are radial, and the abductor digiti minimi is ulnar. Attempts at closed reduction are unsuccessful because of interposition of the volar plate. Additionally, distraction of the MP joint to help dislodge and replace the volar plate simultaneously tightens the tendon noose around the metacarpal neck. Treatment is by relaxation of the noose around the metacarpal neck in the operating room. A dorsal approach is typically used. While protecting the extensor tendon mechanism, the displaced volar plate is identified and divided longitudinally to allow the metacarpal head to be reduced through this division. Alternatively, a volar incision with release of the A1 pulley allows the flexor tendons to be retracted from the metacarpal neck, loosens the noose, and permits reduction of the metacarpal head. Volar approaches have a higher incidence of neurovascular injury than the dorsal approach. After reduction, the MP joint tends to be stable, and immediate active motion can usually be initiated.

Thumb Metacarpophalangeal Joint Dislocations and Fractures (Skier's Thumb)

When compared with finger MP dislocations, injury to the thumb MP joint is common because of its relatively unprotected position. Forced thumb radial deviation during falls can tear the thumb MP ulnar collateral ligament. Patients typically describe the injury clearly and have point tenderness and



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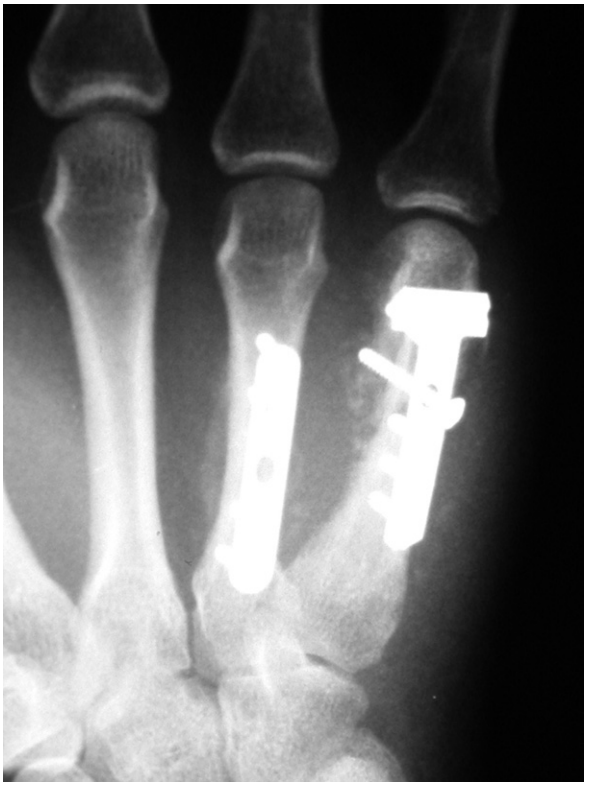

C

Figure 4 • Anteroposterior (**A**) and oblique (**B**) views of fourth and fifth finger transverse fractures of the metacarpal shafts. There is no significant obliquity of the fracture lines to allow for placement of lag screws. **C**, Plate fixation of each fracture with a 2.0-mm miniplate.

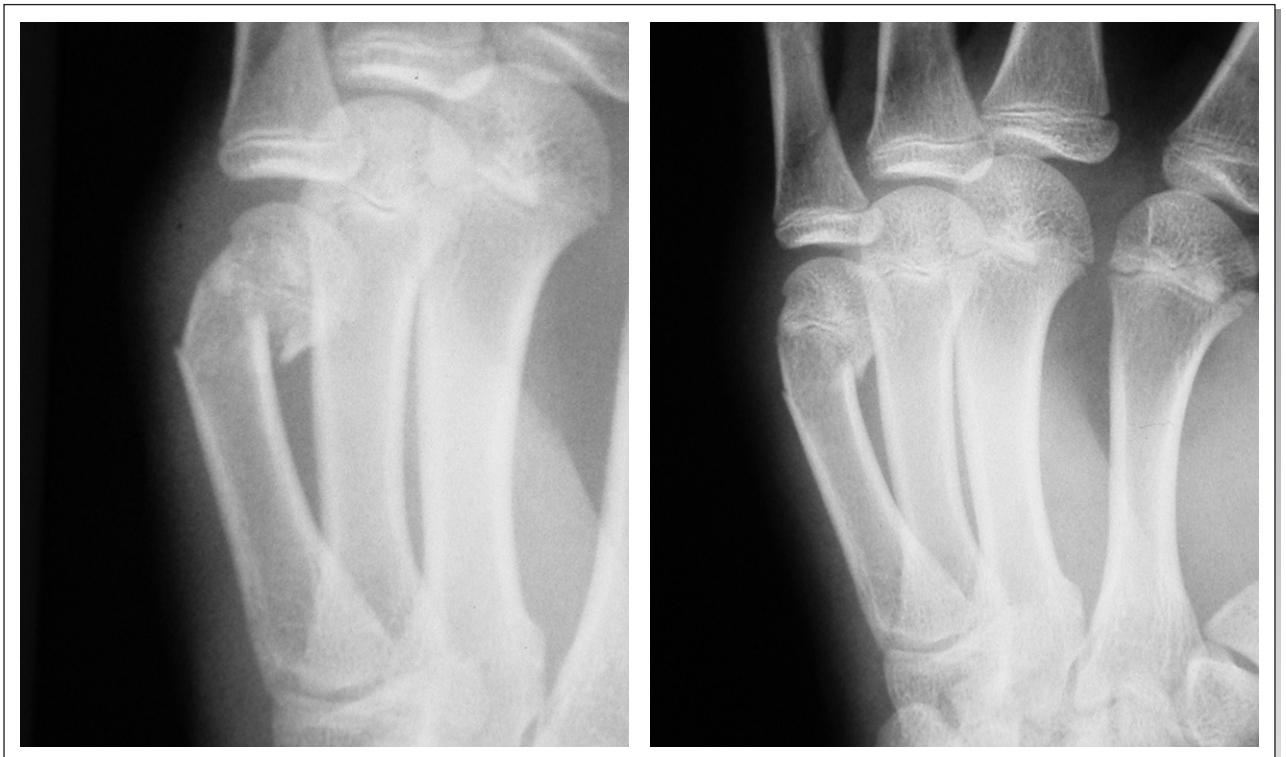

Figure 5 • A, Fifth metacarpal "boxer's fracture," oblique view, before reduction. **B,** Fifth metacarpal "boxer's fracture," oblique view, after the Jahss maneuver (closed reduction).

swelling on the ulnar side of the MP joint. Radiographs may or may not show an articular fracture of the base of the proximal phalanx.

The diagnostic physical examination is aided by median nerve block. Both in full extension and in 45 degrees of flexion, the ability of the thumb to resist radial deviation is tested and compared with that of the opposite hand. Although the numbers vary, opening up of more than 40 degrees total or opening of more than 15 degrees than the opposite side denotes total rupture of the ulnar collateral ligament rather than a partial tear. The "lack of an endpoint" to radial deviation is a finding that one gains with experience on examination and is also an indication of a complete tear. When the patient is well anesthetized, one should palpate firmly for the classic Stener lesion just proximal to the metacarpal head on the ulnar side of the thumb. A Stener lesion consists of avulsion of the displaced end of the collateral ligament off the proximal phalanx to a position superficial to the adductor aponeurosis. The interposition of the adductor aponeurosis precludes successful healing with closed treatment.

Radial abduction forces directed to the thumb also cause intraarticular fractures of the ulnar base of the proximal phalanx (i.e., skier's fractures). Rather than a rupture of the ulnar collateral ligament off the proximal phalanx, the fractured bone remains attached to the collateral ligament. If one sees the fracture well aligned, the collateral ligament is in its proper position, deep to the adductor aponeurosis and adjacent to the joint. Forceful physical examination in patients with skier's fractures can displace the fragment and should therefore be done under fluoroscopic visualization.

TREATMENT OPTIONS AND SURGICAL DECISION MAKING. Patients with partial tears indicated by less than 30 degrees of laxity or a solid endpoint on physical examination have some component of their ligament intact and can therefore be treated with a thumb spica cast for 1 month, followed by splinting in a removable Orthoplast splint until there is minimal pain and no instability with radial abduction. Similarly, patients with fractures in which the joint surfaces line up well are treated for 1 month in a cast and 1 month in a removable splint. The IP joint is left free to allow active IP joint motion.

When total rupture of the collateral ligament is diagnosed, it is difficult to ascertain the position of the collateral ligament. Therefore, the most effective and reproducible solution is operative repair. A curved incision is made on the ulnar side of the joint, with care taken to isolate and protect the radial nerve branches. The adductor aponeurosis is divided and nylon sutures are used to tag the ends for later reapproximation. The collateral ligament is rein-

serted at the ulnar side of the proximal phalanx with bone suture anchors. Although less common, proximal and midsubstance tears of the ligament can occur. Midsubstance tears can be repaired directly. Proximal tears can be repaired with suture anchors placed into the metacarpal head. In tears in all locations, sutures should also be placed between the collateral and the ulnar side of the volar plate to ensure proper positioning. The dorsal joint capsule is then reapproximated to prevent volar subluxation. Finally, if there is any question regarding the quality of the repair or the reliability of the patient, the repair can be protected with a 0.045-inch transarticular K-wire.

When a small bone fragment is present, it can simply be excised if it interferes with the passage of sutures. Larger fractures change the surgical decision making away from ensuring joint stability to preserving articular congruity. A fracture that encompasses more than 20% of the articular surface with more than 1 mm of displacement is an indication for open reduction and internal fixation. Reduction and fixation of the fracture simultaneously restore joint collateral ligament stability.

A few patients have equivocal physical examination findings. These patients are given the choice between a repeat physical examination in 1 week or additional radiology tests (such as ultrasound or MRI) to see the position of the collateral ligament relative to the adductor. Alternatively, they can undergo surgical exploration and repair.

Proximal Phalanx Fractures

Intraarticular Base Fractures

Analogous to a skier's fracture, intraarticular fractures are the result of abduction injuries to the fingers. The collateral ligaments avulse a fragment of bone and articular cartilage off the base of the proximal phalanx. When the fragment involves more than 20% of the articular surface, anatomic alignment of the fracture must be achieved for an optimal outcome. Fixation of the fracture fragment is difficult because of the tight confines between the metacarpal heads. One strategy is to predrill a 0.035-inch K-wire obliquely into the shaft of the proximal phalanx, reduce the fracture under direct vision, and drive the K-wire into the fragment. For larger fragments, screws and intraosseous wires can be used. The MP joints are splinted in full flexion for 4 weeks, at which point the pins are removed and active motion exercises begun. Buddy taping to the adjacent finger helps to protect against a repeat abduction injury to the digit.

Shaft Fractures

Phalangeal shaft fractures do well if active range-of-motion exercises can be started within 3 to 4 weeks of the initial injury. Nondisplaced fractures simply need to be protected for several weeks in a hand-based aluminum splint, with the patient removing the splint to perform nonresisted motion several times a day. Minimal displacement in a fracture without any clinical finger rotation can be treated by a forearm-based plaster splint with the MP joints at 70 degrees and the IP joints in extension. Patients are examined weekly for changes in alignment. At 3 weeks, the plaster splint is switched to an aluminum splint with active nonresisted motion. Fractures requiring emergency room reduction and fractures with an unstable geometry such as comminuted fractures and oblique fractures need even closer follow-up and careful splinting to ensure proper healing and to avoid malunion.

More problematic fractures are those that are unstable, often comminuted, with significant apex volar angulation. The angulation is caused by strong palmar pull on the proximal fragment by the interosseous muscles. If the apparent instability of the fracture is such that it will take longer than 4 weeks to begin motion, operative intervention should be considered. Other indications for surgery include an inability to control finger rotation, long oblique fractures, open fractures, and other associated finger injuries in need of repair.

Surgical options for reduction and fixation depend on fracture geometry, bone comminution, status of the soft tissues, and surgeon preference. The four steps in operative treatment of any fracture are to identify the fracture, reduce the fracture, temporarily hold the fracture to check finger alignment and bone reduction, and fix the fracture. For ease of setup and minimization of soft tissue trauma, percutaneous K-wire fixation is the preferred method, although one must accept less than rigid fixation (Fig. 6). Reduction of the fracture is achieved with longitudinal traction and digit flexion. The reduction is held with percutaneously placed bone clamps or, even more simply, by maintaining traction during pin insertion. Pins are placed through the phalangeal base near the collateral ligaments across the fracture line to rest either in an intramedullary position or across the opposite cortex. Patients are kept in a forearm-based splint until the pins are removed at 4 weeks, at which point unresisted active motion is begun. Gentle assisted PIP motion with stabilization of the proximal phalanx can be initiated before K-wire removal.

Intraosseous wires, K-wires, or some combination may be used for fractures that cannot be reduced and fixed percutaneously. The price for improved exposure and a more anatomic reduction is increased soft tissue swelling and scar formation. Dorsal incisions are preferred, and the extensor tendon mechanism is either split or retracted for fracture exposure. K-wires have great versatility for nearly every fracture pattern and do not require extensive soft tissue dissection for placement. They

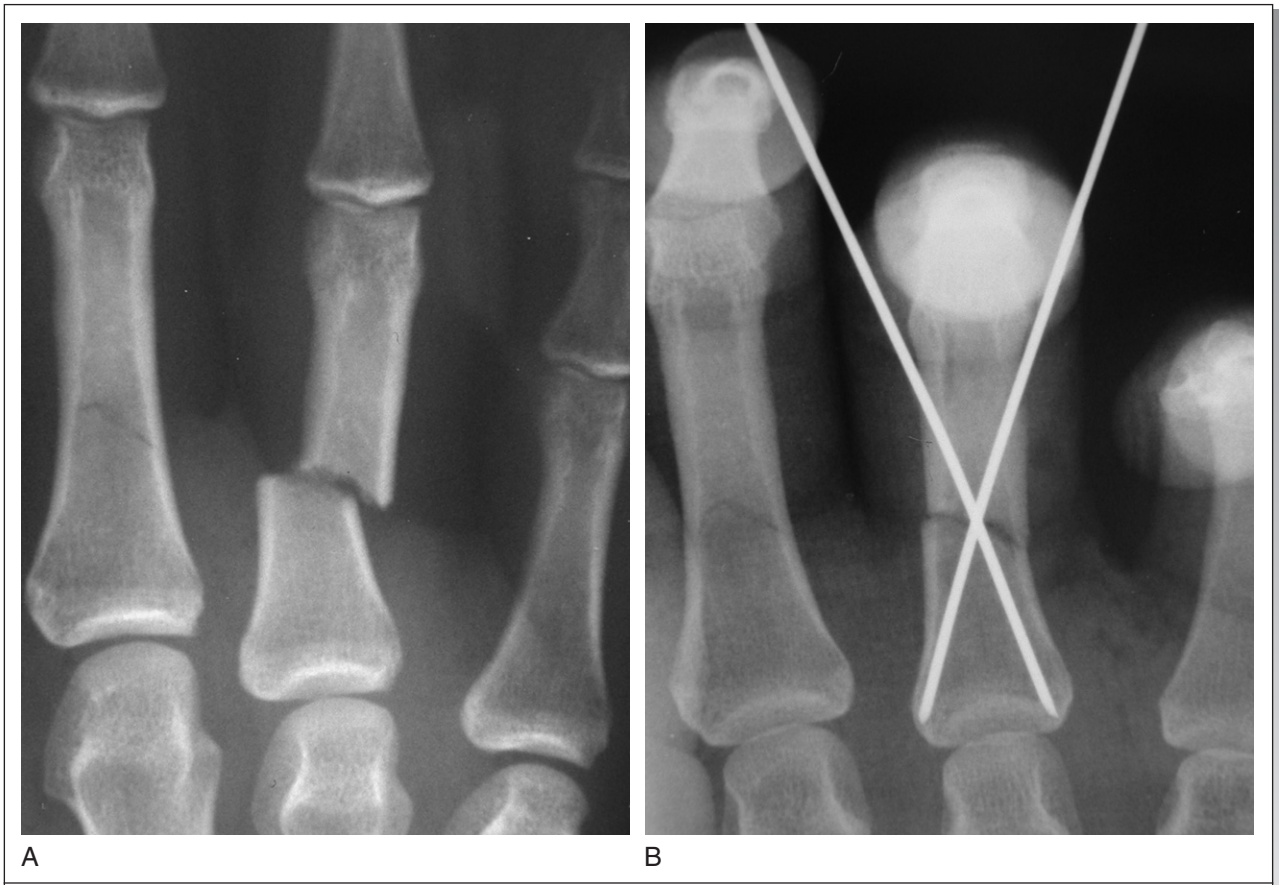

Figure 6 • A, Anteroposterior view of a ring finger proximal phalanx transverse fracture. **B,** Anteroposterior view of the finger in **A** after closed reduction and percutaneous K-wire fixation.

are low profile and facilitate soft tissue closure. Because they are removed percutaneously, no foreign body is left in the digit.

Lag compression screws in the digits provide more rigidity than K-wires do for long oblique fractures. After the fracture has been identified, reduced, and held, the screws are introduced after overdrilling the near cortex. Lag screws require more soft tissue dissection than K-wires. Two and preferably three screws are required for rigid fixation. Screw fixation is technically challenging because of the small working area, the difficulty in temporarily holding the fracture reduction during screw placement, and the tendency for the bone to crack if the fracture line is approached by the screws. Lag screws (either 1.5 or 2.0 mm) are more rigid than K-wires and thus allow immediate active range of motion. The screws are low profile and generally do not require later removal. Similar to other means of fixation, the PIP joints should be splinted in extension to prevent PIP flexion contracture and extensor tendon lag. Finally, plate and screw fixation requires the most soft tissue dissection, is the highest-profile construct, and often needs to be removed at a later time. Plate and screw fixation is associated with some of the highest complication rates; however, it is generally used in the most challenging situations when other more simple methods are insufficient.

Phalangeal Condylar Fractures

Condylar fractures are inherently unstable intra-articular fractures. In keeping with what has been said previously, nondisplaced fractures can be carefully observed, with unresisted motion initiated at 3 weeks. Most of these fractures, however, have some degree of displacement. Displacement greater than 1 to 2 mm requires reduction and fixation. Unfortunately, many of the fractures are impacted and resist attempts at closed reduction.

The fractures are opened, when necessary, through either a dorsal or midlateral approach. Dorsally, the PIP joint can be opened and the articular surface visualized directly between the central slip and one of the lateral bands. In the midlateral approach, the fracture is disimpacted from the lateral cortex, and the effectiveness of the reduction is confirmed fluoroscopically. Fixation is maintained with either K-wires or 1.5-mm screws. The key to a satisfactory result is anatomic reduction and stable fixation to allow early active motion. The patient

should expect to undergo a lengthy course of postoperative therapy to regain full PIP movement, and occasionally tenolysis is necessary.

Proximal Interphalangeal Dorsal Dislocations

Dorsal PIP dislocations are very common. Patients complain of a recent "jammed" finger or question why the PIP joint continues to be swollen and painful months after injury. Frequently, patients arrive in the office with their finger splinted for weeks because of a small volar chip fracture off the base of the middle phalanx.

The PIP joint is best thought of as a box with six sides. The top of the box is the central slip, the sides of the box are the collateral and accessory collateral ligaments, and the front and back of the box are the two articular surfaces. For a dorsal dislocation to occur, the base of the middle phalanx must be torn from its attachment to the volar plate (which remains in its proper position). A fracture, typically small and inconsequential, may occur in the base of the middle phalanx. Tears occur within the substance of the collateral ligaments bilaterally and allow the middle phalanx to dislocate out of its normal position onto the dorsal surface of the proximal phalanx. Often, one of the collateral ligaments tears more than the other and thus leads to angulation of the finger. These common dislocations are in contrast to the much rarer volar PIP dislocation, which represents an injury to the central slip. After closed reduction, volar dislocations should be treated much like an acute boutonnière injury.

The initial treatment of dorsal dislocations is to reduce the dislocation, obtain radiographs, and assess the joint for stability with range-of-motion exercises. PIP joints are reduced in the emergency room with a digital block, longitudinal traction, and pressure applied to the dorsum of the middle phalanx. Radiographs are obtained to ensure that the joint is indeed reduced and to examine the middle phalanx for fractures. The patient is placed in an aluminum splint for a few days for comfort and subsequently seen in the office.

The most critical aspect in management of these injuries in the office is obtaining and maintaining a concentric reduction while allowing early active range of motion. If on range of motion the middle phalanx stays seated on the head of the proximal phalanx, the patient can begin range-of-motion exercises with the finger buddy-taped to the adjacent digit. To counteract the tendency for PIP flexion contracture, the digit should be splinted in full extension at night. The patient is seen at 2 weeks, and if 80 degrees of PIP motion is present, a final check can be scheduled 2 to 3 weeks later. If the patient has either a PIP flexion contracture or less than 70 degrees of total motion, therapy is begun. The patient is seen every few weeks until at least 90 degrees of total PIP motion has been achieved.

Occasionally, because of large middle phalanx fractures, the PIP joint is unstable during range of motion, with the middle phalanx dislocating dorsally. In such cases, the dislocating force must be neutralized as healing takes place. One simple method is to splint the finger and prevent full extension but allow full flexion. Each week, the finger is allowed to extend 10 to 20 degrees more until full motion is achieved. A much more complex treatment is volar plate arthroplasty. The avulsed volar plate is advanced into the area of damaged articular cartilage of the middle phalanx. The volar plate is secured in position with a combination of internal sutures and pullout wires. Postoperatively, the finger is kept from extending and disrupting the repair. Both the simple solution of extension block splinting and the much more complex volar plate arthroplasty run the risk of a postoperative flexion contracture. Volar plate arthroplasty may also result in flexor tendon adhesions.

An alternative solution is ligamentotaxis across the PIP joint with K-wires and rubber bands. The Agee force-couple is one solution to create a downward force on the middle phalanx to prevent redislocation. Ligamentotaxis unloads the joint and assists in realigning the small bone fragments to create a volar lip of bone for the middle phalanx. The K-wires can be placed quickly under digital block anesthesia, and they require no special instrumentation other than an image-intensifying fluoroscopy unit. No scar tissue is created at the PIP joint, and the patient can immediately begin active range-of-motion exercises under the supervision of a hand therapist. The drawbacks to the device are that it is prominent on the finger and it runs the risk of becoming dislodged in a noncompliant patient. After 6 weeks, the K-wires are removed and the patient continues therapy until an acceptable range of motion is achieved.

Pearls and Pitfalls

- Most hand injuries heal with splinting and early active motion.
- Relative and absolute indications for surgical intervention exist for each of the injuries.
- The goal of surgical intervention is to repair deformities and assist the patient in resuming active motion by 3 to 4 weeks after the injury with a minimum of surgical trauma and risk to the hand.
- Many treatment alternatives exist for each injury, and selection depends on multiple factors, including patient and physician preference.

- The sequence in open reduction and internal fixation of fractures is to (1) *find* the fracture, (2) *reduce* the fracture, (3) *hold* the fracture while checking the reduction clinically and radiographically, and (4) *fix* the fracture.

ACKNOWLEDGMENT

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SUGGESTED READINGS

Agee J: Unstable fracture dislocations of the proximal interphalangeal joint. Clin Orthop 214:101-112, 1987.

Amadio PC, Taleisnik J: Fractures of the carpal bones. In Cooney WP, Linsheid RL, Dobyns JH (eds): The Wrist, Diagnosis and Operative Treatment. St Louis, Mosby, 1998, pp 809-864.

Cooney WP: Fractures of the distal radius. In Cooney WP, Linsheid RL, Dobyns JH (eds): The Wrist, Diagnosis and Operative Treatment. St Louis, Mosby, 1998, pp 310-355.

Glickel SZ, Barron OA, Eaton RG: Dislocations and ligament injuries in the digits. In Green DP, Hotchkiss RN, Pederson WC (eds): Green's Operative Hand Surgery. New York, Churchill Livingstone, 1999, pp 772-808.

Kapoor H, Agarwal A, Dhaon BK: Displaced intra-articular fractures of distal radius: A comparative evaluation of results following closed reduction, external fixation, and open reduction with internal fixation. Injury 31:75-59, 2000.

Mayfield JK, Johnson RP, Kilcoyne RK: Carpal dislocations: Pathomechanics and progressive perilunar instability. J Hand Surg 5:226-241, 1980.

Stern PJ: Fractures of the metacarpals and phalanges. In Green DP, Hotchkiss RN, Pederson WC (eds): Operative Hand Surgery. New York, Churchill Livingstone, 1999, pp 711-771.

Hand and Finger Amputations

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Hand and finger injuries are one of the most common reasons for plastic surgeon consultation in the operating room. These injuries should be approached with consideration of the patient's needs and desires. In general, hand function is the most critical consideration, but in some patients, aesthetic considerations should not be understated. This chapter provides a logical approach to hand and finger amputations and outlines the goals of reconstruction.

Etiopathogenesis

Hand and finger amputations are most commonly due to trauma. Crush or avulsion injuries are the most common mechanisms. With severe crushing or maceration of tissues, it is unlikely that salvage options exist. Even with sharp injuries, such as index tip amputations in a heavy laborer, amputation is often the most logical solution.

In addition to trauma, other causes of hand and finger amputations include tumors and vasculitis. Occasionally, severe contractures or other conditions that interfere with overall hand function are best treated with amputations.

Diagnostic Studies

The most important diagnostic tool for hand injuries is an accurate examination. The experienced hand surgeon can pinpoint most injuries and pathology by clinical examination alone. Concomitant injuries to the hand can exist, and they should be fully explored. A complete history, including the mechanisms of trauma, preinjury function of the hand, handedness, work and social requirements, along with patient goals and motivation, must be obtained.

Hand radiographs in three views (anteroposterior, lateral, oblique) must be obtained on all injuries, including dedicated finger views when appropriate. The hand radiographs will rule out additional fractures or foreign bodies. Occasionally, the wrist should be evaluated with CT scanning or MRI.

Reconstructive Goals

Regardless of the anatomic level, the primary goals of amputation include the following:

1. Preservation of functional length
2. Provision of durable coverage
3. Preservation of sensibility
4. Prevention of neuromas
5. Prevention of joint contractures
6. Minimal morbidity
7. Early prosthetic fitting
8. Early return to activities of daily living

A well-planned amputation in the upper extremity should be considered reconstructive rather than ablative. Furthermore, the surgeon should strive for a single definitive procedure so that the need for amputation revision is minimized.

To accomplish these goals, the surgeon must understand and take into account the patient's occupation, functional status, and emotional attitude toward the proposed amputation. Amputations should be deferred in a wavering patient because clear consent must be obtained.

Amputations of the Fingertip

Treatment

If distal phalangeal bone is exposed, the bone may be shortened and the soft tissue either primarily

closed or left to heal by secondary intention. This can be accomplished with a digital block for anesthesia. The digit is exsanguinated with the use of a Penrose drain, which is wrapped at the base of the finger as a tourniquet. The tip is irrigated and any foreign material removed. Digital nerves should be transected under traction to leave the stumps within normal tissue. After bony coverage with viable soft tissue has been achieved via bone shortening, the soft tissue may be appropriately tailored. Primary closure should be performed in a tension-free manner with absorbable sutures. In cases in which the skin cannot be reapproximated, the soft tissue may be allowed to close by secondary intention, which is acceptable if the open wound is less than 1 cm^2. The wound, examined weekly, will close within 3 to 4 weeks.

A lacerated nail bed is often encountered and should be explored. After removal of any damaged nail, the nail bed is repaired under loupe magnification with 6–0 absorbable sutures.

Alternatives

Excessive soft tissue loss may not allow for primary or secondary methods of closure. Skin graft and flap procedures have been described to cover these wounds. In general, skin grafts tend to be less durable and have poorer sensibility. Pedicled flaps are preferred.

A number of local flaps have been developed for coverage of exposed fingertip bone. The Atasoy-Kleinert V-Y flap includes skin and pulp from the volar surface of the digit and can be used only if adequate volar tissue is present. The flap extends from the site of injury to the distal interphalangeal (DIP) crease and is advanced in V-Y fashion. Full-thickness skin is elevated with care taken to preserve the digital nerves and vessels laterally. Hypersensitivity, paresthesias, and cold intolerance have been reported as complications resulting from the use of this flap. In oblique injuries, Kutler lateral V-Y flaps may be used instead. These are laterally based V-Y flaps advanced around the oblique defect. Similar complications to those of the Atasoy-Kleinert flap have been reported.

The cross finger flap uses a laterally based, full-thickness flap from a neighboring digit. In the standard technique, dorsal skin is used to provide skin coverage for a volar tip wound on the adjacent finger. After débridement of the recipient bed, the flap is designed with the use of a template such that it allows easy and comfortable postoperative positioning. The two digits must be positioned properly to prevent torsion of the flap pedicle. Most flaps are based laterally and raised over the middle phalanx (Fig. 1). The paratenon of the extensor tendon is preserved to allow full-thickness skin grafting. Cleland's ligaments may need to be incised to completely mobilize the flap. Twelve to 14 days is sufficient time for neovascularization before division and inset.

A flap from the thenar pad may also be used for coverage of index and middle fingertip amputations with exposed bone (Fig. 2). A proximally based flap is raised just ulnar to the thumb metacarpophalangeal (MCP) skin crease. A "W" design facilitates partial closure of the donor site. Careful placement prevents excessive flexion of the proximal interphalangeal (PIP) joint, which would lead to postoperative stiffness. After division of the flap in 2 weeks, the remaining defect can usually be closed primarily. The thenar flap is not as useful for ring and small finger amputations because these digits do not comfortably flex toward the thenar eminence.

Specific Considerations

NEUROMA FORMATION. Exposed or damaged digital nerves should be transected proximally and allowed to retract within the wound. Such management allows for soft tissue coverage and padding of the nerve end and decreases the risk for painful neuroma formation. Care should also be taken to ensure that the cut end does not lie beneath a skin crease where it may be subjected to compression with flexion of the joint. If a neuroma does develop, it can be treated with a course of desensitization therapy. If the pain persists, reexploration and more proximal transection can be performed.

HOOK NAIL DEFORMITY. A hook nail deformity occurs when the distal phalangeal bone is trimmed proximally to the nail bed. Care should be taken to ensure that the nail bed and distal phalanx remain in the same distal plane. Revision of the nail bed to the appropriate level corrects this deformity.

PRESERVATION OF THE INSERTION OF THE FLEXOR AND EXTENSOR TENDONS. Care should be taken to ensure that skeletal shortening of the exposed distal phalanx does not disrupt the insertion of the flexor digitorum profundus (FDP) or extensor slip. If skeletal shortening compromises the flexor or extensor system of the digit, alternative procedures for length preservation should be considered.

BOXY TIP. If fingertip skin is left redundant laterally, a 'boxy tip" deformity may result. Tapering of the soft tissue at the time of the initial procedure decreases the need for revision of the amputation site.

Amputation from the Distal Interphalangeal Joint to the Metacarpophalangeal Joint

Treatment

Amputations through the DIP joint are treated by skeletal shortening and primary closure. The tech-

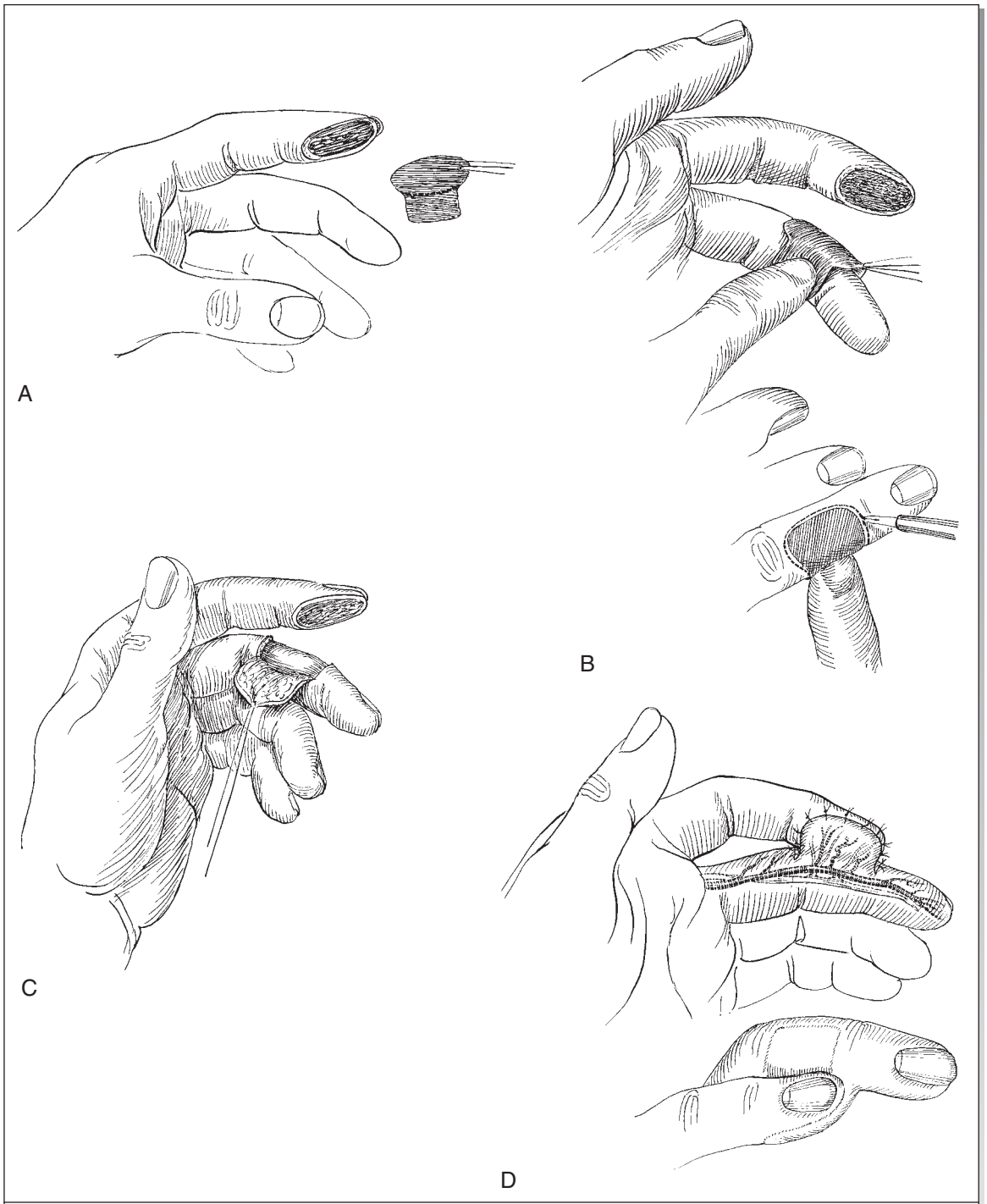

Figure 1 • A, Creation of a template to match the size of the defect. **B,** Tracing of the template over the middle phalanx of the adjoining digit. **C,** Elevation of the flap with preservation of the paratenon. **D,** Inset of the flap with skin grafting of the donor site. (From Chase: Atlas of Hand Surgery. Philadelphia, Saunders, 1973.)

niques are similar to those discussed for tip amputations. The middle phalanx should be rongeured and tapered to approximate the shape of the digital tip. The flexor and extensor tendons should not be sutured together because such suturing results in the quadriga effect (discussed later).

Amputations through the middle phalanx are divided into those distal and those proximal to the insertion of the flexor digitorum superficialis (FDS). When an amputation is distal to the FDS insertion, the remaining middle phalanx may function for grasping, and attempts should be made to preserve

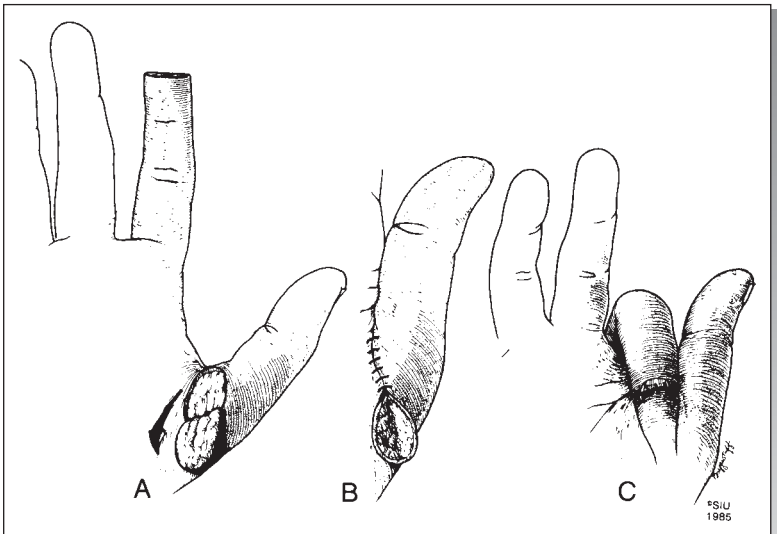

Figure 2 • Thenar crease flap. **A,** A modified thenar flap is radially based and elevated from the metacarpophalangeal joint flexion crease of the thumb. **B,** The donor site is closed primarily by flexing the thumb. **C,** The fingertip is flexed into the flap. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, WB Saunders, 1990.)

the length of the insertion by the use of local flaps for soft tissue coverage of exposed bone. If the amputation is distal, rongeuring and primary or secondary closure may also be used, again while ensuring that the FDS tendon remains intact. There is no need to preserve length when function of the FDS tendon has been compromised by a more proximal amputation, including amputations from the level of the middle phalanx to the MCP joint. In these cases, skeletal shortening and closure serve only cosmetic functions because the PIP joint has lost function. Amputations through the PIP joint are treated similar to those involving the DIP joint in terms of tapering of the condyles to achieve a more natural shape.

Alternatives

REPLANTATION. A single-digit amputation proximal to the FDS insertion is a relative contraindication to microvascular replantation because of the likelihood of adhesions involving both the FDS and FDP tendons. However, single-digit amputations distal to the FDS insertion may be replanted with good to excellent range of motion and function.

RECONSTRUCTION. Amputations of the middle phalanx may require reconstructive procedures to preserve length (and function) of the PIP joint. Local flap alternatives discussed for digital tip amputations may also be applied to the middle phalanx.

Specific Considerations

QUADRIGA SYNDROME. Quadriga is a term describing tethering of the FDP tendons. It originally referred to ancient Roman chariots with four horses driven by a single tethered rein. The FDP tendons of the middle, ring, and small fingers share a common proximal muscle. If the FDP tendon in the amputated digit is tethered to its extensor counterpart, its excursion will be limited by the extensor pull. Therefore, full composite flexion (clenched fist) is not possible if one FDP tendon is tethered. Quadriga is avoided by transecting the FDP tendon and allowing it to retract proximally.

LUMBRICAL-PLUS FINGER. An amputation proximal to the insertion of the FDP tendon may lead to a "lumbrical-plus" digit. The lumbrical muscle arises from the FDP and inserts onto the lateral band of the extensor apparatus. If the distal insertion is transected, the FDP tendon and its attached lumbrical muscle begin to migrate proximally. Such migration places increased tension on the lumbrical tendon, which extends the PIP joint. When flexion is attempted in a lumbrical-plus finger, paradoxical PIP joint extension results because the force is transmitted via the lumbrical to the lateral band of the extensor tendon. This deformity may be addressed by detaching the connection between the lumbrical and the lateral band.

Amputation of the Thumb

All possible efforts are made to replant or revascularize a severely injured thumb; however, with unsalvageable injuries, completion amputation may be performed. Congenital anomalies and some types of tumors may also require amputation of the

thumb. Preservation of thumb length is critical and should be considered before thumb amputation.

Treatment

Distal thumb amputations, or those between the tip and the interphalangeal (IP) joint, leave the patient with sufficient length for acceptable function. Therefore, skeletal shortening with primary or secondary closure may be used, and the principles and techniques follow those previously discussed. With more proximal amputations, skeletal shortening to gain soft tissue coverage should be performed only when the alternatives have been exhausted. At all levels of injury, it is likely that the patient will wish some sort of reconstructive surgery to provide additional length, improve aesthetics, allow greater sensibility, or improve function (or any combination of these goals).

Alternatives

A myriad of procedures have been developed in an attempt to reconstruct the thumb after amputation. Microsurgical techniques allow for replantation at the time of injury and provide reliable soft tissue and bony reconstruction of the injured thumb. These techniques have replaced many of the pedicled flap procedures for the injured thumb. However, several reliable methods are available when microsurgery is not an option. In these instances, the surgical approach to the injured thumb is based on the level of thumb loss: the *distal*, *middle*, and *proximal* zones.

The distal third of the thumb is the zone between the tip and the IP joint. Functional impairment after amputation in this zone is minimal; therefore, lengthening procedures are not generally required. These injuries are often treated in a fashion similar to digital tip amputations. Reliable soft tissue coverage can generally be achieved by means of primary or secondary closure, as well as split-thickness skin grafting. Local skin flaps, including V-Y advancement flaps and the volar advancement flap, have also been advocated. The volar advancement flap is especially suited to the thumb. Injuries involving less than 2.5 cm of volar soft tissue can be reliably covered with this local flap. Flap necrosis and flexion contracture are not as commonly seen in the thumb as in the digits. With more extensive soft tissue loss, distant flaps from the hand, including cross finger flaps, radial sensory innervated cross finger flaps, axial flag flaps, and neurovascular island pedicle flaps, may be used to preserve some amount of sensation to the volar skin of the thumb.

Amputations through the middle zone of the thumb include those between the IP joint and the MCP joint. For purposes of reconstruction, they may be further subdivided into proximal, middle, and distal third amputations. Amputations occurring distally on the proximal phalanx retain sufficient bony support for reasonable thumb function. The guiding principle in reconstruction of these injuries is "relative" lengthening of the first metacarpal and remaining proximal phalanx. Such lengthening is achieved by deepening the first web space to increase the thumb-index interval and thus preserve the functions of pinch and grip. Lengthening is most easily achieved with a four-flap Z-plasty of the first web space (Fig. 3). A number of other techniques have been described, including the use of dorsal hand rotational flaps, radial forearm flaps, and other distant flaps to increase the first web space.

Once amputation reaches the middle third of the thumb (MCP joint), bone length is not sufficient for adequate thumb function. In these cases, lengthening procedures are absolutely indicated because amputation will have a severe effect on postinjury function. Up to 2 cm of bone and soft tissue must be provided to achieve acceptable results. Several sources of bone and soft tissue have been used: osteocutaneous free tissue transfer, transfer of concomitantly damaged digits (pollicization), and metacarpal lengthening by means of osteodistraction.

Amputation of the proximal third (zone) of the thumb is essentially total thumb loss. In these cases, 4 to 8 cm of bone and soft tissue is required to restore adequate function. These injuries have been treated by pollicization of an entire healthy digit, with the index finger most commonly used, and other techniques such as osteoplastic thumb reconstruction and microvascular transfer of the great or second toe.

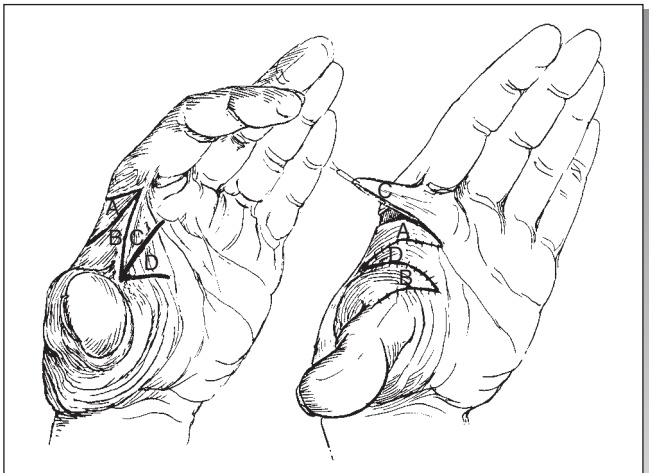

Figure 3 • Application of the four-flap technique for a contracted web space in the hand. **Left,** Design of the four-flap Z-plasty. **Right,** After transfer of the flaps. (After Woolf and Broadbent; from McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990.)

Specific Considerations

MAINTENANCE OF AN ADEQUATE FIRST WEB SPACE. Maintenance of the first web space is critical for allowing free motion of the remaining thumb around the carpometacarpal joint. The four-flap Z-plasty is excellent for releasing the thumb–index finger web space. In addition, significant lengthening is required to release the adduction contracture. Indications include a scarred first web space in which shortness of the thumb amputation stump can be compensated for by deepening the first web space.

In this technique, the skin is marked along the edge of the web. Lines are drawn perpendicular (90 degrees) to this first line. Thereafter, the 90-degree lines are bisected to make a total of four 45-degree flaps. Each line drawn is of equal length. The four flaps, marked sequentially A, B, C, and D, fall into the configuration C, A, D, B after mobilization and transposition (see Fig. 3).

Ray Amputation

Ray amputations may be performed to remove a functionless digit, thereby closing the gap between the remaining fingers. Furthermore, some infections and tumors may require ray amputation to eradicate the infection or excise the tumor with adequate margins.

Treatment

An index ray amputation is performed by making a circumferential incision around the remaining proximal phalanx. The incision is carried proximally along the dorsal aspect of the second metacarpal to the level of the metacarpal head, with skin intentionally left over the proximal phalanx to avoid a postoperative web contracture. The extensor digitorum communis and extensor indicis proprius tendons are transected. The metacarpal is transected distal to the proximal metaphysis and elevated. The first dorsal interosseous and lumbrical muscles to the radial side of the index finger are sectioned at their insertion points. The neurovascular bundles are identified and the vessels ligated. The digital nerves are dissected, transected distally, and tagged for later translocation. The flexor tendons are transected and allowed to retract. The first palmar interosseous muscle is transected along with the remaining attachments, including the volar plate and preosseous bands of the palmar fascia. The digital nerves are translocated between the first and second dorsal interosseous muscles. Care should be taken to avoid excessive mobilization of the radial digital nerve because of the potential for postoperative hyperesthesia.

Two accepted methods can be used for ray amputation of the long finger, both of which aim to close the space that results between the index and ring finger after long finger amputation. To accomplish this goal, the index finger metacarpal can be transposed to the long finger metacarpal. There is, however, a risk of nonunion at the osteotomy site. Alternatively, the deep intervolar plate ligaments between the remaining index and ring fingers can be coapted, thereby closing the potential space.

Both techniques for long finger ray amputation resemble that for amputation of the index finger ray. With transposition, after removal of the long finger ray, an osteotomy of the index finger metacarpal is performed at the metaphyseal flare. The index metacarpal is transposed to the base of the long finger metacarpal and transfixed with either K-wire or miniplate fixation. Immobilization may be required for approximately 4 weeks or until clinical or radiographic evidence of healing is apparent. In the alternative method, the deep intervolar plate ligaments are sutured to one another distally, and a transverse K-wire is used between the index and ring finger metacarpals to maintain alignment. Postoperative immobilization is also required for several weeks.

Ring finger ray amputation follows principles similar to those outlined for long finger ray amputation. Either transposition of the fifth finger metacarpal or direct closure of the deep intervolar plate ligaments can be performed. Fifth metacarpal transposition uses the same general technique as index finger transposition. However, direct closure of the deep intervolar plate ligaments has significant advantages. The ring and fifth fingers share a common attachment to the hamate, which allows 15 to 30 degrees of mobility at their bases. In addition, the ring finger metacarpal does not have tendinous attachments to the hamate as do the other metacarpals to their respective carpal bones. Therefore, the entire ring finger metacarpal may be amputated to allow further mobility of the fifth finger metacarpal with direct closure of the deep intervolar plate ligaments. Postoperative immobilization for both methods is similar to that after ring metacarpal amputation.

Fifth ray amputation is similar to that of the index finger. The proximal fifth metacarpal base must always be left intact, if possible, because it is the site of attachment of the extensor carpi ulnaris tendon. The technique follows that previously described for the index finger, with care taken to leave the hypothenar muscles intact for soft tissue coverage. Of all of the ray amputations, the cosmetic result of fifth ray amputation is the most acceptable.

Alternatives

PROSTHESES. Prostheses are available for proximal finger amputations. They may be customized to fit any type of amputation stump. Color match and overall appearance are excellent, and camouflaging

the appearance of an amputated finger may be of considerable emotional benefit.

Specific Considerations

LOSS OF GRIP STRENGTH. Most ray amputations are electively performed after the functional deficits have been identified and possible alternatives exhausted. A very proximal amputation of the ring or middle finger produces a large defect through which small objects may fall. As such, closure of this "gap" improves the overall functional status of the hand. An index finger proximal phalanx amputation sufficiently shortens the digit so that the long finger becomes preferred for thumb pinch activities. The remaining index digit acts as a physical impediment between the thumb and long finger. Removal of the index ray serves to heighten thumb pinch function in the injured hand. However, ray amputation leads to significant loss of strength in the remaining digits. The overall functional status, occupation, and emotional state of the patient will therefore weigh heavily in the decision-making process.

Amputation through the Carpus

Severe mutilating injuries of the hand may require amputation through the carpus. Large, invasive tumors, severe necrotizing infections, or fungal infections in immunocompromised patients may also necessitate amputation at the level of the carpus.

Treatment

With radiocarpal disarticulation, salvage of the distal radioulnar joint allows for supination and pronation of the distal end of the extremity. Palmar and dorsal flaps are raised distal to the carpus, with as much skin as possible salvaged for closure. The superficial branch of the radial nerve, the median and ulnar nerves, and the dorsal sensory branch of the ulnar nerve should be identified and transected proximally to prevent symptomatic neuroma. The radial and ulnar arteries are suture ligated. All flexor and extensor tendons are divided and allowed to retract into the wound. The radiocarpal joint is entered from the dorsal side, and all ligamentous attachments are divided. Alternatively, the radial, ulnar, and median nerves can be transected proximally in the forearm. The nerves can be buried between the muscle bellies, thereby decreasing the likelihood of symptomatic neuroma.

Alternatives

KRUKENBERG'S OPERATION. A procedure first described by Krukenberg in 1917 converts a distal forearm stump into radial and ulnar rays. These rays, powered by the pronator teres, may be used as opposing forces for grasping activities. The indications for this procedure are controversial and generally include bilateral upper extremity amputations in the blind and juvenile patient population.

Specific Considerations

Patients cannot perform grip or pinch activities after amputation at the carpus. However, sensate skin and wrist pronation and supination at the distal radioulnar joint usually remain intact. These actions allow for some function of the amputated stump. Conversely, more proximal amputations facilitate prosthetic fitting. Generally, at the time of injury, patients are not capable of making decisions regarding this issue. The surgeon should make every effort to salvage as much viable tissue as possible. After healing has occurred, the various options may be presented to the patient and an informed decision made regarding future surgery and the use of prosthetic devices. Preinjury function, age, occupation, and the emotional state of the patient need to be addressed before any definitive treatment is performed.

Previously, patients with proximal amputations of the hand were treated with long below-elbow amputation to allow for prosthetic fitting. However, recent advances in prosthetic manufacturing have made wrist disarticulation an acceptable treatment.

Prostheses

Despite advances in reconstructive microsurgery for complex hand problems, the need for prostheses remains. Prosthetic technology has undergone a revolution in terms of new materials, life-like coloration, and motorized parts. These prosthetic devices can serve a specific, often single function for the patient. The individual needs of each patient must be evaluated before the choice of a prosthesis is made. Patients with similar anatomic defects can often have different goals and expectations, and the prosthesis should be tailored accordingly.

Prostheses for the hand and digits may be categorized into two broad groups: active and passive. An *active* prosthesis extends the injured limb and acts as a clamping device. Each type of active prosthesis is capable of performing a specific, limited type of task. No active prosthesis meets all the needs of the patient, and therefore multiple terminals must be used to gain a wide variety of functions. Active prostheses may be further divided into body powered versus externally powered. A body-powered active prosthesis is controlled by a separate body part, usually the contralateral shoulder. Because the terminal is connected to the active motion of a body part, there is sensory feedback to the central nervous system, thereby improving effectiveness.

Externally powered prostheses are usually driven by myoelectric forces in which the action potentials of remaining muscles are electronically processed and fed to an electric motor. Without visual guidance, these systems do not allow for sensory feedback and subconscious control and are thus less effective.

Passive prostheses serve both a functional and cosmetic role for the amputee. They provide a believable camouflage for the patient and may be adjusted and contoured to hold light objects. Although passive prostheses have no moving parts, they allow for apposition against the remaining normal parts. This is especially true for a partial hand or digit amputee in which the remaining digits are able to work in concert with the passive prosthesis. If the prosthesis is fashioned to replicate the size and shape of the missing parts, sensory feedback pathways may be relied on to accomplish complex activities.

Pearls and Pitfalls

- Despite advances in microsurgical technique and free tissue transfer, amputation of the hand and digits still remains a reliable and safe treatment alternative.
- A well-performed and appropriate amputation should be considered a *reconstructive* procedure.
- The most important variables remain the patient's desires, expectations, and understanding of all possible alternatives and outcomes.

SUGGESTED READINGS

Brown FE: V-Y closure of fingertip injuries. In Blair WF (ed): Techniques in Hand Surgery. Baltimore, Williams & Wilkins, 1996, pp 19-25.

Kleinman WB, Strickland JW: Thumb reconstruction. In Green DP, Hotchkiss RN, Pederson WC (eds): Green's Operative Hand Surgery. Philadelphia, Churchill Livingstone, 1999, p 2068.

Louis DS, Jebson PJ, Graham TJ: Amputations. In Green DP, Hotchkiss RN, Pederson WC (eds): Green's Operative Hand Surgery, Philadelphia, Churchill Livingstone, 1999, p 48.

Parkes A: The "lumbrical-plus" finger. Hand 2:164-167, 1970.

Replantation and Microvascular Repair of Acute Hand Injuries

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As replantation and revascularization have become more commonplace, preservation of tissue viability is no longer the sole measure of success. Careful patient selection and meticulous surgical technique allow return of an acceptable level of function, which is now the goal of treatment. Whether to proceed with replantation of an amputated part is no longer an issue of technical feasibility, but one of functional practicality.

Patient Selection

A successful outcome begins with appropriate patient selection. After taking into account the mechanism of injury, the location and level of amputation, and the general health of the patient, the decision to proceed is made by the surgeon. A complete history and physical examination of the patient and the amputated parts is mandatory when assessing the potential for replantation. The likelihood of a successful functional outcome must exceed the potential morbidity incurred by prolonged anesthesia and the risks associated with anticoagulation. The patient's age, sex, occupation, and wishes regarding treatment also influence the decision to perform replantation.

Before undergoing replantation, the patient should be informed of the anticipated perioperative and postoperative course, including the lengthy nature of the procedure, the potential need for surgical revision, and the anticipated length of stay in the hospital. The patient must be committed to participate in the months of occupational therapy and undergo the additional surgery needed to achieve an optimal functional outcome. In addition, the patient should be aware that cold sensitivity and stiffness may persist indefinitely and must be willing to abstain from nicotine for at least 6 months after replantation.

Accepted *indications* for replantation include the following:

Thumb—All attempts should be made to salvage the thumb. Even in cases of avulsion, replantation allows preservation of length and sensitivity and therefore achieves a better result than completion amputation. Despite possible diminished range of motion at the interphalangeal or metacarpophalangeal (MP) joints (or both), dexterity and appearance are superior after replantation than after completion of amputation.

Single digit distal to the insertion of the flexor digitorum superficialis (FDS)—Preservation of the FDS tendon allows return of useful function after replantation. Although motion is often limited at the distal interphalangeal (DIP) joint, it is preserved at the MP and proximal interphalangeal (PIP) joints. Replantation of digits amputated distal to the DIP joint is possible if sufficient skin (4 mm) proximal to the nail bed remains to allow identification of adequate dorsal veins for anastomosis. Even when not available, successful replantation has been accomplished with isolated arterial revascularization alone. Methods such as leeches and the application of heparinized pledgets can be used to manage excess of blood until venous neovascularization occurs.

Multiple digits—The potential loss of function is so great with multiple digital amputation that

replantation of all possible digits should be attempted. Replantation of an amputated digit in a different position is possible and gives the surgeon greater flexibility in restoring the vital functions of pinch and grip.

Partial hand—It is possible to restore satisfactory range of motion, grip, and pinch after replantation at the palmar level. Replantation is more successful with injuries proximal to or at the level of the superficial or deep palmar arch than at the level of the origin of the digital vessels. Preservation of blood supply to the intrinsic musculature results in a more functional hand than when ischemic fibrosis occurs.

Wrist/forearm—Patients with amputations at this level are generally good candidates for replantation. The mechanism of injury frequently involves a sharp blade resulting in a guillotine-type amputation. Vessels and nerves are large at this level and easily repaired. Lengthy therapy is required, but the appearance and function achieved after replantation of the wrist or forearm exceeds that provided by a prosthetic.

Pediatric population—Children are candidates for replantation of all amputated parts. Epiphyseal growth is not adversely affected by replantation, and children often adapt well to diminished range of motion. Although anastomosis of the smaller nerves and vessels may be technically challenging, replantation should be performed whenever possible and frequently results in a satisfactory functional outcome.

Elbow/upper part of arm—Replantation of the upper part of the arm remains controversial. Although replantation at the elbow or humeral level may result in limited hand function, it may be beneficial in allowing conversion of an above-elbow amputation to a more functional below-elbow amputation. Note should be made of the time of injury and careful attention paid to the ischemic time of the amputated part. The musculature of the arm and forearm does not tolerate as long a period of ischemia as the musculature of the hand.

Absolute contraindications to replantation include associated life-threatening injuries or systemic illness that renders a long anesthetic a prohibitive risk. Relative contraindications include the following:

Crush or avulsion mechanism—Sharp amputations have much higher success rates for replantation than crushed or avulsed parts. In such injuries, the zone of vascular injury can extend proximally and distally well beyond the level of gross tissue injury. Long segments of the neurovascular bundle may be visualized emanating from the amputated part and are consistent with avulsion. A linear red streak along the skin overlying the neurovascular bundle is also indicative of a shear injury of the underlying vessel.

Multiple-level injury—The extremity proximal to the amputation and the amputated segment itself should be examined closely for additional injuries. Injuries to structures at multiple levels may impair inflow or outflow despite the presence of patent anastomoses. Such injuries may also preclude the restoration of useful function after replantation.

Injury proximal to the level of insertion of the FDS—Any single-digit amputation proximal to the insertion of the FDS tendon (zone 2) should be considered a poor candidate for replantation. These patients, even after additional surgery, often do not regain significant function after replantation to justify the procedure.

Systemic disease—Patients with diabetes, atherosclerosis, and other vasospastic conditions should be carefully evaluated to determine their systemic tolerance and the adequacy of vessels for replantation. Such conditions increase the technical difficulty of replantation.

Smokers—Smoking should be prohibited after replantation. The patient must be compliant with this requirement for replantation. Neither the patient needing replantation nor the surgeon performing the operation should underestimate the addictive properties of nicotine.

Prolonged warm ischemia time—Fingers are devoid of muscular tissue and can therefore survive relatively lengthy periods of ischemia. Proximal and upper arm amputations must be reperfused within 6 hours to avoid irreversible damage to muscle. Prolonged warm ischemia time or improper preoperative preservation of the amputated part, however, may preclude successful replantation. The metabolic consequences of replantation after lengthy ischemic periods include permanent end-organ damage as well as life-threatening electrolyte and acid-base disturbances.

Mentally unstable patient—Special consideration needs to be given to any amputation in a mentally ill patient. The patient should be carefully evaluated by both the replant surgeon and a mental health specialist to determine whether replantation is appropriate. Once the decision has been made to proceed with replantation, the replant surgeon and mental health specialist need to cooperate to ensure optimal care and therapy.

Initial Management

After the replant team has been notified of a potential candidate, steps must be taken to ensure proper

preservation of the amputated part. The amputated part should be wrapped in moist saline gauze and placed in a bag. The bag is placed on ice, but ice should never come in direct contact with the amputated extremity. The patient should receive appropriate antibiotic and tetanus prophylaxis. Active bleeding should be controlled without blind clamping or suturing. The patient should be stabilized and transferred in an expedient manner.

On arrival, the patient and amputated part are examined. The degree of contamination is assessed, as is the nature of the skeletal injury and the quality of the surrounding soft tissues. Radiographs of the proximal and distal amputation sites are obtained to rule out other injuries. Intraarticular involvement and the quality of the adjacent bones influence, in part, the functional outcome of replantation. Blood is crossmatched as needed.

The surgeon must counsel the patient appropriately and carefully weigh whether function would be better with replantation than with revision amputation and a possible prosthesis. Expectations of the patient should be addressed, and the patient should be informed of the need for postoperative surgery. If the patient is unwilling to accept the treatment recommendation of the surgeon, it may be necessary to obtain or offer a second opinion from a colleague.

Surgical Technique

Replantation is often performed under general anesthesia; an axillary block should also be used to provide a sympathectomy effect. The patient is placed in the supine position, although the prone position can be useful for replantation of the thumb. A tourniquet should be applied and used as needed. Because the operative time may be prolonged, pressure points should be padded to prevent pressure breakdown. Sequential compression devices are applied to the lower extremities for prophylaxis of deep venous thrombosis before induction of anesthesia.

The operative sequence may vary depending on the level of injury and surgeon's preference. In general, replantation incorporates, in one sitting, elements from all of the procedures routinely performed by the hand surgeon.

In the case of multiple-digit amputations, all possible fingers should be replanted if the mechanism and level are appropriate. The thumb, middle finger, and ring finger should be given priority over the index and small fingers. Transposition of an amputated digit may be necessary to preserve pinch, grip strength, or the width of the hand. Such transposition allows the least damaged amputated part to be reattached to the most useful site. To minimize ischemic time and fatigue, structure-by-structure repair is performed on all fingers simultaneously.

Preparation of the Amputated Part and Stump

A two-team approach should be used. The amputated part should be taken to the operating room for preparation while evaluation of the patient is taking place in the emergency room. The amputated part should be scrubbed and cleansed. Devitalized or contaminated tissues are débrided. In arm amputations, exposure is achieved with midlateral incisions. Radical débridement of muscle is often necessary. Anterior/posterior compartment fasciotomies of the arm, carpal tunnel/Guyon's canal, and the hand compartments are performed. The lacerated muscle/tendons, vessels, and nerves are tagged, and bones are shortened.

For digital amputation, longitudinal midlateral incisions are made of sufficient length to provide wide exposure. With the use of loupe magnification or the operating microscope, the neurovascular bundles are identified and dissected clear from the zone of injury. Dorsal veins are identified and tagged in the plane between the reflected skin flap and the extensor tendon. Flexor and extensor tendons are identified in the amputated segment and cut cleanly. Sutures placed in the tendon can be used for identification and the repair. The amputated bone is shortened if appropriate and prepared for fixation. The proximal amputation site is simultaneously prepared in a similar manner by the second team.

Osteosynthesis

The amputated and recipient segments are brought into the same field and osteosynthesis achieved. Sufficient bone shortening (0.5 to 1 cm in a digit, 2 to 8 cm in an arm) should be performed to facilitate tension-free primary repair of uninjured vessels/nerves and closure of the skin. The angle of the osteotomy can be adjusted when shortening the bone to maximize surface contact. Although a variety of methods may be appropriate, double Kirschner wire or interosseous wire fixation (or both) is frequently used. Plating and screw fixation methods often necessitate wide exposure and further devascularization and are therefore avoided. In certain instances, the surgeon may elect to defer shortening of the bone to preserve an articular surface. If the joint is irreparable, immediate arthrodesis or even arthroplasty may be considered. Periosteum should be reapproximated whenever possible.

Tendon Repair

Tendon repair is performed after osteosynthesis. Extensor tendons can be coapted with interrupted mattress sutures. The flexor tendon sheath may be divided to facilitate identification of the flexor digitorum profundus and superficialis tendons, but the A2 and A4 pulleys should be preserved. The Tajima

suture technique is useful for repair of the flexor tendon when it is necessary to preserve the exposure afforded by maintaining the digit in extension for vascular and nerve repair. Sutures placed in each segment of the tendon are tied after repair of the vessels and nerves. Flexor tendon repair should be reinforced with an epitendinous suture to provide additional strength.

Vascular Repair

The order of vascular repair is decided by surgeon preference. Dorsal veins are easier to identify when they become distended after restoration of arterial inflow. However, performance of venous anastomosis before arterial repair avoids congestion of the replanted part and can be accomplished in a bloodless field. It is, however, more technically demanding. If ischemic time has been prolonged, the arterial anastomosis should be performed first. In the case of forearm or upper arm amputation, the arterial anastomosis should always be performed first because bleeding from the veins of the amputated part allows dissipation of toxic lactic acid and also lessens the potassium burden following restoration of outflow.

Regardless of which anastomosis is performed first, a low threshold should be maintained for the use of interposition vein grafts to ensure a tension-free anastomosis. The arterial anastomosis should not be performed until the surgeon visualizes a normal-appearing intima both proximally and distally and confirms pulsatile blood flow from the proximal vessel. Vein grafts can be readily obtained from the forearm or the dorsum of the foot and should be reversed before anastomosis. One or two digital arteries should be repaired along with at least two veins. Veins may need to be mobilized or rerouted to ensure tension-free anastomosis.

The amputated segment should appear perfused immediately after completion of the arterial anastomosis. Topical agents (papaverine, lidocaine) are used to diminish vasospasm. A persistent cadaveric appearance necessitates careful inspection of the anastomosis for technical error and revision as needed. A blue appearance and brisk capillary refill suggest poor venous outflow, and the venous anastomosis should be inspected.

In the case of thumb replantation in which the patient is supine, positioning of the digit for the arterial anastomosis can be awkward. A vein graft is useful in this circumstance. It is technically easier to perform the distal anastomosis of the vein graft to the dominant ulnar digital artery on the back table. The princeps pollicis artery or radial artery at the snuff box are used for the establishment of inflow.

Nerve Repair

Careful and accurate nerve repair cannot be overstressed. Poor results with regard to sensation can negate an otherwise good result. Furthermore, a painful replanted digit is always more of a handicap than is an amputation. Nerves must be trimmed back both proximally and distally until a normal fascicular pattern can be visualized under the microscope. Careful fascicular alignment is achieved, and only a few sutures are required in most cases. It is important to perform nerve repairs without tension, and grafting, when required, should be performed. The posterior interosseus nerve, median antebrachial cutaneous nerve, and sural nerve are potential nerve donor sites.

Closure

Because edema increases as the case proceeds, once the necessary repairs are completed, each side of the hand is closed before proceeding with the repairs on the remaining side. Closure should be loose to avoid compression of the anastomoses. Frequent monitoring with pulse oximetry or Doppler during closure is useful to identify any compression that would result in vascular compromise. A loose, bulky dressing and splint are applied.

Postoperative Management

General Measures

After replantation, care is aimed at preserving perfusion of the replanted extremity. The patient's body temperature should be maintained by keeping ambient room temperature at no less than 75° F. Attempts are made to minimize patient discomfort and its associated sympathetic response with adequate analgesia and sedation. Brachial plexus and axillary catheters can be used for both pain control and sympathectomy. The patient should be kept with nothing by mouth for at least 24 hours after the procedure to avoid problems with administration of anesthesia should a return to the operating room be required. Adequate hydration is maintained with intravenous fluids.

Pharmacologic Management

Most surgeons use antithrombotic agents after microsurgical repair of vessels. Aspirin, an irreversible platelet antagonist, along with low-molecular-weight dextran (10%), a platelet and fibrin antagonist and volume expander, can be given. A test dose of dextran is given to exclude potential allergic reaction, and continuous infusion at 30 mL/hr is initiated in the operating room and maintained for 3 to 5 days postoperatively. Systemic heparin is occasionally used in cases in which there has been difficulty maintaining patency of the vascular anastomosis.

Monitoring

The most reliable method of postoperative monitoring of perfusion is direct inspection for color, capillary refill, and temperature. Experienced personnel should examine the replanted part both as a baseline and hourly for the first 48 hours. A temperature probe or pulse oximetry monitor attached to the tip of the digit can be useful in detecting subtle changes in perfusion that may indicate vascular compromise.

Changes in appearance of the replanted part may be obvious or subtle. A pale, flaccid digit with delayed capillary refill suggests diminished arterial inflow. Brisk capillary refill and a blue hue are indicative of venous congestion or outflow obstruction. In either case, the dressing should be removed and the replanted part examined. Surgeons must maintain a low threshold for exploration if there is any doubt. For mild venous congestion, leech therapy can be used while new venous channels are being formed.

Therapy

Standard tendon repair protocols are not useful in cases of replantation. These protocols do not account for simultaneous flexor and extensor repairs, along with vascular, nerve, and bone repairs. Each case must be approached individually. The surgeon must take into account the rigidity of the fixation, the security of the vascular anastomosis, and the quality of the soft tissue and tendon repairs.

In general, guarded passive flexion and extension is initiated at 1 to 2 weeks. This is done in a hospital setting, and vascularity is evaluated after mobilization. At 3 weeks, light active and passive flexion/extension is allowed. At 5 weeks, full flexion/extension is allowed. Strengthening exercises are started at 10 weeks.

Complications

Perioperative complications include loss of arterial inflow or venous outflow. Pressure sores may develop if adequate padding is not used during the replantation procedure. More proximal replantations or those with a prolonged warm ischemic time may be associated with severe metabolic disturbances after reperfusion. Subsequent rhabdomyolysis and resultant acidemia and hyperkalemia may result in permanent renal damage. In addition to a risk for gastrointestinal hemorrhage, the use of perioperative anticoagulation is associated with a risk for hematoma at the operative site or at the site of puncture for induction of regional anesthesia.

Replantation is usually performed on contaminated wounds. Débridement of devitalized or contaminated tissue must be sufficient to minimize the risk of postoperative infection. Replant loss after the first week is often the result of infection and not technical difficulty with vascular restoration.

Late complications that affect function of the replanted part may occur weeks to months after replantation. Malunion, nonunion, and diminished range of motion can be related to the type of fracture fixation and the associated period of immobilization. Tenolysis is deferred until the surrounding soft tissue is healed and supple and the patient's progress from therapy has plateaued. In more proximal amputations, impaired blood flow to intrinsic musculature can result in fibrosis and ischemic contracture, which may require subsequent release or débridement.

Most patients do not recover normal sensation in the affected part after replantation, and thus attainment of protective sensation is a more realistic goal. Sufficient bone shortening that allows primary nerve repair and strict adherence to meticulous technique are necessary to optimize sensory recovery and minimize neuroma formation. Cold sensitivity after replantation is a frequent complaint but often improves with time.

Pearls and Pitfalls

- Replantation remains a demanding procedure, and a successful functional outcome requires experienced surgical judgment, as well as meticulous technique.
- Errors regarding patient selection increase the technical difficulty of the procedure and may ultimately result in a well-perfused digit that is painful and nonfunctional.
- A long-term commitment by the surgeon, patient, and therapist is essential for success.
- The potential for restoration of the patient's sense of self makes replantation one of the most rewarding surgical procedures.

SUGGESTED READINGS

Goldner RD, Urbaniak JR: Replantation. In Green DP (ed): Operative Hand Surgery, vol 1. New York, Churchill Livingstone, 1998, pp 1139-1157.

Idler RS, Steichen JB: Complications of replantation surgery. Hand Clin 8:3, 1992.

Lee WP, May JW Jr: Free flaps for soft-tissue coverage. In Peimer CA (ed): Surgery of the Hand and Upper Extremity, vol 2. New York, McGraw-Hill, 1996, pp 1878-1880.

May JW Jr, Toth BA, Gardner M: Digital replantation distal to the proximal interphalangeal joint. J Hand Surg [Am] 7:161, 1982.

Scheker LR, Hodges A: Brace and rehabilitation after replantation and revascularization. Hand Clin 17:473, 2001.

Soucacos PN: Indications and selection for digital amputation and replantation. J Hand Surg [Br] 26:572, 2001.

Peripheral Nerve Repair and Transfers

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The processes of nerve regeneration and target reinnervation are complex and involve multiple factors. The timing of the repair, the level of the injury, the extent of the zone of injury, the technical skill of the surgeon, and the method of repair or reconstruction contribute to the functional outcome after nerve injury. This chapter reviews the principles and techniques of nerve repair and discusses options for direct repair, including nerve grafts, conduits, and nerve-to-nerve transfers.

Etiopathogenesis

The causes of peripheral nerve injury are multifactorial and may be congenital or acquired. Trauma, tumors, vascular lesions, and iatrogenic injuries are causes of most nerve injury.

Pathologic Anatomy

A detailed history and physical examination are often all that is required to diagnose peripheral nerve injury and to ascertain the probable pathologic anatomy. Information regarding both the premorbid function of the patient and the mechanism of injury is critical in the evaluation of a nerve injury. Information on whether the injury was open or closed or whether it involved a stretch or a crush injury assists in assessment of the degree of injury. A complete sensory and motor examination of the injured extremity should include two-point discrimination of the digits and pinch and grip testing. Tinel's and provocative testing should begin at the cervical spine and proceed to the wrist. In addition to evaluation of innervation density with static and moving two-point discrimination, threshold testing (vibratory stimulus of a 256-Hz tuning fork or Semmes-Weinstein monofilaments) and evaluation of protective sensation with pain and temperature testing help to give a complete and accurate picture of the pathology. A simple qualitative 10/10 test rapidly and accurately provides an excellent estimate of sensibility.

First-degree injuries (*neurapraxia*) represent a localized conduction block at the site of trauma. The site of trauma may appear normal by physical examination, or evidence of ecchymosis or hematoma may be apparent. Tinel's sign is absent because there is no axonal discontinuity. Recovery can vary from days to 12 weeks. When the conduction block improves, recovery is immediate, in contrast to the slower progression of neural recovery observed in more severe degrees of injury. Nerve conduction studies demonstrate no action potential at the area of the conduction block; however, a normal response to stimulation is seen above and below the level of the block.

Second-degree injuries (*axonotmesis*) are the least serious of the degenerative injuries. Axons are disrupted and wallerian degeneration occurs; however, the endoneurial sheaths are preserved, so axonal contact and guidance of the regenerating growth cones are unimpeded by scar or topographic mismatch. Tinel's sign is present because of axonal injury; it progresses distally with the regenerating axons at a rate of 1 to 3 mm/day. The axonotmesis recovers according to the sequential reinnervation of target sensory and motor end-organs in a logical proximal-to-distal manner. This finding contrasts with neurapraxia (first-degree injury), which recovers relatively completely within 3 months. Excellent spontaneous complete (or nearly complete) recovery is expected.

Third-degree injuries also represent an axonotmesis in that the axons are disrupted; however, the architectural disturbances are more extensive and include the endoneurial sheath, the basal laminae of Schwann cells, and other connective tissue disruptions within the boundaries of the perineurium. The endoneurial contents are no longer able to guide axonal regrowth, and mismatch of fibers occurs in a proximal-to-distal direction. Some of the fibers fail to grow across the injury and may form a disorganized conglomeration of nerve fibers, Schwann cells, and scar tissue. The results of third-degree injury vary depending on the extent of intrafascicular scarring and the fascicular topography at the level of the injury that results after topographic mismatch and the ensuing axonal misdirection. Despite this variability, it is generally accepted that microsurgical repair with grafting is less successful than the results seen with spontaneous recovery of a third-degree lesion. Recovery of a third-degree injury may be improved with surgical decompression and neurolysis if the conduction block is localized to a known area of nerve compression. A third-degree injury causing significant pain may therefore be better treated by excision of the injured nerve section and nerve grafting. Treatment of a third-degree injury with surgical intervention is not considered outside the standard of care.

Fourth-degree injuries (*neuroma in continuity*) consist of intact epineurium, but all the subepineurial layers have undergone complete disruption and replacement with scar. No nerve regeneration is possible, and all nerve elements in the area are replaced by scar (neuromas). Tinel's sign may be present, but it fails to progress distally. As with other injuries, a fourth-degree injury requires a period of observation for reliable diagnosis, although the severity of the injury mechanism and the absence of progression of Tinel's sign are early, ominous warnings. A fourth-degree injury requires excision of the area of injury and microsurgical repair with grafting or a conduit technique.

Fifth-degree injuries (*neurotmesis*) are defined as complete division of the nerve. No recovery is possible without repair. These injuries can follow a severe stretch and avulsion injury, but more commonly they are associated with open trauma.

Sixth-degree injuries were added to Sunderland's original classification to address a mixed nerve injury. A nerve may undergo a combination of different degrees of injury. These injuries are challenging because of the possibility of poor function of the less severely injured portions of the nerve. It is important to differentiate fourth- and fifth-degree injured fascicles from less severely injured or uninjured ones. Knowledge of fascicular anatomy and correlation with the pattern of observed recovery can be helpful in this regard but may not provide a complete picture. Intraoperative electrodiagnostic studies can help to complete the topographic picture.

Diagnostic Studies

Electrodiagnostic testing includes nerve conduction studies and electromyography. These studies are useful in defining the distribution of an injury, as well as the extent of the injury (partial versus complete). Electrodiagnostic testing can also be helpful in documenting the recovery of nerve function. In the evaluation of a closed nerve injury, it is important to understand that nerve conduction studies and electromyography performed before 4 to 6 weeks after a nerve injury yield indeterminate results because it takes this long for muscle to demonstrate detectable denervational changes on electromyography. By 12 weeks, motor unit potentials should be present, at least in the proximally innervated muscle, if functional recovery is to be anticipated.

Both nerve conduction studies and electromyography are useful adjuncts to physical examination. The combination of a detailed clinical evaluation and electrodiagnostic studies provides the surgeon with an excellent guide for planning a reconstructive strategy.

Reconstructive Goals

The goals of reconstruction are to maximize nerve recovery and useful function in a timely fashion. In the case of multiple nerve injuries, such as those seen in a brachial plexus injury, surgical priorities must be established. First priority should be given to restoration of elbow flexion, followed by shoulder abduction and external rotation. Sensory priorities should focus on sensibility of the ulnar thumb and radial index finger and the ulnar border of the hand.

Treatment

Open injuries, in general, require early exploration. If the injury is a sharp laceration and conditions are optimal, the nerve can be repaired primarily. The proximal and distal extent of the zone of injury can be difficult to appreciate in anything other than an injury from a knife or glass. Definitive primary nerve repair is not recommended for injuries associated with significant soft tissue damage because the longitudinal zone of injury is not initially apparent. In this situation, delayed repair better allows the surgeon to define the zone of injury.

If primary repair is not appropriate at the time of initial exploration, the proximal and distal ends of the nerves should be tagged and sutured to full length to prevent significant retraction at a secondary procedure. If possible, the nerve should be approximated by using fascicular patterns to aid in topographic alignment and to maximize the

definitive repair. At 3 weeks, or when the patient's overall condition and wound permit, the nerve should be reexplored with the goal of achieving a definitive repair.

Gunshot wounds must be considered an exception to the general rule of early exploration of open injuries. Although these injuries are technically considered to be open, the mechanisms of nerve damage are predominately heat and shock effects. Consequently, many of these injuries recover spontaneously and should more appropriately be treated as closed or blunt trauma.

Closed or blunt injuries are initially managed conservatively. If anatomic recovery is not seen at 6 weeks, electrodiagnostic studies should be obtained for assessment and baseline evaluation. If postinjury recovery has not occurred at 12 weeks, additional clinical and electrical assessment should be performed. If motor unit potentials are identified with electromyography, spontaneous reinnervation of that muscle should be anticipated, and these patients should continue to be treated expectantly. However, the absence of clinical or electrical evidence of reinnervation at 3 months indicates the need for exploration. The type of surgical procedure that is ultimately performed is determined after exploration and intraoperative nerve conduction testing. A nerve that has been completely divided or that fails to conduct an action potential intraoperatively should be managed by resection of the neuroma and microsurgical repair of the nerve gap with interpositional nerve grafting.

An in-continuity lesion that is able to be stimulated electrically can be more of a challenge. Neurolysis proximal and distal to the neuroma allows identification of functioning fascicles by using a nerve stimulator for the motor fascicles and nerve-to-nerve intraoperative recording for sensory fascicles. The nonfunctioning nerve fascicles can be transected proximally and distally and grafts placed without disrupting the partially functioning neuroma.

Surgical release of any known areas of distal compression can also be useful. For example, release of the carpal tunnel in a proximal median nerve injury can help to facilitate regeneration across the wrist, and release of the peroneal nerve at the fibular head and the tibial nerve at the tarsal tunnel can assist in peroneal and tibial nerve recovery in association with more proximal recovering sciatic nerve injuries.

The *principles of nerve repair* include the following:

1. Quantitative preoperative assessment of motor and sensory systems
2. Microsurgical technique, including magnification, instrumentation, and microsutures
3. Tension-free repair
4. Use of an interpositional nerve graft when a tension-free direct repair is not technically possible
5. Primary repair when conditions permit
6. Delay in repair for approximately 3 weeks in cases in which primary repair is not optimal (for example, a severe crush, stretch, or loss of nerve tissue)
7. Early protected range of movement to allow nerve gliding
8. Occupational and physical therapy to maintain range of motion and assist in postoperative sensory and motor reeducation and rehabilitation to maximize the clinical outcome

Microsurgical techniques are required for a technically optimal nerve repair. The operating microscope should be used whenever possible; however, surgical loupes (at least 4× magnification) are adequate in areas where placement of the microscope is difficult. Repairs should be performed with either 9–0 or 10–0 nylon (interrupted) sutures. Nerve ends are prepared sharply by using either a No. 15 blade on a rigid background or sharp, straight microscissors. The first suture should always be placed intentionally loose to facilitate optimal alignment of the nerve ends with the remaining sutures. Any outwardly pointing fascicles should be gently trimmed so that they can properly align themselves within the repair.

Any tension at the nerve repair site must be managed by nerve grafting. Postural maneuvers to decrease tension should be avoided; they cause gapping and scarring at the nerve repair site when the joint is mobilized and may result in stiffness of the immobilized joint. Mobilization of the nerve ends proximally and distally for short distances of 1 to 2 cm can provide some relief of tension without negatively affecting blood flow, but *extensive* mobilization of the nerves should be avoided. It is important to recognize that under unfavorable conditions, axonal regeneration is more successful across two tension-free neurorrhaphy sites than across one tight repair site.

Conduits are an alternative to an interpositional nerve graft. Research on the use of alternatives to interpositional nerve grafts has been driven by issues associated with donor nerve harvest, availability, and associated morbidity. The use of conduits to facilitate nerve regeneration across a nerve gap is an area of considerable research.

It is well established that the influence of the distal stump can be exerted equally well through a conduit or a nerve graft over short distances of less than 3 cm. However, current research has not succeeded in developing a conduit that is successful for long nerve gaps (greater than 3 cm). Currently, the use of conduits by the authors is limited to gaps less than 3 cm in noncritical, small-diameter areas of sensation and in patients who decline autogenous nerve graft harvest.

Nerve grafting techniques follow the same principles as previously described for direct repair. In

general, nerve grafting should be delayed for 3 weeks until the extent of nerve damage can be more fully appreciated. Primary nerve grafts may be justified in areas of extensive trauma involving injuries to multiple structures that may be difficult and dangerous to reexplore. In these instances, generous débridement of the nerve both proximally and distally should be performed to ensure that the resection is well outside the zone of injury. The size of the defect is measured with joints maximally extended to ensure that an adequate length of graft is obtained to facilitate tension-free repair. The graft should be oriented in a reverse fashion from its native position so that the regenerating fibers are not diverted from the distal neurorrhaphy site and the distal stump. Surgically created misalignment can be avoided by placement of the grafts in the same sequence proximally and distally. As with direct repair, knowledge of the intraneural topography of the peripheral nerves is essential to obtain a good clinical outcome. The distal stump of the nerve can be exposed distally until the motor and sensory branching pattern is clarified. With the appropriate motor and sensory areas defined distally, appropriate fascicles can be visually followed proximally to their location in the distal nerve stump. This technique avoids the time and physical manipulation of the nerve necessary with surgical neurolysis. The fascicular patterns of the proximal stump are thus assessed. If the nerve gap is small, corresponding fascicular organizations matching the distal stump can be identified. In longer gaps, the cross-sectional anatomy is less likely to match the distal stump, and the techniques of awake stimulation or histochemical staining may be useful. In general, knowledge of the intraneural topography is most commonly used to align the nerve grafts. This knowledge is gained by taking the opportunity, case by case, to electrically stimulate normal nerves with a nerve stimulator to identify the motor/sensory topography.

Nerve transfers are increasingly being used for reconstruction of proximal nerve injuries. The use of nerve transfers in the upper extremity has been limited to brachial plexus avulsion injuries in which no proximal source of viable nerve is available for nerve grafting. However, nerve grafting across a proximal nerve injury is associated with poor functional outcomes. The distance to the target muscle may be too far from the regenerating nerve fibers to provide timely and useful reinnervation. In contrast, distal nerve transfers provide regenerating axons to the injured nerve at a site close to the target muscle, thereby avoiding the long delay for reinnervation. In addition, many of these procedures can be carried out without the need for an interpositional nerve graft. When a nerve graft is required, it is generally short and can be harvested from the ipsilateral extremity.

A nerve transfer therefore converts a high-level nerve injury to a low-level nerve injury by recruiting expendable nerve fascicles from the donor nerve to innervate critical nerves close to their target end-organs. Donor nerves are preferentially selected according to their proximity to the motor end-plate of interest, and the neurorrhaphy is carried out in a tension-free manner without the need for a nerve graft. The criteria for motor and sensory nerve transfers include an expendable donor nerve, a donor nerve with a large number of pure axons (motor or sensory), a donor nerve near the target organ, and preferably a donor motor nerve that innervates a muscle that is synergistic to the target muscle.

Motor nerve transfers to restore elbow flexion have been the subject of considerable interest. The authors initially preferred the medial pectoral nerve to motor the musculocutaneous nerve. Approximately 6 years ago, the procedure was modified so that a redundant portion of the normal ulnar nerve supplying branches to the flexor carpi ulnaris (FCU) was transferred directly to the biceps branch of the musculocutaneous nerve. This transfer supplies motor input more distally in the arm, at a level that is close to the motor end-plates of the biceps muscle. It does not require an interpositional nerve graft. In this procedure, the ulnar nerve is identified at the midarm level, adjacent to the biceps branch of the musculocutaneous nerve. Redundant fascicles to the FCU are identified by internal neurolysis and the use of a nerve stimulator. These branches, typically located on the lateral aspect of the ulnar nerve, account for approximately 20% to 25% of the nerve. Adequate intrinsic hand function with the remaining portion of the ulnar nerve must be confirmed before dividing the selected portion of the ulnar nerve. Alternatively, or simultaneously, the motor fibers from the medial pectoral muscle are transferred to the brachialis branch of the musculocutaneous muscle with an interpositional nerve graft to maximize the strength of elbow flexion.

Other options for biceps reconstruction include a portion of the flexor carpi radialis motor fascicles of the median nerve, the entire thoracodorsal nerve, and the intercostal nerves. Dissection of the intercostal nerves under the ribs can be tedious, and the nerves tend to be small, with a limited number of motor axons. When using the intercostals, an attempt should be made to include one of the larger segmental nerves that innervate the rectus abdominis muscle because they provide more motor fibers. Interpositional nerve grafts are often required with intercostal nerve transfers, especially in obese patients.

Shoulder abduction reconstruction using nerve transfers concentrates primarily on reconstruction of the suprascapular nerve (SSN), as well as the axillary nerve. Injury to the SSN results in weakness of the supraspinatus and infraspinatus muscles during initial arm abduction and external rotation. Injuries to this nerve should preferentially be repaired with nerve grafts if the proximal C5-6 root is available. The short distance of the motor end-

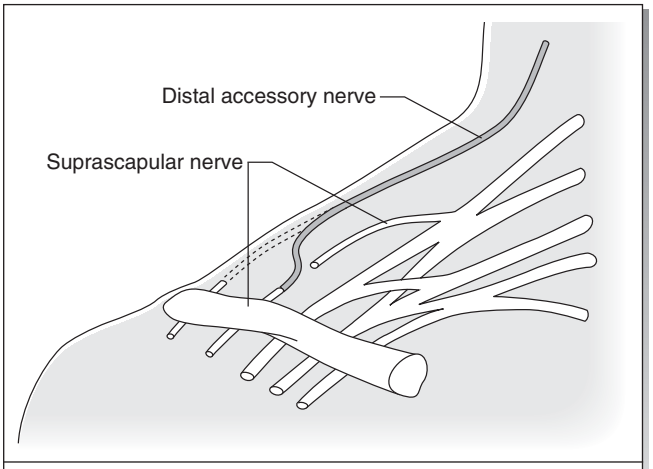

Figure 1 • Distal accessory nerve transfer to the suprascapular nerve. The distal portion of the accessory nerve is used after it has given off its branches to the upper portion of the trapezius.

plates to the root level of these nerves results in an excellent prognosis with nerve grafting. When no suitable proximal nerve is available for reconstruction, the distal portion of the accessory nerve (DAN) can be used. The DAN is identified and traced distally to preserve the upper trapezius branches, which are important in shoulder function. The SSN is identified behind and below the clavicle. This nerve can be difficult to identify. It is the only branch of the upper trunk and can be located by following the lateral border of the upper trunk. Alternatively, the SSN can be exposed with blunt finger dissection. In this approach, a blunt finger dissects inferiorly through the neck incision, as well as superiorly through the infraclavicular exposure to the brachial plexus. The SSN is palpated under the clavicle and delivered into the neck incision. The neurorrhaphy of the DAN to the SSN is completed through the neck incision (Fig. 1).

The medial pectoral nerve (MPN) can also serve as a donor for the SSN. No neck incision is required, and the SSN is identified via the infraclavicular approach as described earlier.

Injury to the axillary nerve results in weakness of the deltoid muscle, which contributes significantly to shoulder abduction. The MPN and a portion of the triceps branch to the radial nerve are the usual nerve donors for reconstruction of the axillary nerve. The axillary nerve is quite large relative to either of these donor nerves, and a significant size mismatch can be seen. It is important to preferentially innervate the superolateral portion of the axillary nerve where the motor fibers of the deltoid muscle are located. The MPN and a triceps branch can also be used in combination to provide greater motor input. Identification of branches of the MPN is easily accomplished after standard exposure of the infraclavicular brachial plexus; this maneuver involves reflection of the pectoralis major muscle and division of the pectoralis *minor* muscle. A nerve stimulator is applied to the undersurface of the pectoralis minor to identify two to four branches of the MPN, which are divided as distally as possible. A short nerve graft is still occasionally required to achieve a tension-free repair.

Use of a triceps branch can often be accomplished without a nerve graft because the main triceps branch separates from the radial nerve quite proximally. It is traced distally until it further branches into three terminal branches, one of which is selected and neurolysed proximally as far as possible. Care is taken to leave sufficient function of the triceps muscle with the remaining two branches. The donor branch of the triceps is sutured directly to the superolateral portion of the axillary nerve. Other transfers for deltoid reconstruction include the thoracodorsal nerve and the intercostal nerves.

Intrinsic hand function (*ulnar nerve*) reconstruction using distal nerve transfers is highly desirable given the poor outcomes after high ulnar nerve injuries. Nerve transfers provide a closer source of motor axons to the target muscles and avoid the prolonged time needed for reinnervation observed in high injuries. When the median nerve is intact, the distal branch of the anterior interosseous nerve (AIN) to the pronator quadratus can be transferred to the deep motor branch of the motor nerve. The deep motor branch of the ulnar nerve is first identified in Guyon's canal and traced proximally to the midforearm region. The AIN is identified and traced distally into the midpoint of the pronator quadratus muscle where branching occurs. Direct repair of the AIN to the motor branch of the ulnar nerve can always be accomplished in a tension-free manner without the need for a nerve graft.

Sensory nerve transfers can be performed for both the ulnar and median nerve. When the ulnar nerve requires reconstruction, the common digital nerves to the third web space (median nerve) can be used. In complete, high ulnar nerve injuries, the transfer can be accomplished in the distal end of the forearm, at the same level where the motor nerve transfer of the AIN to the deep motor branch of the ulnar nerve is performed. The most ulnar fascicles of the median nerve supply sensation to the third web space. Identification can be made by careful intraneural neurolysis in the forearm and usually confirmed by following the fascicles. Intraoperative nerve stimulation is performed to ensure that no motor fibers are involved. To minimize donor morbidity, the distal end of the donor nerve may be sutured in an end-to-side fashion to the median nerve where the sensory fascicles to the second web space reside. Alternatively, an end-to-side neurorrhaphy of the cut end of the sensory ulnar nerve to the side of the median nerve can be performed to provide ulnar sensation.

Reconstruction of the median nerve to provide critical sensation to the first web space is possible

when the ulnar nerve is intact. The donor is the portion of the ulnar nerve that supplies the noncritical area of the fourth web space. The common digital nerves are dissected and transected distally. The nerves of the fourth web space are transposed to the first web space, and an end-to-end neurorrhaphy is accomplished without the need for a nerve graft. The distal end of the donor ulnar nerve is sutured end to side to the ulnar digital nerve to the fifth digit to provide sensation to the donor ulnar distribution of the fourth web space.

Postoperative Care

All nerve repairs, grafts, and transfers require early range of motion. Dressings are removed by postoperative day 3, the wounds are examined, and the patient begins range-of-motion exercises. In the area of the nerve repair or transfer, the repair sites are protected from extreme ranges of motion by using protective splints or slings for approximately 2 weeks. After the short period of protection, restricted movements are started. Mobilization begins with active, active-assisted, and finally passive range-of-motion exercises at the site of the nerve repair. The rehabilitation goals in the early postoperative period are to regain full passive range of motion and prevent joint stiffness and contractures.

Later stages of rehabilitation focus on motor or sensory reeducation (or both). Successful motor function requires not only successful motor reinnervation but also restoration of the central mechanisms controlling motor function. In the case of nerve transfer, the transferred nerve is now innervating a different muscle, a concept similar to the reeducation required after tendon transfers. Rehabilitation is accomplished by having the patient attempt to contract both the donor muscle and the reinnervated muscle at the same time. For example, flexion of the wrist while attempting to flex the elbow is important in retraining of the FCU-to-biceps transfer. Sensory reeducation requires cortical remapping through sensory input from the newly innervated area and begins as soon as the patient is able to perceive any type of sensory stimulus.

Pearls and Pitfalls

- It is important to document preoperative pain for baseline comparison with postoperative symptoms.
- If pain is an issue preoperatively, the use of a Bier block containing bretylium and lidocaine (Xylocaine) is helpful, even when general anesthesia is used. In addition, preoperative and postoperative oral gabapentin (Neurontin) and a postoperative pain pump are useful.
- Ask advice for complicated nerve cases by consulting individuals with greater experience. It is usually "when" to operate that is at issue, not so much "what" operation to perform.
- Avoid the tendency to find "anatomic anomalies" during surgery. The anatomy is usually consistent, and "anomalies" generally represent misunderstanding of the anatomy.
- Encourage early postoperative movement.

SUGGESTED READINGS

Mackinnon SE (ed): Peripheral Nerve Surgery. Clin Plast Surg 30, 2003.

Mackinnon S, Novak C: Nerve transfers: New options for reconstruction following nerve injury. Hand Clin 15:643, 1999.

Nath R, Mackinnon S: Nerve transfers in the upper extremity. Hand Clin 16:131, 2000.

Novak C, Mackinnon S: A distal anterior interosseous nerve transfer to the deep motor branch of the ulnar nerve for reconstruction of high ulnar nerve injuries. J Reconstr Microsurg 18:459-463, 2002.

Oberlin C, Beal D, Leechavengvongs S, et al: Nerve transfer to biceps muscle using a part of ulnar nerve for C5-6 avulsion of the brachial plexus: Anatomical study and report of four cases. J Hand Surg [Am] 19:232, 1994.

Tung T, Mackinnon S: Flexor digitorum superficialis nerve transfer to restore pronation: Two case reports and anatomic study. J Hand Surg [Am] 26:1065, 2001.

Brachial Plexus Injuries

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In the latter part of the 19th century, Duchenne provided the anatomic basis of brachial plexopathy by ascribing specific nerve injury to specific muscle dysfunction. Further refinement of brachial plexus anatomy was derived from electrical stimulation studies performed by Erb and colleagues in 1874. The first attempted surgical repair of brachial plexus injury was performed by Thorburn in 1900. However, equivocal outcomes and high perioperative morbidity dampened enthusiasm for surgical intervention until a resurgence of interest brought on by the plethora of wartime plexus injuries. In 1947, Seddon described surgical repair with nerve grafts, and a decade later, Sutherland published an analysis of the pathologic features of brachial plexus injury with accompanying strategies for surgical treatment. The advent of microsurgical technique further promoted the treatment of these injuries.

The modern philosophy of evaluation and treatment of brachial plexus injury relies on technology-aided diagnosis, precise surgical planning, creative application of grafts and neurotization, aggressive therapy, and lesion-specific splinting techniques.

Pathologic Anatomy

Function of the upper extremity is the result of neuromuscular communication between the somatosensory afferent and somatomotor efferent fibers of the fifth through eighth cervical and first thoracic spinal nerves. The five spinal nerves exit the posterior triangle of the neck behind the anterior scalene and form the three trunks of the brachial plexus on the anterior surface of the middle scalene (Fig. 1). Before merging to form the upper (superior) trunk of the plexus, the fifth and sixth cervical nerves give rise to the nerve to the subclavius muscle, the dorsal scapular nerve, and the suprascapular nerve. The dorsal scapular nerve to the levator scapulae and rhomboids arises from the fifth cervical root. The suprascapular nerve to the infraspinatus and supraspinatus and the nerve to the subclavius originate at the junction of the formation of the superior trunk. The seventh cervical nerve becomes the middle trunk after giving a contribution to the long thoracic nerve from C5 and C6 to the serratus anterior. The lower (inferior) trunk is formed at the superior margin of the thoracic inlet by the eighth cervical nerve and the first thoracic nerve and assumes a position just superior to the first rib. Six divisions of the plexus, three anterior and three posterior, result from bifurcation of the previously described trunks. The divisions traverse lateral to the thoracic inlet and posterior to the subclavian artery. The cords of the plexus lie immediately behind the pectoralis minor adjacent to the second portion of the axillary artery. The anterior divisions of the superior and middle trunks unite lateral to the axillary artery to give rise to the lateral cord. The posterior divisions of all three trunks merge to form the posterior cord posterior to the artery, whereas the medial cord is a continuation of the anterior division of the inferior trunk on its medial aspect.

In the axilla, the lateral cord gives rise to the lateral pectoral nerve to the pectoralis major muscle. The posterior cord contributes to the upper and lower nerves of the subscapular muscle and also to the nerve to the latissimus dorsi (thoracodorsal nerve). Cutaneous sensory information of the medial aspect of the arm and forearm is relayed through respective nerves to the medial cord. Innervation of the pectoralis major and minor muscles from the medial pectoral nerve also originates from the

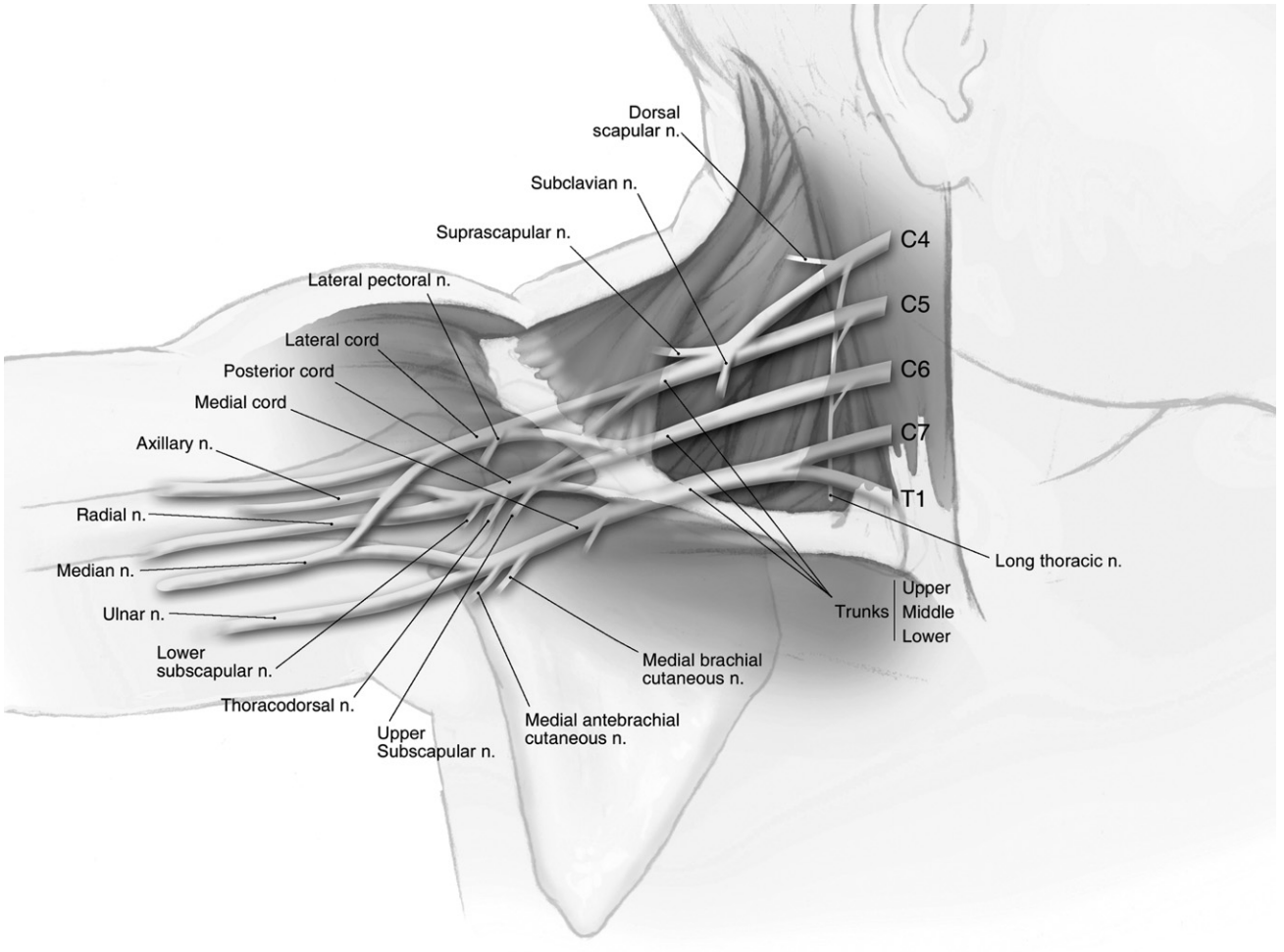

Figure 1 • Brachial plexus anatomy.

medial cord. The terminal branches of the cords give rise to the musculocutaneous nerve (lateral cord), radial and axillary nerves (posterior cord), and the ulnar nerve (medial cord). The median nerve is formed from the terminal branches of the lateral cord (lateral portion) and medial cord (medial portion). The terminal branches innervate the various muscles of the upper extremity that are involved in sensory and motor function of the shoulder, elbow, wrist, and hand.

The major caveat to the anatomy of the brachial plexus is the varying contributions of the fourth cervical and second thoracic spinal nerves. A prefixed or postfixed brachial plexus occurs when the entire plexus originates one cervical level higher or lower, respectively, than normal. Anatomically, a prefixed plexus is farther away from its innervated structures in the upper extremity, thereby allowing greater tension. Conversely, a postfixed plexus has its lower trunk surrounding the first and second ribs and is therefore more susceptible to thoracic injury.

Etiopathogenesis

Insults to the brachial plexus are the result of direct compression, traction, or penetrating trauma. Injuries are classified as open versus closed and acute versus insidious in onset. Open injuries are usually the result of projectiles penetrating the overlying muscles with direct trauma to the plexus or subsequent traction on the nerve roots. Supraclavicular injuries predominate in this scenario, whereas infraclavicular injuries are associated with thoracic penetrating wounds. Closed compression injuries result from direct force applied to the neck. Such injuries most commonly result from the chronic use of backpacks and shoulder harnesses, which allows the clavicle or the soft tissue structures to transfer direct force to the nerves. Hematoma or injury to the subclavian or axillary arteries may also apply direct pressure on the cord structures.

Traction injuries occur with cephalad or caudal force applied to the arm and also with hyperextension and lateral flexion of the cervical spine. The majority of cases are caused by injuries associated with motor vehicle accidents. However, contact sports may result in high impact torsion of the upper roots of the plexus. In children, particularly neonates, upper root injury is the result of obstetric-related injuries during difficult or instrumented vaginal deliveries; macrosomia, gestational diabetes, and shoulder dystocia predispose newborns to this type of injury. Traction on the lower portion of the plexus is related to excessive or swift abduction and external rotation of the arm, which may occur in dragging or hanging incidents. Upper plexus injuries are more common because of a greater degree of exposure to both open and closed trauma in the unprotected cervical region.

At the cellular level, disruption of the nerves, fascicles, and the nerve root complex may occur with varying degrees of complexity. Avulsion of the nerve root involves preganglionic disruption of the dorsal and ventral roots of the spinal nerve. Rupture of the spinal nerve takes place at any location distal to the dorsal root ganglion. Peripheral nerve injury can be additionally classified by the degree of axonal damage and demyelination (Fig. 2). *Neurapraxia* implies stretch injury without frank disruption of the axon. Demyelination may occur and result in a conduction block, or Sunderland's first-degree injury. Damage to the axon, or *axonotmesis*, is a second-degree injury in which the connective tissue sheath of the nerve is left intact. Neurapractic and axonotmetic lesions resolve with minimal functional impairment. *Neurotmetic* injuries, which comprise Sunderland's third- through fifth-degree injuries, imply progressive disruption from endoneurium to perineurium to epineurium. Spontaneous regeneration is disorganized and functional recovery is incomplete at best. Surgical intervention is generally warranted for the more severe injuries. Mackinnon's sixth-degree injury is any combination of the aforementioned lesions along the length of the same nerve.

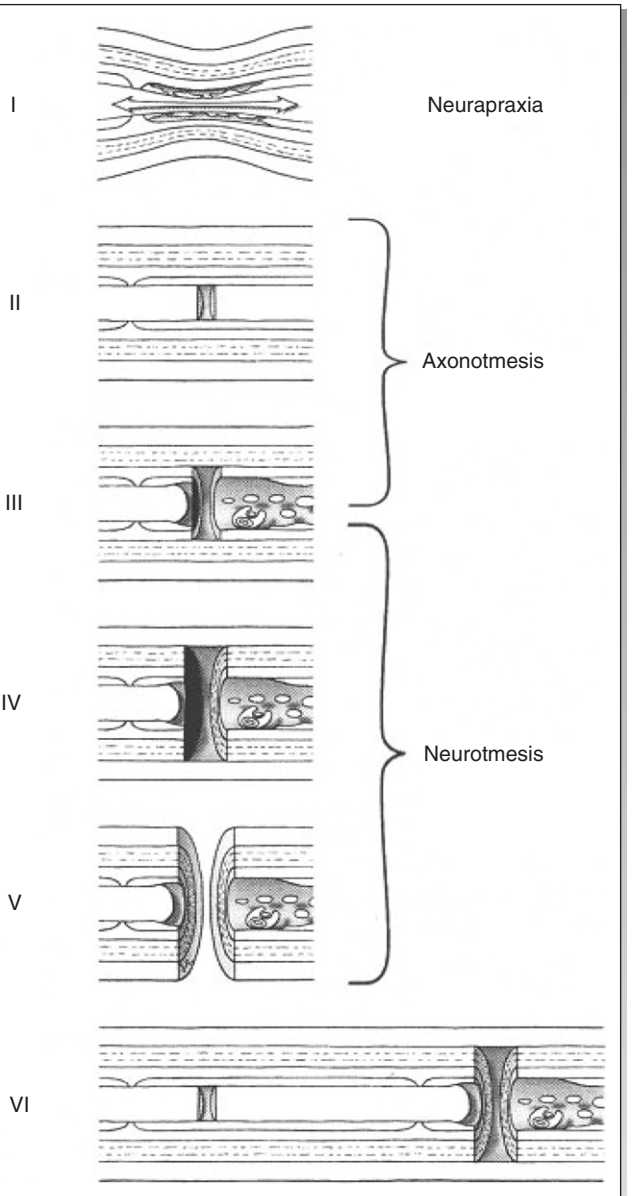

Figure 2 • Classification of peripheral nerve injuries. (Adapted from Brandt KE, Mackinnon SE: Microsurgical repair of peripheral nerves and grafts. In Aston SJ, Beasley RW, Thorne CHM [eds]: Grabb and Smith's Plastic Surgery, 5th ed. Philadelphia, Lippincott Raven, 1997.)

Diagnostic Studies

A complete history and physical examination are paramount and often of higher yield than diagnostic studies in assessing potential injury. However, identifying the site and deciphering the extent of nerve damage frequently require operative exploration with intraoperative electrophysiologic studies.

The mechanism and velocity of the injury provide insight into the location of the nerve injury. Muscle atrophy and contracture vis-à-vis the opposite limb give clues to the pattern of nerve involvement. The British Medical Research Council scale is used to standardize the extent of paralysis (Table 1).

Motor examination is aimed at identifying the exact nerve root deficits. The use of diagrams that refer the axonal distribution to the function of the nerve roots (see Fig. 1) obtained during initial assessment of the injury is crucial to planning repair. A schematic of the plexus that takes into account motor and sensory deficits assists in deciphering an avenue for rehabilitation and reconstruction. At the glenohumeral joint, abduction of the arm, shoulder extension and external rotation are relayed through C5, adduction and shoulder flexion through C6, and internal rotation through C6 and C7. At the elbow, forearm flexion and supina-

TABLE 1 British Medical Research Council Muscle Grading System

MUSCLE GRADE	OBSERVATION
1	No contraction
2	Flicker or trace of contraction
3	Active movement with gravity eliminated
4	Active movement against gravity
5	Active movement against gravity and resistance
6	Normal power

tion are the result of stimulation from C5, pronation from C6, and extension from C7. The wrist and hand receive input from C7 through T1, wrist and finger extension from C7, and wrist and finger flexion and contraction of the intrinsic muscles of the hand from C8 and T1.

Sensory examination is undertaken by assessment of pain, temperature, and proprioception modalities corresponding to the dermatomal distribution of the nerve roots. The sensory afferent roots of the medial aspect of the hand, forearm, and arm are received at C8, T1, and T2, respectively. On the lateral aspect, C5 and C6 receive information from the forearm and arm and C7 from the dorsum of the hand.

Radiographic Studies

The high-impact nature of closed injuries, especially those that are the result of direct trauma from falls and motor vehicle accidents, warrants complete evaluation of the cervical spine. Cervical radiographs are essential in assessment of trauma and may also reveal fractures and subluxations that damage the spinal cord and nerve roots as they exit their foramina. Plain radiographs of the chest, shoulder, and humerus are useful for excluding fractures and dislocations of the bony structures that may suggest associated nerve or vascular injury. Contrast-enhanced computed tomography (CT) and magnetic resonance imaging of the cervical soft tissues are the least invasive techniques for analyzing the anatomic relationships of deformed trunks, divisions, and cords of the plexus. Identification of impinging lesions such as hematomas or aneurysms and localization of scar neuromas in delayed evaluation are best achieved with these diagnostic tools. The use of CT-myelography, which produces a contrast-enhanced image of the subarachnoid space, can lend insight into the possibility of root avulsion. An avulsion injury damages both the nerve root and meninges and causes diverticula, which are referred to as traumatic meningoceles or pseudomeningoceles.

Despite the enhanced anatomic information gained from radiographic studies, interpretation of the functional data gained from physical examination is of extreme importance and ultimately governs decision making for surgical repair.

Electrophysiologic Assessment

An acutely injured nerve may be electrically stimulated within the first 72 hours before complete degradation of the neurotransmitters at the motor end-plate. After this period, fascicular discrimination can be undertaken microsurgically, with particular attention paid to the internal topology. In most instances, it takes 2 to 3 weeks for the characteristic changes of nerve injury and regeneration to be seen on electromyograms. Before this period, fibrillations and altered wave patterns are not specific. In the operating room, a conduction drop across neuromas gives insight into the functional status of the nerve and provides prognostic data for potential recovery. Somatosensory evoked potentials and sensory nerve action potentials are used to discriminate preganglionic and postganglionic injuries when nerve root avulsion is suspected. When evoked potentials are elicited from areas of anesthesia, a preganglionic or dorsal root injury is assumed. Additional electrophysiologic assessment is outlined in Table 2.

Treatment

Primary Exploration

Exposure of the brachial plexus is obtained by either *supraclavicular* or *infraclavicular* incisions. The

TABLE 2 Electrophysiologic Findings after Nerve Injury

	NORMAL	NEURAPRAXIA	AXONOTMESIS/ NEUROTMESIS
Electromyography	Normal muscular response	Absent muscular response Fibrillations absent	Absent muscular response Fibrillations present
Nerve conduction study	Normal action potentials Normal latency	Reduced motor and sensory action potential Prolonged latency	Absent motor and sensory action potential Latency absent
Somatosensory evoked potentials	Normal cortical response	Decreased conduction Increased latency	Conduction decreased >50% Latency increased >10%

roots, trunks, and early branches of the plexus are exposed above the clavicle by making an incision on the posterior border of the sternocleidomastoid. Dissection is carried through the sternoclavicular fossa to the anterior scalene muscle. During dissection, the external jugular vein is ligated, but care should be taken to avoid injury to the internal jugular vein and to preserve the cutaneous branches of the cervical plexus and the spinal accessory nerve. Retraction of the omohyoid and the lateral edge of the sternocleidomastoid assists in identification of the phrenic nerve on the ventral aspect of the anterior scalene muscle. Following the phrenic nerve superiorly leads to identification of the upper roots of the plexus. The remainder of the dissection consists of exposing the plexus in an inferolateral fashion while preserving the suprascapular, transverse cervical, and dorsal scapular arteries.

The cords and terminal branches are best exposed through an *infraclavicular* incision beginning at the superior margin of the clavipectoral groove and directed laterally and obliquely inferior to the anterior axillary fold. Access to the cords and terminal branches adjacent to the axillary artery is achieved by reflection of the pectoralis minor or caudal retraction of the clavicle and subclavius muscle.

Repair

After adequate operative exposure is obtained, the suspected avulsions, disruptions, and neuromas are identified for additional evaluation and primary repair. Electrophysiologic assessment is performed to determine whether exposed roots are in continuity with the spinal cord. Somatic evoked potentials from stimulation of muscle groups are measured by using electrodes placed on the parietal aspect of the scalp. Nerve conduction across an area of injury, including neuromas, is measured. The lesion is repaired if there is significant conduction loss (greater than 50%). Those with less conduction loss are usually first- or second-degree injuries that will improve with time. Microsurgical neurolysis is indicated for any neuroma that has some degree of nerve conduction (neuroma in continuity). Intraneural compression from fibrosis is relieved by meticulous decompression with evidence of fascicular release. If after adequate dissection there is still no conduction or incomplete conduction—as assessed by the degree of muscle contraction—the defect is assumed to be a grade III or IV lesion and the neuroma is excised. The proximal nerve is resected back to normal-appearing fascicles. The viability of an avulsed nerve stump can be assessed by intraoperative frozen-section analysis or by direct electrical stimulation. The presence of dorsal root ganglion cells by frozen section or response to stimulation excludes the possibility of avulsion. This nerve root can be used as an intraplexal motor donor.

Nerve conduits (nerve grafts, veins, and bioabsorbable tubes) can be used to restore continuity and promote regeneration by direct interposition. Common sites for nerve graft harvest include the sural, sensory branch of C4, great auricular, superficial radial, lateral femoral cutaneous, and medial antebrachial cutaneous nerves. If there is inferior avulsion of the plexus with confirmed preganglionic injury, the ulnar nerve may be used as a nerve transfer or as a free vascularized nerve graft for intraplexal repair. Establishment of the internal orientation is accomplished with the aid of distal neurolysis, awake stimulation, intraoperative immunohistochemical stains, and precise attention to anatomy.

When avulsion injuries are encountered, nerve transfers from functional nerve roots (neurotization) are performed to allow motor or sensory input to damaged nerves. Ruptured nerve roots provide an intraplexal donor for interposition grafting to distal lesions. Extraplexal motor donors available for transfer include the phrenic, hypoglossal, and spinal accessory nerves; the motor and sensory branches of the cervical plexus; and the third through sixth intercostal nerves. The contralateral spinal accessory nerve and the nerve roots of C7 through cross-thoracic transfer with interposition nerve grafts provide additional motor donor nerves.

Reconstruction is designed to provide maximal useful function of the limb rather than complete repair of all structures involved. Injuries resulting from complete avulsion of all the roots and multiple disruptions of the distal portions of the plexus make recovery unpredictable. Reconstruction should be aimed at restoration of at least two muscle groups that can provide functional ability for carrying objects, retracting the limb from danger, and stabilizing the limb. Restoration of muscle function should be prioritized. The highest priority is preservation of elbow flexion. Next in importance is stabilization of the shoulder—possibly by nerve transfers to the supraspinatus and deltoid muscles. The third priority is reconstruction of median-innervated motor and sensory function. Because recovery of the intrinsic function of the hand is usually poor, repair of the ulnar nerve is of low priority.

Posttraumatic Brachial Plexus Reconstruction

A few generalizations can be made about the timing and approach to repair, but each brachial plexus injury requires individual assessment and specific treatment. Acute open injuries should be explored immediately to assess for associated vascular injury and to repair sharply transected nerves. These injuries have the best recovery if repaired within 24 hours. In closed injuries in which vascular damage is not suspected, nerve function can be reassessed within 2 to 3 weeks. Determination of nerve root avulsion is essential because this form of injury does not connote a good prognosis without surgical intervention (Fig. 3).

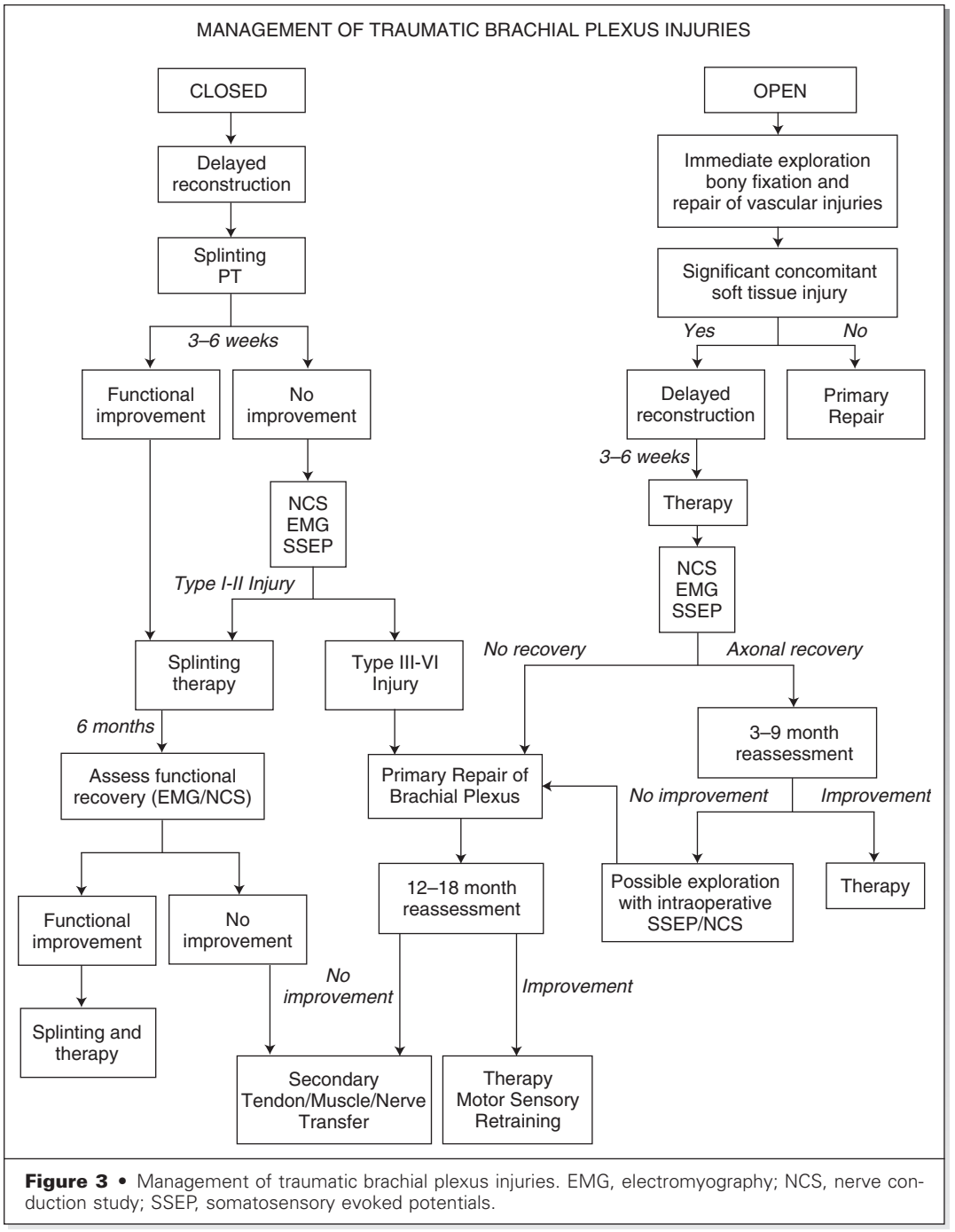

Figure 3 • Management of traumatic brachial plexus injuries. EMG, electromyography; NCS, nerve conduction study; SSEP, somatosensory evoked potentials.

Considerations during microsurgical repair are as follows: (1) avulsed roots require neurotization of their distal defects, (2) postganglionic ruptures are coapted with viable proximal stumps via nerve grafts, (3) similar-caliber nerve transfers and conduits are used for maximal reconstructive results, (4) motor and sensory topographic integrity is maintained during neurorrhaphy, and (5) restoration of elbow flexion and shoulder stabilization is given reconstructive priority.

TOTAL AVULSION. Root avulsion of C5-T1 allows minimal options for reconstruction because of the lack of intraplexal donors. To stabilize the shoulder and allow elbow flexion, the supraspinatus, deltoids, and biceps take priority in repair. Direct neurotization of the suprascapular nerve with the spinal accessory nerve and transfer of three intercostal nerves to the anterior division of the upper trunk will allow elbow flexion and protective sensation of the hand via the musculocutaneous and the lateral

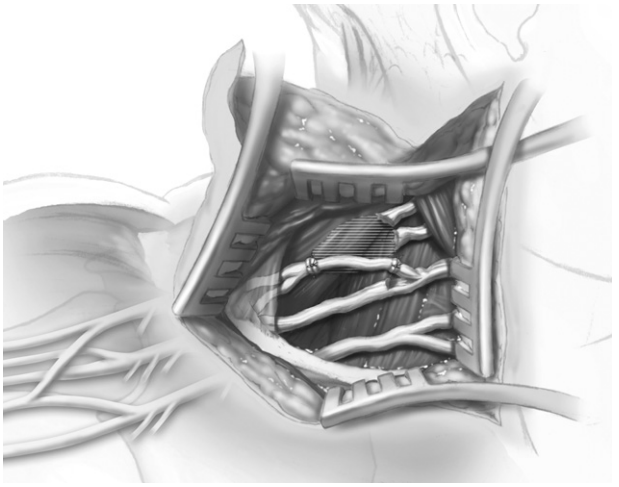

Figure 4 • Brachial plexus repair following avulsion and rupture of C5-C6. The suprascapular nerve is neurotized by the spinal accessory nerve. The functional C7 root is split and used as a donor for the anterior and posterior divisions of the upper trunk via multiple sural nerve grafts.

portion of the median nerve. Alternatives include direct intercostal neurotization of the musculocutaneous nerve and the use of contralateral C7 or ipsilateral cervical plexus motor donors to neurotize the axillary and medial nerves.

UPPER ROOT AVULSION. Reconstruction for combined preganglionic injury to C5/C6 or C5/C6/C7 involves the same principles applied to restoration of shoulder stabilization and shoulder flexion. The suprascapular nerve is neurotized by the ipsilateral spinal accessory, and priority is directed toward reconstruction of the musculocutaneous, axillary, and medial nerves with intercostal nerves. If the C7 root is intact, it can be completely or partially sacrificed (Fig. 4) to reconstruct the upper trunk. The distal C7 targets are neurotized by extraplexal donors (ipsilateral cervical motor nerves, intercostal nerves). When the C7 root is damaged, a viable ulnar nerve can also be used for distal transfer to the musculocutaneous nerve.

LOWER ROOT AVULSION. Patients with avulsion of the C8/T1 roots must be counseled early that lower root injuries portend a poor prognosis and restoration of hand function may necessitate multiple operative procedures. The intercostal nerves are used to neurotize the medial cord and the motor portion of the medial or ulnar nerves. If the C7 root is avulsed, it must be repaired with multiple intercostal grafts, and the lower defects are repaired as described earlier.

RUPTURE. Postganglionic lesions usually occur with concomitant avulsions of other roots of the plexus. A common pattern of proximal brachial plexus injury secondary to traction is lower root avulsion with upper root rupture. In this scenario, the viable upper roots can be used to relay information by nerve grafts to the cords or the proximal divisions of the plexus and by neurotization of the lower trunks via the intercostal nerves and the suprascapular nerve via the spinal accessory nerve. Neurotization of the lower plexus is accomplished in a fashion that allows continuity with the middle trunk if there is a successful nerve graft to the upper trunk. Otherwise, direct neurotization to the musculocutaneous nerve is performed. The defect from a rupture at C5 can be repaired with cabled sural nerve grafts from C5 to the superior trunk of the plexus.

If avulsion of the C5 and C6 donor nerves has occurred, alternative measures such as contralateral C7 transfer may be considered. Moreover, priority may be given to neurotization of the musculocutaneous nerve with transfers from fascicles of the ulnar nerve or intercostal nerves.

In addition to operative repair of primary brachial plexus injuries, secondary reconstruction is performed to compensate for irreparable nerve injuries, such as in patients who have sustained massive destruction of the plexus, who have failed primary repair, and who are initially seen with an unrepaired primary injury within 12 months of injury. Secondary procedures consist of transferring functional muscle groups to positions that effectively restore the resultant nerve deficit. Such transfers are performed mostly to restore elbow flexion and external rotation of the shoulder. Transfer of the insertion of the latissimus dorsi, pectoralis major, or triceps muscles onto the biceps tendon can augment flexion at the elbow.

Shoulder dysfunction stems from a combination of scar contracture around the joint capsule and loss of the external rotators supplied by the upper plexus. Release of the contracture and removal of the fibrosis may be of initial benefit. Simultaneously, transfer of the origins of the latissimus dorsi and teres major to a posterior position on the humerus allows external rotation of the shoulder. Free functional muscle grafts are useful options for restoration of elbow flexion and hand function; the contralateral latissimus dorsi, rectus, and gracilis are used as functional donors. At the time of exploration, the ipsilateral spinal accessory nerve, the intercostal nerves, or the contralateral C7 root (via cross-thoracic grafts) can be banked in the medial or lateral antecubital fossa for later neurotization with free muscle flaps.

Obstetric Brachial Plexus Reconstruction

Primary reconstruction is attempted in any patient who presents at 3 months of age with complete paralysis, has two muscle group deficits (shoulder rotators, elbow flexor, etc.), or demonstrates weakness that has not improved by 6 months (Fig. 5).

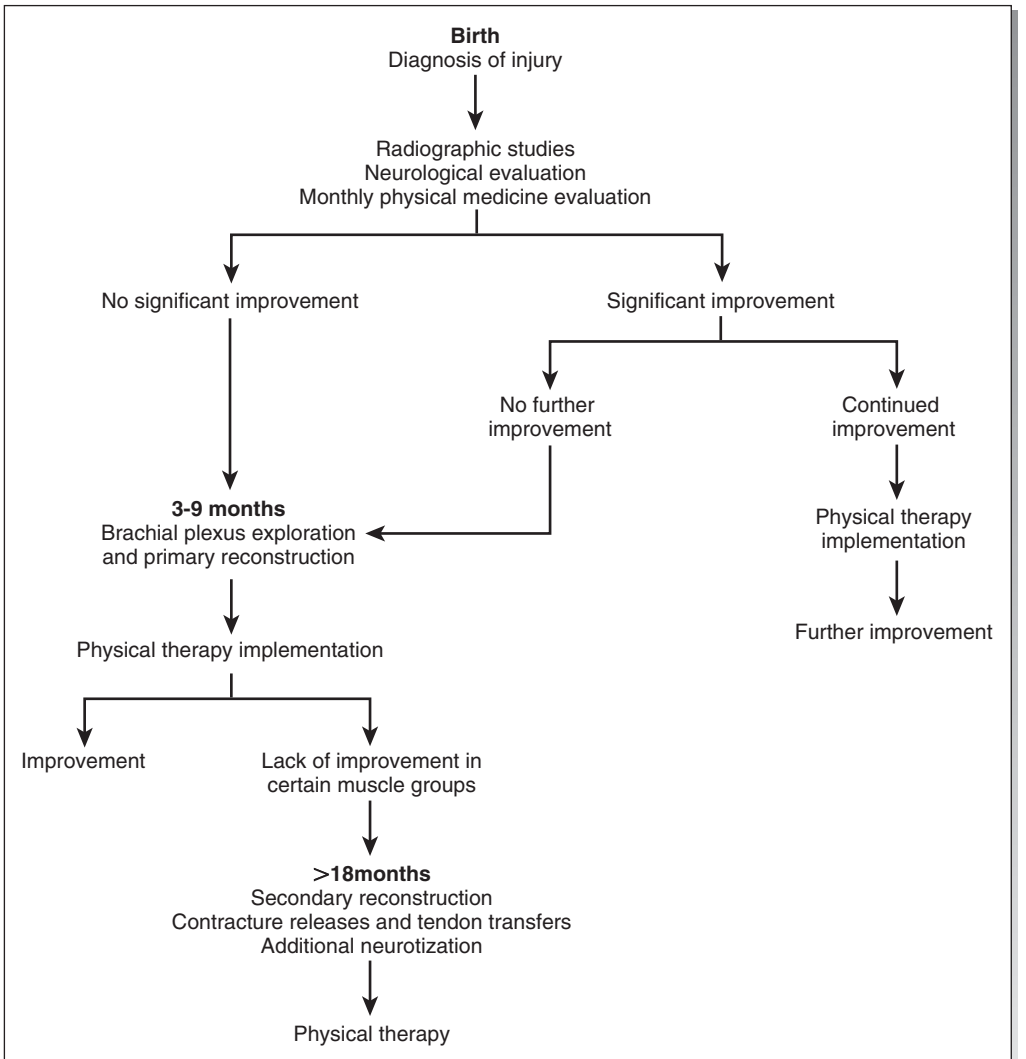

Figure 5 • Management of obstetric brachial plexus injuries.

Neuromas are excised and viable nerve roots are identified for neurotization. Intact roots assessed by somatosensory evoked potentials are neurotized with multiple sural nerve grafts to the upper and middle trunk or their respective anterior and posterior divisions.

The lesions that result from brachial plexus injuries predominantly involve the upper roots. If the C5 root is avulsed, the ipsilateral accessory nerve is connected to the suprascapular nerve. Rupture of the upper trunk is corrected by multiple nerve grafts to the corresponding divisions from intact C5 and C6 roots. If the C7 root is damaged, the C6 root is joined to the middle trunk, the C5 root is attached to the upper trunk, and the suprascapular nerve is neurotized with the accessory nerve. When avulsions are severe and involve the entire plexus and when intraplexal donors are unavailable, extraplexal donors are used (e.g., the intercostal, phrenic, hypoglossal, and contralateral C7 and axillary nerves.

When recovery after primary reconstruction or nonsurgical management is incomplete, secondary deformities resulting from contracture may arise; because of the nature of upper plexus deficits, patients are usually fixed in an internally rotated, adducted position. Secondary reconstruction, performed at least 12 months after primary reconstruction or at 18 months of age, is designed to release the periglenoid contracture and restore abduction and external rotation with muscle transfers.

Correction of the majority of secondary defects can be accomplished with release of the subscapularis and pectoralis at the humerus. The axillary nerve is freed of scar by microneurolysis, followed by transfer of the teres major and latissimus dorsi to the posterior aspect of the humerus to allow external rotation (Fig. 6). Elbow flexion is restored with partial triceps transfer and lengthening. Tendon transfers in the forearm are also performed to repair hand deficits. A schematic of possible secondary procedures is outlined in Table 3.

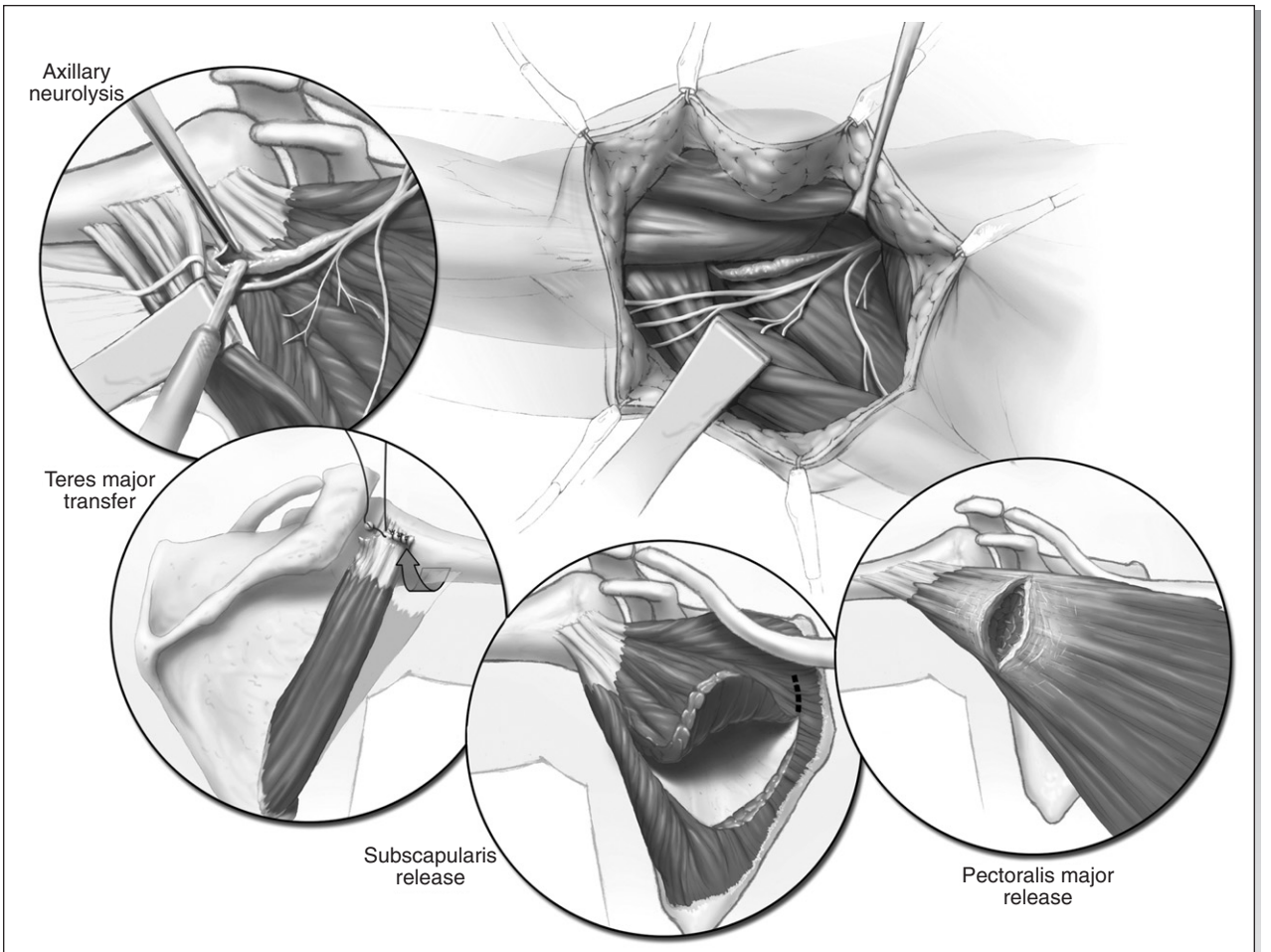

Figure 6 • Secondary repair, the quad procedure: (1) release of the subscapularis and pectoralis at the humerus, (2) axillary nerve microneurolysis, (3) tendon transfer of the teres major and (4) latissimus dorsi to the posterior aspect of the humerus.

Outcomes

Functional recovery is related to the type of injury, the age, and the timing and type of repair. In posttraumatic reconstructions, the prognosis is best in young patients with short denervation times (<6 months). Favorable outcomes in both traumatic and obstetric injuries are seen in patients who sustain upper plexus injuries with fewer avulsions and no associated bony or vascular injury. The better prognosis with upper root palsies than with lower root palsies is the result of preserved hand function and closer muscle targets. Concomitant injury to the subclavian vessels results in ischemia to the limb and excessive scar formation that worsens the nerve injury.

Intraplexal donors are superior to extraplexal donors. Microneurolysis and interposition grafting are more successful than coaptation grafting. Direct neurotization with specific targets has produced results superior to those of intraplexal nerve grafts. Coaptation of the spinal accessory with the suprascapular nerve together with neurotization of the musculocutaneous nerve with the intercostal nerves provides reproducible restoration of shoulder and elbow function. The prognosis is improved with the use of similar-caliber donor and recipient nerves and with distal transfers. Coaptation closer to the muscle end-plate allows less dispersion of donor nerve fibers and provides more specific muscle targets.

In obstetric brachial plexus repair, the greatest improvement in deltoid, biceps, and triceps function occurs with primary brachial plexus repair. Upper plexus (C5/C6) injuries have a better prognosis than lower plexus (C7/C8/T1) injuries, and the number of avulsions is inversely correlated with the amount of improvement after surgery.

In secondary reconstructions, there is a marked improvement in abduction and external rotation after surgery for obstetric brachial plexus injuries. Success is independent of whether previous therapy was instituted, including primary reconstruction.

TABLE 3 Secondary Reconstruction: Indications and Methods

SECONDARY RESIDUAL DEFORMITY	SECONDARY QUAD PROCEDURE
Internal rotation	Muscle transfers to the teres minor
Shoulder adduction	Latissimus dorsi
± Scapular winging	Teres major
± Biceps contracture	Muscle releases
	Subscapularis
	Pectoralis major and minor
	Axillary nerve neurolysis and decompression
Poor elbow extension	Nerve exploration and neuroplasty
	Radial nerve
	±Tendon transfers
Poor extension of the wrist and digits	Tendon transfers
Poor elbow extension	PT → ECRB
	FCR → EDC
	PL → EPL
	± Wrist capsulodesis
Poor elbow flexion	Nerve exploration and neuroplasty
Poor supination	Radial nerve
	Musculocutaneous nerve
	± Nerve transfers
Elbow flexion contracture	Biceps lengthening after failed serial cast splinting
Poor flexion of the wrist and fingers	Nerve exploration and neuroplasty
	Median nerve
	Ulnar nerve
	± Free muscle transfers

ECRB, extensor carpi radialis brevis; EDC, extensor digitorum communis; EPL, extensor pollicis longus; FCR, flexor carpi radialis; PL, palmaris longus; PT, pronator teres.

Pearls and Pitfalls

- Repair of brachial plexus injuries depends on the time between initial evaluation and injury and requires multimodal therapy to restore useful function to the upper extremity.
- Acute open injuries are explored immediately to repair bone and vascular injuries. Nerve repair at that time is performed only if easy tension-free coaptation can be accomplished between cleanly transected nerves. Otherwise, secondary exploration is performed after failure of recovery or neurophysiologic testing.
- Closed injuries undergo evaluation for 3 weeks; they are explored if no recovery is gained and reassessed monthly if partial recovery is evident.
- Patients who fail primary repair or are initially evaluated after 1 year may still have hope for recovery with secondary muscle transfers.

SUGGESTED READINGS

Alnot JY: Traumatic brachial plexus lesions in the adult: Indications and results. Hand Clin 11:623, 1995.

Leffert RD: Brachial Plexus Injuries. New York, Churchill Livingstone, 1985.

Merrell GA, Barrie KA, Katz DL, Wolfe SW: Results of nerve transfer techniques for restoration of shoulder and elbow function in the context of a meta-analysis of the English literature. J Hand Surg [Am] 26:303-314, 2001.

Millesi H: Brachial plexus injuries: Management and results. Clin Plast Surg 11:115, 1984.

Millesi H: Brachial plexus injury in adults: Operative repair. In Gelberman RH (ed): Operative Nerve Repair and Reconstruction. Philadelphia, Lippincott, 1991.

Shenaq SM, Berzin E, Lee R, et al: Brachial plexus birth injuries and current management. Clin Plast Surg 25:527-536, 1998.

Shenaq SM, Kim JYS, Armenta AH, et al: The surgical treatment of obstetric brachial plexus palsy. Plast Reconstr Surg 113:54E-67E, 2004.

Sunderland S: Nerves and Nerve Injuries. New York, Churchill Livingstone, 1978.

Terzis JK: Brachial plexus reconstruction in 204 patients with devastating paralysis. Plast Reconstr Surg 104:1221-1240, 1999.

Terzis JK, Papakonstantinou KC: The surgical treatment of brachial plexus injuries in adults. Plast Reconstr Surg 106:1097-1112, 2000.

Tumors of the Hand and Wrist

PETER M. MURRAY

Approximately 7000 malignant musculoskeletal neoplasms are diagnosed annually in the United States, but neoplasms of the hand and wrist are uncommon. It is estimated that 4% of malignant and 15% of benign musculoskeletal neoplasms involve the hand and wrist. It is imperative that clinicians be aware of the characteristics of musculoskeletal malignancy so that these potentially fatal lesions are not overlooked. Because of the rarity of these malignant neoplasms, periodic review of their characteristics and treatment protocols is incumbent on all who treat hand and wrist complaints. Most practicing hand surgeons come in contact with few hand and wrist malignancies during their careers. Therefore, it is recommended that community surgeons refer malignant neoplasms of the hand and wrist to medical centers accustomed to treating such patients.

Progress in chemotherapy, radiation therapy, oncologic surgical procedures, and free tissue transfer techniques has ushered in an era of limb salvage for malignant hand and wrist neoplasms. Fortunately, most benign neoplasms of the hand and wrist can be treated by marginal surgical resection without fear of recurrence. The focus of this chapter is to outline treatment principles of the more common hand and wrist neoplasms and the reconstructive alternatives available for limb salvage surgery.

Diagnostic Studies

Neoplasms of the hand and wrist can be characterized as either benign or malignant. Ultimately, histologic grade based on the tumor's malignant characteristics finalizes diagnosis (Table 1).

The first step in the evaluation of any suspected neoplasm is a complete physical examination, including inspection and measurement of the mass and identification of any overlying skin changes. Masses larger than 5 cm with associated pain and recent, rapid growth are particularly worrisome. The evaluation should proceed with a neurovascular examination of the affected limb to assess compression of the nerves and vessels. Patients identified with primary bone tumors should be evaluated by serum and protein electrophoresis to rule out myeloproliferative disorders such as multiple myeloma.

TABLE 1 Histologic Grade and Tumor Characteristics

Grade 0	Benign	
Grade 1	Low grade	Few mitotic figures, mostly mature cells
Grade 2	High grade	Many mitotic figures, many immature cells, tumor necrosis

From Enneking WF, Spanier SS, Goodman MA: A system for the surgical staging of musculoskeletal sarcoma. Clin Orthop 153:106-120, 1980.

The evaluation should include biplanar radiography with imaging of the hand, wrist, and forearm, including the joints above and below the tumor. Benign soft tissue lesions do not typically involve bone; however, cortical erosion of the metacarpals or phalanges is not unusual in some slow-growing lesions. High-grade soft tissue sarcomas frequently invade surrounding bone. Primary tumors of bone show either distinct or nondistinct margins, and "cortical breakthrough" is an ominous sign suggesting extension of bone tumor into soft tissue (Fig. 1). Such extension is generally seen with malignant tumors or locally aggressive benign tumors. Bones with tumors destroying more than 50% of the cortex are at substantial risk for pathologic fracture.

Suspected malignant tumors require additional imaging studies. If the lesion is a primary tumor of bone, a non–contrast-enhanced computed tomographic (CT) scan is helpful in determining tumor location and destruction. It is important for the

surgeon to specify the limits of the CT scan so that the entirety of the tumor is included in the study. CT scans should be obtained in the axial, coronal, and sagittal planes with 1-mm cuts; a three-dimensional reformat may also be helpful.

Magnetic resonance imaging (MRI) is the study of choice for soft tissue tumors. The lesion is examined with T1 axial, T1 best long axis, T2 axial, proton density, STIR (short tau inversion recovery), and fat-suppressed post–gadolinium-enhanced T1 sequencing. T1 imaging provides enhanced soft tissue detail, whereas blood, pus, and malignant tumors show signal enhancement on T2 imaging. Malignant soft tissue lesions have an inhomogeneous appearance on MRI. Fat enhances on both T1 and T2 sequencing. In the case of a suspected vascular tumor, conventional arteriography is generally necessary to define the often tortuous and complex anatomy of the lesion. In some instances, selective embolization of vascular channels may be indicated to facilitate tumor resection or even biopsy.

If the imaging studies support the possibility of a malignant neoplasm, a metastatic workup should be performed, including a bone scan and chest and abdominal CT scans. If malignancy seems likely, consultation with a medical oncologist should be initiated prior to biopsy of the tumor.

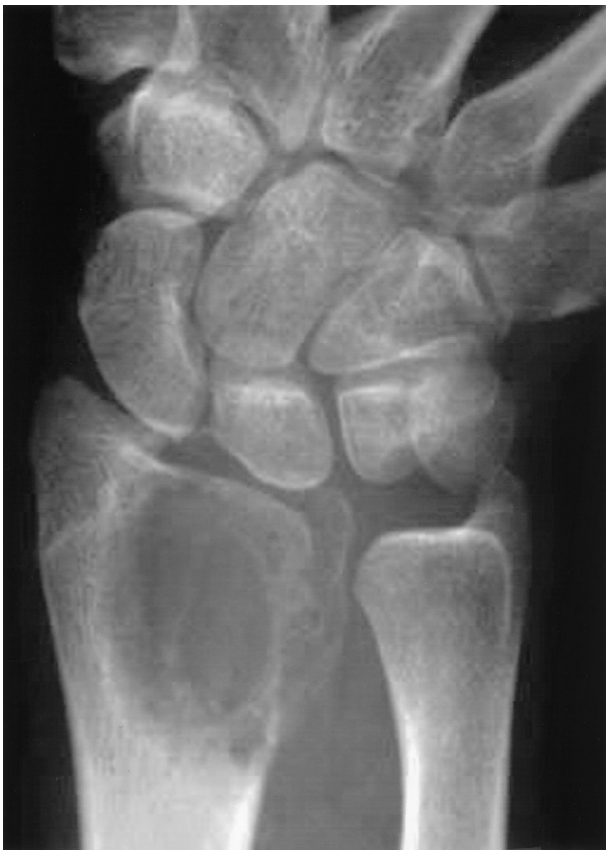

Figure 1 • Giant cell tumor of the distal end of the radius demonstrating cortical breakthrough.

The tumor workup should help the surgeon identify the most appropriate anatomic site for biopsy, usually where the most representative tumor specimen can be obtained with the least difficulty. If metastatic lesions have been identified, the biopsy may be quite removed from the hand and wrist.

Benign lesions of the hand and wrist can be treated by excisional biopsy in the community setting. Biopsy of the suspected lesion is the most important step in establishing the diagnosis and initiating treatment, and the biopsy should be undertaken at the institution performing the definitive surgery.

Surgical Technique

The biopsy incision must be linear and reserved. Extended, transverse incisions are unacceptable because if malignant, the entirety of the biopsy tract is contaminated with tumor cells and must be excised at the time of tumor resection. Transverse incisions can complicate and jeopardize attempts at limb salvage. Meticulous hemostasis must be observed during biopsy.

If the surgeon is confident that the tumor is benign, regional or even local anesthesia can be considered. When performing the biopsy of a suspected malignancy, general anesthesia is preferred over regional anesthesia because needle puncture of the extremity can facilitate spread of malignant tumor by contaminating the intervening soft tissue structures as the needle passes through vascular or lymphatic channels containing tumor cells. Exsanguination of the extremity is *not* performed in the presence of a suspected malignant or aggressive benign lesion for fear of disseminating tumor cells. The arm is elevated for 1 to 2 minutes before inflating the tourniquet to 250 mm Hg.

For primary bony lesions, the cortex of the bone is perforated with a fine drill, creating an oval, thereby placing the least amount of stress on the bone. Small curets are then used to remove a specimen, or a syringe is used if the tumor is liquid in consistency. In addition, cultures should be obtained because many soft tissue infections can be mistaken for tumor.

Reconstructive Goals

The ultimate goal of hand and wrist tumor surgery is complete removal of the lesion while preserving as much function as possible. In general, hand and wrist reconstruction is accomplished by two distinctly different surgical methods: amputation and limb salvage. The ablative procedure required (Table 2) is dependent on the extent of tumor infiltration and the functional demands of the patient. For most

TABLE 2 Tumor Procedure Required for Reconstruction

PROCEDURE	DISSECTION METHOD	CONDITION OF MARGIN
Intralesional resection	Debulking or curettage of lesion or amputation through lesion	Tumor present at margin
Marginal resection	En bloc resection/amputation within reactive zone	No gross tumor, microextension of tumor
Wide resection	En bloc resection/amputation with perimeter of normal tissue removed	Normal tissue at margin. Micro–skip lesions may be present
Radical resection	En bloc resection/amputation of compartment	Normal tissue

Adapted from Enneking WF, Spanier SS, Goodman MA: A system for the surgical staging of musculoskeletal sarcoma. Clin Orthop 153:106-120, 1980.

benign lesions, intralesional or marginal resection is all that is necessary. For malignant lesions, wide or radical resection is typically performed. The terms "wide resection" and "radical resection" are difficult to apply to hand and wrist surgery because the hand and wrist are not compartmentalized in the classic anatomic sense. Many structures in the hand and wrist are contiguous with structures that traverse through the forearm, thereby defining compartmental resection of the hand as an above-elbow amputation. Certain principles of limb salvage surgery, however, can be applied.

Digits

Malignant tumors involving any portion of a digit are best treated by ray amputation, with the exception of malignant tumors at the level of the distal phalanx, which can be treated by transverse amputation. Partial amputation proximal to the level of the flexor digitorum superficialis insertion is often functionally and cosmetically undesirable. For example, amputation of the index finger at the proximal interphalangeal joint often results in the patient "bypassing" the digit for the intact long finger. Amputation of the long or ring finger at the metacarpophalangeal level hinders fine manipulative tasks.

Wrist

At the wrist, the reconstructive goal is to maintain pain-free stability. If stability with mobility cannot be accomplished after resection of the tumor, wrist arthrodesis may be the best surgical option for maintaining function. For example, if a substantial portion of the distal end of the radius or ulna is removed with the bony tumor or if portions of the wrist extensor tendons are sacrificed during tumor resection, wrist arthrodesis is the favored reconstructive option. A stable wrist held in approximately 15 degrees of extension is paramount in regaining prehensile function of the digits. An unstable, painful, or malpositioned wrist adversely affects the patient's grip and digital function.

Coverage and Bone Defect Reconstruction

In wide or radical tumor resection about the hand and wrist, vital structures may become uncovered. Additionally, epitenon, periosteum, epineurium, and vascular adventitia are not suitable for supporting skin grafts. If appropriate coverage is not provided for these structures, hand and wrist function may be severely compromised. In this setting, complications such as infection or nonhealing wounds can occur and result in amputation. With an exposed nerve or tendon, a fasciocutaneous flap provides durable tissue and a gliding surface. For the dorsum of the hand and wrist and for the palm, one can consider a pedicled radial forearm fascial flap. Free tissue coverage alternatives can include a temporoparietal fascial flap, a serratus anterior free muscle transfer, and a rectus abdominis free flap. No matter what option is chosen, immediate coverage of all major neurovascular structures, bone, and tendon is necessary.

Bone defects frequently result from removal of either benign or malignant bone tumors. Excision of benign lesions such as giant cell tumors of bone and aneurysmal bone cysts may create substantial bone defects. In addition, malignant tumors requiring wide resection can leave massive bone defects. The success of limb salvage surgery rests on the ability of the surgeon to reconstruct these defects. Bone defects smaller than 6 cm in length are reconstructed with conventional bone grafting techniques, such as an autogenous iliac bone graft or bone substitutes. Allografts are less desirable alternatives. Defects larger than 6 cm are reconstructed with vascularized bone flaps such as a free fibula transfer.

Treatment

Treatment of tumors of the hand and wrist is individualized according to the neoplasm. With few exceptions, benign tumors may be treated by marginal resection. Malignant tumors are treated by

wide or radical resection with preservation of as much function as possible. In the hand, such a resection may include a ray or double ray amputation. Patients treated in this manner are generally candidates for adjuvant or neoadjuvant treatment. Alternatively, above- or below-elbow amputation can be considered.

Benign Tumors of Soft Tissue

Ganglion cysts account for 60% of all tumors of the hand and wrist. Women are three times more likely to be affected, and they are unusual in children. The cause of ganglions is unknown. The most common location is the dorsal aspect of the wrist, with the majority of such ganglions emanating from the scapholunate interosseous ligament. Other common sites for ganglion cysts are the volar radial aspect of the wrist and the flexor tendon sheath. Treatment options include observation, aspiration, and excision. Pain, limited motion, or recurrence after aspiration is considered an indication for excision.

Ganglion cyst surgery can be performed under local anesthesia with intravenous sedation. A transverse or linear incision is used to approach a cyst on the dorsum of the wrist, whereas a linear incision is preferred for a volar radial ganglion. Bruner's incision is used for ganglions of the flexor tendon sheath. The key to successful ganglion surgery is complete dissection and removal of the stalk, along with a small portion of the adjacent joint capsule. In the case of a dorsal wrist ganglion, the stalk usually arises from the capsule of the radiocarpal joint. The defect created in the capsule is left open, the tourniquet is released, hemostasis is achieved, and the wound is closed. Ganglion cysts excised in this manner have recurrence rates as low as 4%, versus a 50% recurrence rate associated with aspiration alone.

Giant cell tumor of the tendon sheath is the second most common tumor of the hand and wrist region and most commonly occurs in women between the ages of 30 and 50. Histologic features include multinucleated giant cells, histiocytes, and hemosiderin pigmentation. This benign lesion does not metastasize but recurs in 5% to 10% of cases. In larger lesions, MRI of the affected digit is helpful to define the location because the tumor can arise dorsally or volarly and spread around the margins of the finger. A volar Bruner's approach is preferred, but a straight dorsal or midlateral approach can also be used (Fig. 2). The key to removal of a giant cell tumor of the tendon sheath is awareness that the lesion may extend into the proximal or distal interphalangeal joints. Removal of intraarticular extensions, as well as isolated, adjacent lesions in the surrounding tissue, is an important factor in preventing recurrence. Marginal resection is preferred for these tumors.

Epidermal inclusion cysts are the result of traumatic impregnation of epithelial cells beneath the skin of the fingertips or directly into the distal phalanx. The cysts grow slowly and painlessly, although painful lesions can occur. The lesions most commonly affect the thumb and the long finger in men in the third and fourth decades of life. Simple excision of the cyst may be performed under local or regional anesthesia. Extended incisions over the pad of the finger are to be avoided.

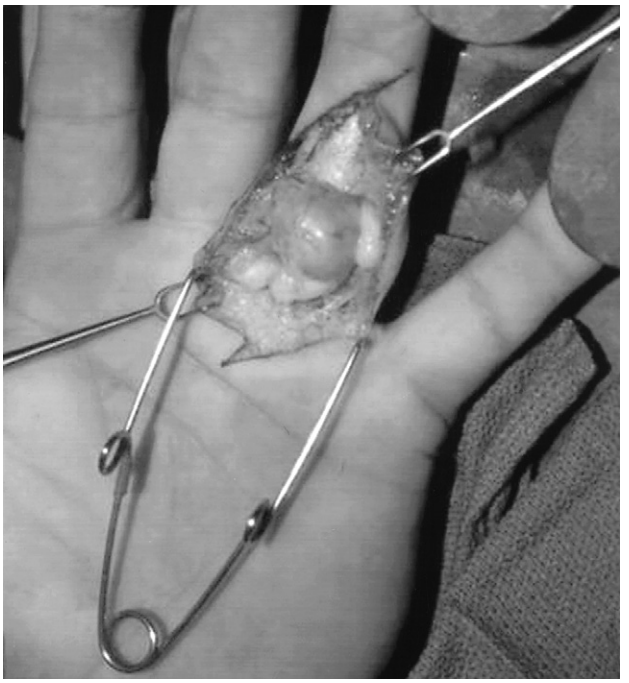

Figure 2 • Bruner's incision to expose a giant cell tumor of the tendon sheath.

Lipomas of the hand may be large in size. These tumors are usually painless; however, when they extend into the carpal tunnel, signs and symptoms of carpal tunnel syndrome may occur. Treatment is excision. Exposure of the superficial palmar arch, the common digital arteries, and the common digital nerves is required. Preoperative MRI is helpful in determining the anatomic extent of these lesions. Recurrence is unusual.

Neurilemoma is the most common peripheral nerve sheath tumor. It is twice as common in the upper extremity as in the lower extremity. Neurilemomas are more frequently seen on the flexor surface of the hand and forearm and can be mistaken for ganglion cysts. Patients usually have a painless mass, although some (50%) have paresthesias; Tinel's sign can often be elicited over the lesion. The lesion arises from Schwann cells and is slow growing. Lesions are easily dissected from the adjacent nerve. Resection is best performed with the aid of loupe magnification or the operating microscope. In most cases, these lesions can be successfully removed with only a small risk for postoperative neurologic deficits.

Hemangiomas are composed of proliferations of endothelial cells and are generally present by the first month of life. Involution of hemangiomas occurs during childhood, with 70% resolving by 7 years of age. Some lesions persist into adulthood, frequently with overlying skin changes. The lesions can be distinguished from vascular malformations by the history and clinical examination. Arteriography, contrast-enhanced CT scanning, or MRI can be helpful. When symptomatic, hemangiomas are treated by marginal excision, with care taken to isolate and ligate all vascular tributaries. Diffuse lesions have a much higher recurrence rate, and large lesions with multiple recurrences may require amputation.

Glomus tumors are a rare tumor that can occur in the subungual area. They originate from the myoepithelial cells of an arteriovenous anastomosis. Small tumors present as a painful nail exacerbated by exposure to cold. They can be extremely tender. Larger tumors give a blue hue to the nailbed and can deform the nail. Diagnosis is by history; plain radiographs may show contour changes in the distal phalanx. Further diagnosis is made by MRI showing an enhancing subungual mass. Treatment is by excision, with care taken to preserve the nail bed.

Benign Tumors of Bone

An *aneurysmal bone cyst* (ABC) is a primary bone tumor believed to result from hemodynamic abnormalities. Approximately 80% of ABCs develop within the first 2 decades of life. The hand and wrist region account for only 5% of all ABCs, and the metacarpals and phalanges are the most common site. Patients typically have pain and swelling. On radiographic examination, cortical expansion is often seen with a sclerotic rim on the remaining cortex (Fig. 3). Marginal curettage of the bony cavity has resulted in recurrence rates approaching 60%. Enhanced cure rates have been reported with the addition of adjunctive cryosurgery to standard marginal curettage. Cryosurgery (liquid nitrogen) causes tissue necrosis through rapid freezing to destroy any remaining tumor cells. The remaining defect is filled with autogenous bone graft. Recurrent lesions may necessitate removal of the entire phalanx or metacarpal and reconstruction with an autogenous, tricortical iliac bone graft or a nonvascularized fibular graft. A less desirable option is the use of allograft bone or bone substitute.

Enchondroma is the most common bone tumor of the hand and wrist and accounts for more than 90% of all hand and wrist bone tumors. The tumors are asymptomatic and usually discovered by coincidence or after pathologic fractures. The lesions are radiographically located in the medullary portion of the bone and are well circumscribed; cortical expansion and punctate calcifications are seen frequently. The presence of multiple enchondromas in the same extremity typically implies the diagnosis of Ollier's disease, whereas Maffucci's syndrome is a rare condition with multiple enchondromas and hemangiomas. Malignant transformation of solitary enchondromas is very rare, but malignant transformation of enchondromas has been reported in as many as 30% of patients with Ollier's disease or Maffucci's syndrome.

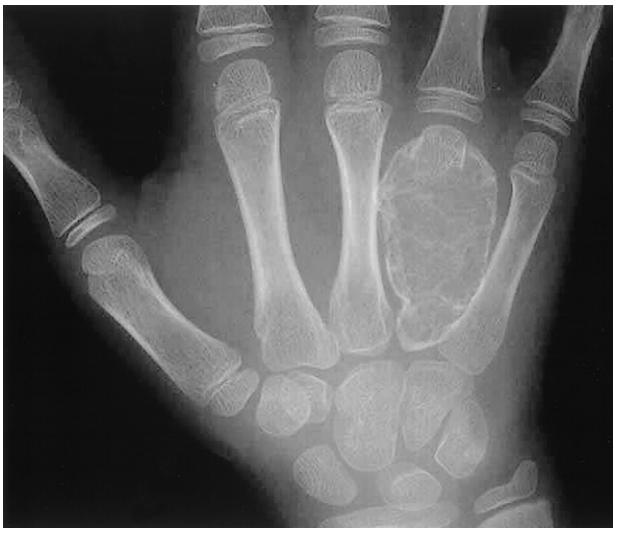

Figure 3 • Cortical expansion caused by an aneurysmal bone cyst in the fourth metacarpal of a child.

For enchondroma lesions compromising greater than 50% of the cortex, impending pathologic fracture is a concern. In patients with a pathologic fracture, splint immobilization is usually adequate to obtain healing. In some instances, open reduction/internal fixation or closed reduction/percutaneous pin fixation of the fracture may be necessary. After healing is achieved, resection and autogenous bone grafting are indicated. Failure to address the enchondroma places the patient at risk for recurrent pathologic fractures.

The enchondroma is removed by intralesional curettage. Lesions of the proximal phalanges and the metacarpals are generally approached dorsally through a midline incision. Recurrence rates for lesions treated in this fashion are 4.5%. Alternatively, excellent results have been reported after using allograft bone or leaving the defect devoid of material. Healing of the defect after curettage and grafting can be expected in approximately 6 weeks.

Giant cell tumor of bone in the hand is uncommon, but approximately 14% of all giant cell tumors occur in the wrist. Although these neoplasms are benign, they may display aggressive characteristics such as local recurrence, bone destruction, and distant metastasis. When identified in the hand and wrist, the lesions are typically painful and can cause local swelling. On radiographic examination, the tumor is lytic with cortical enlargement and indistinct boundaries (see Fig. 1).

Traditional treatment by simple intralesional curettage and bone grafting has resulted in recurrence rates approaching 80%. When used in conjunction with either cryosurgery or cementation, however, curettage of the lesion results in reduced recurrence rates, but the lowest recurrence rates have been reported with wide excision and appropriate reconstruction.

The classic giant cell tumor of bone in the wrist occurs in the distal end of the radius. Wide excision is accomplished through a dorsal approach, with preservation of the extensor tendons. The distal third of the radius is osteotomized and removed along with the pronator quadratus muscle. The wrist is reconstructed by arthrodesis. For defects measuring 6 cm or less, a conventional autogenous iliac crest or nonvascularized fibula graft can be used. For larger defects, a vascularized fibula transfer is preferred. Another alternative for larger defects is conversion to a one-bone forearm. The advantage of the free fibula transfer reconstruction is the potential preservation of forearm rotation. For giant cell tumors of the bones of the hand, optimal results have been achieved after wide excision with bone reconstruction or ray amputation.

Malignant Tumors of Soft Tissue

Despite differences in cell origin, soft tissue sarcomas can be considered a related group of lesions by virtue of their similar clinical behavior. Features such as the mitotic index, extent of tumor necrosis, and cellular anaplasia are predictive of the clinical behavior of these tumors. The more common soft tissue sarcomas seen in the hand are *epithelioid sarcoma*, *synovial cell sarcoma*, and *malignant fibrous histiocytoma*, with epithelioid sarcoma considered the most common. Patients with soft tissue sarcomas may have either a painless or a painful mass. Rapid growth is characteristic, and soft tissue sarcomas can become substantial in size; however, epithelioid sarcoma may occur as a relatively small nodule. It may also ulcerate and thereby lead to a misdiagnosis of infection. MRI is extremely helpful in the evaluation of soft tissue sarcomas. The most common metastatic location of soft tissue sarcomas is the lung.

The foundation of treatment of soft tissue sarcomas is wide surgical resection. Adjuvant radiotherapy is considered highly effective in the treatment of soft tissue sarcomas because the local recurrence rate is reduced by approximately 50%. In contrast, chemotherapy is currently of uncertain benefit in the treatment of most soft tissue sarcomas. The overall 5-year survival rate approaches 50% with aggressive management.

Malignant Tumors of Bone

Chondrosarcoma is a slow-growing tumor of cartilage and is regarded as the most common malignant tumor of the hand and wrist. Patients often have a slowly enlarging, long-standing painful mass. Chondrosarcoma most commonly arises in the metacarpals and the proximal phalanges. On radiographic examination, cortical erosion with extension of tumor into soft tissue is usually seen. The hallmark of chondrosarcoma is punctate calcifications within the lytic lesion of bone.

Chondrosarcoma does not respond to chemotherapy or radiotherapy, and therefore surgery is the only treatment. Wide local excision is the treatment of choice, but ray amputation is preferred for lesions of the metacarpals and proximal phalanges (Fig. 4). One study of chondrosarcomas of the hand treated in this fashion reported only a 11% recurrence rate and a 0% metastasis rate.

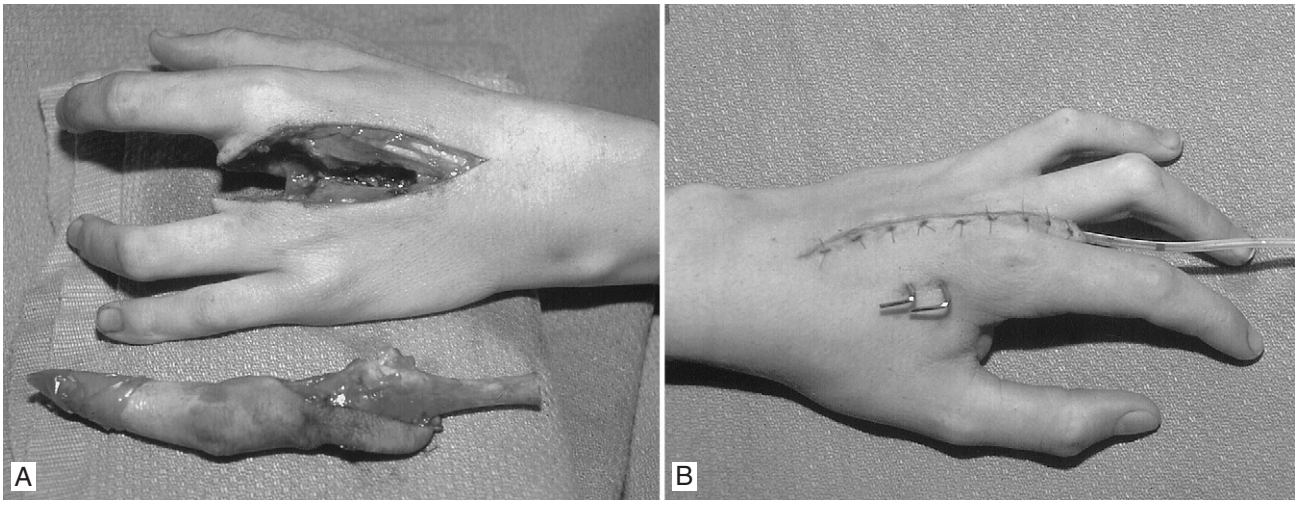

Figure 4 • Ray resection of the third digit. **A,** After resection. **B,** After closure and percutaneous pinning.

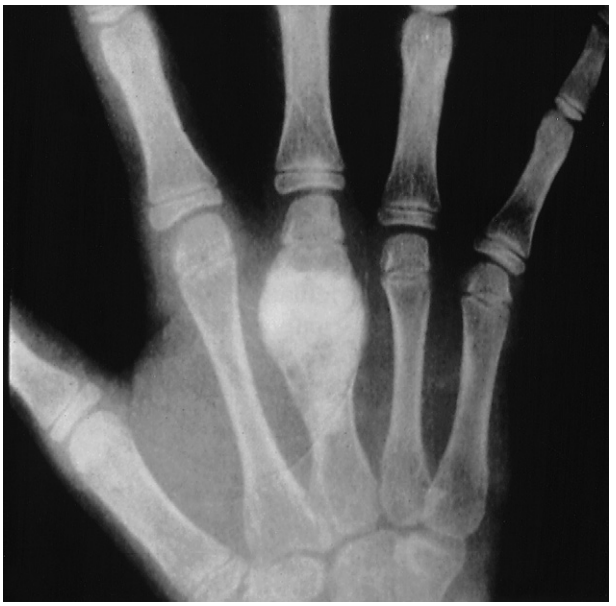

Figure 5 • Osteosarcoma of the third metacarpal in a child. Note the dense, sclerotic, osteoblastic nature of the neoplasm.

Osteosarcoma is the most common primary malignant tumor of bone. However, it is rarely seen in the hand. When present, it usually occurs in older adults and typically affects the metacarpals and proximal phalanges. Patients generally have a rapidly enlarging, firm, fixed, and painful mass. Radiographically, the lesions may be lytic, or they have dense new bone formation (Fig. 5); extensive cortical destruction is common. Treatment of hand and wrist osteosarcoma includes wide excision or amputation along with adjuvant chemotherapy. Delays in initiating treatment have been shown to decrease survival.

Metastatic involvement of the bones of the hand is exceedingly rare, but metastatic spread to the bones of the hand may be the initial finding of previously undiagnosed cancer. Patients with metastatic disease in the hand may or may not complain of pain, and pathologic fracture may be the earliest manifestation. The most common primary sites of metastatic disease in the hand are the lung, head/neck, and kidney. The bladder, breast, colon, esophagus, prostate, ovary, and thyroid have also been reported as origins for cancer metastases to the hand. The metastatic lesions may be lytic or osteoproliferative on radiographic study. The metacarpals and distal phalanges are the most common locations. The majority of patients with metastatic tumors of the bones of the hand have a life expectancy of less than 6 months. Therefore, treatment of these patients centers on pain relief and preservation of hand and wrist function. For lesions of the distal phalanx, transverse amputation is appropriate, whereas more proximal lesions are treated by ray amputation. If the metastatic tumor involves more than 50% of the cortex of the bone, impending pathologic fractures are a concern, and prophylactic treatment is effective. Patients with less than 3 months' life expectancy can be treated by marginal resection of the tumor supplemented by internal stabilization. Radiation therapy can be considered for lesions in rapidly deteriorating patients.

Principles of Limb Salvage in the Hand and Wrist

Because of the continuity of tenosynovium from the hand to the elbow, true radical excision of malignant tumors cannot be accomplished short of an above-elbow amputation. Radical or even wide tumor resections around the hand and wrist can be functionally impractical. What constitutes radical and what constitutes a wide resection in the hand and wrist region remain contentious. Some have suggested that 3- to 5-cm margins around the tumor are appropriate if the lesions are sensitive to adjunctive therapy. Margins such as these can afford a reasonable attempt at salvage surgery.

For malignant lesions of the distal phalanx, a transverse amputation at the level of the middle phalanx, with sparing of the flexor digitorum superficialis insertion if possible, can be considered. For malignant tumors at the level of the middle or proximal phalanx, ray amputation is appropriate in most circumstances. For malignant bone tumors of the fifth metacarpal or soft tissue sarcomas of the hypothenar eminence, ray amputation may be appropriate if an adequate radial margin can be obtained. A double ray amputation, including the fourth and fifth ray, may be necessary.

The sensitivity of the tumor to adjunctive therapy is an important factor in determining the surgical margin. Similar principles can be applied to malignancies of the second, third, and fourth metacarpals or the intervening web spaces. In such situations, maintenance of the thumb ray permits reasonable fine motor skills, even after a double ray amputation. Ray amputations of the fourth ray, the fifth ray, or both the fourth and fifth rays substantially diminish grip strength. For malignancies of the thenar eminence, the first web space, or the first metacarpal, first and second ray amputations may be necessary to obtain adequate margins. Double ray amputation of the first and second rays has profound effects on fine motor skills, but reasonable grip strength is maintained. Thumb reconstruction procedures such as pollicization should be considered after first ray amputations. For first and fifth ray amputation, coverage may be an issue. Although local flaps are an option, free muscle transfer is often a superior alternative, especially if postoperative radiation therapy is necessary.

In certain advanced cases of bone or soft tissue sarcoma, hand salvage surgery may not be possible, and below-elbow or even above-elbow amputations are necessary. An effort should be made to preserve

as much length as possible with a below-elbow amputation. Preservation of forearm length can enhance below-elbow prosthetic fitting. Every attempt should be made to preserve the elbow in upper extremity amputations as patients with above-elbow amputations do not often tolerate prosthesis.

Postoperative Care

Postoperative care of the patient is individualized and depends on the nature of the tumor, the extent of resection, and the complexity of the reconstruction. Tumor defects managed by free muscle transfer are treated according to the standard postoperative free flap protocol. Drains are left in place 24 to 48 hours, and dressings are not changed for 3 to 5 days if a split-thickness skin graft was used. If a bone graft or free osseous transfer was used in reconstruction of bone defects about the wrist, strict postoperative immobilization is necessary until radiographic signs of healing are present. Digital motion and shoulder motion are encouraged during periods of wrist immobilization. After a 3- to 5-day period of immobilization, active and passive range of motion can be instituted following ray amputations. For small benign soft tissue lesions, complete return to all activities can generally be anticipated shortly after suture removal. For benign tumors of bone treated with bone grafting, a 4- to 6-week period of immobilization is necessary before the initiation of digital and wrist mobilization.

The most important aspect of postoperative management of malignant bone or soft tissue sarcoma is follow-up evaluation, especially by a musculoskeletal oncologist. For soft tissue sarcomas, follow-up should include MRI and a CT scan of the chest and abdomen. For sarcomas of bone, a plain bone radiograph, CT scan of bone, bone scan, and CT scans of the chest and abdomen are necessary.

Pearls and Pitfalls

- The greatest pitfall in musculoskeletal tumor oncology is misdiagnosis.
- Aggressive sarcomas in the hand and wrist region are often overlooked because of the rarity of diagnosis.
- Masses in the hand, although commonly benign, must be taken seriously.
- Especially suspicious are lesions larger than 5 cm, lesions displaying rapid growth, and lesions that are painful.
- MRI is a valuable diagnostic adjunct for soft tissue lesions. The workup for suspected malignant tumors should always include a bone scan, chest and abdominal CT scans, and a complete laboratory panel.
- Most lesions metastasizing to the hand arise from previously undiagnosed cancer.

SUGGESTED READINGS

Athanasian EA: Bone and soft tissue tumors. In Green D (ed): Operative Hand Surgery, vol 2, 4th ed. New York, Churchill Livingstone, 1998, pp 2223-2253.

Athanasian EA, Wold LE, Amadio PC: Giant cell tumors of the bones of the hand. J Hand Surg [Am] 22:91-98, 1997.

Bauer RD, Lewis MM, Posner MA: Treatment of enchondromas of the hand with allograft bone. J Hand Surg [Am] 13:908-916, 1988.

Glowacki KA, Weiss AP: Giant cell tumors of tendon sheath. Hand Clin 11:245-253, 1995.

Kehoe NJ, Reid RP, Semple JC: Solitary benign peripheral-nerve tumours. Review of 32 years' experience. J Bone Joint Surg Br 77:497-500, 1995.

Marcove RC, Sheth DS, Takemoto S, Healey JH: The treatment of aneurysmal bone cyst. Clin Orthop 311:157-163, 1995.

McGrath BE: Management principles of aggressive tumors of the hand and forearm. In Light TR (ed): Hand Surgery Update 2, 2nd ed. Rosemont, IL, American Academy of Orthopaedic Surgeons, 1999, p 369.

Musculoskeletal neoplasms. In Kasser J (ed): Orthopaedic Knowledge, vol 5. Rosemont, IL, American Academy of Orthopaedic Surgeons, 1996, pp 133-148.

Musculoskeletal oncology. In Beaty JH (ed): Orthopaedic Knowledge Update 6: Home Study Syllabus. Rosemont, IL, American Academy of Orthopaedic Surgeons, 1999, pp 167-189.

Okada K, Wold LE, Beabout JW, et al: Osteosarcoma of the hand. A clinicopathologic study of 12 cases. Cancer 72:719-725, 1993.

Palmieri TJ: Chondrosarcoma of the hand. J Hand Surg [Am] 9:332-338, 1984.

Thornburg LE: Ganglions of the hand and wrist. J Am Acad Orthop Surg 7:231-238, 1999.

The Rheumatoid Hand

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Rheumatoid arthritis is an inflammatory arthropathy that affects bone, cartilage, ligaments, tendons, subcutaneous tissue, and skin. Rheumatoid arthritis frequently affects the upper limb, and specific problems involving the upper extremity are best managed in conjunction with a rheumatologist, hand therapist, and hand surgeon. The aims of treatment of the rheumatoid hand are guided by the patient's needs and activities, as they can vary greatly in patients with similar clinical disease and deformity.

Etiopathogenesis

The specific cause of rheumatoid arthritis remains unknown. HLA-DRB1 genes on chromosome 6 and the major histocompatibility complex class II alleles, which are associated with rheumatoid arthritis, appear to affect susceptibility to the disease.

Hand-related disease in rheumatoid arthritis begins with soft tissue synovitis. Each of the succeeding phases in the disease process can occur as a discrete event, but more often they tend to occur together at varying rates of progression. These events include synovial thickening (often with pannus formation), degradation of articular cartilage and the underlying bone, ligamentous attenuation and laxity, and eventually, joint deformity and subsequent contracture. In the hand, evidence of disease often extends beyond the joints: rheumatoid nodules, stenosing tenosynovitis, tendon rupture, and compression neuropathies. Furthermore, the medications used to treat rheumatoid arthritis can have deleterious effects on the skin and soft tissues of the hands.

The synovium in rheumatoid arthritis is inflamed, with an increased number of lymphocytes in the perivascular areas surrounded by macrophages and plasma cells. There is a proliferation of synovial lining cells in association with marked angiogenesis. The substances released by the synovium along with physical expansion of the surrounding structures lead to the other pathologic aspects in the disease. Pannus refers to a thickened area of synovium that behaves in a locally aggressive manner and causes destruction of ligaments, articular surfaces, tendons, and bones. The articular surface is damaged not only by the direct effect of the hypertrophic synovium but also by prostaglandins and several cytokines released by the inflammatory cells. The cytokines tumor necrosis factor-α (TNF-α) and interleukin-1 are present in high quantities in rheumatoid synovial fluid and lead to a multitude of detrimental effects on the articular surfaces and underlying bones. The cytokines also act as stimulants for the production of matrix metalloproteinases, which have a direct effect on matrix-rich tissues such as bone and cartilage.

The hyperplastic synovium and inflammatory cells tend to expand and degrade the ligaments surrounding affected joints. This impairs their ability to maintain integrity of the joint and leads to additional destruction of the joint surface, pain, and eventual loss of the normal, congruous gliding of that joint, especially during loading. The force of the musculotendinous units across the destabilized joint subsequently leads to deformity of the joint and resultant tightening of the previously lax ligaments and tendons. Contracture of the joint may evolve and ultimately force the articular surfaces into a subluxed or dislocated position.

Synovial and joint pathology also cause other effects on local tissues. The tendons can degrade to the extent of rupture, not only because of the effect of the inflamed hyperplastic synovium but also because of the abnormal forces and line of pull caused by joint subluxation. In addition, the excess synovium can exert pressure on various structures that pass through narrow regions and cause compression neuropathies and stenosing tenosynovitis.

TABLE 1 Pathologic Anatomy

ANATOMIC SITE	ANOMALY
Wrist	Dorsal subluxation of the ulnar head
	Radial subluxation, palmar flexion, ulnar translocation, supination, and palmar subluxation of the carpus on the distal end of the radius
Extensor tendons	Tendon rupture
	de Quervain's stenosing tenosynovitis
Flexor tendons	Stenosing tenosynovitis
	Tendon rupture, especially the flexor pollicis longus and flexor digitorum profundus tendons to the index finger
Metacarpophalangeal joints of the fingers	Ulnar deviation
	Volar subluxation
	Intrinsic shortening
Fingers	Swan neck deformity
	Boutonnière deformity
Thumb	Boutonnière deformity
	Swan neck deformity
	Radial deviation at the first metacarpophalangeal joint

Pathologic Anatomy

A rheumatoid hand can have varying degrees of deformity. Early disease may simply show signs of synovial thickening and pannus formation. In later stages, patients may have severely deformed digits, hands, and wrists with debilitating contracture, pain, and joint disruption. The anatomic changes often seen in a rheumatoid hand are summarized in Table 1 and Figure 1.

Diagnostic Studies

The diagnosis of rheumatoid arthritis rests on clinical assessment with confirmation by appropriate laboratory studies. Criteria for the disease were formally established by the American College of Rheumatology in 1988 (Table 2). The clinical activity of the disease has an impact on the timing of surgical intervention. Clinical assessment of the hand, including complete functional evaluation, is critical for planning both physical therapy and surgical treatment. This assessment is often made in conjunction with a hand therapist. A directed physical examination of a rheumatoid hand is outlined in

TABLE 2 Revised Criteria for Classification of Rheumatoid Arthritis (Traditional Format)*

CRITERIA	DEFINITION
1. Morning stiffness	Morning stiffness in and around the joints that lasts at least 1 hour before maximal improvement
2. Arthritis of 3 or more joint areas	At least 3 joint areas simultaneously have had soft tissue swelling or fluid (not bony overgrowth alone) observed by a physician. The 14 possible areas are the right or left PIP, MCP, wrist, elbow, knee, ankle, and MTP joints
3. Arthritis of the hand joints	At least 1 area swollen (as defined above) in a wrist, MCP, or PIP joint
4. Systemic arthritis	Simultaneous involvement of the same joint areas (as defined in 2) on both sides of the body (bilateral involvement of the PIP, MCP, or MTP joints is acceptable without absolute symmetry)
5. Rheumatoid nodules	Subcutaneous nodules over bony prominences or extensor surfaces or in juxtaarticular regions that are observed by a physician
6. Serum rheumatoid factor	Demonstration of abnormal amounts of serum rheumatoid factor by any method for which the result has been positive in < 5% of normal control subjects
7. Radiographic changes	Radiographic changes typical of rheumatoid arthritis on posteroanterior hand and wrist radiographs; must include erosions or unequivocal bony decalcification localized in or most marked adjacent to the involved joints (osteoarthritis changes alone do not qualify)

*For classification purposes, patients are said to have rheumatoid arthritis if they satisfy at least four of these seven criteria. Criteria 1 through 4 must have been present for at least 6 weeks. Patients with two clinical diagnoses are not excluded. Designation as classic, definite, or probable rheumatoid arthritis is *not* to be made.

MCP, metacarpophalangeal; MTP, metatarsophalangeal; PIP, proximal interphalangeal.

From Arnett FC, Edworthy SM, Bloch DA, et al: The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. Arthritis Rheum 31:315-324, 1988.

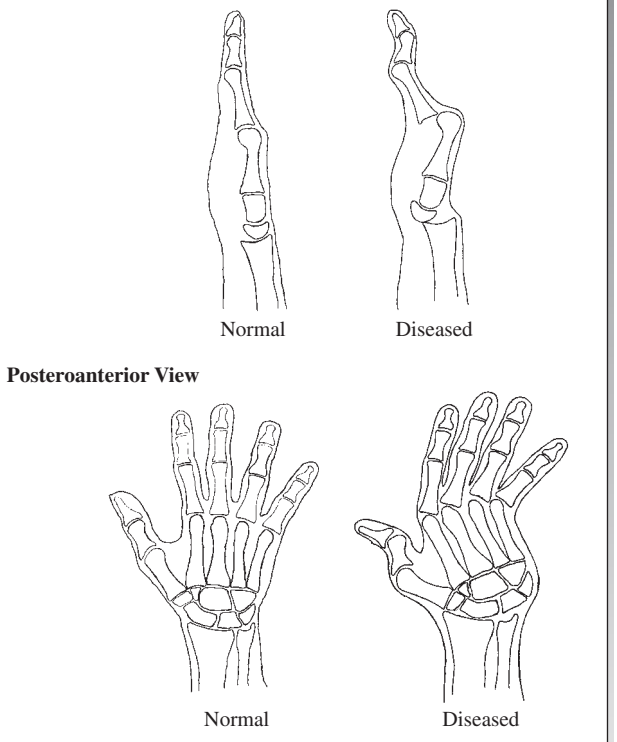

Figure 1 • The rheumatoid hand and wrist.

Table 3. Plain radiographs of the digits, hand, and wrist are valuable for evaluating the pathology and planning surgical interventions. Other useful diagnostic studies include computed tomography, electromyography, ultrasonography, and magnetic resonance imaging. Needle aspiration of joints, ganglions, and nodules for cytologic and bacteriologic study is occasionally indicated.

Reconstructive Goals

Reconstructive surgery of a rheumatoid hand should achieve four goals. Different surgeons, however, disagree with their order and priority. Although more operations are performed to relieve pain, the first priority should be prevention of disease progression, a goal more commonly achieved with medical therapy than with surgery. When medical therapies fail to halt progression of the disease, it is appropriate to consider surgical procedures. Thus, the reconstructive goals, in order of importance, are as follows:

1. Prevention of local disease progression
2. Relief of pain
3. Improvement of function
4. Improvement of appearance

Treatment

Medical Therapy

The use of disease-modifying antirheumatic drugs (DMARDs) has made a major impact in the management of a large proportion of patients, especially those with more advanced disease. However, because articular destruction is now known to occur

TABLE 3 Features on Physical Examination

ANATOMIC SITE	FEATURE OR SIGN PRESENT
Wrist	"Piano key sign"—depress the prominent ulnar head and elicit significant movement and pain along with springing of the ulnar head back into position when the pressure is released
	Decreased range of motion, often with pain and crepitus
	Radiocarpal instability
Extensor tendons	Loss of extension of all fingers and thumb
	Synovial thickening over the dorsum of the hand, wrist, and distal end of the forearm
Flexor tendons	Loss of the deep long flexors to the thumb or fingers
	Synovial thickening throughout the length of the flexor tendon sheaths
	Triggering digits
Metacarpophalangeal joints of the fingers	Synovial thickening
	Abnormal position—ulnar deviation and palmar subluxation
	Intrinsic testing shows tightness
Fingers	Synovial thickening
	Abnormal position—boutonnière and swan neck deformities
	Loss of range of movement, joint instability, crepitus
Thumb	Synovial thickening
	Abnormal position—boutonnière and swan neck deformities and adducted first web space
	Loss of range of movement, joint instability, crepitus
Others	Altered sensation in the distribution of the median, ulnar, or radial nerves
	Rheumatoid nodules—subcutaneous border of the ulna and fingers

early in the disease process, the trend at present is early combination DMARD therapy inasmuch as recent studies have shown an improved prognosis with such treatment. Nonsteroidal antiinflammatory drugs and corticosteroids are also commonly used along with intraarticular corticosteroid injections. Methotrexate is one of the most commonly used DMARDS, although the use of leflunomide is increasing. More recent research has been directed at cytokine (TNF-α) inhibitors and angiogenesis inhibitors.

Physical Therapy

The occupational therapist and physical therapist may see rheumatoid sufferers at various times during the course of their disease. They may be involved early to provide splinting and gentle exercise programs to help with pain relief. Assistance may also be provided for the patient's activities of daily living. Several aids have been developed for arthritis sufferers, and rheumatoid patients may benefit from these. Therapists provide services for patients who do not require surgery and play a crucial role in every rheumatoid patient who undergoes any form of hand surgery. The successful outcome of surgery ultimately relies on excellent postoperative therapy.

Surgical Treatment

In a patient with active disease, surgical intervention is of little value. Synovectomy and nerve decompression may be necessary, but in most cases, reconstructive procedures are reserved for patients with medically controlled or "burnt out" disease. The use of corticosteroids and cytotoxic agents is thought to increase surgical complications, but it is our practice not to alter medications in patients undergoing surgery. One must be alert for a possible increased need for corticosteroids in the immediate perioperative period because patients may have a degree of adrenal suppression and cannot increase their secretion of cortisol at times of physiologic stress.

This chapter does not provide a comprehensive summary of the surgical techniques used in a rheumatoid hand but emphasizes only the most useful techniques. A well-established principle in a patient with disease at several joint levels is to perform surgery in a proximal-to-distal direction because proximal deformities influence distal joint mechanics.

EXCISION OF THE ULNAR HEAD. Excision of the ulnar head is a common procedure in a rheumatoid hand (Fig. 2). The main indication is ulnar-sided wrist pain, particularly with forearm rotation. Prominence of the ulnar head dorsally (*caput ulnae*) can lead to attritional rupture of the extensor tendons to the ulnar digits (the so-called Vaughn-Jackson lesion); therefore, this procedure is often combined with tendon transfers. The surgical approach is through a dorsal midline incision, with care taken to protect the dorsal branch of the ulnar nerve. The retinaculum of the sixth extensor compartment is opened, and synovectomy of the exten-

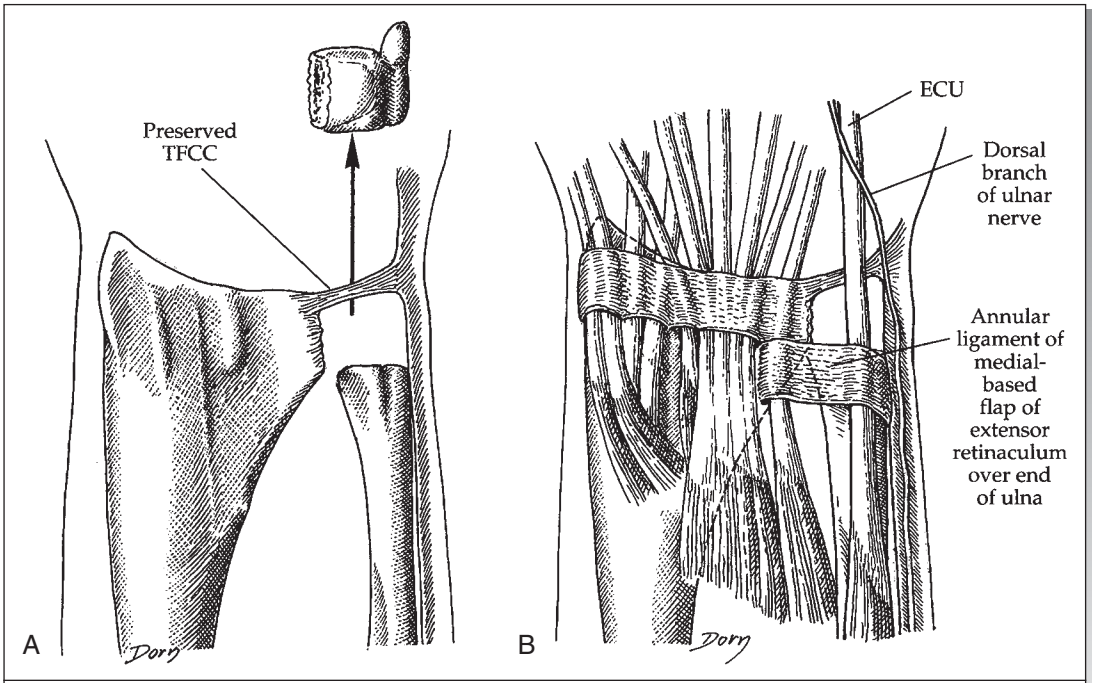

Figure 2 • Ulnar head excision and stabilization. ECU, extensor carpi ulnaris; TFCC, triangular fibrocartilage complex. (From Beasley RW: Beasley's Surgery of the Hand. New York, Thieme, 2003.)

sor carpi ulnaris tendon is performed. An incision is made in the floor of the sixth compartment to allow access to the ulnar head and the space between the ulna, carpus, and radius. Approximately 2 cm of ulnar head is removed with a sagittal saw after subperiosteal dissection. Sufficient head is excised, while remaining just proximal to the sigmoid notch, to prevent contact with the distal end of the radius in most of the arc of forearm rotation. Residual contact between the two bones at the extremes of rotation is acceptable. The distal ulnar stump is smoothed and contoured. In the gap that is present after excision of the ulnar head, the triangular fibrocartilage complex can be tightened and sutured to the dorso-ulnar region of the distal end of the radius to correct carpal supination on the radius. The preserved ligamentous tissue around the cut end of the ulna is plicated to prevent distal ulnar instability. The extensor carpi ulnaris is often subluxed volarly, and it must be repositioned by placing a sling of the proximal extensor retinaculum around the tendon and suturing it back on itself. The two critical parts of the operation are stabilization of the distal end of the ulna and securing of the extensor carpi ulnaris tendon in a dorsal position over the ulna (see Fig. 2). After careful wound closure over a suction drain, a dressing and above-elbow plaster are applied with the forearm placed in supination. The drain is removed the following day, and the splint is maintained for a period of 4 weeks. Finger and thumb movement is encouraged throughout the period of recovery.

Occasionally, fusion of the distal radioulnar joint is performed with formation of a pseudarthrosis more proximally in the ulna. This technique, known as the Sauve-Kapandji procedure, is sometimes used in rheumatoid patients with ulnocarpal ligamentous laxity to prevent further ulnar carpal translation and supination of the carpus.

TOTAL WRIST ARTHRODESIS USING A CONTOURED PLATE. When all other surgical procedures have failed or are not appropriate, severe deformity and pain can be reliably treated by complete fusion of the wrist, particularly in high-demand patients. Several authors have suggested that in significantly osteopenic patients, fusion can be accomplished with one or two Steinmann pins. A drawback to this procedure is that the wrist can be fused only in neutral position. Alternatively, contoured prebent titanium plates allow various wrist positions. The final position of the wrist is determined by the plate selected, the most common being neutral or slight flexion and 10 degrees of medial deviation.

Resection of the ulnar head is usually performed through a longitudinal incision as previously described, particularly with distal radioulnar joint involvement. The third dorsal compartment is opened, and the incision should extend onto the third metacarpal. The dorsal wrist capsule is raised in both directions. The distal end of the radius is exposed subperiosteally along with the proximal half of the third metacarpal. The dorsal cortices of the distal end of the radius, carpal bones, and base of the third metacarpal are removed with an osteotome. What is left of the cortical surfaces should be removed with a small powered bur, with special attention paid to the radioscapholunate, scapholunate, scaphocapitate, lunocapitate, and third carpometacarpal joint surfaces while maintaining their volar cortical relationships. Cancellous iliac bone graft is morselized and packed into the decorticated joints. The plate is secured with appropriately sized screws starting from the most distal position and proceeding proximally. The plate should be aligned so that the capitate is fixed to it. Some compression can be applied during placement of the first screw into the radius if care is taken to avoid a rotational deformity of the third metacarpal. The plate's position and screw length should be checked by fluoroscopy. Additional cancellous bone graft is packed into the remaining spaces. The dorsal wrist capsule is closed along with the third dorsal extensor compartment. The extensor pollicis longus tendon is transposed so that it sits dorsal to the extensor retinaculum. A drain is placed and the wound closed. A soft dressing and a plaster splint are applied. The plaster splint can be changed to a functional splint in 2 weeks when the swelling has subsided.

WRIST IMPLANT REPLACEMENT ARTHROPLASTY. Several authors have revisited wrist replacement arthroplasty for rheumatoid sufferers because many patients are pleased with the improvement in pain and the benefit of maintaining some wrist motion. Although a previous wrist implant arthroplasty makes total wrist fusion slightly more technically difficult, the possible benefit of painless, stable motion is probably worthwhile in select patients with rheumatoid arthritis, particularly low-demand patients who would otherwise require bilateral arthrodeses.

The Menon universal wrist replacement implant (Fig. 3) is one of the most reliable implants. Sizing is determined with preoperative radiographs. In general, a smaller implant should be used so that if the sizer does not seem large enough, a larger implant can be placed. A midline dorsal incision is used to approach the joint, with careful preservation of the superficial radial nerve and the dorsal branch of the ulnar nerve. An extensor retinacular flap is elevated, starting from the sixth dorsal compartment and continuing until the septum between the first and second dorsal compartments is reached. The extensor tendons are retracted and synovectomy performed if needed. The dorsal wrist capsule, including the periosteum of the distal end of the radius and the joint capsule of the distal radioulnar joint, is raised in a proximal-to-distal direction. The ulnar head is typically resected. A small hole is made

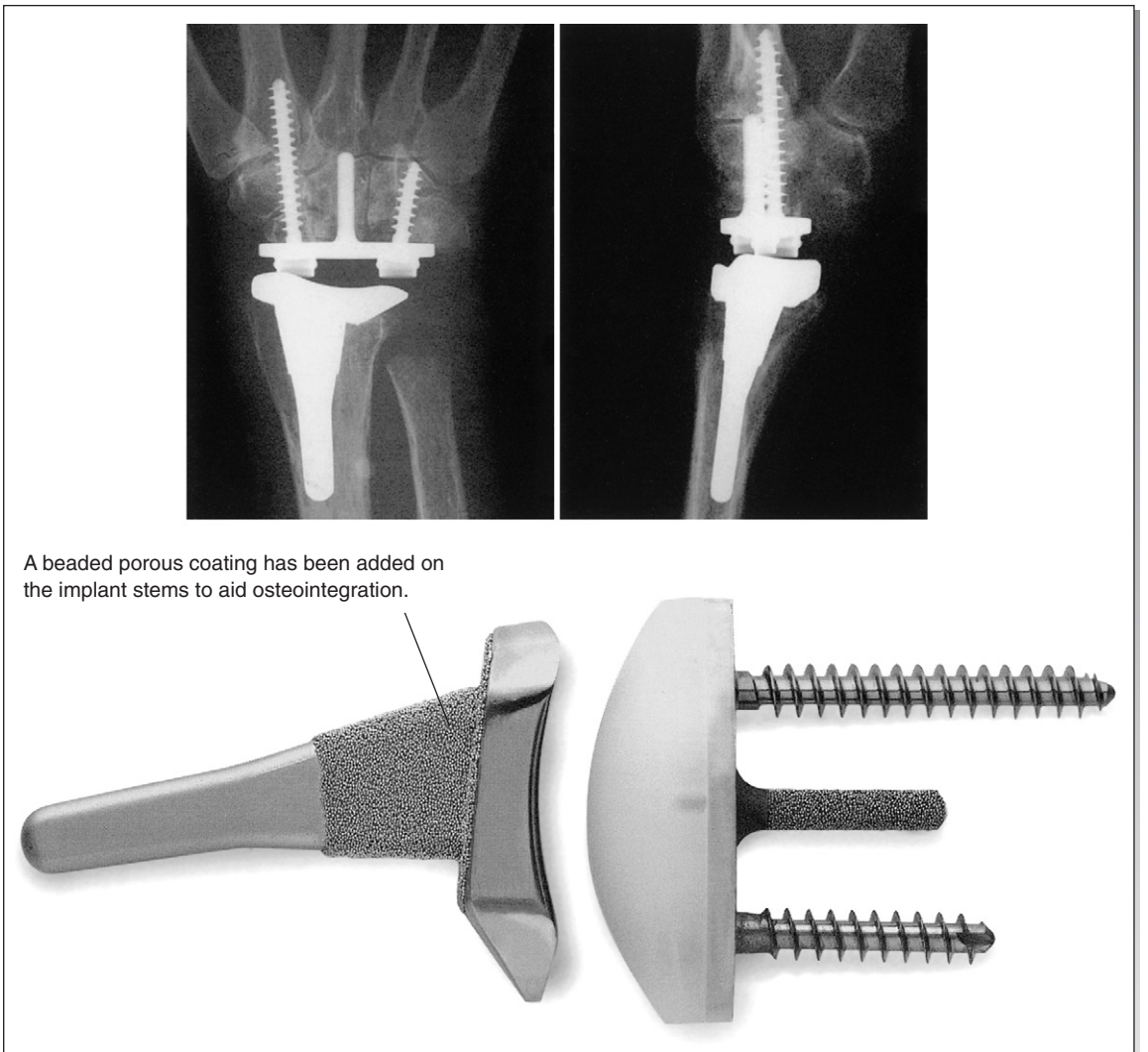

Figure 3 • Universal 2 Total Wrist Implant System. (Courtesy of Kinetikos Medical Inc., Carlsbad, CA.)

in the end of the radius, 5 mm from the dorsal lip, near Lister's tubercle. The radial alignment guide rod is passed down the radius and its position verified with fluoroscopy. The radial guide bar is passed over the rod until it abuts the radius. The radial cutting guide block is placed into the guide bar. The dorsal lip of the radius may be smoothed to allow the cutting guide block to sit well. Two 1.1-mm Kirschner wires are placed in the central row of holes in the guide block. The guide rod and guide bar can be removed so that radial osteotomy can be performed. The wires and cutting guide block are removed, and the alignment rod is placed back into the radius. The broach is slid over the alignment rod and used to make a hole for the stem of the radial implant. The trial component is inserted to check its fit.

For the carpal osteotomy, the scaphoid and triquetrum must be wired distally to the capitate. The lunate is removed with rongeurs. The special drill guide is fitted with the guide plate and placed up against the base of the capitate. The rest of the drill guide is placed against the skin over the third metacarpal, and the guide plate is positioned over the dorsal surface of the capitate. The hole is drilled and countersunk. The carpal guide bar is placed in the hole and the carpal cutting guide slotted into the bar. The cutting guide is secured to the carpus with K-wires so that the carpal guide bar can be removed. The osteotomy is performed through the base of the hamate, as well as through the scaphoid, capitate, and triquetrum. The trial carpal component is inserted, and the holes on the radial and ulnar sides are drilled by using the appropriate guide for each of the holes. The radial hole should cross into the second metacarpal shaft, but the ulnar hole should cross only into the hamate and not into the fourth metacarpal.

The wound is irrigated and the definitive implants inserted with cement in the stem holes.

The carpal component is placed first along with the ulnar and radial screws. The radial component is inserted. The polyethylene trial component is placed next because its size may need to be altered if the implant is unstable or motion is restricted. The definitive polyethylene insert is placed. The remaining K-wires are removed and the intercarpal joints fused. The dorsal capsule is repaired to cover the implant, but a portion of the extensor retinaculum may also be needed to cover the proximal end of the implant. A wrist splint is used for 10 days, after which gentle wrist mobilization is commenced.

METACARPOPHALANGEAL JOINT REPLACEMENT ARTHROPLASTY. The main indication for this procedure is to treat painful joints that have undergone degeneration with ulnar drift of the digits and volar subluxation of the joints. The procedure is performed through a transverse curvilinear incision and the joint opened longitudinally through the ulnar dorsal hood, some distance from the extensor tendon. The collateral ligaments and the bone proximal to the articular surface of the metacarpal head are exposed, and a transverse osteotomy is performed with an oscillating saw as close as possible to the head. The head, ulnar collateral ligament, and synovium are removed to reveal what is left of the articular surface of the base of the proximal phalanx. Attention must be paid to preserving the radial collateral ligament. If the intrinsic muscles are tight, they may be divided. The base of the proximal phalanx may occasionally need to be cut if the base is eroded in an asymmetric fashion. The articular surface remaining on the base of the proximal phalanx should be removed with bone rongeurs. On the dorsoradial aspect of the metacarpal, a small hole should be made for reattachment of the radial collateral ligament. Implant sizers should be used to determine the correct implant for each joint. The implant should be sufficiently large for the hinge portion to extend beyond the bony extent of the metacarpal osteotomy. If the volar plate is tight or the implant seems too tight, the volar plate may be divided transversely. Before insertion of the definitive implant, 3–0 nonabsorbable braided suture is used to secure the remnant of the radial collateral ligament, with one limb of the suture being passed through the cortical hole previously made in the metacarpal. The sizer is replaced and the suture temporarily tensioned to ensure that the ligament is snug and neither too tight nor too loose. The final implant is placed first into the metacarpal end and then into the proximal phalanx end. The collateral ligament suture is tied. If the tendon is not centralized over the implant, the radial hood should be plicated. The skin is closed over a soft rubber drain, and a soft dressing and plaster splint are applied. After 1 week, an outrigger thermoplastic splint is fashioned to allow for controlled finger motion. Motion is slowly increased over time, and after 10 weeks splinting can be discontinued.

RECONSTRUCTION OF THE OBLIQUE RETINACULAR LIGAMENT FOR SWAN NECK DEFORMITY. This procedure is indicated for milder forms of swan neck deformity (type I) before established joint changes have occurred. Each affected finger is opened through a midline dorsal incision centered over the proximal interphalangeal joint. The ulnar lateral band of the extensor system is divided proximally and mobilized distally while maintaining its attachment to the distal phalanx. Through a Bruner incision, the finger is opened volarly centered over the proximal interphalangeal joint. The flexor tendon sheath is exposed while preserving the A2 pulley. The slip of extensor tendon is tunneled distally around the ulnar side of the digit to allow it to pass volar to Cleland's ligaments and thus volar to the axis of flexion, and it is sutured to the A2 pulley on the ulnar side of the flexor tendon sheath. The reconstruction is tensioned so that the distal joint is extended to more than its original position and the proximal joint is flexed to 15 degrees, thereby preventing proximal interphalangeal joint hyperextension, in addition to augmenting distal interphalangeal joint extension.

TENDON RECONSTRUCTION FOR BOUTONNIÈRE DEFORMITY. Established boutonnière deformity can be treated by tendon reconstruction if the interphalangeal joint surfaces are preserved and the deformity can be corrected passively (Fig. 4). Passive motion may need to be achieved with serial splinting and occasionally even by surgical joint release. The technique combines both distal tenotomy and centralization of the lateral bands. A longitudinal incision centered over the proximal interphalangeal joint in the dorsal midline of the involved digit is used. An extensor tenotomy is performed midway between the distal and proximal interphalangeal joints so that the oblique retinacular ligament can still provide some degree of distal phalanx extension through its tenodesis effect. The transverse retinacular ligament is divided at the volar edge of the malpositioned lateral bands, and this release is continued proximally and distally to allow the lateral bands to be sutured together over the proximal interphalangeal joint. A Kirschner wire is placed across the proximal interphalangeal joint in full extension before suturing the lateral bands, and this wire is left in place for 3 to 4 weeks postoperatively (see Fig. 4). Controlled distal joint flexion is encouraged during the early postoperative period.

PROXIMAL INTERPHALANGEAL JOINT ARTHRODESIS. The primary indication for proximal interphalangeal joint arthrodesis is pain and instability, both of which cause significant functional deficits. Through a longitudinal incision over the dorsum of the proximal interphalangeal joint, the joint is opened by dividing the central slip and carefully preserving the lateral bands. The collateral ligaments are divided to allow for adequate visual-

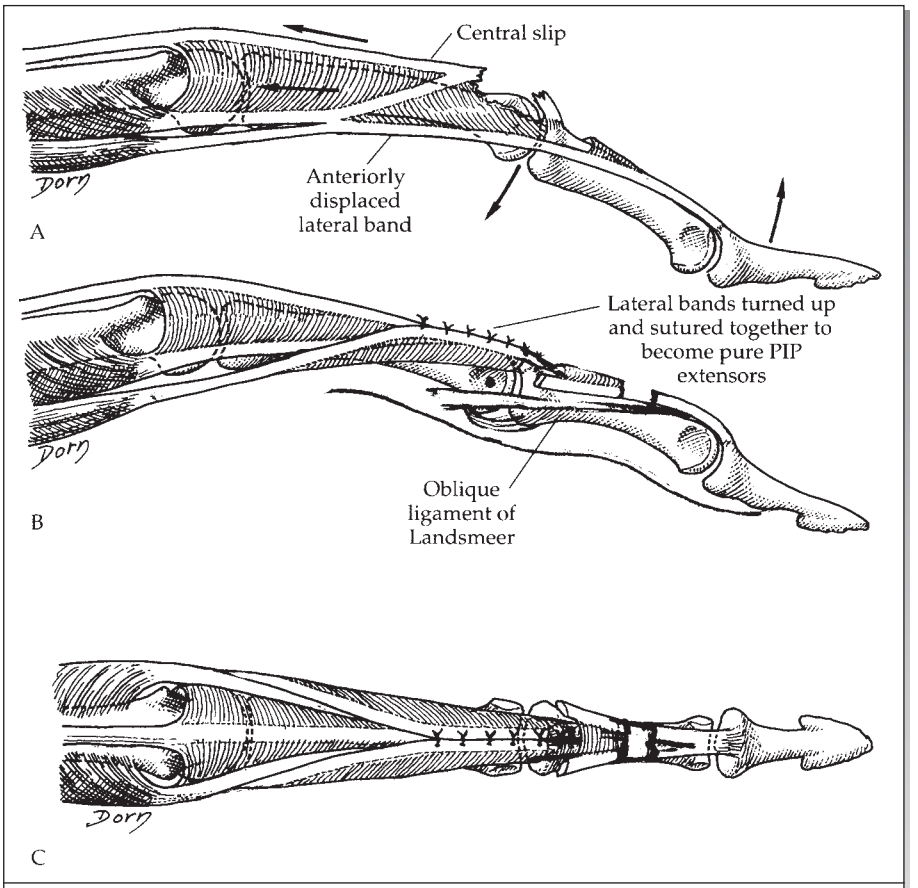

Figure 4 • A, Basic pathology of a boutonnière deformity. **B** and **C,** Reconstruction for an established boutonnière deformity converts all finger interphalangeal (IP) extensor forces solely to the proximal interphalangeal (PIP) joint with release of the distal interphalangeal joint hyperextension by tenotomy of the lateral bands. The anterior margin of the elevated lateral bands at the PIP joint are turned up dorsally and sutured together over the PIP joint. (From Beasley RW: Beasley's Surgery of the Hand. New York, Thieme, 2003.)

ization of the joint surfaces. A synovectomy is performed. The base of the middle phalanx and the head of the proximal phalanx are removed with an oscillating saw. The angle of the osteotomies is calculated so that the resultant angle of the fused joint is approximately 25 degrees for the index finger with a gradual increase in the joint angle so that the little finger is at approximately 45 degrees. One axial and two oblique double-ended K-wires are placed into the middle phalanx and brought out through the skin so that the points are flush with the osteotomy. The surgeon holds the joint in correct position and the assistant advances the wires into the proximal phalanx while maintaining firm compression. The wires are left in place for at least 8 weeks and are removed only after radiologic confirmation of fusion.

INTERPHALANGEAL AND METACARPOPHALANGEAL JOINT FUSION OF THE THUMB. Several techniques have been described to achieve fusion of the joints of the thumb. The cannulated Acutrak compression screw is most useful, however, for the interphalangeal and metacarpophalangeal joints of the thumb. For the interphalangeal joint, a dorsal H-shaped incision is made over the joint. A small oscillating saw is used to remove the articular surfaces, and the appropriate resultant angle of the joint fusion is determined by the angles of the osteotomies. This technique generally produces approximately 10 degrees of flexion. A central Kirschner wire is placed from the site of the osteotomy distally so that the wire exits through hyponychial tissue. The bone ends are apposed and the wire driven into the proximal phalanx under fluoroscopic control. The required length of the cannulated mini Acutrak screw is estimated. A small incision is made around the central wire at the tip of the thumb to allow insertion of the drill and screw. Under fluoroscopic guidance, the cannulated drill is used over the K-wire that has been inserted. An appropriately sized screw is placed while stabilizing the phalanges to prevent rotation.

The metacarpophalangeal joint is opened through a longitudinal incision, and the extensor tendon is

split. The articular surfaces are exposed and the appropriate osteotomies performed to give a resultant angle of approximately 20 degrees for the fusion. As opposed to the interphalangeal joint, the Kirschner wire is placed in an oblique direction across the joint in a proximal-to-distal direction. Fluoroscopy is used to check the position of the wire. The "standard" sized Acutrak screw is used for this joint. A second K-wire may be placed parallel to the original wire to prevent rotation during drilling and placement of the screw. After a small incision is made in the skin, the cannulated drill is placed over the central wire and drilling is performed under fluoroscopic guidance. The appropriately sized cannulated Acutrak screw is placed across the joint until it is in correct position and compression is achieved across the joint fusion site. Both wires are removed and the wounds closed.

Postoperative Care

In the early postoperative phase, strict elevation and well-fitted, comfortable dressings and splints are essential. After a period ranging from a few days to several weeks, the dressings and original splint are removed. Continued splinting is usually required, and the hand therapist is generally skilled in making the required splint from lightweight thermoplastics. A dynamic splint may be of benefit to allow controlled movement, as is the case after metacarpophalangeal joint replacement. Careful wound care is required because the patients are often taking medications that may inhibit healing, although wound problems and dehiscence are surprisingly uncommon.

The hand therapist's role in postoperative care is crucial. After wound healing, the patient must return to at least the preoperative level of functioning and, optimally, improve on it. This goal often involves relearning basic hand maneuvering and thus involves a prolonged process that necessitates persistence by the patient, therapist, and surgeon.

Early postoperative complications include bleeding, infection, and wound breakdown. Infections may be localized to the skin but may also include pin tract infection and implant infection/extrusion. Bone healing may be prolonged because rheumatoid sufferers often have significant osteopenia and osteoporosis. The long-term complications of surgery include recurrence of the original deformity, often as a result of the continuing rheumatoid disease. Silastic implants are particularly prone to failure, fracture, and dislodgement. Arthrodesis usually provides a solution for most complications and, as such, remains the gold standard for treatment.

Pearls and Pitfalls

- Individualized, patient-specific goals of treatment remain central to management of a rheumatoid hand.
- Early medical treatment is crucial, and early surgical intervention should be considered for all patients. Such management has become increasingly important because it is now well established that damage to tissues and articular surfaces occurs early in the disease process, long before deformity develops.
- One must remember to address all deformities of the upper limb by proceeding proximally from the shoulder to the distal interphalangeal joints.
- The aim of restoring function is important, but one must remember that pain is far more disabling than loss of motion. Reduction of pain is something that surgeons can often reliably offer to patients, and such pain reduction facilitates improvement in function.
- When motion-sparing procedures have failed, joint fusion is a reliable option for reducing pain and increasing function.

SUGGESTED READINGS

Beasley RW: Beasley's Surgery of the Hand. New York, Thieme, 2003.

Feldon P, Terrono AL, Nalebuff EA, Millender LH: Rheumatoid arthritis and other connective tissue diseases. In Green DP, Hotchkiss RN, Pederson WC (eds): Green's Operative Hand Surgery, 4th ed. Philadelphia, Churchill Livingstone, 1999, pp 1651-1739.

Lee DM, Weinblatt ME: Rheumatoid arthritis. Lancet 358: 903-911, 2001.

Lister GD: Rheumatoid disease of the wrist and hand. In Jurkiewicz MJ, Krizek TJ, Mathes SJ, Ariyan S (eds): Plastic Surgery. Principles and Practice, vol 1. St. Louis, Mosby, 1990, pp 909-930.

Millender LH, Nalebuff EA: Reconstructive surgery in the rheumatoid hand. Othop Clin North Am 6:709-732, 1975.

Ruby LK, Cassidy C: Rheumatoid arthritis of the hand and wrist. Hand Clin 12(3), 1996, pp 463-628.

Small Joint Contracture

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ALBERT PANNOZZO

Joint stiffness is the most frequent and disabling long-term complication after a hand injury. Much of the morbidity is prevented by adhering to several key principles, including early and appropriate skin coverage, alignment and rigid stabilization of bony injuries, splinting of the hand in an anatomically "safe" position, and early mobilization. Acquired posttraumatic stiffness, which sets in after injury or after inappropriate immobilization, and its surgical management are the focus of this chapter.

Etiopathogenesis

Stiffness is defined as restriction of normal mobility of a given joint. Limitation of flexion or extension may be active, passive, or both. When either flexion or extension is impaired, a contracture exists.

An understanding of the myriad causes of joint stiffness directs proper therapy. Joint stiffness may result from a local cause (e.g., direct injury to a joint) or a systemic condition (e.g., rheumatoid arthritis) or may have an indirect cause (e.g., intrinsic or extrinsic tendon adhesions, tendon imbalance, improper immobilization, posttraumatic edema, reflex sympathetic dystrophy, Dupuytren's contracture, burns, scars).

Limitation of joint motility may be due to articular or extraarticular causes. Articular causes include adhesions, incongruent joint surfaces, and contracture of the capsule or ligaments. Extraarticular causes include adhesions within the gliding planes, blocking of tendons by exostoses, or tendon nodules.

Small joint contractures may be classified as acquired or congenital (Tables 1 and 2). Trauma to the joint or neighboring skin, bone, or tendon is the most common cause of acquired joint stiffness. In general, acquired posttraumatic stiffness usually results in an extension contracture of the metacarpophalangeal (MCP) joint and a flexion contracture of the proximal interphalangeal (PIP) joint for reasons outlined in the next section.

Pathologic Anatomy

Joint stiffness follows predictable patterns that are best appreciated by an understanding of normal anatomy. Both the interphalangeal (IP) and MCP joints have collateral ligaments, palmar fibrocartilage, and a loose dorsal capsule guarded by an extensor expansion. However, the MCP and IP joints have anatomic differences of clinical significance.

The MCP joints are capable of side and rotatory movements when the joints are in extension and are therefore classified as *condyloid* joints. When the joints are flexed, neither abduction nor adduction is possible because the metacarpal head is broader volarly and in the sagittal plane the head has the shape of a cam, with the distance from the axis of motion to its volar surface greater than the distance to its dorsal surface. The collateral ligaments are eccentrically attached to the sides of the head of the metacarpals and originate dorsal to the flexion and extension axes of joint motion. As a result, the collateral ligaments are lax when the joint is extended but tightly stretched over the condyles of the metacarpal head when the joint is flexed. Therefore, it is important to maintain the MCP joint in flexion during immobilization to prevent shortening of the collateral ligaments.

The PIP joints are considered *hinge* joints because movements are restricted to flexion and extension by the configuration of the joint surfaces; the bicondylar distal end of the proximal phalanx fits congruently into two concave surfaces on the middle phalanx that are separated by a prominent median ridge. The articular surface of the proximal phalangeal head, unlike that of the metacarpal head, has a constant radius in the sagittal plane. The point of origin of each collateral ligament is on the

TABLE 1 Etiology of Small Joint Contracture of the Hand

CONGENITAL CAUSES	ACQUIRED CAUSES
Arthrogryposis	Trauma
Camptodactyly	Burns
	Connective tissue disease
	Spasticity/paralysis
	Volkmann's ischemia

axis of joint motion. As a result of these anatomic features, the collateral ligaments of the IP joints are under almost equal tension in both flexion and extension and do not dictate the best immobilization posture for this joint.

The MCP and IP joints also differ at the volar plate. The IP volar plate is inelastic; it maintains comparable dimensions in flexion and extension, thus necessitating translation of the plate in a proximal-to-distal direction with joint movement. Any limitation of the proximal volar plate results in restriction of motion. The proximal part of the volar plate, which is thin and membranous, is attached to the volar aspect of the proximal phalanx by check ligaments (two resistant fibrous bands that fix the lateral borders of the volar plate to the middle phalanx). In an uninjured joint they prevent hyperextension. In an injured, inappropriately immobilized joint they hypertrophy, shorten, and may prevent both flexion and extension of the joint by the formation of checkrein ligaments.

At the MCP joint the volar plate is firmly attached distally but loosely attached proximally, thereby allowing hyperextension of the joint. The volar plate of the MCP joint is relatively elastic, which allows it to shorten with flexion. There is no tendency for the development of checkreins. At the distal interphalangeal (DIP) joint, the checkrein ligaments are shorter, less taut, and more laterally placed, thus allowing for more hyperextension.

As a result of these differences, collateral ligament stiffness plays an important role in the genesis of stiffness at the MCP joints with development of the more typical extension contracture. In the IP joints, the collateral ligaments may adhere to the sides of the condyles, from which they may need to be freed, but the checkrein ligaments are the primary pathologic structure requiring release for full correction of the more typical IP flexion contracture.

Preoperative Assessment

The pathology of posttraumatic contracture is scar formation or adhesion of injured tissues, or both. Skin, soft tissues, ligaments, and tendon structures around the joint may also be involved. Management of patients with posttraumatic contracture commences with a complete history, functional evaluation, and physical examination.

Functional Evaluation

Any evaluation must take into consideration the injured joints, as well as the function of the whole hand. Surgical procedures rarely produce a return of normal joint motility. The aim of surgery should be to achieve functional improvement. As a rule,

TABLE 2 Common Causes of Small Joint Stiffness

LIMITATION OF MCP JOINT FLEXION	LIMITATION OF MCP JOINT EXTENSION
Thickening of the dorsal capsule	Long-standing intrinsic tightness
Contracture of the collateral ligament	Dupuytren's contracture
Extensor tendon/hood adhesions	Volkmann's contracture
Contracture of skin (e.g., dorsal burn)	Burns/palmar scarring
Bony block within the joint	Crush injuries
	Spasticity
	Sclerodactyly
LIMITATION OF PIP JOINT FLEXION	**LIMITATION OF PIP JOINT EXTENSION**
Contracture of the dorsal skin	Scarring of the volar skin
Extensor muscle tightness or tendon adhesion	Dupuytren's contracture
Intrinsic tightness	Scarring of the flexor tendon sheath
Contracture of the capsular ligaments, especially the collateral ligaments	Flexor muscle tightness or tendon adhesions
Bony block or exostosis	Contracture of the volar plate
	Adherence of the collateral ligaments
	Bony block or exostosis

MCP, metacarpophalangeal; PIP, proximal interphalangeal.

when the arc of motion of the joint is within 30 degrees of normal, it is unlikely that surgery will benefit the patient. *Functional* range of motion, based on common activities of daily living, averages 61 degrees at the MCP joint and 60 degrees at the PIP joint. Although the MCP joint provides a large percentage of the overall flexion arc, the portion attributed to the PIP joint is most important functionally because it is responsible for grip strength and completion. Functional requirements are not the same for each finger. For example, the functional arc of motion of the ring and little fingers can tolerate a loss of up to 40 degrees of extension, but adequate flexion is required. The index and middle fingers, however, both require satisfactory extension for effective pinch and grasp. At times, the arc of motion should be altered to a position of greater *functional* value to the patient even if the *absolute* value is not changed.

Physical Examination

Physical examination should proceed in a systematic manner. The following should be evaluated in every patient:

- Skin and soft tissues: They should be well healed, supple, and free of any residual inflammation or infection. When skin shortening is the cause of the contracture, passive joint motion stretches the scarred skin and causes blanching. In this setting, the scarred skin is assumed to be the causative pathology, although the degree of involvement of the deeper soft tissues cannot be adequately assessed without operative evaluation.
- Alignment/stability: Releasing a contracted joint is not indicated in patients with an unstable skeleton or misaligned digits. Successful release of a joint contracture demands immediate postoperative mobilization, and any concomitant procedure that prevents such mobilization can reduce the quality of the result.
- Assessment and documentation of neurovascular status: Surgery in an insensate finger is unlikely to produce significant functional improvement. Surgery in a digit with marginal vascularity may be hazardous. Fingers with poor neurovascular inflow might be better served by arthrodesis or amputation.
- Range of movement: Accurate measurements are necessary to monitor the patient's response to nonoperative measures.
- Tenodesis effect: Most often, there is no single cause for a joint contracture. A combination of joint tightness and other external constraints, such as muscle-tendon tightness and soft tissue contractures, is responsible. The dynamic tenodesis effect is negative when the pathology is intraarticular or periarticular, including tendon adhesions close to the joint. When tendon adhesions are located proximal to the affected joint, the dynamic tenodesis effect is positive (Fig. 1). The principle involved in assessing the tightness of any musculotendinous unit is to reverse its action. If it is not possible to place the hand in a posture that fully reverses its action, there must be tightness of the involved structure. In other words, when a tendon adhesion is present, specific movements distal to the area of adherence are lost. Specific passive movements are limited because the adherent tendon acts as a checkrein.

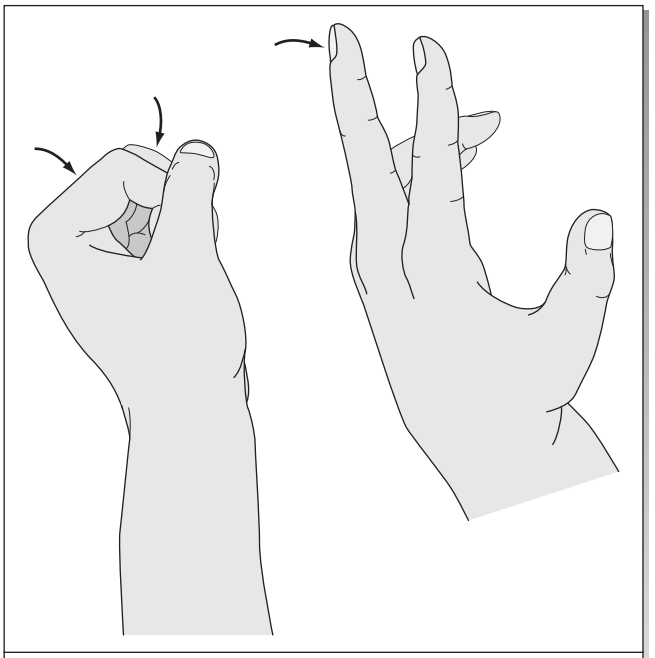

Figure 1 • Dynamic tenodesis. Intrinsic tightness is present if the range of interphalangeal joint flexion is limited when the metacarpophalangeal joint is extended or hyperextended as opposed to when it is flexed.

Special Tests

Radiographs are useful to visualize the integrity of the articular surfaces, assess skeletal alignment, and exclude exostoses.

Treatment

Treatment of established posttraumatic contracture of the hand begins with a thorough assessment, physical examination, and selected imaging studies. The key to a rational treatment plan is an understanding of the exact location and pathology of the contracture.

Nonsurgical

The majority of MCP/PIP joint contractures are successfully treated nonoperatively. It is imperative that therapy be commenced as soon as possible after an injury. Dynamic splinting or serial progressive static splinting maintained for 12 to 24 hours a day determines what can be achieved with conservative treatment. If the patient demonstrates sustained progress, operative release should be delayed. Surgery is indicated if a functional limitation persists and there is no progress in active or passive joint motion after several months of well-supervised therapy.

Surgical

Surgery is offered only if nonoperative treatment has failed to restore adequate function. Variables associated with improved outcomes include (1) younger age of the patient; (2) single, well-identifiable pathology; (3) well-healed skin with soft, pliable tissues; (4) intact musculotendinous units; (5) uninvolved joint surfaces; and (6) cooperative patients with access to an experienced hand therapist.

Although careful clinical assessment can help to determine the site of pathology, the exact cause of restricted joint motion is not always predictable preoperatively. The surgeon should anticipate the need for additional procedures.

The use of local anesthesia for patient input during the procedure offers the ability to intraoperatively verify the adequacy of the release and demonstrate to the patient the increase in motion achieved intraoperatively. If local anesthesia is not an option, a traction check will be necessary if extrinsic tendon adhesions are suspected, and observation of the degree of active motion is necessary. A proximal incision is made with passive pulling of the involved tendons to document range of motion.

Before performing capsulectomy/soft tissue release of the MCP/PIP joints, it is important that all potential causes for the loss of joint motion be considered and identified (see Table 1).

The following are absolute contraindications to soft tissue surgical procedures:

1. Severe joint destruction because soft tissue procedures may improve motion, but not pain, in these joints
2. Unreconstructible tendons

RELEASE OF METACARPOPHALANGEAL JOINT EXTENSOR CONTRACTURE (DORSAL CAPSULOTOMY). A longitudinal incision is made over the MCP joint to expose the entire extensor hood mechanism as far as the level of the intrinsics on each side of the joint. A transverse incision may be used if all four joints are involved. The extensor tendon is freed from its adhesions while preserving the periosteum. If extensor adhesions are the primary restriction to passive flexion, extensor tenolysis alone may prevent the need for capsulectomy. The extensor tendon is split longitudinally to expose the capsule, and the capsule is incised transversely. The thickened dorsal capsule and synovium are excised. The joint surface and collateral ligaments are inspected.

Joint tightness is tested in ulnar/radial deviation. One or both collaterals may require release. The collateral ligaments are freed from any adhesions and their attachment released from the metacarpal head if an attempt to passively flex the MCP joint fails. The ligaments are sectioned, if needed, in a dorsal-to-palmar fashion in small increments until full passive joint motion is restored. If possible, a portion of the radial collateral ligament is preserved to prevent ulnar drift with pinch and power grasp.

Full passive flexion of the MCP joint should now be possible. If the MCP joint opens like a book instead of the proximal phalanx gliding under the head of the metacarpal, a periosteal elevator is passed around the volar aspect of the head of the metacarpal to free adhesions between the metacarpal head and the volar plate (Fig. 2). Active range of motion is now examined. If full active motion is still not possible, persistent shortening of musculotendinous units is the most likely culprit because of an overlooked flexor tendon adhesion. Extensor tendon repair is then performed.

METACARPOPHALANGEAL JOINT FLEXION CONTRACTURE. Flexion contractures are almost exclusively caused by factors extrinsic to the joint and its capsule (see Table 2). Because of its specific anatomic structure, recovery of full extension seldom requires joint capsular release after the primary pathology has been addressed.

RELEASE OF PROXIMAL INTERPHALANGEAL JOINT FLEXION CONTRACTURE. A volar Y-V incision is generally used. If a skin contracture is present, a Z-plasty is more suitable. The radial and ulnar neurovascular bundles are isolated, and the position of the transverse branch of the digital artery is ascertained and used as a guide to identify the checkrein ligaments. The artery passes just distal and dorsal to their proximal insertions. It should be preserved because it supplies the vinculum breve of the superficialis and the vinculum longum of the profundus tendon. If the mobile sheath between the A2 and A4 pulleys is significantly contracted and adherent, excision of a window of sheath is required to provide adequate release. The C1 portion of the flexor sheath is excised between the A2 and A3 pulleys to allow retraction and tenolysis of the tendons.

The checkrein ligaments are identified and divided, and an attempt is made to extend the joint passively. If full extension is not possible, the volar plate is released from its attachment to the accessory collateral ligament on both sides of the joint. The volar synovial pouch and the inner portion of

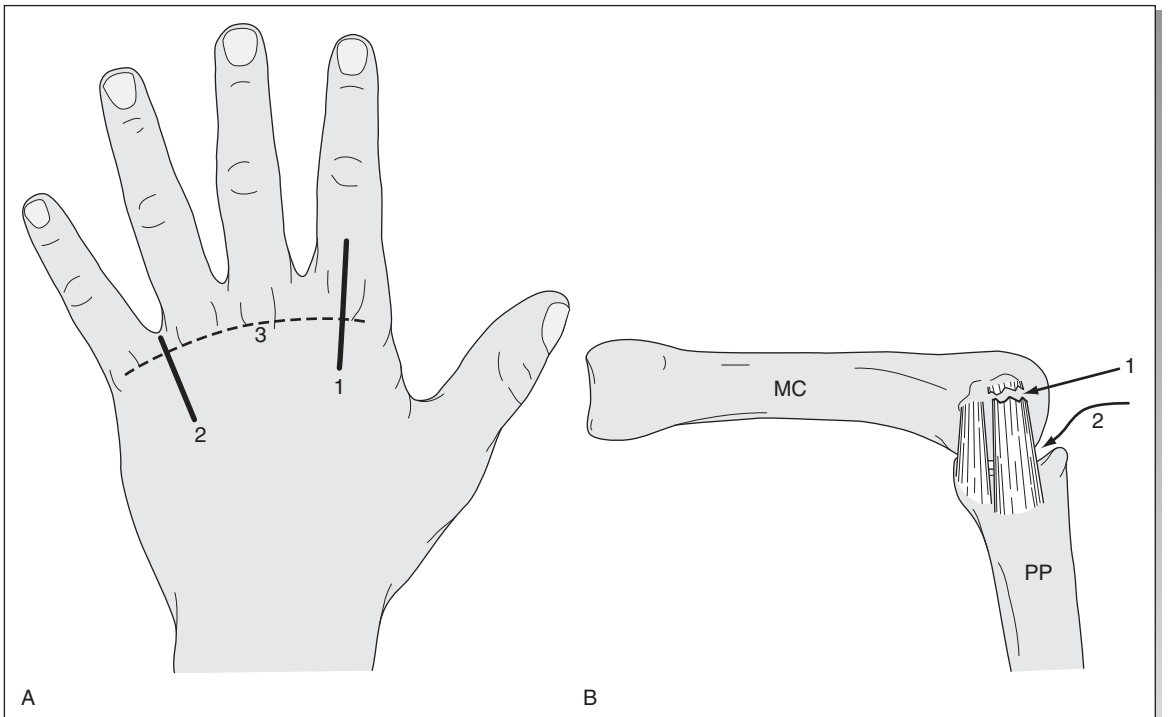

Figure 2 • A, Exposure for metacarpophalangeal (MCP) capsulectomy. 1. Midline dorsal approach (single joint). 2. Web space incision (adjacent joints). 3. Transverse incision (multiple joints). **B,** Technique for dorsal capsulectomy. 1. Dorsal capsule excision and release of the "cord" portion at the point of attachment to the metacarpal; the accessory collateral ligament is left intact. 2. An elevator is used to release adherent volar capsule and re-create a normal pouch beneath the metacarpal head. MC, metacarpal; PP, proximal phalanx.

the collateral ligaments are freed of any adhesions (Fig. 3). If passive extension of the joint is still not possible, the collateral ligaments are recessed from their origin in the head of the proximal phalanx. The release should be performed sequentially in a palmar-to-dorsal direction until full extension is achieved. Adequate release is confirmed by allowing the patient to move the finger. A discrepancy between active and passive range of movement is probably due to problems with excursion of either the flexor or extensor tendon system, and tenolysis is required. An "extensor check" is performed; a competent extensor mechanism will result in a tenodesis effect of the long extensor tendon and lead to full PIP joint extension when both the wrist and MCP joints are placed in flexion. In cases of long-standing and severe flexion contracture, overstretching of the extensor apparatus develops with attenuation of the central slip. Central loop reconstruction may prove effective, but it is seldom indicated because careful postoperative splinting will most often lead to retightening of the central slip.

RELEASE OF PROXIMAL INTERPHALANGEAL JOINT EXTENSION CONTRACTURE. A dorsal or midlateral incision is used to expose the extensor mechanism (Fig. 4). The transverse retinacular fibers are identified and incised longitudinally to permit retraction of the lateral band and exposure of the dorsal capsule and both collateral ligaments (see Fig. 4). A complete dorsal arthrotomy is performed just dorsal to the collateral ligaments by releasing the dorsal capsule from its attachment to the head of the proximal phalanx. Care is taken to avoid injury to the central extensor tendon. Excursion of the joint is evaluated. If the joint has a tendency to hinge open, a periosteal elevator is used to mobilize the recess between the collateral ligaments and the head of the proximal phalanx and to release any adhesions in the palmar capsular recess/retrocondylar space. If full passive flexion is still not possible, the collateral ligaments proper are released from their attachment to the proximal phalanx. Recession of the collateral ligaments should be performed in stages, beginning dorsally and progressing volarly until full digital flexion is achieved. Active range of motion is checked, with particular attention paid to intrinsic tightness. Central tracking of the extensor tendon also needs to be confirmed. If required, unilateral repair of the transverse retinacular fibers to the lateral band may be undertaken.

Postoperative Care

After surgical release of a contracted joint, the hand is placed in a well-padded dressing with a dorsal

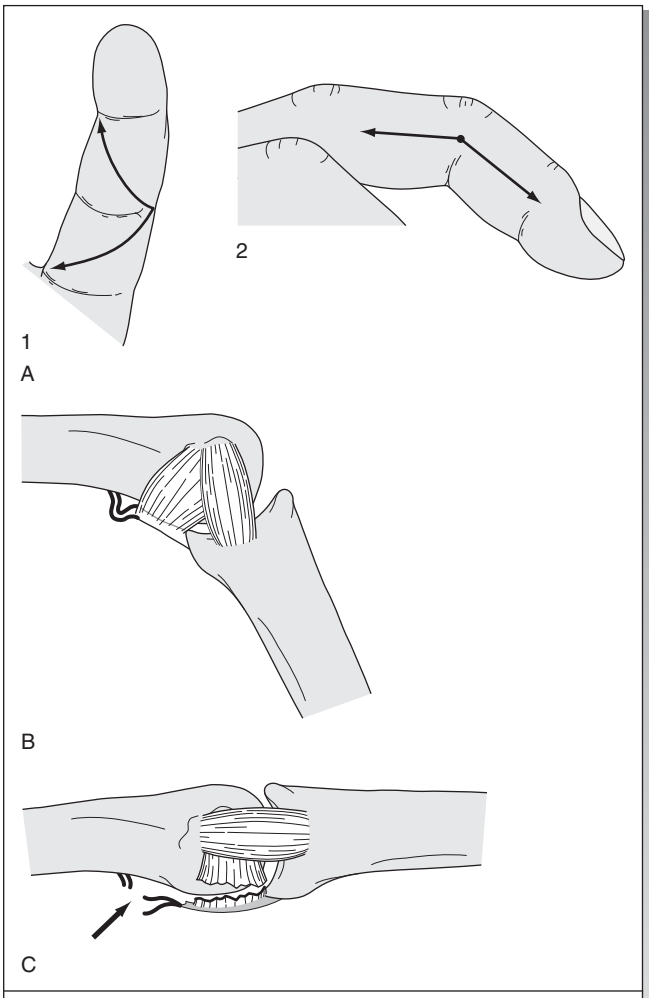

Figure 3 • A, Approach to the volar aspect of the proximal interphalangeal (PIP) joint. 1. Volar zigzag Bruner incision. 2. Midlateral incisions can be used on both sides of the finger. **B** and **C,** Release of flexion contracture of the PIP joint by division of the checkrein ligaments and the accessory collateral ligaments.

splint holding the MCP joint in approximately 60 to 80 degrees of flexion and the IP joints in extension. It is important to maintain the gains achieved at surgery with a rehabilitation program that includes immediate motion. Therapy is initiated within 24 to 48 hours of surgery. Dressings are removed on the last postoperative visit. Active, active-assisted, and passive range-of-motion exercises are commenced at this stage. The hand is generally splinted in the "safe" position between therapy sessions until decreased swelling and pain permit light use of the hand. Dynamic splints may be used if problems in maintaining passive motion are encountered. After functional use has been established for light activities, a strengthening program can be instituted. Vigilance for recurrence of contractures is mandatory, and static and dynamic splinting is commonly required for a period of up to 3 months.

Complications

Complications include overcorrection, instability leading to early joint degeneration, inadequate correction, recurrence of the contracture, and damage or disruption of the extensor or flexor system. Damage to the neurovascular bundle may occur with release of PIP flexion contractures. Ischemia may result if a long-standing PIP flexion contracture is splinted in full extension immediately after surgery. The most frequent complication is failure of the operation to return useful motion on a permanent basis. It is known that long-term results often show a loss of intraoperative gains.

Pearls and Pitfalls

- Successful surgical management of small joint contractures of the hand requires an under-

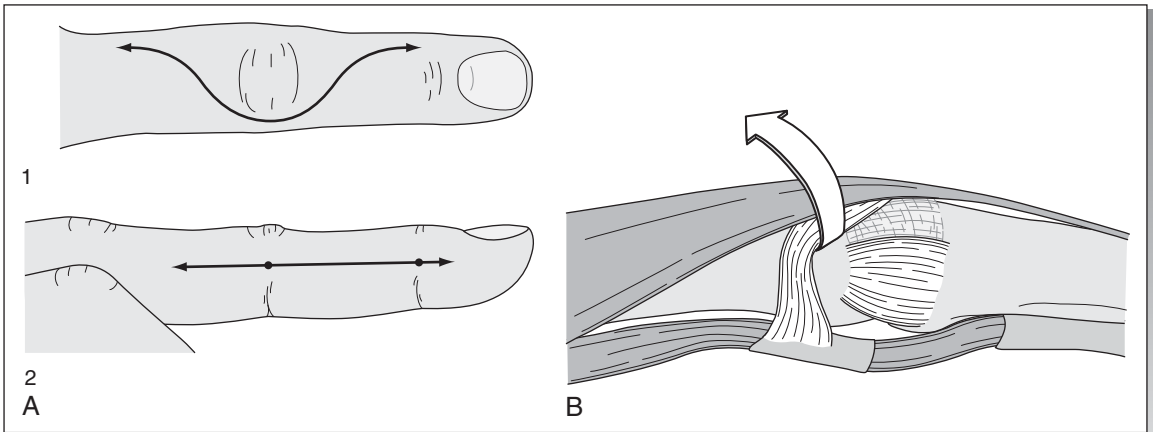

Figure 4 • A, Dorsal approach to the proximal interphalangeal (PIP) joint. 1. Dorsal incision. 2. Midlateral incisions can be used on both sides of the finger. **B,** Exposure of the lateral and dorsal aspect of the PIP joint after incision and elevation or retraction of the transverse retinacular ligament.

standing of the multiplicity of lesions that may be present.

- A significant increase in function may be achieved despite a minimal increase in motion if the range of motion is shifted to a more functional position.
- Excellent improvement should be expected in a young patient with a single identifiable cause for the contracture.

SUGGESTED READINGS

Curtis RM: Capsulectomy of the interphalangeal joints of the fingers. J Bone Joint Surg Am 36:1219-1232, 1954.

Diao E, Eaton R: Total collateral ligament excision for contracture of the proximal interphalangeal joint. J Hand Surg [Am] 18:395-402, 1993.

Gould JS, Nicholson BG: Capsulectomy of the metacarpophalangeal and proximal interphalangeal joints. J Hand Surg 4:482-486, 1979.

Harrison DH: The stiff proximal interphalangeal joint. Hand 9:102-108, 1977.

Sprague BL: Proximal interphalangeal joint contractures and their treatment. J Trauma 16:259-265, 1976.

Young VL, Wray C, Weeks PM: The surgical management of stiff joints in the hand. Plast Reconstr Surg 62:835-341, 1978.

Dupuytren's Contracture

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Dupuytren's disease remains an enigmatic condition, and despite decades of research, its cause and optimal treatment remain unclear. The demographics of aging in the industrialized world, where most of those afflicted live, dictate that the number of patients with this condition will increase. Treatment remains surgical, although enzymatic fasciotomy of involved cords has recently produced encouraging short-term results. In terms of surgical intervention, the pendulum has swung back from radical attempts to eradicate all traces of involved tissue to a more measured, conservative approach. Significant advances have been made in understanding this condition at the cellular level, and these advances may soon be harnessed for therapeutic benefit. Refinements in genetic testing may allow reliable prediction of disease severity.

Etiopathogenesis

In the long interval since the first description of this condition, there has been much controversy regarding its cause, pathophysiology, and treatment. Dupuytren's disease may be familial, but the genetic mechanism is still unclear. Few formal studies of inheritance have been undertaken; however, an autosomal dominant cause with variable penetrance has been suggested. It is possibly polygenic, or there could be an interaction between environmental and genetic factors.

The disease is characterized by benign fibroproliferative involvement of the palmar and digital fascial structures, but the underlying cause of the palmar fascial fibrosis remains unclear. Some immunologic abnormalities have been described in Dupuytren's disease, thus suggesting that the immune system has a role in disease pathogenesis. Histologic examination of tissue affected by Dupuytren's disease reveals excess collagen and extracellular matrix deposition. A high proportion of collagen type III, instead of the usual collagen type I, has been reported in the nodules. A specialized cell type, the myofibroblast, has been observed in the nodules and is thought to be the source of the altered collagen production and, thus, the key cell involved in the disease process. Myofibroblasts are probably differentiated fibroblasts with phenotypic characteristics between those of fibroblasts and smooth muscle cells, the latter capable of producing smooth muscle actin isoforms and several growth factors. The nodules have some histologic features of neoplasia, including a high proportion of mitotic cells. Cells from fibromatous nodules have been reported to show chromosomal aberrations, including trisomies and unbalanced translocations, but the clinical significance of these findings is unclear. Mechanical forces in the tissues and growth factors such as transforming growth factor-β1 appear to play important roles in regulating the proliferation and apoptosis of myofibroblasts.

Pathologic Anatomy

Success in protecting the neurovascular bundle from iatrogenic injury in Dupuytren's surgery is made possible only by knowledge of normal hand anatomy and the characteristic pattern in which the anatomy changes with disease progression. The palmar fascia is a complex structure containing bands of thickened collagen that allows longitudinal structures to pass through without interference. It is a fibrous skeleton with guide channels and retinacular restraints for longitudinally running fibers that anchor the skin and allow digital motion. In Dupuytren's contracture, the most frequently affected structures in the palm are the pretendinous bands with the formation of pretendinous cords. Spiral bands, natatory ligaments, and digital fascial structures are less

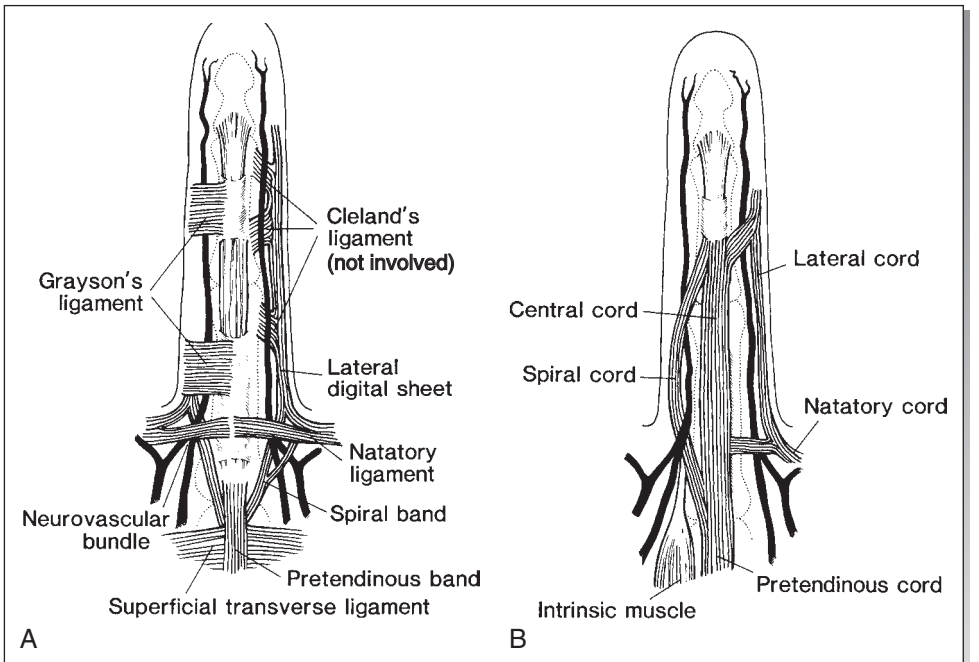

Figure 1 • A, The components of normal fascia that become diseased. **B,** The diseased cords that develop from the normal fascial structures. A spiral cord can originate from the pretendinous cord or from an intrinsic muscle. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990.)

frequently affected. The neurovascular bundle is at greatest danger in the palmar digital area, where it may be severely displaced by a spiral cord. The spiral cord typically begins proximally as the pretendinous cord passing deep to the neurovascular bundle distal to the metacarpophalangeal (MCP) joint. The spiral cord runs lateral to the neurovascular bundle. It involves the lateral digital sheath and may again become superficial as it joins Grayson's ligament (Figs. 1 and 2). The radial side of the hand, including the commissural bands, can be involved in severe cases. The ligaments of Cleland, transverse retinacular ligaments, oblique retinacular ligaments, transverse ligaments of the palmar aponeurosis, and the septa of Legueu and Juvara are not involved in the disease process.

Diagnosis

Although the diagnosis of Dupuytren's disease in its classic form is not subtle, its clinical features in the early stages may mimic other conditions. Dupuytren's nodules should be differentiated from other soft tissue lesions such as localized pigmented villonodular synovitis, palmar ganglions, inclusion cysts, or rarely, epithelioid sarcoma. A surgeon treating a patient for "trigger finger" may be dismayed to witness the progressive contracture of Dupuytren's disease postoperatively. Clinical features that suggest a poor prognosis include young age, severe proximal interphalangeal (PIP) joint contracture, Garrod's knuckle pads over the PIP joints, and extensive skin involvement.

The effect of the disease on joint motility is the major determinant for surgical intervention. Both active and passive range of motion of joints in involved digits is measured with a goniometer. Indications for surgical intervention fall into subjective and objective criteria. Subjectively, the patient may request treatment when the degree of contracture makes normal hand function difficult. MCP joint contracture can be completely corrected nonsurgically, and thus surgical intervention may be delayed. However, patients usually find that MCP contracture of 40 degrees or more causes sufficient restriction to warrant correction. Correction of PIP joint contracture, in contrast, becomes increasingly difficult as disease duration and the degree of joint contraction progress. PIP joint contracture of 25 degrees or more is a clear indication for surgical correction.

Plain radiographs may be useful in documenting the degree of contracture or assessing degenerative articular changes. Involvement of the distal interphalangeal joint or the presence of a boutonnière deformity indicates advanced disease. A clinicopathologic staging system based on disease activity and histologic appearance has been described; this system divides affected tissue into proliferative, involutional, and residual disease.

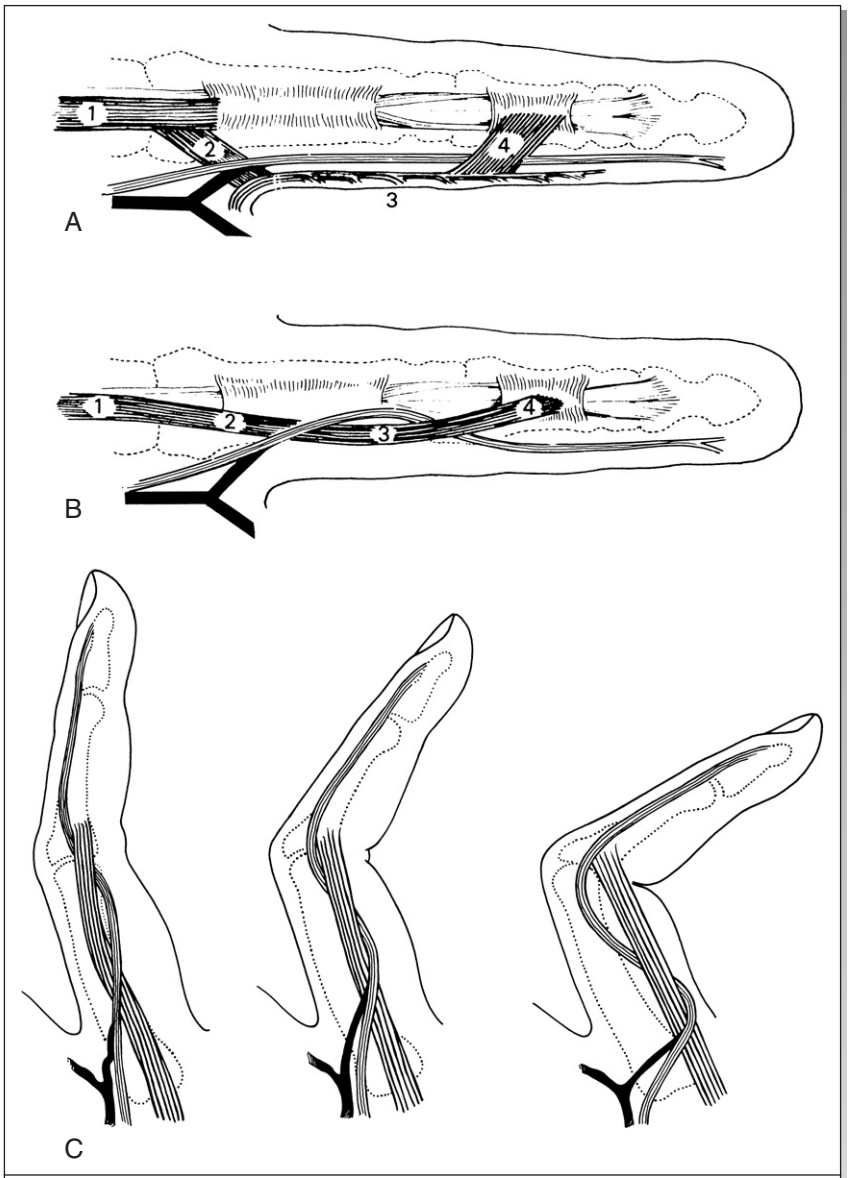

Figure 2 • Mechanism of displacement of the neurovascular bundle by a spiral cord. **A,** The normal structures that produce a spiral cord in disease—the pretendious band (1), the spiral band (2), the lateral digit sheet (3), and Grayson's ligament (4). **B,** As the four structures become diseased, the complex shortens and straightens, and the neurovascular bundle is displaced toward the midline of the finger. **C,** With increasing proximal interphalangeal joint contracture, the neurovascular bundle is displaced closer to the midline, and the point of displacement becomes more proximal and more superficial. (From McCarthy JG [ed]: Plastic Surgery. Philadelphia, Saunders, 1990.)

Treatment

Surgical

Surgical intervention for Dupuytren's contracture has evolved from the enthusiastic radical excision of the disease in the 1970s (with inherently higher complication rates) to the current practice of focused, limited excision. The importance of emphasizing functional outcomes over reliance on objective clinical measurements is the key to success in surgery for Dupuytren's contracture. Treatment of severe PIP joint contracture is challenging. Preoperative skeletal traction with an external fixator allows stretching and lengthening of the diseased tissue, but the traction technique must be followed quickly by definitive surgery or the contracture will

rapidly recur. The extent of PIP joint contracture release after excision of diseased tissue remains controversial. The addition of capsuloligamentous release permits greater intraoperative correction. However, the correction is not usually maintained postoperatively, and the final result is comparable to that in patients who undergo aponeurectomy alone. Incomplete extension results in less functional loss than does restricted flexion as a result of overzealous PIP joint release.

Management of Dupuytren's disease, which is predominantly palmar and causes only MCP joint contracture, may be treated by open fasciectomy or less invasive techniques. Subcutaneous fasciotomy was first advocated by Dupuytren in 1832. It is best suited for patients with thin palmar fascial cords mainly affecting the MCP joints and for patients in whom more extensive procedures are contraindicated. It may also be used as a preliminary procedure in patients requiring more extensive fasciectomy to facilitate subsequent skin incisions and dissection. Palmar segmental aponeurectomy has demonstrated satisfactory long-term results. Small curved incisions are made along the cord for excision of intermittent lengths of cord while leaving the intervening fascia intact. An advantage of this technique is that it does not violate the proximal finger compartment, thus making dissection easier in cases requiring reoperation at a later stage. The incidence of nerve damage is similar to that in open techniques.

Percutaneous needle fasciotomy is occasionally useful in medically unstable patients and can be performed with minimal morbidity in an outpatient setting. Under local anesthesia, a 19-gauge needle is used to sever the palmar cords while the digit is extended. However, it is controversial because of the potential for neurovascular injury and high rates of recurrence.

The mainstay of treatment of combined palmar and digital contracture remains limited fasciectomy with an open palm technique or local skin advancement (or both) by means of a Z-plasty or V-Y technique. The open palm technique involves transverse incisions in the palm and the palmar digital area. After contracture release there may be a gap of up to 4 cm. The phalangeal incisions are closed, but the palmar wound is allowed to heal secondarily. The advantage of this technique is that early motion may be commenced and many complications such as hematoma formation, edema, pain, and joint stiffness may be avoided. The technique does, however, require approximately 2 to 3 weeks longer for wound healing to occur.

Dermatofasciectomy involves removal of the fascia as well as the overlying skin. The excised skin is replaced with full-thickness skin grafts. Excellent results have been reported with this method; however, it has the drawbacks of poor graft sensibility and contour, as well as increased stiffness from prolonged immobilization. Although recurrence is rarely seen at the site of the skin graft, contractures can recur at the edges of the graft, as well as in ungrafted areas. These factors, combined with donor site morbidity, relegate dermatofasciectomy to recurrent cases or younger patients with aggressive disease.

Restoration of range of motion and absence of recurrent disease are important outcome measures in surgery for Dupuytren's contracture. Motion has been reported to increase from 46% of normal preoperatively to 96% postoperatively with use of the open palm technique; these gains are maintained over the long term. However, Dupuytren's disease can recur in up to 74% of patients. Patients who have surgery at an earlier age and those with severe PIP joint or small finger involvement have higher rates of recurrence. Patients who have been treated by dermatofasciectomy rather than simple fasciectomy also have recurrent disease, but secondary surgery is required in only 15% of cases.

Nonsurgical

The history of treatment of Dupuytren's contracture has been characterized by numerous attempts at successful nonoperative therapy. Physical modalities such as physical therapy, splinting, and ultrasound have demonstrated little benefit. Medical treatments such as oral corticosteroids, dimethyl sulfoxide, allopurinol, and interferon similarly are not successful and are burdened with significant side effects. Radiotherapy shows moderate short-term improvement, but its routine use for a benign condition cannot be justified.

The use of enzymatic (clostridial collagenase) fasciotomy may represent a significant advance in the treatment of this condition. The enzyme targets the excessive collagen deposition in Dupuytren's disease and attempts lysis and rupture of the finger cords. Although early results have been encouraging, long-term follow-up in a larger series of patients is needed before this therapy achieves routine use.

Postoperative Care

The postoperative protocol is tailored by the complexity of the procedure and the age of the patient. Hand elevation is indicated in the early postoperative phase. The role of the hand therapist is critical in instituting a postoperative program to maximize the functional outcome. It has been stated that 50% of the operative results depend on effective postoperative management. In general, postoperative rehabilitation should be initiated after cessation of the initial inflammatory phase, when the wound can tolerate active motion without reactive flare. Splinting should be started when active range of motion is begun. Scar management and edema control should be tailored to the patient's needs. It is important that the therapist and physician recognize and

expeditiously treat inflammatory flare-ups, which are occasionally seen postoperatively, particularly in female patients. This can be a difficult challenge for the patient and surgeon alike, but flare-ups can be ameliorated by pain control, judicious use of steroids, and gentle therapy to maintain active range of motion.

Pearls and Pitfalls

- Dupuytren's disease can be controlled, but not cured by surgery. It is important to counsel the patient about the natural history of the condition, the likelihood of multiple operations, and the considerable risk for complications.
- The goal of treatment is to correct the functional loss caused by contracture rather than to eliminate all evidence of the condition.
- Beware of a postoperative "flare reaction," particularly in younger female patients. It is characterized by marked inflammation with pain and rapid recurrence. This complication, although difficult to treat, is improved by pain control, oral steroids, and hand therapy to maintain range of motion.
- When operating on patients with recurrent disease, one must expect that the nerves and vessels may have been damaged during the primary procedure. Clinical examination should include two-point discrimination testing and a digital Allen test. Doppler examination or angiography may also be indicated.

SUGGESTED READINGS

Badalamente MA, Hurst LC, Hentz VR: Collagen as a clinical target: Nonoperative treatment of Dupuytren's disease. J Hand Surg [Am] 27:788-798, 2002.

Kloen P: New insights in the development of Dupuytren's contracture: A review. Br J Plast Surg 52:629-635, 1999.

McCash CR: The open palm technique in Dupuytren's contracture. Br J Plast Surg 17:271-280, 1964.

McFarlane RM: On the origin and spread of Dupuytren's disease. J Hand Surg [Am] 27:385-390, 2002.

McGrouther DA: The microanatomy of Dupuytren's contracture. Hand 14:215-236, 1982.

Peripheral Nerve Compression

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Peripheral nerve compression is a clinical problem that results from chronic pressure on a nerve. Nerve entrapment and nerve compression are equivalent, but nerve compression implies a chronic condition. Acute nerve compression is not the subject of this chapter. Treatment requires correct diagnosis and staging of the degree of nerve compression. Based on staging, either nonoperative or surgical treatment is indicated.

Etiopathogenesis

An increase in pressure on the nerve fascicle of greater than 20 mm Hg decreases venous flow within the nerve. The pressure can be applied externally by a factor that increases the volume within the anatomic confines of the nerve (e.g., the synovium within the carpal tunnel) or by something that increases the fluid within the epineurium (e.g., the glucose to polyol pathway in a diabetic patient). After 2 months of such pressure, fluid leaks from the endoneurial microvessels to create endoneurial edema. By 6 months, demyelination begins and progresses. In time, axonal loss occurs. The electrophysiologic parallels of these changes are an increase in latency and slowing of conduction velocity as the myelin thins, and a decrease in amplitude as axonal loss progresses. The parallels that occur in neurosensory testing are increasing cutaneous pressure or vibratory thresholds in the early stages and an increase in two point–discrimination distance in the later stages. The clinical parallels are a perception of tingling or pins and needles in the early stage for the sensory component and weakness for the motor component of the peripheral nerve. In the later stage of chronic nerve compression, there is persistent sensory disturbance and numbness for the sensory component and wasting or atrophy for the motor component of the peripheral nerve.

Pathologic Anatomy

For each peripheral nerve, there are one or more anatomic sites at which the nerve is susceptible to compression. The sites may be normal anatomy, abnormal anatomy, or normal anatomy that has become abnormal through injury. Injury can cause swelling, which, when combined with the immobilization needed to treat a fracture/dislocation, can result in chronic compression.

Diagnostic Criteria

Differential Diagnosis

To make a diagnosis of chronic nerve compression, the physician must first include this possibility in the differential diagnosis. If a patient complains of tingling in the right thumb and index finger, the differential diagnosis must begin with a lesion of the left postcentral gyrus and, proceeding distally, could include compression of the C6 nerve root in the vertebral foramen, compression of the upper trunk of the brachial plexus by the anterior scalene muscle, compression of the median nerve in the forearm by the deep head of the pronator teres or a fibrous origin of the flexor superficialis to the ring or middle finger, or more distally, compression of the median nerve in the carpal tunnel.

History

The patient's history is critical in making the correct diagnosis. To continue with the example cited earlier, nighttime awakening in a patient with symptoms in the thumb and index finger clearly points to the carpal tunnel as a source of the symptoms. If symptoms occur when the neck is turned, in association with shoulder pain, cervical disk pathology would be likely. If the patient has a history of a motor vehicle accident with whiplash, a cervical disk and brachial plexus compression should be considered.

A peripheral nerve can be compressed at more than one site (i.e., the double-crush syndrome). It is most often seen with the combination of a C6 nerve root and carpal tunnel syndrome or with cubital tunnel syndrome and compression of the lower trunk of the brachial plexus. It is less commonly seen in compression of the radial nerve in the radial tunnel and the radial sensory nerve in the forearm or in compression of the median nerve in the carpal tunnel and the median nerve in the forearm (pronator syndrome).

Physical Examination

Physical examination is critical in diagnosing chronic nerve compression. The skin territory and the muscles innervated by the suspected nerve must be individually examined. For example, the finding of abnormal sensibility of the little finger pulp could mean that the ulnar nerve is compressed at the wrist or the elbow. Finding abnormal sensibility over the dorsal ulnar aspect of the hand or weakness of the flexor profundus to the little finger identifies the source as being proximal to the wrist.

Two other critical physical examination techniques are used to make the diagnosis of chronic nerve compression. The first involves variations of external compression that produce symptoms. For example, if the nerve crosses a joint at the site of compression, flexion of that joint will increase pressure on the nerve and elicit its symptoms. Examples are wrist flexion for carpal tunnel syndrome (Phalen's sign) or elbow flexion to identify cubital tunnel syndrome. Provocative tests that invoke the muscle are also helpful, such as forearm pronation to identify radial sensory nerve compression or elevation of the hands above the shoulders to identify compression of the brachial plexus in the thoracic inlet. Direct pressure may be applied to the suspected site of nerve entrapment, such as pressure over the median nerve in the forearm or the brachial plexus beneath the scalene muscle. The second critical maneuver is to percuss the nerve at the suspected site of compression, usually at the region of anatomic narrowing (Tinel's sign). The sign as originally described noted distal progression of tingling related to nerve regeneration over time after a nerve injury. However, with respect to chronic nerve compression, the pathophysiology just described creates a mechanical sensitivity to percussion that permits identification of the site of compression.

Electrodiagnostic and Neurosensory Testing

Electrodiagnostic testing, although traditionally used in patients suspected of having nerve entrapment, is critical only for patients thought to have a nerve root problem (i.e., a cervical or lumbosacral disk cause). Such patients give a history of pain that initiates in the neck or lower part of the back, and selected electromyographic evaluation is necessary. Reasons against performing routine electrodiagnostic testing are that (1) it has a high false-negative rate, (2) it is unable to identify certain nerve compressions, (3) it is painful, (4) it is expensive and often costs more than a surgical decompression procedure, and (5) it cannot identify superimposed nerve compression in a patient with neuropathy.

Alternatively, neurosensory testing is reliable, valid, and painless. It measures the cutaneous perception threshold for temperature, vibration, and pressure and gives a definitive measurement of either small fiber function (thermal threshold, C and A-delta fibers) or large fiber function (vibration and pressure, A-beta fibers). The instrument of choice is the Pressure-Specified Sensory Device. It is useful for identification and documentation of peripheral problems, including nerve compression, neural regeneration, and neuropathy.

Neurosensory testing provides the basis for treatment of peripheral nerves. Based on the measurements, the degree of nerve compression can be staged. Examples of numerical staging systems are given for the median, ulnar, and posterior tibial nerves in Tables 1 to 3. With the use of staging or numerical grading, appropriate treatment can be planned.

Treatment

Nonoperative Therapy

When the degree of nerve compression is mild, specifically grades 1 to 4, the nerve may recover without surgical decompression (Fig. 1). Nonoperative treatment varies depending on the nerve compressed, but the principle is to "unload" the nerve where the pressure occurs. For example, with compression of the median nerve in the carpal tunnel, splinting the wrist in neutral position at night and during daily activities such as typing "unloads" the compression associated with wrist flexion. For cubital tunnel syndrome, wrapping the arm with a

TABLE 1 Numerical Grading Scale for the Median Nerve at the Wrist Level

NUMERICAL SCORE		DESCRIPTION OF IMPAIRMENT
Sensory	Motor	
0	0	None
1		Paresthesia, intermittent
2		Abnormal pressure threshold (Pressure-Specified Sensory Device) <45 years old ≤3 mm at 1.0–20 g/mm^2 ≥45 years old ≤4 mm at 2.2–20 g/mm^2
	3	Weakness, thenar muscles
4		Abnormal pressure threshold (Pressure-Specified Sensory Device) <45 years old ≤3 mm at >20.0 g/mm^2 ≥45 years old ≤4 mm at >20.0 g/mm^2
5		Paresthesias, persistent
6		Abnormal innervation density (Pressure-Specified Sensory Device) <45 years old ≥4 mm to <8 mm at any g/mm^2 ≥45 years old ≥5 mm to <9 mm at any g/mm^2
	7	Muscle wasting (1–2/4)
8		Abnormal innervation density (Pressure-Specified Sensory Device) <45 years old ≥8 mm at any g/mm^2 ≥45 years old ≥9 mm at any g/mm^2
9		Anesthesia
	10	Muscle wasting (3–4/4)

towel at night prevents sleeping with the elbow flexed and can help to relieve symptoms and avoid surgery.

Operative Treatment

Surgical treatment is directed at removal of any structure that can cause compression of the peripheral nerve. The techniques have been extensively described and are not reviewed here. However, certain specific technical points deserve to be outlined for some of the less common nerve entrapment sites.

The median nerve in the forearm is best approached with a straight or S-shaped, 6- to 8-cm incision on the volar aspect of the forearm. Through this incision, the forearm fascia is divided, including the lacertus fibrosus. The same approach should be used for both pronator syndrome and anterior interosseous nerve compression. It is important to avoid injury to the medial and lateral antebrachial cutaneous nerves. The site of compression is almost always the deep head of the pronator teres or the fibrous arch of the superficialis muscles. The median nerve should be completely freed through the areas of these structures. The site of compression is almost never proximal to the elbow, and a long zigzag incision is not necessary. If it is believed that there is an entrapment site proximal to the elbow, a separate small incision above the medial humeral epicondyle can be used.

The radial nerve at the elbow is approached with a curved or straight incision over the extensor muscle mass in the forearm distal to the lateral

TABLE 2 Numerical Grading Scale for the Ulnar Nerve at the Elbow Level

NUMERICAL SCORE		DESCRIPTION OF IMPAIRMENT
Sensory	Motor	
0	0	None
1		Paresthesia, intermittent
	2	Weakness in pinch/grip (lb) Female: 10–14/26–39 Male: 13–19/31–59
3		Abnormal pressure threshold (Pressure-Specified Sensory Device) <45 years old ≤3 mm at 1.0–20.0 g/mm^2 ≥45 years old ≤4 mm at 1.9–20.0 g/mm^2
	4	Weakness in pinch/grip (lb) Female: 6–9/15–25 Male: 6–12/15–30
5		Paresthesia, persistent
6		Abnormal innervation density (Pressure-Specified Sensory Device) <45 years old ≥4 mm to <8 mm at any g/mm^2 ≥45 years old ≥5 mm to <9 mm at any g/mm^2
	7	Muscle wasting (1–2/4)
8		Abnormal innervation density (Pressure-Specified Sensory Device) <45 years old ≥8 mm at any g/mm^2 ≥45 years old ≥9 mm at any g/mm^2
9		Anesthesia
	10	Muscle wasting (3–4/4)

TABLE 3 Numerical Grading Scale for the Posterior Tibial Nerve at the Ankle

NUMERICAL SCORE		DESCRIPTION OF IMPAIRMENT
Sensory	Motor	
1		Paresthesia, intermittent
2		Abnormal pressure threshold (Pressure-Specified Sensory Device) <45 years old ≤6.3 mm at 6.8–30 g/mm^2 ≥45 years old ≤8.3 mm at 25–40 g/mm^2
	3	Weakness, thenar muscles
4		Abnormal pressure threshold (Pressure-Specified Sensory Device) <45 years old ≤6.3 mm at >30.1 g/mm^2 ≥45 years old ≤8.3 mm at >40.1 g/mm^2
5		Paresthesias, persistent
6		Abnormal innervation density (Pressure-Specified Sensory Device) <45 years old ≥6.3 mm to <10 mm at any g/mm^2 ≥45 years old ≥8.3 mm to <11 mm at any g/mm^2
	7	Muscle wasting (1–2/4)
8		Abnormal innervation density (Pressure-Specified Sensory Device) <45 years old ≥10.1 mm at any g/mm^2 ≥45 years old ≥11.1 mm at any g/mm^2
9		Anesthesia
	10	Muscle wasting (3–4/4)

humeral epicondyle. The same approach should be used for both radial tunnel syndrome and the posterior interosseous nerve. The radial and posterior interosseous nerves should be identified and freed. If compression is suspected above the elbow, a separate incision is used. It is important to avoid injury to the posterior cutaneous nerve of the forearm. It is also important to identify and divide the fibrous edge of the extensor carpi radialis brevis and the supinator fascia (arcade of Fröhse).

The ulnar nerve at the elbow is often compressed by an accessory origin of the medial head of the triceps, which can cross over the nerve. If present, it should be divided. When performing anterior transposition of the ulnar nerve, care should be taken to divide the fascia from the medial head of the triceps to the medial intramuscular septum. In addition, it is important to remove a segment of the medial intermuscular septum and divide the periosteal origin of the flexor carpi ulnaris from the ulna at the distal ulnar nerve transposition point. Patients should begin early elbow movement postoperatively.

Peroneal nerve compression can occur at several sites. For the common peroneal nerve at the fibular neck, the fascia superficial to the peroneus longus muscle is divided, the muscle is elevated, and the fibrous bands deep to the muscle are evaluated. Bands are present in approximately 20% of cadavers, but in 80% of patients coming to surgery. Care must be taken to observe the entrance of the small motor fascicles into muscle just past these bands. For the superficial peroneal nerve in the leg, approximately 20% of patients have a proximal division of the nerve, thus mandating evaluation and fasciotomy of both the anterior and the lateral compartments. The anterior tarsal tunnel is never a site of entrapment unless the ankle has been crushed, fused, or skin grafted. If the deep peroneal nerve is compressed distally, beneath the extensor hallucis brevis tendon, the tendon must be excised.

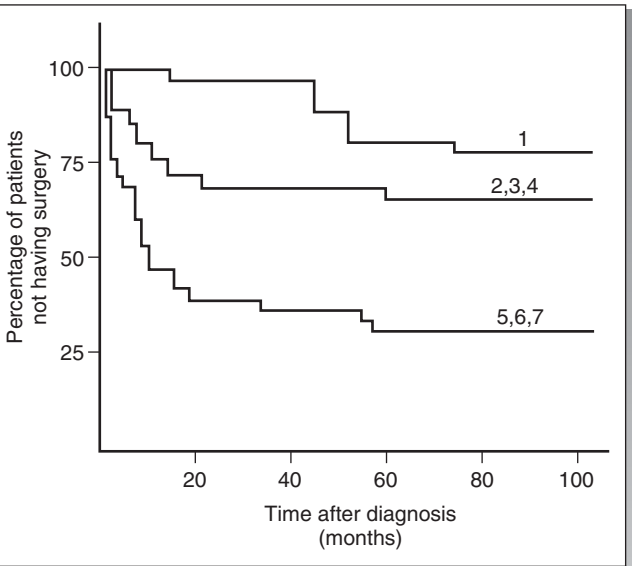

Figure 1 • Life table analysis of the results of nonoperative management of cubital tunnel syndrome. For statistical purposes, a successful outcome of nonoperative management was defined as not including a surgical procedure. From the graph, 89% of patients with intermittent symptoms only (grade 1), 67% of patients with intermittent symptoms and an increased sensorimotor threshold (grades 2, 3, and 4), and 38% of patients with persistent symptoms and signs of degeneration (grades 5, 6, and 7) did not undergo surgery. Patients with grades higher than 7 had surgery without a trial of nonoperative treatment. These differences were significant at the $P < .001$ level. (Redrawn from Dellon AL, Hament W, Gittelsohn A: Non-operative management of cubital tunnel syndrome: Results of an eight year prospective study. Neurology 43:1673-1677, 1993.)

With regard to the tibial nerve at the ankle, the tarsal tunnel is *not* the site of compression in tarsal tunnel syndrome. The flexor retinaculum is invariably loose unless there has been previous surgery or immobilization for an ankle injury. The tarsal tunnel must be opened to identify variations in the calcaneal nerves and in the tibial nerve itself, which may have a high division and require internal neurolysis, depending on the degree of fibrosis identified intraoperatively. An incision must be planned across the abductor hallucis brevis muscle, centered on the lateral plantar tunnel, to provide access to the medial and lateral plantar and calcaneal tunnels. It is the more distal tunnels in which a pressure increase causes symptoms. The fascia of the abductor must be released superficially, the muscle retracted, and the medial and lateral plantar tunnels identified. Each can be cannulated to demonstrate its tightness and identify the course of the tunnel, which is toward the navicular for the medial tunnel and directly across the foot for the lateral tunnel. The roof of each tunnel is incised, and the septum between the tunnels is cauterized and excised to create one large tunnel. The fascia from the roof of the lateral tunnel is followed posteriorly to identify the calcaneal tunnel. Fifty percent of patients have a 1-mm sensory branch to the skin of the arch that arises from the medial plantar nerve and crosses the vessels. The sensory nerve passes directly beneath the incision.

Postoperative Care

Mobilization of the decompressed peripheral nerve is the most important aspect of treatment. The technique used to decompress the peripheral nerve must permit early movement of the joint across which the nerve travels or the nerve will become adherent in its newly decompressed position. For carpal tunnel surgery, the splint is worn for only the first week to ensure the flexor tendons stay within the carpal tunnel and to avoid bowstringing. During this week, finger flexion and extension permit the median nerve to glide. No splint is used for cubital tunnel surgery, but rather, immediate postoperative elbow range of motion is encouraged, avoiding complete elbow extension. The latter is permitted after the first week. For radial nerve surgery, forearm supination and pronation, as well as elbow movement, are encouraged. For tarsal tunnel surgery, the patient is placed in a large, bulky, Robert Jones–type dressing to permit immediate postoperative ambulation. A walker instead of crutches is used because a walker permits the tibial nerve to glide and prevents heel cord contracture. For peroneal nerve surgery, immediate ambulation is permitted. After brachial plexus neurolysis and scalenectomy, immediate head turning and shoulder shrugs are encouraged on an hourly basis for the first week.

Pearls and Pitfalls

- The history and physical examination can be used to diagnose peripheral nerve compression in 90% of patients. The diagnosis can be documented with neurosensory testing.
- Electrodiagnostic testing has such a high false-negative rate that it is reserved for patients suspected of having nerve root compression.
- Patients with diabetic neuropathy have numbness, tingling, and pain that can be relieved by decompression of the peroneal nerve at the knee and dorsum of the foot and by decompression of the four medial ankle tunnels.
- More than one nerve entrapment may be present, and the second entrapment may be situated at more than one site along the course of the same peripheral nerve.
- Avoid injuring a cutaneous nerve at the incision site to prevent painful neuromas postoperatively.

SUGGESTED READINGS

Dellon AL: Treatment of symptoms of diabetic neuropathy by peripheral nerve decompression. Plast Reconstr Surg 89:689-697, 1992.

Dellon AL: Clinical grading of peripheral nerve problems. Neurosurg Clin N Am 12:229-240, 2001.

Lundborg G: Nerve Injury and Repair. Edinburgh, Churchill Livingstone, 1988.

Mackinnon SE, Dellon AL: Experimental study of chronic nerve compression: Clinical implications. Clin Hand Surg 2:639-650, 1986.

Mackinnon SE, Dellon AL: Surgery of the Peripheral Nerve. New York, Thieme, 1988.

Vascular Insufficiency and Ischemia

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Vascular insufficiency of the upper extremity is a relatively rare problem that can be caused by a wide variety of clinical conditions. Acute ischemia of any cause is a surgical emergency that demands immediate evaluation and treatment. Vascular insufficiency of more insidious onset may not cause symptoms until late in the disease process because of the relatively low metabolic demands of the upper extremity. Regardless of the cause, vascular insufficiency can ultimately result in tissue loss, intolerable pain, or both. Successful treatment can be a formidable surgical challenge.

Etiopathogenesis/Causative Factors

Causes of vascular insufficiency of the upper extremity can be broadly categorized into three groups: *trauma*, including iatrogenic injuries; *atherosclerosis*; and *connective tissue* or *vasospastic diseases*. Thromboembolic arterial occlusion can result from any of these events and is frequently the cause of the initial symptoms.

Iatrogenic upper extremity vascular occlusion can be secondary to a variety of medical procedures, but any procedure that cannulates an artery can result in arterial occlusion. Brachial artery puncture for cardiac catheterization results in occlusion in approximately 1% of cases. In addition, dialysis access procedures may culminate in hand ischemia as a result of arterial steal.

Acute penetrating trauma of an artery can result in the formation of a pseudoaneurysm, which generally manifests as a pulsatile hematoma. Contusion or repetitive blunt trauma can damage the arterial media layer and cause dilatation and true aneurysm formation.

Atherosclerotic disease can occur anywhere along the arterial tree and can cause symptoms either by occlusion of the artery at the site of disease or by embolic occlusion of a distal vessel. Atherosclerotic disease can also result in aneurysm formation.

The list of systemic diseases that can result in vascular ischemia of the upper extremity is varied (Table 1). Connective tissue disorders cause deposition of antigen-antibody complexes in the vascular endothelium, thereby leading to occlusion of small arteries. In the hand, such deposition usually occurs at the level of the proper digital arteries. Several inflammatory diseases can also result in hand ischemia. Inflammation of part of or the entire vascular wall can lead to spasm or thrombosis and, ultimately, vascular insufficiency. Buerger's disease (thromboangiitis obliterans) is caused by inflammation of all layers of the vessel wall and may be due to a reaction to tobacco smoke.

The main vasospastic disorders are Raynaud's disease and Raynaud's syndrome. Raynaud's disease is a primary diagnosis of vasospasm, whereas Raynaud's syndrome or phenomenon is a secondary diagnosis assigned to vasospasm resulting from an underlying disease. Both can cause decreased nutritional flow and reduced oxygen delivery resulting in ischemia and cell death. In addition to pain and cold intolerance, patients may complain of weakness and decreased sensation in their fingers. The examiner may detect loss of dermal ridges, color changes (pallor, cyanosis, rubor), coolness, abnormal hidrosis, or tissue breakdown.

A number of conditions are associated with Raynaud's phenomenon: thoracic outlet syndrome, dialysis shunting, vibration-induced injures, frostbite, toxins (tobacco, lead, vinyl chloride, stimulants), scleroderma, rheumatoid arthritis, cancer, hypothyroidism, and blood dyscrasias

TABLE 1 Systemic Causes of Vascular Insufficiency of the Upper Extremity

Atherosclerosis
Connective tissue disorders
Lupus
Scleroderma
Rheumatoid arthritis
Inflammatory disorders
Thromboangiitis
Giant cell arteritis
Takayasu's arteritis
Uremic arteritis
Polyarteritis nodosa
Dermatomyositis
Sepsis
Deep venous thrombosis

(cryoglobulinemia, polycythemia vera); however, most patients have no associated disorder.

Anatomy

The axillary artery becomes the brachial artery at the inferior border of the teres major muscle. As the brachial artery courses distally, it travels from the medial to the anterior aspect of the humerus before terminating as the radial and ulnar arteries at the level of the neck of the radius. The ulnar artery runs deep to the flexor muscle bellies and becomes more volar in the distal end of the forearm. Branches of the ulnar artery in the forearm include muscular branches, recurrent branches around the elbow and wrist, and the common interosseous artery. The common interosseous artery divides anteriorly and posteriorly. The median artery is a branch of the former. Nutrient and muscular branches, as well as anastomotic branches about the wrist, arise from the anterior and posterior interosseous arteries. The ulnar artery terminates as the primary contributor to the superficial palmar arch.

The radial artery travels laterally in the forearm and is slightly smaller than the ulnar artery. Similar to the ulnar artery, it begins near the neck of the radius and runs deep to the muscle bellies; it becomes more superficial in the distal part of the forearm. Radial artery branches include the muscular and recurrent arteries in the forearm. The dorsal or posterior branch of the radial artery enters the dorsum of the hand at the radial styloid process under the tendons of the first dorsal compartment. A branch passes through the two heads of the first dorsal interosseous artery to the palm and joins the deep palmar arch. The radial artery is the main contributory artery of the deep palmar arch; a superficial volar branch from the radial artery on the abductor pollicis brevis connects to the superficial palmar arch.

The ulnar artery enters the hand at the lateral aspect of the pisiform bone and contributes to the superficial palmar arch. A deep branch passes dorsally to the hypothenar muscles and sends a branch to the deep palmar arch. A branch is also given to the ulnar aspect of the little finger. The convexity of the superficial arch runs approximately with Kaplan's line, dorsal to the palmar aponeurosis. Three common digital arteries pass volar to the second, third, and fourth lumbricals and divide into two proper digital arteries in the second, third, and fourth web spaces.

The deep palmar arch lies at the proximal aspect of the metacarpal bones and is deep to the flexor tendons and adductor pollicis muscle. The princeps pollicis artery is the first volar metacarpal branch of the deep palmar arch. It most often provides the radial and ulnar digital arteries to the thumb and often the radial digital artery to the index finger.

Cadaver dissections have demonstrated that the superficial palmar arch is "complete" in approximately 80% whereas the deep arch is complete in 97%. Despite a significant amount of variance, radial artery dominance is more common. There appears to be a low rate of clinical radial-ulnar independence inasmuch as only 7% of ulnar and 2% of radial arteries fail to perfuse the hand within 6 seconds.

Diagnostic Studies

In a setting of acute vascular insufficiency, it is important to determine the mechanism and circumstances of the injury or event, the duration of symptoms, treatment before initial evaluation, past medical history, medications, tobacco use, occupation and occupational exposure, repetitive injury, and previous similar occurrences or injury. A patient with a history of chronic vascular insufficiency should be questioned about timing, frequency, treatments, environments, or activities that make symptoms worsen or improve; family history and past medical history, with a specific focus on autoimmune disorders, blood dyscrasias, and diseases affecting the microvasculature (e.g., diabetes mellitus, renal disease, malignancies); age at onset; the presence of unilateral or bilateral disease; and changes in sensation or strength.

Physical examination should include the entire upper extremity, including the neck. It is necessary to compare both arms. Documentation is made of atrophy, edema, skin quality, previous surgical or injury sites, soft tissue or skeletal deformities, and ulcerations or gangrene. When assessing the hand, particular attention is paid to color, posture, mottling, and calluses or masses. Nail growth, signs of infection, deformities, and nail bed color are evaluated. The extremity is examined for temperature changes, hidrosis, pulses, capillary refill, digital

turgor, and palpable masses. Any mass should be evaluated for pulsations and compressibility. Neurologic status, both sensory and motor, is recorded.

An Allen test is usually part of the evaluation of blood supply to the hand. The radial and ulnar arteries are occluded at the wrist while the patient clenches the fist to exsanguinate the hand. The result is considered abnormal if release of pressure over either of the arteries results in greater than a 6-second delay in digital reperfusion. Allen's test is useful as a quick screening test but has been shown to have a significant rate of false-positive results.

An external Doppler ultrasound probe can be used to provide a quick, noninvasive method for assessment of adequate flow and vessel integrity. Besides the major vessels, pulses can be evaluated at the palmar arch, common and proper digital arteries, and the superficial radial branch. Doppler signals should be assessed at various arterial landmarks with digital compression on others to detect audible discrepancies from the normal triphasic flow pattern.

The gold standard for evaluating the macrovasculature of the upper extremity is angiography. Information regarding the direction of flow, patency of vessels, and collateral circulation may be assessed by this method. Complications of angiography include allergic reaction to contrast media, hematoma or damage (or both) to the vessel at the injection site, renal failure, brachial plexus injury, aortic dissection, and dislodgment of emboli.

Radionuclide imaging using technetium 99m pertechnetate to tag red blood cells is useful for assessing vascular insufficiency states. This minimally invasive technique provides dynamic and static information regarding vascular perfusion and anatomy.

Magnetic resonance angiography is a noninvasive technique for assessing vasculature. Among its advantages is the ability to visualize all layers of a blood vessel without a dye load or radiation exposure. Computed tomographic angiography is another recently developed technology that also has the advantage of avoiding arterial cannulation.

A number of other techniques may be available for evaluating the vasculature of the upper extremity, although they usually add little to the evaluation of a patient with severe ischemia. Digital plethysmography, color duplex imaging, transcutaneous partial pressure of oxygen, intramuscular pH measurements, indicator dilution techniques, laser Doppler fluxmetry, and skin surface temperature recordings all provide information regarding flow or perfusion (or both) of the digits, but they add little to understanding of the arterial anatomy. Cold stress testing and vital capillaroscopy may be helpful in the evaluation and treatment of vasospastic disorders, but they do not delineate vascular pathology.

Standard laboratory testing is undertaken when assessing a patient for possible vascular insufficiency. Such tests include a complete blood count, erythrocyte sedimentation rate, and a comprehensive metabolic panel. Depending on the history and physical examination, plain radiographs (hand and thoracic outlet) may be appropriate.

Reconstructive Goals

The goals in treating vascular insufficiency of the upper extremity are to provide the patient with a well-perfused, nonpainful, functional, sensate, and normal-appearing upper extremity.

Treatment

Treatment of suspected or known vascular compromise is dependent on the diagnosis. Treatment may also be determined by the extent of injury, the involved vessel, and the presence of collateral circulation.

Embolism

The sudden onset of hand ischemia is often indicative of embolization. The cause may be elucidated by the history and physical examination. Angiography is helpful in defining the location of the pathology and noting the presence of collaterals. The upper extremity is the destination of 15% of emboli; the heart is the source more than half the time. Cardiac emboli tend to be larger than arterial emboli and consequently cause occlusion of proximal arm vessels or at the ulnar/radial artery takeoff. Potential causes of emboli include atrial fibrillation, myocardial infarction, thoracic outlet syndrome, arteriosclerotic plaque, vascular grafts, trauma, and aneurysms. Symptoms such as pain, pallor, paresthesias, pulselessness, and paralysis suggest a relatively proximal source. Patients who have Raynaud's-type symptoms generally have emboli from atherosclerosis or thrombi that have showered the arterial tree more distally.

Although some emboli or thrombi may spontaneously dissolve, ischemic changes and potential gangrene necessitate more aggressive intervention. Thrombolytic agents are used when surgical reconstruction is not possible (e.g., very distal numerous lesions or an unstable patient). Thrombolytic agents may be also considered in cases of nonembolic thrombosis, the most common causes of which are atherosclerosis, low-flow states, or intraarterial injections.

EMBOLECTOMY. To perform embolectomy, the artery of concern is accessed via surgical exposure. An arteriotomy is made in the vessel proper or a side branch. The size of the arteriotomy incision

should be sufficiently large to pass the appropriately sized Fogarty catheter (2.0- or 3.0-mm catheters for the forearm, 1.0- or 1.5-mm catheters for the distal part of the palm). In cadaveric studies it was impossible to displace emboli more distally with Fogarty catheters. The catheter passes either through the embolus or between it and the arterial wall. Pressure far greater than surgically necessary would need to be exerted to cause arterial rupture or dissection of plaques. The Fogarty catheter is gently fed into the artery until resistance is encountered. The balloon on the catheter is cautiously inflated with saline and pulled back toward the arteriotomy site. It is important to adjust the tension on the balloon to the point of mild resistance. The embolus is evacuated through the arteriotomy site. The process is repeated until no further evidence of extractable clot is found. Thrombolytic therapy is added in patients with persistent hypoperfusion distally.

The arteriotomy is closed under magnification. The vascular repair may be monitored by external or internal Doppler; fluorescein injections may be used to assess distal perfusion. Monitoring for compartment syndrome should also be performed. Dextran and heparin infusions may be started and continued for approximately 1 week. Oral anticoagulants are generally started perioperatively and continued for 3 months.

THROMBOLYTIC THERAPY. Urokinase and streptokinase convert plasminogen to plasmin and thereby cause fibrin clot degradation. Intraarterial delivery is usually performed with concomitant intravenous administration of heparin to prevent pericatheter thrombosis. Fibrinogen levels are monitored at least every 12 hours; infusion rates are halved for fibrinogen levels less than 150 mg/dL and stopped if less than 100 mg/dL, until levels of fibrinogen have normalized (165 to 365 mg/dL).

The combination of a powerful thrombolytic agent with an invasive delivery system can lead to major complications. Patients receiving such therapy need to be monitored closely, and patient selection is extremely important. It must be emphasized that if there is an injured arterial segment, thrombolysis is only a temporizing measure until excision of that segment can be performed.

Traumatic Arterial Occlusion

Direct or repeated trauma to an extremity can result in arterial injury and thrombus formation. In the upper extremity this most commonly occurs at the ulnar artery in the palm ("hypothenar hammer" syndrome), but it can also occur at the radial artery in the snuffbox, the digital arteries, or more proximally in the axillary or brachial arteries as mentioned previously. Digital artery thrombosis can be seen with the use of heavy vibratory machinery such as jackhammers and has also been reported in baseball catchers as a result of repeated trauma to the base of the index finger.

It is important to recognize that simple occlusion of a single digital artery or even a major vessel such as the radial or ulnar artery rarely causes ischemia because of the rich collateralization usually found in the hand. However, removal of the clot plus excision of the injured vessel is required to eliminate a source of potential emboli.

Arterial Reconstruction

When thrombus formation or trauma results in intimal damage, the injured arterial segment is removed. Primary anastomosis may be undertaken if there is no tension at the arterial repair site. It is important that the injured artery be excised. The possibility of thrombus canalization must be ruled out because it may compromise the repair if not removed.

Grafts are often necessary to bridge healthy artery to healthy artery. Donor veins may include superficial leg (saphenous or dorsal foot), cephalic, or distal forearm veins. Vein grafts are flushed with heparinized saline before the anastomosis. The proximal anastomosis may be performed first to assess blood flow through the graft and to ensure that the graft is not twisted and that it is reversed.

Arteries are occasionally used as grafts for arterial reconstruction. Although "expendable" artery is generally a misnomer, the radial and internal mammary arteries are increasingly being used as donor arteries for cardiac revascularization. In the upper extremity, arterial grafts can occasionally be harvested from a nonreplantable part or a nonreconstructible artery.

Size mismatch of vessels can be accommodated for by a number of methods. The donor and recipient ends can be cut to match sizes. Oblique cuts or longitudinal incisions into the vessel increase the diameter. To decrease the diameter of a vessel, a V-shaped excision of vessel can be closed primarily. Excellent suture technique is critical in this lumen-narrowing procedure. The end-to-side technique may also compensate for size mismatch. A sleeve anastomosis may be used when the proximal vessel is smaller than the distal vessel. This technique is also beneficial in cases in which the recipient vessel is severely calcified. The smaller vessel is telescoped into the larger vessel by placing partial-thickness sutures in the proximal vessel and full-thickness arterial wall sutures inside to outside in the distal vessel. Sleeve anastomoses take less time and have been reported to have patency rates comparable to those of the end-to-end technique.

Aneurysms

Arteries with aneurysmal formation may be ligated if the collateral circulation is sufficient, although proximal aneurysms of the upper extremity should

be treated by resection and vascular reconstruction. Treatment of those located distally in the forearm and hand is dependent on the preoperative and intraoperative findings. Ligation of the affected artery can be performed if adequate collateral circulation is noted despite occlusion of the affected vessel. Reconstruction is undertaken when it is technically feasible and when the presence of backbleeding is noted intraoperatively.

An ulnar artery aneurysm in the palm is treated by resection and ulnar nerve decompression in Guyon's canal. The artery can occasionally be repaired primarily, although a short-segment vein graft is usually required. There remains some controversy regarding the need for reconstruction of the ulnar artery in such cases. Those arguing against reconstruction point to reported cases of recurrent aneurysm formation at the repair sites, relatively poor patency rates of the reconstructed artery, and the fact that the benefit of reconstruction has not been clearly demonstrated. However, reconstruction of a short segment of the ulnar (and for that matter the radial) artery in the wrist or hand is nearly always justified.

Sympathectomy

Peripheral sympathectomy alone or in conjunction with arterial reconstruction may increase distal perfusion in the upper extremity. Removal of the direct sympathetic innervation of arterial smooth muscle results in diminution of vasoconstriction. In addition, peripheral sympathectomy appears to correct abnormal arteriovenous shunting, thus improving nutritional blood flow to the ischemic areas. Peripheral sympathectomy may be more beneficial than more centrally placed sympathectomy (cervicothoracic) for several reasons. By excising the sympathetic input at the end-organ, sympathetic fibers that bypass the sympathetic trunk are included. This technique lessens the opportunity for alternative pathways to affect end-organs and decreases receptor upregulation. It is also thought that direct elimination of the dense concentrations of sympathetic fibers at arterial bifurcations by adventitial excision improves the results of surgery. Sympathectomy has been shown to benefit patients with Raynaud's phenomenon, as well as those with multilevel unreconstructible occlusive disease.

The specific indications for and details of the surgical technique of peripheral sympathectomy are still evolving. Patients who have persistent vasospasm resulting in cold intolerance, pain, and ulceration, despite medical management and changes in social habits, are candidates for surgical sympathectomy.

Arterialization of the Venous System

Arterialization of the venous system is a procedure first proposed at the beginning of the last century as an alternative means of providing arterial blood flow to ischemic tissue. It is a salvage technique used when there is no way to restore antegrade arterial flow. In the upper extremity, the in situ technique is used to anastomose an artery to a vein on the dorsum of the distal end of the forearm or hand. Venous side branches are ligated to direct flow distally to the fingers, and valvulotomies are performed to overcome the valves to the level of the dorsum of the hand. More distally, the valves eventually become incompetent with resultant improved perfusion of the fingers. In addition, arterialization of the venous system appears to stimulate neovascularization. Patients with extensive necrosis or infection should not be treated by arterialization of the venous system because the initial postprocedure edema may cause worsening of necrosis and infection. The initial edema gradually resolves, and wounds heal with an impressive reduction in pain (Fig. 1).

Microvascular Transplantation

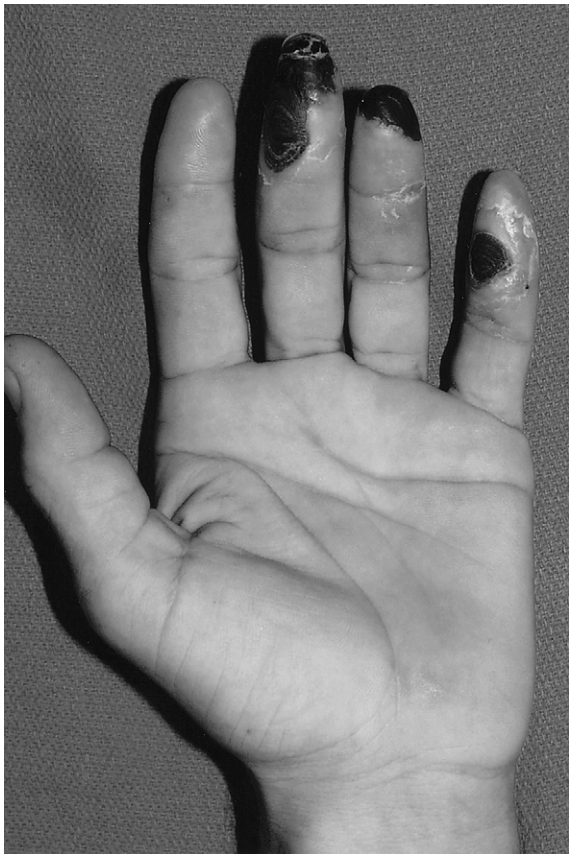
Microvascular tissue transplantation is another procedure that can improve the distal blood supply in patients in whom bypass or in situ vein grafting is not feasible. It is based on the observation that well-vascularized tissue results in improved perfusion of the surrounding tissue. Omental or thin pliable flaps have been reported to reduce pain and improve wound healing in the hand and permit tendon gliding. The flap is placed in skin pockets or covered with a split-thickness skin graft.

Revascularization of Amputated Parts

Acutely amputated digits or upper extremities are usually revascularized directly by repair or by reconstruction of the injured artery or arteries. As in reconstruction after thrombus removal, damaged arteries need to be débrided as far back as uninjured vessel. In an attempt to gain length, the artery may be mobilized from the surrounding tissue, reversed vein grafts may be used to bridge arterial segments, and arteries from unreplantable digits may be used as donor grafts. Heparinized saline catheter arterial shunts may be used in injuries involving a significant muscle component with a prolonged ischemia time.

Nonsurgical Treatment

A wide variety of pharmacologic agents are used to treat vascular insufficiency, including calcium channel blockers (nifedipine, verapamil, diltiazem), sympathicolytics (phentolamine, phenoxybenzamine, guanethidine), angiotensin-converting enzyme inhibitors (captopril), serotonin receptor antagonists (ketanserin), endogenous neuropeptide vasodilators (calcitonin gene–related neuropeptides), drugs altering prostaglandin metabolism (alprostadil), and rheologic agents (aspirin, pentoxifylline).



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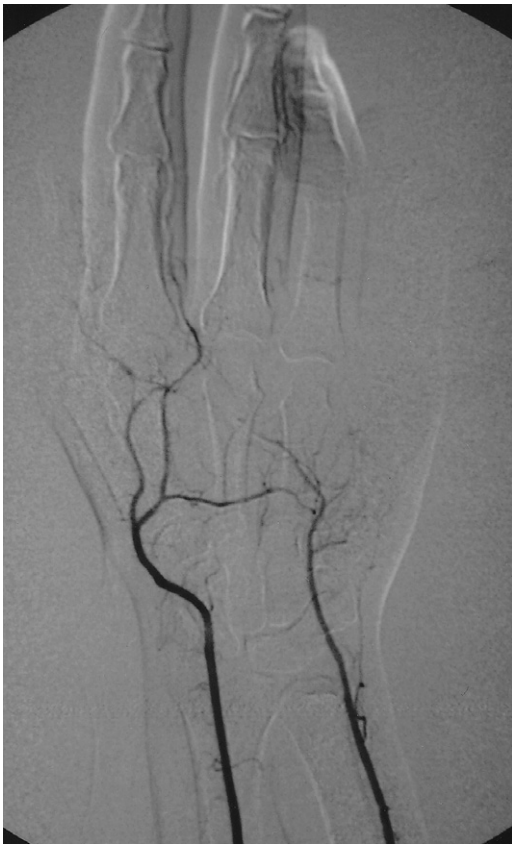

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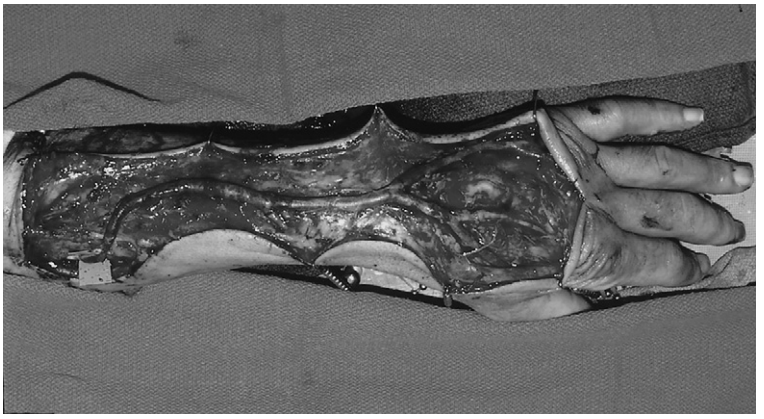

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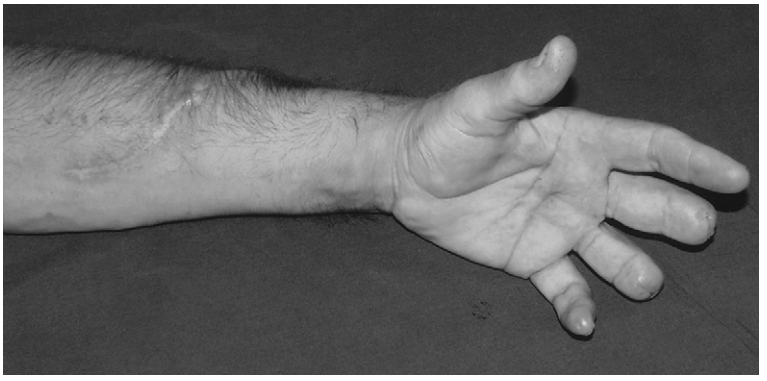

D

Figure 1 • A, The hand of a 39-year-old man with end-stage renal disease and long-standing insulin-dependent diabetes mellitus. The patient had necrosis of the fingers and severe pain. **B,** Arteriogram of the hand showing a lack of reconstructible digital vessels. **C,** Intraoperative view showing end-to-side anastomosis of the cephalic vein to the radial artery. **D,** The hand 2 months postoperatively. The wounds are healed with a marked reduction in pain.

Antidepressants (tricyclic antidepressants, selective serotonin reuptake inhibitors) have also been used.

The clinical efficacy of the aforementioned medications remains difficult to prove. Calcium channel blockers, specifically nifedipine, are the most commonly prescribed pharmacologic intervention for vasospastic disorders of the upper extremity. By reducing smooth muscle action, they decrease the vasoconstriction caused by sympathetic input. Nifedipine has also been shown to be efficacious treatment for patients with Raynaud's syndrome.

Care must be taken when deciding on the appropriate pharmacologic intervention. It is important to understand that many of the agents suggested for the management of vasospasm are not necessarily specifically approved for these disorders.

Biofeedback therapy depends on the ability of patients to voluntarily control their autonomic reflexes. Patients visualize and concentrate on improving blood flow and increasing the temperature of their hand or involved upper extremity.

Postoperative Care

The wide variety of diseases and treatments discussed require specific postoperative care. However, a few generalizations can be made. Anticoagulation is not routinely required after vascular reconstruction or microvascular transplantation. However, if thrombectomy is performed or if there is known or suspected clot in the vascular tree, a period of anticoagulation is usually indicated to minimize clot propagation. The period of anticoagulation required is somewhat arbitrary; if embolectomy is performed or if there is thrombus in smaller vessels, the patient is treated with warfarin (Coumadin) for 3 months.

Pearls and Pitfalls

- Vascular ischemia of the upper extremity lends itself poorly to shortcuts in treatment. Accurate diagnosis is the most expeditious way to determine a beneficial treatment plan.
- A combination of surgical approaches (vascular bypass or reconstruction plus sympathectomy, or both) is often indicated in patients with upper extremity vascular insufficiency.
- Incisions should be planned carefully, as this patient population has a high incidence of wound complications.

SUGGESTED READINGS

Flatt A: Digital artery sympathectomy. J Hand Surg 5:550, 1980.

Koman LA, Ruch DS, Smith BP, et al: Vascular disorders. In Green, DP, Hotchkiss RN, Pederson WC (eds): Green's Operative Hand Surgery, 4th ed. Philadelphia, Churchill Livingstone, 1999, pp 2254-2302.

Merritt W: Comprehensive management of Raynaud's syndrome. Clin Plast Surg 24:133, 1997.

Taylor RS, Belli AM, Jacob S: Distal venous arterialization for salvage of critically ischaemic inoperable limbs. Lancet 354:1962, 1999.

Troum S: Management of vasospastic disorders in the hand. Clin Plast Surg 24:121, 1997.

Congenital Anomalies of the Upper Extremity

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BRIAN LABOW

Treatment of congenital malformations of the upper limb provides challenge and opportunity for the reconstructive hand surgeon. The subtle differences in each affected hand and the broad spectrum of anomalies encountered defy standardized treatment protocols. Diagnosis is made more difficult by the small size and complexity of the pediatric hand. Experience in the field is cumulative; however, useful outcome information cannot be obtained until the child has approached skeletal maturity. This chapter highlights the recent technologic and surgical advancements that have contributed greatly to the treatment of these children.

Classification

Most early attempts at classifying congenital hand deformities were based on a descriptive approach and lacked an embryologic or anatomic basis. Although the embryology of the upper extremity has unfolded over the past century, proper classification systems and treatment schema have come into focus only since the 1960s. Unlike other branches of hand surgery, in which acquired deformities can be classified anatomically, congenital hand deformities may be categorized in a number of ways (Table 1). An ideal classification system should be simple and descriptive and permit easy recording and cross-specialty discussion. Unfortunately, any single classification system will be incomplete because the basic understanding of congenital limb anomalies is continuing to evolve at the cellular and molecular levels.

The classification system in place today is derived from classifications that grouped conditions based on the anatomic parts affected by a certain embryonic failure. Within the various categories, there are classification systems that have withstood the test of time because they have an effective impact on both communication among hand surgeons about each category and on treatment of individual anomalies. However, the large number of different classification systems makes diagnosis and treatment perplexing for the uninitiated (Table 2).

Polydactyly

Polydactyly, an excess of digits or parts, is the most common congenital anomaly of the upper limb (incidence of 2 to 19 per 10,000 live births). The anomaly may be radial (thumb), central (index, long, or ring rays), or ulnar (fifth ray). These terms are preferred over preaxial, central, and postaxial. In the white and Asian populations, radial polydactyly is most frequent (1 in 3000 live births). In African American and Native American populations, ulnar polydactyly predominates (13 in 1000 live births). Most polydactyly cases are not true duplications because the extra digit or digits are abnormal in both size and shape. The extra digit may have a skeletal connection to its partner at any level (distal, middle, or proximal phalangeal or metacarpal) or have none at all (floating or rudimentary); it usually has two or three phalangeal segments.

The Iowa classification system used by most surgeons reflects the variability (Table 3). Different types of thumb polydactyly are correlated by the level of branching from the skeletal axis and are classified I through VI. Rare or unusual types of duplications that include two or more metacarpals with and without triphalangeal components are placed in an additional category, group VII (Table 4). Although cumbersome, the system is logical and may be applied to other portions of the hand (Table 5).

TABLE 1 Modified Embryologic Classification

CATEGORY	SUBCATEGORY	EXAMPLE
Failure of formation	With regeneration	Phocomelia Radial and ulnar deficiencies Symbrachydactyly
	Without regeneration	Terminal transverse deficiency
Abnormal number of digits	Slight impairment of mesenchymal tissue	Simple syndactyly, polydactyly, thumb duplication, cleft hand (typical)
	Moderate to severe impairment	Complicated syndactyly, thumb duplication Congenital constriction band syndrome
Generalized skeletal abnormities	—	Multiple hereditary exostosis, dwarfism
Failure of differentiation	—	Radioulnar synostosis, metacarpal synostosis
Overgrowth or undergrowth	—	Macrodactyly, limb hypertrophy

TABLE 2 Genetic Classification of Congenital Hand Anomalies

MOLECULAR	SYNDROME	LIMB DEFECT	GENE
Transcription factor	Holt-Oram	Radial deficiency	*TBX5*
	Synpolydactyly	Syndactyly, brachydactyly	*HOXD13*
	Townes-Brock	Polydactyly	*SALL1*
	Hand-foot-genital	Brachydactyly	*TWIST*
	Pallister-Hall	Ulnar deficiency, polydactyly	*GL13*
	Greig	Syndactyly, polydactyly	*GL13*
Signaling protein	Grebe	Brachydactyly	*CDMP1*
	Hunter-Thompson	Brachydactyly	*CDMP1*
	Aarskog	Brachydactyly	*FGD1*
Receptor protein	Apert	Brachysyndactyly	*FGFR2*
	Pfeiffer	Syndactyly, brachydactyly	*FGFR2*
Unknown	Split hand/foot	Syndactyly, clefting	—
	Nager	Posterior limb deficiency	—
	Tarsal-carpal	Brachydactyly, fusions	—

The cause of polydactyly has not yet been defined. Current theories suggest an imbalance between mitotic activity and apoptosis within the apical ectodermal ridge and underlying mesoderm. Animal models exist in which maternal rats exposed to cytosine arabinoside during early pregnancy frequently give birth to offspring with radial polydactyly. A gene responsible for a certain type of radial polydactyly with a triphalangeal thumb has been localized to chromosome 7q36.

Treatment of polydactyly varies from simple to complex, depending on the location and the degree

TABLE 3 Iowa Classification of Thumb Polydactyly

TYPE	LEVEL OF POLYDACTYLY
I	Distal phalanx
II	Interphalangeal joint
III	Proximal phalanx
IV	Metacarpophalangeal joint
V	Metacarpal
VI	Carpometacarpal joint
VII	Various forms of polydactyly at the carpometacarpal joint, including triphalangeal components and thumbs at any level II through VI

TABLE 4 Classification of Triphalangeal Thumb

TYPE	CLINICAL FEATURES
I	Rudimentary phalanx. Thumb length is normal with minor ulnar deviation
II	Short triangular middle phalanx. The thumb is longer with ulnar (occasionally radial) deviation
III	Trapezoidal middle phalanx. Thumb length is longer, and the metacarpal may contain two epiphyses. The thumb is often supinated and adducted
IV	Long rectangular middle phalanx. The thumb is longer. Also called the five-fingered hand

TABLE 5 Classification of Ulnar Polydactyly

TYPE	CLINICAL CHARACTERISTICS
I	Soft tissue mass only; no skeletal connection
II	A digit containing normal structures articulating to a fifth finger
III	Complete digit with a metacarpal

of skeletal involvement. The principles of treatment have not changed in 20 years, and all revolve around creation of the best digit from the available parts (Fig. 1). In radial polydactyly, duplications of the distal phalanx (Iowa types I and II) are easiest to treat. The radial distal phalanx is trimmed in conjunction with reconstruction of the collateral ligament and soft tissue and eponychial contouring. Most surgeons perform the skeletal and soft tissue corrections simultaneously at or before 1 year of age.

Proximal phalangeal duplications (Iowa types III and IV) are the most common, and treatment goals are similar. Additional pitfalls that exist with these types of duplications include interphalangeal (IP) and metacarpophalangeal (MP) joint stiffness, proximal phalangeal angulation, and limited thumb extension secondary to the checkrein effect of scarring. Meticulous soft tissue dissection is essential for preservation of a periosteal sleeve for reconstruction of the collateral ligament and prevention of adherence of the extensor mechanism to bone. A closing wedge osteotomy is safer and heals more predictably than does an opening wedge osteotomy with a bone graft.

Duplications at the metacarpal level (Iowa types V, VI, and VII) are highly variable and should be considered one tissue at a time. Problems with repair are similar to those of type IV corrections, but only more acute. The difficulty of reconstruction and the potential for untoward results are directly proportional to the level of the duplication, with diffi-

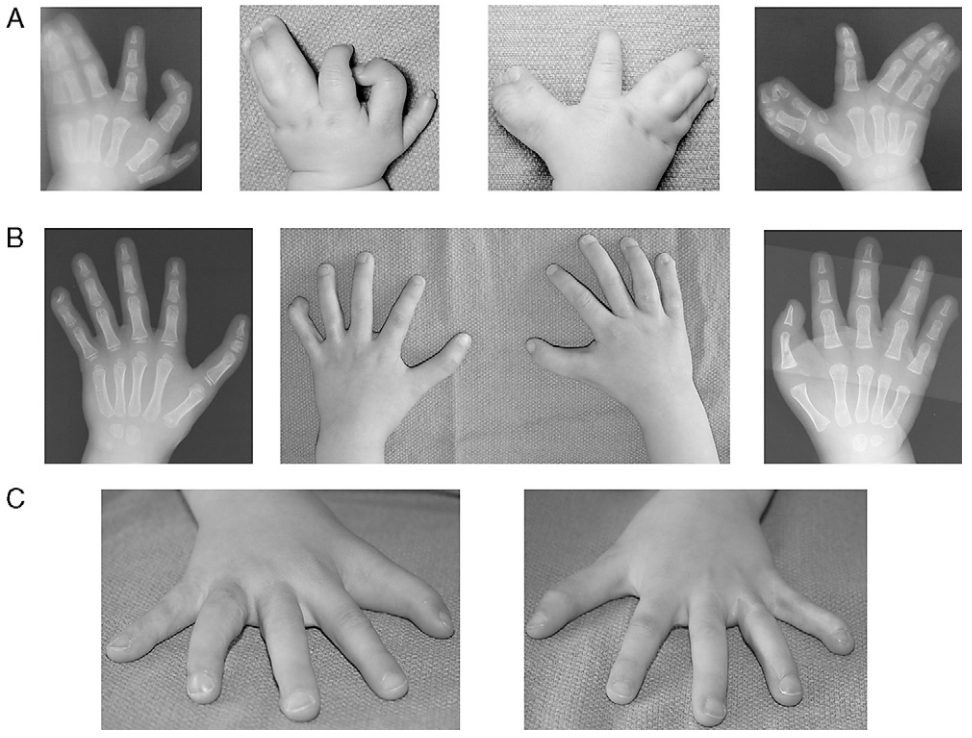

Figure 1 • Polydactyly and syndactyly, early aggressive surgery. **A,** Child with bilateral deformities. The left hand has complete simple syndactyly of the ulnar three digits, a normal index finger, and a triphalangeal ulnar thumb with a biphalangeal radial thumb. The right hand has a similar syndactyly, a normal index digit, and a more complicated thumb polydactyly. Note the dorsal buckling of both ring proximal interphalangeal joints by 6 months of age. **B,** Early release of all webbing plus correction of both thumbs was accomplished at 10 months of age. The left thumb was reconstructed with a closing wedge osteotomy of the central phalanx and excision of the radial aspect of the thumb with transfer of the thenar muscles. On the right side, the central thumb ray was retained and the distal portion of the ulnar portion transposed on top of the proximal phalanx of the central thumb. **C,** The postoperative appearance at 3 years, with no revisions. The intradigital web spaces and commissure flaps are well maintained, and the contour and pigmentation match of the skin grafts are excellent. The clinodactyly will be corrected when the child is older.

culty increasing as the duplications proceed distally to proximally. The constructed thumbs are never normal, and with growth, both soft tissue and skeletal corrections are often needed. The first web space is frequently deficient and should be addressed at the initial reconstruction. In triphalangeal thumb duplications, the goal remains to make the best thumb from all available parts. In over half of type VII cases, the triphalangeal thumb or a part thereof is discarded. If both thumbs are equal, the partner with the strong ulnar collateral ligament at the MP joint is preserved. On occasion, the triphalangeal thumb is well aligned and stable and the biphalangeal thumb is deviated. In such cases, the triphalangeal thumb is retained.

Syndactyly

Syndactyly, one of the most common congenital hand anomalies, can occur sporadically or may be inherited. Furthermore, the condition may be an isolated finding or can occur as part of a syndrome. The current understanding of the embryologic origin of syndactyly suggests that most digital fusions occur as a result of failure of apoptosis within the developing limb bud between the digital rays. However, syndactyly can also result from extrinsic forces, as seen in constriction ring syndrome.

The classification of syndactyly includes complete, incomplete, simple, and complex categories (Table 6). In addition, the term *complicated* has been added to denote hands with disorganized and abnormal neural, muscular, vascular, or skeletal elements, as seen with polysyndactyly or acrosyndactyly. In such cases, a separate classification schema may be used.

Many techniques have been described for repair of syndactyly. There has been a movement away from dorsal triangular and rectangular flaps toward the use of dorsal island flaps. There is no single best technique. Although new techniques are continually emerging, the principles of repair are well established and include the following:

TABLE 6 Classification of Syndactyly

TYPE	CLINICAL CHARACTERISTICS
Simple	Soft tissue webbing only
Incomplete	Does not extend to fingertip
Complete	Extends to tip of involved digits
Complex	Skeletal fusion, extra skeletal parts
Complicated	Skeletal fusions plus extra delta phalanges, transverse phalanges, soft tissue interconnections, abnormal extrinsic tendons, abnormal intrinsic insertions

- Lining the commissure with full-thickness flaps
- Palmar zigzag incisions
- Use of full-thickness skin grafts
- Equal distribution of grafts between digits
- Early skeletal correction without physical injury
- Meticulous surgical technique
- Secure postoperative immobilization

The surgeon should be comfortable with one or two methods and learn to use them well (Fig. 1). Several technical points are emphasized. Regardless of the exact series of incisions planned, separation of the two digits should include isolation and excision of the intradigital fibrous band or bands that are characteristic of all types of syndactyly. Identification of the neurovascular structures is mandatory. Although the digital nerve bifurcation may be deepened by neurolysis, the arterial bifurcation should preferably be preserved. Dorsal flap design must be sufficient to establish a commissure. One recent innovation describes an island transfer of dorsal skin into the commissure, as opposed to the traditional pedicled rectangular or hourglass-shaped flap. Numerous alternative donor sites for skin grafting have appeared in the literature. Although preputial skin has garnered much attention, the authors believe that this skin is prone to infection and hyperpigmentation. In syndactyly conditions that require larger amounts of skin to correct, such as the Apert hand or mitten hand, a transversely oriented graft may be harvested from the lower abdominal fold. This site provides adequate skin of satisfactory quality and color match without much potential hair.

Postoperative management with an emphasis placed on adequate immobilization to prevent skin graft shear and wound separation remains an important aspect of treatment. Graft loss and other wound complications that lead to healing by secondary intention produce contractures and contribute significantly to web creep. To prevent this complication, an above-elbow cast is worn for 3 weeks. At that time, a custom interdigital splint is fabricated and worn for another 3 weeks. Outcome studies are variable and usually incomplete. Recurrent syndactyly or "web creep" occurs in at least 15% to 20% of patients who are monitored through adolescence. Some recurrence is caused by epithelialization of open wounds, usually as a result of inadequate postoperative immobilization. Other cases are secondary to the scar tissue not expanding as much as the other tissue as the child grows.

In addition to the typical syndactyly patients who have single–web space involvement, there are several classes of patients with complicated syndromic syndactyly. Treatment of the Apert hand and

those with complex central synpolydactyly remains challenging. The Apert hand is subclassified by the degree of complexity. Separation of rays plus skeletal correction within the first year of life is advocated. Emphasis is placed on thumb lengthening, creation of an adequate first web space, and excision of the metacarpal synostosis so that the fifth ray may oppose the thumb for pinch and gripping functions. At present, there is no solution to enable motion at the site of phalangeal fusion (symphalangism) within the central three rays of the hand. Revisions are often necessary but well appreciated by patients, who function well with their short digits and thumbs.

Central synpolydactyly is fortunately rare but, when present, has a high degree of penetrance within family pedigrees. The goal of creating central digits of adequate length that are neither rotated nor angulated is often foiled by the abnormal and asymmetric growth of the skeletal parts. Secondary revisions are frequently required as the children grow into adults.

Vascular Malformations

Diagnosis as well as treatment of vascular anomalies of the upper extremity is no longer a "black hole" within hand surgery. The classification system proposed by Mulliken and Glowacki almost 2 decades ago has categorized this disparate group of anomalies and provided a clear explanation of our current knowledge and treatment. The anomalies are now classified as either (1) hemangiomas, lesions that involute within 7 years of life, or (2) malformations, lesions that do not involute. The endothelial cell of origin further identifies the malformations: capillary (C), venous (V), lymphatic (L), arteriovenous (AV), and arterial (A). In addition, the lesions are designated as slow flow, typically venous malformations (VMs), lymphatic malformations (LMs), or lymphaticovenous malformations (LVMs), or fast flow when they have an arterial component, typically arteriovenous fistulas (AVFs) or shunts. This system is clinically applicable and makes treatment more rational and less complex.

Upper extremity lesions are much less common than those occurring in the head and neck and lower extremity regions. Surgery for rapidly enlarging hemangiomas is rarely indicated. Large lesions, which typically involve the axilla, thorax, and head and neck regions, respond 80% of the time to steroids. Two thirds of nonresponders regress with the administration of interferon alfa. At present, there is no predictable pharmacologic treatment of malformations.

Treatment of vascular malformations has recently been clarified. Surgical treatment is predictable in all slow-flow groups. Surgical debulking is most commonly performed in VMs, LMs, or mixed lymphaticovenous (LVM/capillary lymphatic venous malformation [CLVM]) malformations. Optimal outcomes occur in those whose malformation is confined to the subcutaneous and fascial tissue planes and the worst in those with diffuse muscular involvement. Major short-term problems are related to hematoma formation and skin flap viability. Long-term adverse results are caused by neuroma formation, scar contracture after extensive dissection, and persistent malformation. The most difficult group of patients to treat is those with fast-flow lesions and multiple AVFs. More than half of operative procedures are preceded by either sclerotherapy (slow flow) or embolotherapy (fast flow) by an interventional radiologist.

Radial Clubhand (Radial Deficiency)

Longitudinal failure of formation within the upper limb is subclassified as radial, central, and ulnar (Tables 7 to 10). Descriptive synonyms are radial clubhand, cleft hand, and ulnar hemimelia. Radial anomalies are seen most frequently. The more comprehensive term "radial deficiency" should be used because there are usually associated abnormalities of the wrist and thumb. In fact, the proximal portion of the limb is also deficient in more severely affected limbs. The true incidence of longitudinal deficiencies is not known, but a range of 1 to 3 per 100,000 live births is cited in the literature. Most radial clubhands occur sporadically, and less than a fourth of affected children have a syndromic association such

TABLE 7 Classification of Radial Deficiency

TYPE	RADIOGRAPHIC FINDINGS	CLINICAL FEATURES
I (short radius)	Distal growth plate present but delayed and narrow. Mild radial shortening without bowing. Thumb present.	Minor deviation
II (radius in miniature)	Both distal and proximal growth plates present but abnormal growth of both. Ulna thickened and bowed.	Moderate deviation
III (partial)	Partial absence of the radius: hypoplasia; distal > middle > proximal ulna, which is short, thick, bowed. Wrist unsupported.	Moderate to severe deviation
IV (absence)	No radius. Ulna thick, short and bowed. Most common type.	Severe deviation, often dislocated

TABLE 8 Modified Classification of the Entire Spectrum of Radial Longitudinal Deficiencies

TYPE	THUMB	CARPAL	DISTAL RADIUS	PROXIMAL RADIUS
N	Absent or hypoplastic	Normal	Normal	Normal
0	Absent or hypoplastic	Absent, hypoplasia	Normal	Normal, radioulnar synostosis, radial head dislocation
1	Absent or hypoplastic	Absent, hypoplasia, coalition	>2.0 mm shorter than ulna	Normal, radioulnar synostosis, radial head dislocation
2	Absent or hypoplastic	Absent, hypoplasia, coalition	Hypoplasia	Hypoplasia
3	Absent or hypoplastic	Absent, hypoplasia, coalition	Physis absent	Variable hypoplasia
4	Absent or hypoplastic	Absent, hypoplasia, coalition	Absent	Absent

as VACTERL (vertebral, anal, cardiac, tracheoesophageal, renal, and limb anomalies), Holt-Oram syndrome (congenital heart and limb deficiency), TAR (thrombocytopenia and absent radius), phocomelia (commonly associated with thalidomide ingestion), and Fanconi's anemia (pancytopenia).

The degree of deformity varies, and the same classification has been used for 40 years. In general, the amount of bone (radius) missing corresponds directly with the soft tissue deficiencies. The degree of radial deviation of the hand and carpus is also associated with palmar subluxation extending to dislocation. The hypoplastic or aplastic extensor muscles are weaker than the flexors, which course radial to the central axis of rotation of the wrist. Consequently, finger flexion is accompanied by wrist radial deviation and flexion. Restoration of the muscular imbalance is one of the goals of treatment.

The large number of recommended procedures for correction of radial clubhand is a testimony to the difficulty associated with this problem. One of the reasons for poor outcomes is that only skeletal solutions were considered and the effect of soft tissues was ignored. The hand and carpus were positioned on top of the ulna, and carpectomies were performed, even if there was insufficient room during the "centralization" procedures. A major concern was placed on not damaging the blood supply to the distal ulnar epiphysis. Subsequently, "radialization" procedures placed the ulna beneath the radial carpal bones and included tendon transfers to counter the radial pull of the strong digital flexors. The extensor carpi ulnaris is shortened, and the flexor carpi ulnaris with or without detached radial wrist flexors (if present and mobile) is transferred to the extensor. Mobility is greater because no carpal bones need to be excised. This positioning is also facilitated by preoperative distraction, which stretches soft tissue sufficiently to allow precise positioning (Figs. 2 and 3). Most experienced surgeons now favor early radialization within the first year of life for a radial clubhand (Fig. 4). Vascularized second toe metatarsophalangeal joint transfer to the radial side of the hand and carpus is a creative solution (Fig. 5).

The outcome of the aforementioned procedures is always suboptimal. Motion is limited, strength is diminished, and recurrent radial deviation with or without flexion is common, especially in patients without a radius and with poor-quality soft tissues. Thumb pollicization always enhances function and appearance. As preteens or teenagers, most youngsters request distraction lengthening, which is both predictable and effective. Lengthening of the forearm is best performed as a two-stage process: (1) application of a distraction device and osteotomy followed by (2) removal of pins, intercalary bone grafting, and internal plate and screw fixation (see Fig. 3).

TABLE 9 Classification of Ulnar Deficiencies

TYPE	GRADE	CLINICAL CHARACTERISTICS
I	Hypoplasia	Growth plates present, minimal shortening, growth deficient
II	Partial aplasia	Absence of distal or middle third of ulna
III	Aplasia	Complete absence of ulna
IV	Synostosis	Absence of ulna, humeroradial fusion

TABLE 10 Classification of Ulnar Deficiencies by First Web Space

TYPE	GRADE	CLINICAL CHARACTERISTICS
A	Normal	Normal first web space and thumb
B	Mild	Mild first web space deficiency, mild thumb hypoplasia, normal extrinsic tendons
C	Moderate to severe	Moderate to severe first web space deficiency, thumb in same plane as the hand, thenar muscles hypoplastic, extrinsic tendons abnormal
D	Absent	Aplasia of the thumb

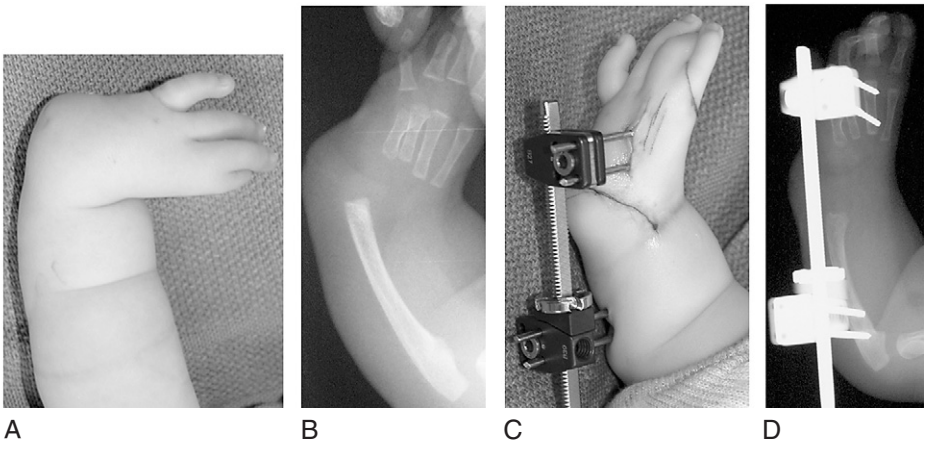

Figure 2 • Distraction techniques and radial dysplasia. As a technique, distraction lengthening has been useful in patients with partial or complete radial agenesis. **A,** At birth, the left hand and carpus are dislocated relative to the distal end of the ulna. Type IV arms are the most difficult to treat. **B,** The radiograph was taken with maximal longitudinal traction on the hand after 6 weeks of aggressive stretching and splinting by the family and occupational therapists. **C,** In a separate surgical procedure before radialization, the tight radial soft tissues were released and a distraction apparatus applied. **D,** Within 3 weeks sufficient length was achieved to permit easy repositioning of the hand and wrist over the distal part of the ulna.

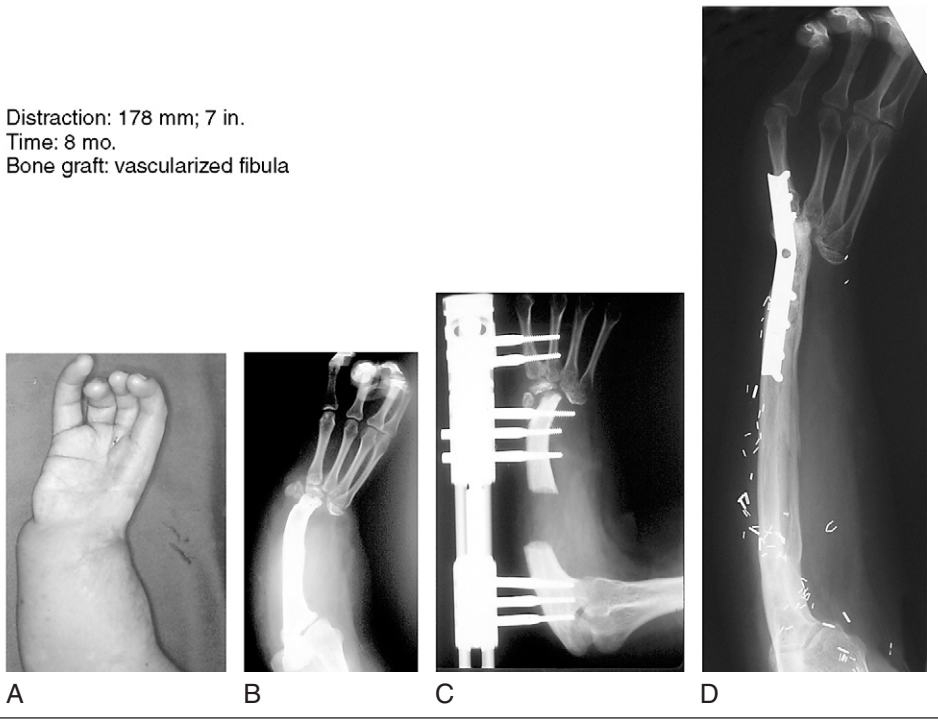

Figure 3 • **A,** A patient with radial agenesis treated by centralization at 5 years of age. **B,** At 28 years, he requested a longer forearm. He has a functional hand with very limited wrist motion. **C,** The uniplanar external fixation apparatus must stabilize the hand as well as the ulna on both sides of the osteotomy. It is often impractical to expect these large gaps, 178 mm in this case, to heal by "callostasis" alone. **D,** A vascularized fibular graft with internal fixation allowed much earlier removal of the apparatus and return to normal function.

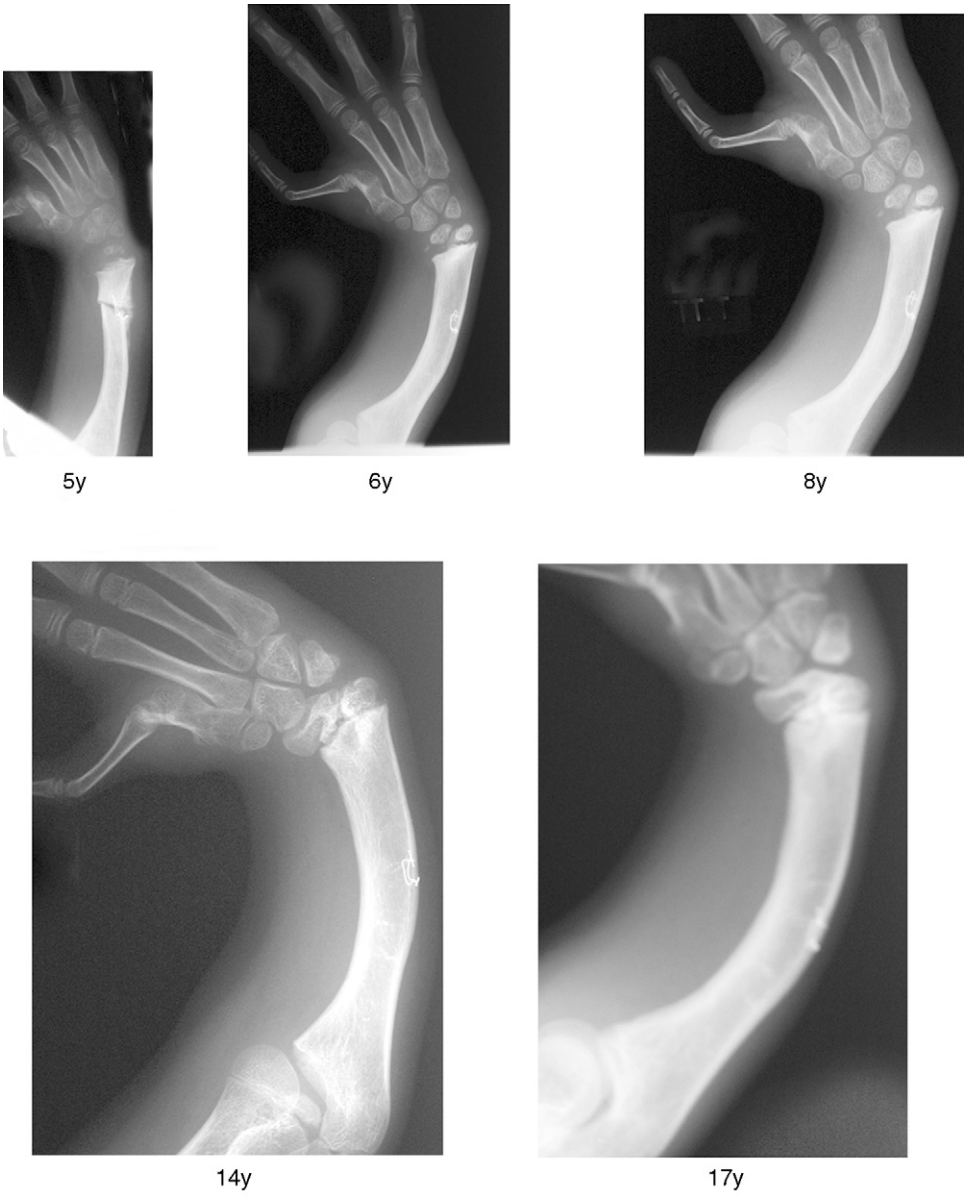

Figure 4 • Growth of the radial clubhand. Within the first year of life this child, born with complete agenesis of the radius, underwent centralization of the hand and wrist over the distal end of the ulna without carpal bone excision. Pollicization of the index finger was completed on the same hand 1 year later. At 5 years, 6 months of age, osteotomy of the distal end of the radius was performed to correct bowing and clubhand posturing. The interosseous wire used to secure the closing wedge osteotomy can be used to document longitudinal growth of both the proximal and distal portions of the ulna over a 12-year period. Note the persistent ulnar bowing that has occurred with time and widening of the distal end of the ulna, which at skeletal maturity looks more like a radius than an ulna. With time and growth, the hand has moved into more radial deviation and slight flexion. The muscular imbalance caused by the strong pull of the extrinsic flexors is responsible for the persistent distortion with growth.

Constriction Ring Syndrome

Constriction ring (amniotic band) syndrome was described by Hippocrates, but its cause is still debated. It is believed to result from entanglement of the digits or more proximal portions of the extremity in fibrous bands that have sheared from the amniotic sac in utero. These cases occur sporadically (1 in 15,000 live births), and there is no evidence of genetic involvement.

Both the upper and lower extremities may be involved. The findings in two extremities are never exactly the same, as is often seen in genetic hand

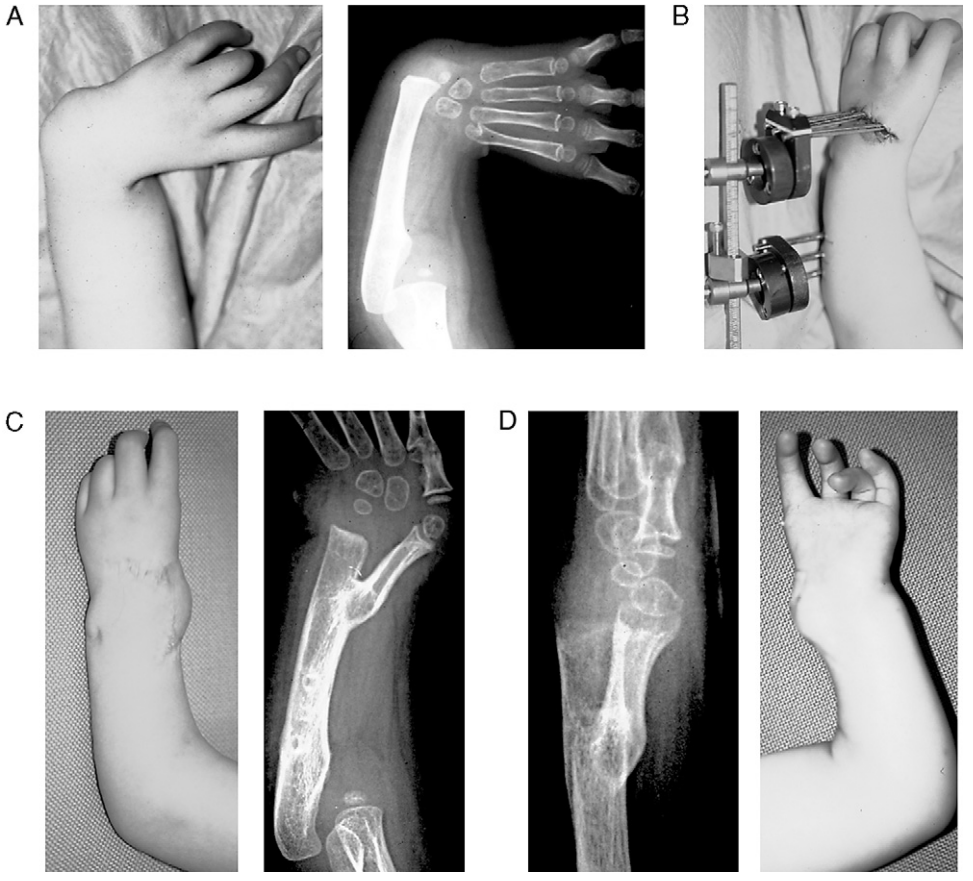

Figure 5 • Second toe transfer for radial clubhand. **A,** Clinical appearance and radiograph of a type IV deformity with complete absence of the radius. **B,** Preliminary distraction is used to position the hand and carpus over the distal end of the ulna. **C,** A microvascular second toe transfer with an intact metatarsophalangeal joint and overlying soft tissue provides radial stability to the hand. As growth occurs at the distal end of the ulna, the radial buttress will grow at both the metatarsal head and the proximal phalangeal growth center. **D,** The appearance is shown 3 years after transfer. Hand function has not changed, and the neutral position has been maintained. (Courtesy of Simo Vilkki, MD, of Iceland.)

anomalies. Although the digits, in particular the long finger, are most frequently involved, constriction rings may affect the wrist, forearm, and arm as well. In addition, the depth and circumferential extent of the lesion may vary. In mild cases, there may a slight indention on one side of the digit producing minimal edema distal to the lesion. In more severe cases, the distal portion of the digit may be severely congested because of impeded lymphatic and venous drainage. In severe cases, fusion of multiple digits may occur and lead to mild or severe acrosyndactyly. For others, autoamputation may have occurred in utero. In all cases, structures proximal to the constriction ring are normal. Most series quote a 40% to 60% incidence of associated malformations.

Treatment has changed little over the past decade. Although superficial rings producing little constriction may not need therapy, most patients with distal swelling require release. Even in relatively mild cases, simple excision of the ring is inadequate because the resulting circumferential scar contracts and reproduces the lesion, albeit in a milder form. Instead, lateral Z-plasties on either side of the digit with a straight-line closure over the more visible dorsal and volar surfaces are recommended. The skin and subcutaneous tissue should be raised separately to allow excision of excess fat and facilitate contouring of the digit. Care must be taken when excising the ring to preserve the digital nerve branches, which may lie in close proximity to a deep constriction ring. Although many authors have advocated circumferential release of the digits, caution must be exercised, especially in correction of deep bands.

In more severe cases in which multiple digits are fused, early release, especially of the border digits, is mandatory to avoid buckling or other growth disturbances. As with other complex types of syndactyly, it is preferable to have all digits liberated by 1 year of age. In the case of severe proximal con-

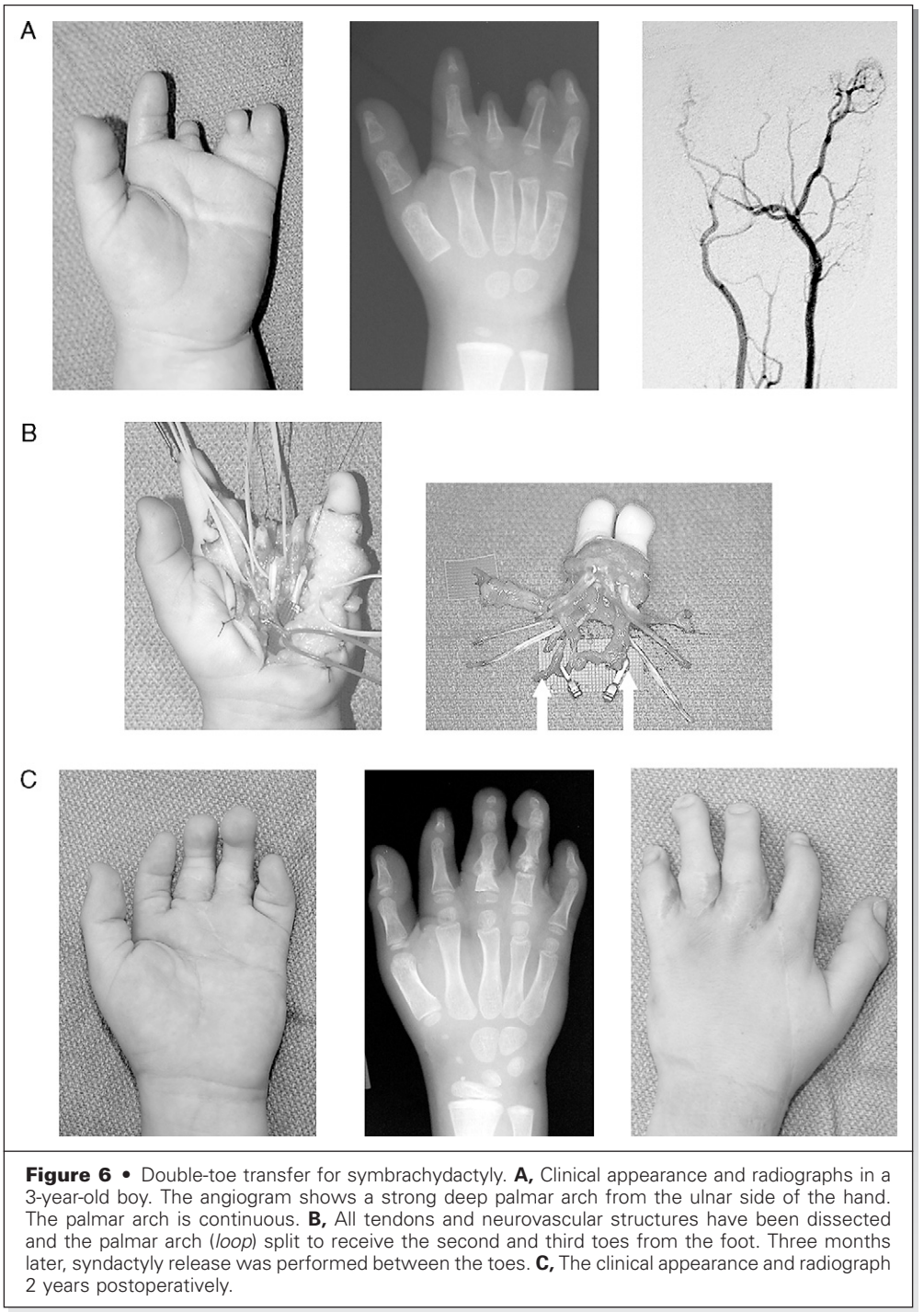

Figure 6 • Double-toe transfer for symbrachydactyly. **A,** Clinical appearance and radiographs in a 3-year-old boy. The angiogram shows a strong deep palmar arch from the ulnar side of the hand. The palmar arch is continuous. **B,** All tendons and neurovascular structures have been dissected and the palmar arch (*loop*) split to receive the second and third toes from the foot. Three months later, syndactyly release was performed between the toes. **C,** The clinical appearance and radiograph 2 years postoperatively.

striction bands or autoamputated digits, single or multiple toe transfers have also been described.

Microvascular toe transfers for congenital amputations seen in the constriction ring syndrome have greatly improved the treatment of these children. The normal anatomy proximal to the site of the constriction ring or amputation makes such transfers technically easier to plan and the outcomes more predictable. Sensation is normal, and extrinsic flexor and extensor motion is better in these patients than in those with transfers for other congenital deformities (Fig. 6).

Thumb Hypoplasia/Aplasia

A hypoplastic thumb is relatively common and accounts for anywhere from 11% to 37% of all congenital hand anomalies. Although it can occur in iso-

lation, thumb hypoplasia is found in a wide variety of syndromes (e.g., Holt-Oram) and is associated with other malformations (e.g., VACTERL). In the past, discussion of the hypoplastic thumb was based simply on radiographic evaluation of the osteoarticular column. However, such variables as size, position, relationship to the other digits, osseous components, joint integrity and stability, intrinsic and extrinsic musculature, and first web space depth and breadth should also be considered. Hypoplasia exists when any structure that contributes to a normal thumb is deficient or absent. All skeletal and soft tissues are absent in an aplastic thumb.

The classification system (Table 11) contains five types with one subdivision (types IIIA and IIIB). Understanding the anatomic differences that distinguish the various types of hypoplasia and their relationship to the carpus and forearm is fundamental to the discussion of treatment.

Type I thumbs are the most minimally affected and have a slightly shorter, more slender appearance than a "normal thumb." The thenar musculature is present, but hypoplastic. The first web space may be narrowed.

Type II thumbs are moderately hypoplastic with skeletal deficiencies involving the phalanges, metacarpal, trapezium, trapezoid, scaphoid, and to a lesser extent, the lunate. The first web space is narrow, and the thumb is held in an adducted position. The ulnar collateral ligament of the MP joint is lax and the thenar muscles underdeveloped or absent. Moreover, numerous muscular and tendon anomalies may be found with type II thumbs (as well as type III thumbs). In addition to abnormal posture and position, absence of IP and MP flexion or extension creases is an important clue to these abnormalities.

TABLE 11 Classification of Thumb Deficiency

TYPE	ANATOMIC FINDINGS
I	Minor hypoplasia, intact skeleton, good thenar muscles
II	Skeletal hypoplasia, thenar hypoplasia, collateral ligament laxity, deficient first web space
III	Same as type II plus greater skeletal hypoplasia, extrinsic muscle/tendon deficiency, greater thenar deficiency
A	Intact CMC joint
B	Absent CMC joint with deficient thumb metacarpal
IV	Floating thumb ("pouce flottant") with no skeletal connection
V	Absence of thumb

CMC, carpometacarpal.

Type III thumbs are severely hypoplastic. Skeletal shortening and narrowing are more pronounced, and the hand and wrist may be deviated as a result of hypoplasia or aplasia of the radial carpus. In fact, the distal end of the radius itself may be smaller and the radial styloid absent. This group is subdivided into type IIIA thumbs, which contain a complete metacarpal and an intact carpometacarpal (CMC) joint, and type IIIB thumbs, which have a tapered metacarpal but no CMC joint. In both groups, the thenar musculature is severely hypoplastic or absent. The MP joint is quite lax, with either the collateral ligament or the volar plate missing. A wide variety of musculotendinous and skeletal defects exist—abnormal anatomy is the rule. One common finding is "pollex abductus," in which the thumb is deviated away from the palm at the MP joint level. The anomaly is a result of abnormal intertendinous connections between the extrinsic flexor and extensor in conjunction with a weak ulnar collateral ligament. In addition, the motor branch of the median nerve may be absent and only one neurovascular bundle may be present.

Type IV thumbs are often referred to as "floating" thumbs or "pouce flottant." These thumbs have a small nail plate and contain two small phalanges; the latter are connected to the hand by a narrow skin stalk containing a neurovascular pedicle.

Type V thumbs are completely absent, and roughly half have a deficient radius as well. In fact, radial dysplasia may occur in conjunction with any of the aforementioned thumb types. The entire family of radial underdevelopment should be viewed as a spectrum rather than a series of unique entities.

Treatment options are numerous and vary with the extent of hypoplasia. However, a number of specific goals should be kept in mind:

- A mobile, stable CMC joint with an intact metacarpal
- A supple first web space of adequate dimension
- Mobility in at least two of the three joints (CMC, MP, IP)
- A stable MP joint
- Adequate motors for strong MP or IP flexion and extension
- The capacity to be placed in a palmar abducted position for pinch and grasp

Most type I patients do not require surgical correction, and indeed they (and their parents) may be unaware of the finding. When surgical correction is necessary, a first web space release that includes a four-flap Z-plasty, in addition to fascial release from the underlying musculature, is preferred. In type II patients, release of the first web space is usually accompanied by a stabilization procedure for the MP joint with or without a tendon transfer to assist in palmar abduction or opposition. Management of

TABLE 12 Cleft Hand Classification

CLINICAL FINDINGS	TYPICAL CLEFT	ATYPICAL CLEFT "SYMBRACHYDACTYLY"
Involvement	Bilateral, hands/feet	Unilateral, hand only
Inheritance	Familial	Spontaneous
Syndactyly	Common	None
Polydactyly	Present	Absent
Cleft configuration	"V shaped"	"U shaped"
Anatomic findings:		
Arterial	3 arteries to the ring finger	Minimal vessels to the central 3 digits
Tendon	2 or more flexors	Vestigial tendon to the ring finger
Skeletal	Hypertrophy adjacent	Hypoplasia to cleft

type III thumbs depends greatly on the presence or absence of a CMC joint (i.e., IIIA or IIIB). In type IIIA patients, all of the aforementioned maneuvers (first web space deepening, MP joint stabilization, and opponensplasty) are generally carried out in one stage. The major variable becomes the status of the flexor mechanism, which may require a staged approach. Surgical correction of type IIIB thumbs is controversial. Some surgeons (and parents) wish to preserve the severely hypoplastic thumb even at the expense of function. In such cases, staged reconstruction involving a vascularized joint transfer, such as the second metatarsal from the foot, has been used to establish a neo-CMC joint. The authors believe that pollicization is the treatment of choice because of its superior functional result. In type IV and type V reconstructions, pollicization is the treatment of choice.

The timing of treatment may be controversial. In the absence of other organ system complications, the authors prefer to address the hypoplastic thumb between 10 and 18 months of age. In cases of radial dysplasia, pollicization at 1 year is preceded by wrist centralization at 5 to 8 months. All treatment plans must be individualized and must take into account the experience of the surgeon, the parents' desires, and the overall condition of the child.

Cleft Hand

After much debate, distinction has been made between a typical cleft hand and an atypical cleft hand, now termed symbrachydactyly. The typical cleft is distinguished by a V-shaped central cleft, syndactyly, polydactyly, and bilateral hand and often foot involvement. Treatment frequently involves no surgery in patients with the most bizarre malformations because these children adapt so ingeniously. For most patients, however, surgery is required for separation of digital webbing, release of flexion contractures, removal or repositioning of extra parts, and especially repositioning of the index ray, which is often in a "no man's land" between a mobile thumb and somewhat functional ring and small fingers. Incisions for the digital transpositions are carefully designed for the creation of an adequate first web space lined with full-thickness flap tissue. Preservation of an adductor pollicis originating from a third metacarpal is crucial for pinch strength. Analysis of outcomes in these children and adults indicates that the hands are functional but pinch and grip strength are always deficient.

The broad category of symbrachydactyly ("together," "short," "digit") includes a teratologic spectrum between hands with a metacarpal and three phalanges smaller than normal and hands with minimal metacarpals and no phalanges or digits (Tables 12 to 14). The distinguishing characteristics include unilateral involvement, absence of lower extremity involvement, a "U-shaped" cleft (if present), and the presence in most hands of a digital remnant with a nail. Treatment of each hand must be individualized for the particular deficiency. One of the most common manifestations is a hypoplastic hand with a small but mobile and functional thumb, a satisfactory first web space, and four soft tissue remnants representing digits located distal to the four metacarpals. Diminutive extrinsic flexor and extensor tendons cause the nubbins to retract and move. Most parents refuse vascularized toe transfers for their children but consent to nonvascular-

TABLE 13 Classification of Symbrachydactyly

1. Short finger type	Short phalanges, including reduction up to aplasia of one or more phalanges, combined with simple syndactyly
2. Cleft hand type	One or more medial fingers missing
3. Monodactyly type	Thumb present and all digits as vestigial nubbins with nails
4. Peromelic type	Hand stump with or without small humps representing digits

TABLE 14 Modified Classification of Symbrachydactyly

1. Triphalangia type	Hand has no missing bones, digits have three phalanges, middle phalanx may be small
2. Diphalangia type	Hand with one phalanx (usually middle) missing in one or more digits
3. Monophalangia type	Hand with one or more digits with all three phalanges missing
4. Aphalangia type	Hand with all three phalanges missing in one or more digits
5. Ametacarpia type	Hand with missing metacarpal and all three phalanges in one or more rays
6. Acarpia type	Hand with absence of all digits and partial or complete absence of carpals
7. Forearm amputation	Arm with absence of the distal portion of the forearm; rudimentary nubbins may represent digits

ized phalangeal transfers if the soft tissue permits. Long-term outcomes are best if the transfers are performed before 12 to 16 months of age and if the periosteum is intact and the collateral ligaments and volar plate are attached to the supporting metacarpal. The bones grow as they were programmed on the foot. Even in experienced hands, partial or complete resorption of transferred phalanges can occur in up to a third of cases.

Overgrowth: Macrodactyly

Macrodactyly is pathologic enlargement of both the soft tissue and skeletal parts of the digit. The term *gigantism* has also been used in patients with concomitant enlargement of the more proximal portions of the hand and forearm. In actuality, the category of overgrowth anomalies is the most poorly defined and understood group of malformations seen by the pediatric hand surgeon. The present classification of lipofibromatosis (I), neurofibromatosis (II), digital hyperostosis (III), hemihypertrophy (IV), and Proteus syndrome (V) will ultimately be changed after more basic science information is obtained (Table 15).

The most common form of digital or hand enlargement is associated with lipomatosis and skeletal deviation along nerve territories (nerve territory–oriented macrodactyly). Grotesque digital enlargement in newborn and very young children is best treated by amputation. Digits worth salvaging are treated by aggressive reduction of soft tissue, preservation of neurovascular bundles, corrective osteotomies, and early epiphysiodeses. However, the digits will be stiff, have reduced sensitivity, and are subject to premature osteoarthritis.

Enlargement associated with some form of vascular malformation is the second most common form of overgrowth. The term Klippel-Trenaunay (KT) syndrome unfortunately has been interchanged with all types of limb, arm, and hand enlargement. KT syndrome describes a limb with multiple abnormal vascular elements: capillary, lymphatic, venous malformations (CLVMs). The enlargement is usually seen in the presence of abnormal amounts of fat in all tissue planes and premature osteoarthritis. These are slow-flow vascular malformations, in contrast to limbs with Parkes Weber (PW) syndrome, which are defined as those with enlargement and a fast-flow arteriovenous malformation with multiple AVFs. Lower limb involvement by both of these lesions is four to five times more common than involvement of the upper limb. They are both difficult to treat. Most limbs with KT syndrome are treated by sclerotherapy for the venous and lymphatic components, surgical debulking, and joint repositioning with arthrodeses. PW limbs are

TABLE 15 Classification of Overgrowth Deformities

TYPE	DESIGNATION	CHARACTERISTICS
I	Lipofibromatosis	Histology shows epineural and perineural fibrosis and fatty infiltration of the nerve; nerve territory–oriented deviation
II	Neurofibromatosis	Skin lesions present; histology shows epineural and perineural fibrosis and no fatty infiltration; sarcomatous change can occur
III	Hyperostosis	Present at birth; excess growth is both longitudinal and transverse; osteochondral nodules common at joint levels
IV	Hemihypertrophy	Digital overgrowth less extreme than types I and II; commonly incorrectly called Proteus syndrome
V	Proteus syndrome	Digital or limb overgrowth, linear epidermal nevus, thickened ("cerebriform") palms and soles, lipomas, exostoses

treated by embolization of fast-flow shunts and lesions, followed by surgical debulking, among other individualized skeletal procedures. In both, coagulation parameters may be grossly abnormal, especially within the lesions. Patients with KT syndrome are susceptible to lethal pulmonary emboli. Patients with PW syndrome frequently have some form of cardiac overload and incipient congestive heart failure or failure to thrive as babies. The fast-flow lesions often persist in the more proximal portions of the affected limb after amputation of the distal nonfunctional, painful parts. In the future, this group of vascular lesions will be treated with specific pharmacologic therapy targeted to the abnormal endothelial cells.

Toe Transfers

Over the past 4 decades, autotransplantation of a toe to the hand has progressed from the laboratory to an invaluable reconstructive technique for congenitally and traumatically deficient hands. Single-digit transfer of either the second toe or the great toe is the most common, but numerous variations exist. The trimmed toe, modified great toe, twisted toe, and wraparound modifications have been described. In addition, the second toe has been a commonly used digit either in isolation or as a monobloc transfer with two or even three other digits. Although technical improvements have paved the way for a wide array of transfers, the indications and outcomes of such transfers are only now coming into focus.

Creation or restoration of a thumb is the most common indication for toe transfer. However, the absence of a stable post against which the thumb ray can oppose (e.g., symbrachydactyly, constriction ring syndrome, one-digit hand with ulnar deficiency) is also a frequent indication (see Fig. 6). Less frequently, digits are transferred to a partially functional hand to augment grip, provide pinch, or improve appearance.

A number of important differences are seen between toe transfers in a posttraumatic patient who has lost a digit or digits and transfers in the pediatric population with congenital hand anomalies.

In patients with congenital deformities, there is no ideal time for toe transfer. Some proponents of early surgery have suggested that early transfer allows for better acceptance of the digit and improved function. Most surgeons have not found this to be the case and typically wait until 2 to 4 years of age when the hand and toe vessels are larger and the child is more cooperative. In children with no digits or thumbs, toe transfer to the thumb position has been performed at 1 year of age.

It is essential that the surgical team be familiar with the anatomy of the foot and its common vascular variations. A retrograde approach to toe dissection is preferred to rapidly identify whether the dorsal or plantar system is dominant. Preservation of the epiphyses is essential for growth, and the use of C-wires for osteosynthesis is preferable to interosseous wires or plate and screw fixation.

Secondary procedures are common. In double-toe monobloc transfers, the web space is deepened several months after the transfer. Scar revisions, tenolyses, or osteotomies may be required and are usually performed several years after transfer. Every effort should be made to complete all major reconstruction before the child enters formal schooling between the ages of 5 and 6 years.

Results reported in large series demonstrate satisfactory acceptance of transferred toes in most patients. Sensory return in children is excellent, with two-point discrimination of 3.0 to 4.0 mm. Most patients (>80%) incorporate the digits into daily activities within 6 to 12 months after transfer. Acceptance of the donor site defect and appearance of the hand has also been reported to be satisfactory. Functional problems with walking, running, and jumping are minimal if four of five metatarsal heads are preserved in their normal position. However, patient selection and proper counseling of the parents remain paramount in achieving a satisfactory outcome.

Distraction Lengthening

Distraction lengthening can be an effective reconstructive option for correction of the skeletal deficiencies seen in congenital upper limb anomalies. The concept is not new, and applications in the long bones of the lower extremity have been well publicized over the past 60 years. More than 20 years ago the original upper limb applications were described by Matev for traumatic thumb losses and by Kessler for congenital ray deficiencies. However, little has been published in the congenital hand literature.

The primary goal of distraction lengthening is to gain length for enhancement of mechanical advantage, prehension, grip, and pinch—all functional considerations. In most patients, the appearance of the limb, hand, or digit is unquestionably improved. The major advantage of this treatment modality is preservation of sensation in the lengthened part; the primary disadvantage is the prolonged and sometimes complicated process of distraction.

Indications in congenitally deficient limbs are still evolving. For example, humeral lengthening for deficiencies such as phocomelia creates limbs that are more functional and sufficiently long to support a functional prosthesis. Elbow distraction has been disappointing in terms of creation of pseudarthroses for movement, but it has been helpful in repositioning of limbs with humeroradial synostosis, in which the wrist and hand are facing posteriorly. Forearm lengthening for restoration of radius or ulna length in patients with hereditary multiple exostosis has

been effective and predictable. It is important to note that more than half of patients with severe radial deficiencies who have been corrected by either centralization or radialization desire lengthening as teenagers. The distractions usually achieve an improvement in length of 7.0 to 8.0 cm (see Fig. 3). For successful results, it is essential that the forearms be unscarred. Children born with cutis aplasia associated with in utero Volkmann compression injuries do not do well with these techniques because of excessive scarring of the muscle and soft tissues within the forearm.

Distraction is effectively used at the wrist level to lengthen severe radial clubhand before formal centralization (see Fig. 2A). The most predictable distractions are achieved for brachymetacarpia and for short thumb in the constriction ring syndrome. Enhancement of digital length has been performed for the short Apert thumb later in childhood and for short digits in many congenital deficiencies. The techniques have also been effective in moving skeletal parts more distally in complex anomalies such as constriction ring syndrome, central synpolydactyly, and many unclassified complex hand malformations. However, there must be sufficient bone stock for placement of pins without damage to the physes in all cases. Distraction should be discouraged, for example, in patients with Poland's syndrome and symbrachydactyly associated with normal MP and proximal and distal IP joint motion. In these cases, although length may be gained, function is always compromised.

Complications of this technique are directly proportional to the length of time that the pins and distraction frame have been in place. Early problems include infection, pin tract cellulitis, soft tissue breakdown, and distal vascular compromise. Late problems include joint stiffness, nonunion, malunion, or poor bone generation and secondary fracture. Digits are always longer but are thin and stiff.

Despite its limitations, this powerful technique should be considered for the treatment of deficiencies seen in central synpolydactyly and symbrachydactyly or for short thumbs in constricted ring syndrome. Many parents simply do not want a complex microvascular toe-to-hand transfer or any type of "spare parts" surgery that harvests tissue from another part of the child's body.

SUGGESTED READINGS

Buck-Gramcko D: Congenital Malformations of the Hand and Forearm. London, Churchill Livingstone, 1998.

Ezaki M, Kay SBJ, Light TR, et al: Congenital Hand Deformities. New York, Churchill Livingstone, 1999.

Flatt A: The Care of Congenital Hand Anomalies. St Louis, Quality Medical Publishing, 1994.

Gupta A, Kay SPJ, Scheker LR: The Growing Hand. Diagnosis and Management of the Upper Extremity in Children. London, Mosby, 2000.

Smith P: Lister's The Hand: Diagnosis and Indications. London, Churchill Livingstone, 2002.

Upton J: Congenital Anomalies of the Hand and Forearm. Philadelphia, Saunders, 1990.

Acute Infections of the Upper Extremity

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L. SCOTT LEVIN

The hand is vulnerable to infection because of its unique anatomy, including multiple separated spaces that are easy targets for opportunistic organisms. Infections of the hand and upper extremity can result in permanent disability. The leading causes of a poor outcome are inadequate surgical débridement and failure to recognize the polymicrobial nature of hand infections. Only meticulous wound management and adequate antibiotic therapy can limit complications such as recrudescence of infection, stiffness, septic arthritis, and osteomyelitis.

Etiopathogenesis/Causative Factors

Infections of the upper extremity predominantly begin in the hand after penetration of the skin barrier and inoculation of tissue. Causative factors include trauma (60%), human bites (25%), drug abuse (10%), and animal bites (5%). Most acute hand infections are pyogenic bacterial infections that are polymicrobial in nature. In addition to gram-positive aerobic organisms such as *Streptococcus* and *Staphylococcus*, an increasing number of anaerobic organisms are present in hand infections. An example of a nonpyogenic infection is herpetic infection of the distal phalanx (herpetic whitlow). Acute infections must be differentiated from chronic infections such as atypical tuberculosis and fungal colonization, as well as noninfectious conditions such as gout, collagen vascular diseases, and acute soft tissue calcification.

Pathologic Anatomy

It is mandatory that the surgeon be familiar with the potential spaces of the hand that are capable of harboring and enclosing bacteria: the dorsal subcutaneous space, the thenar space, the midpalmar space, the synovial sheaths, the articular surfaces, and the fascial planes of the forearm. These spaces permit gliding for motion but also allow rapid extension of an infection. A second factor favoring infections in the hand is the ease with which enclosed and septate structures become ischemic as a result of venous and lymphatic congestion; this results in local arterial insufficiency secondary to tissue reaction and swelling. A tissue tension becomes critical, necrosis results from microvascular thrombosis and the newly devitalized tissue promotes infection.

Diagnostic Studies

Laboratory tests should include a complete blood count, blood chemistry analysis, and sedimentation rate. Plain radiographs should be obtained to rule out foreign bodies, gas collection, osteolysis, and sequestrum. All wound swabs and drainage, as well as aspirates, should be sent for anaerobic and aerobic culture and Gram stain.

Reconstructive Goals

The goals of treatment are to arrest bacterial proliferation, remove purulent and necrotic tissue, and restore function. The treatment plan includes four fundamental principles: (1) adequate débridement and drainage, (2) appropriate antibiotic therapy, (3) a period of immobilization and elevation, and (4) early mobilization. Algorithms outlining the principles of treatment of the most common infections of the hand, as well as the usual dosages of antibiotics, are summarized in Table 1 and Figures 1 and 2.

TABLE 1 Antibiotic Recommendations for Hand Infections

ANTIBIOTIC AGENT	DOSAGE
Dicloxacillin	250-500 mg PO q6h
Ampicillin-sulbactam	1.5 mg IV q6h
Cefazolin	1 g IV q8h
Gentamicin	Loading dose of 2 mg/kg of body weight, then follow serum levels
Nafcillin	1-2 g IV q4–6h
Penicillin	2-4 million U IV q4-6h

Specific Treatment and Techniques

Cellulitis

Cellulitis is a nonsuppurative inflammation of subcutaneous tissue characterized by widespread swelling, erythema, tenderness, warmth, and throbbing pain without definite localization. A variety of aerobic and anaerobic organisms can produce cellulitis, but in the majority of cases it is caused by *Streptococcus* and *Staphylococcus*. Treatment is nonsurgical and consists of elevation, immobilization, and antibiotics (dicloxacillin or nafcillin). If the inflammatory swelling fails to subside after 48 to 72 hours of antibiotic therapy, an abscess or closed space collection should be considered and drained if present.

Lymphangitis

Lymphangitis is an inflammation of the lymphatic channels and lymph nodes and is a common sequela of bacterial infections with *Streptococcus* and *Staphylococcus*. It is generally visible as an erythematous streaking proximal to a finger or hand infection. Patients usually have fever and a general feeling of malaise. Lymphangitis may spread within hours, and therefore treatment should begin

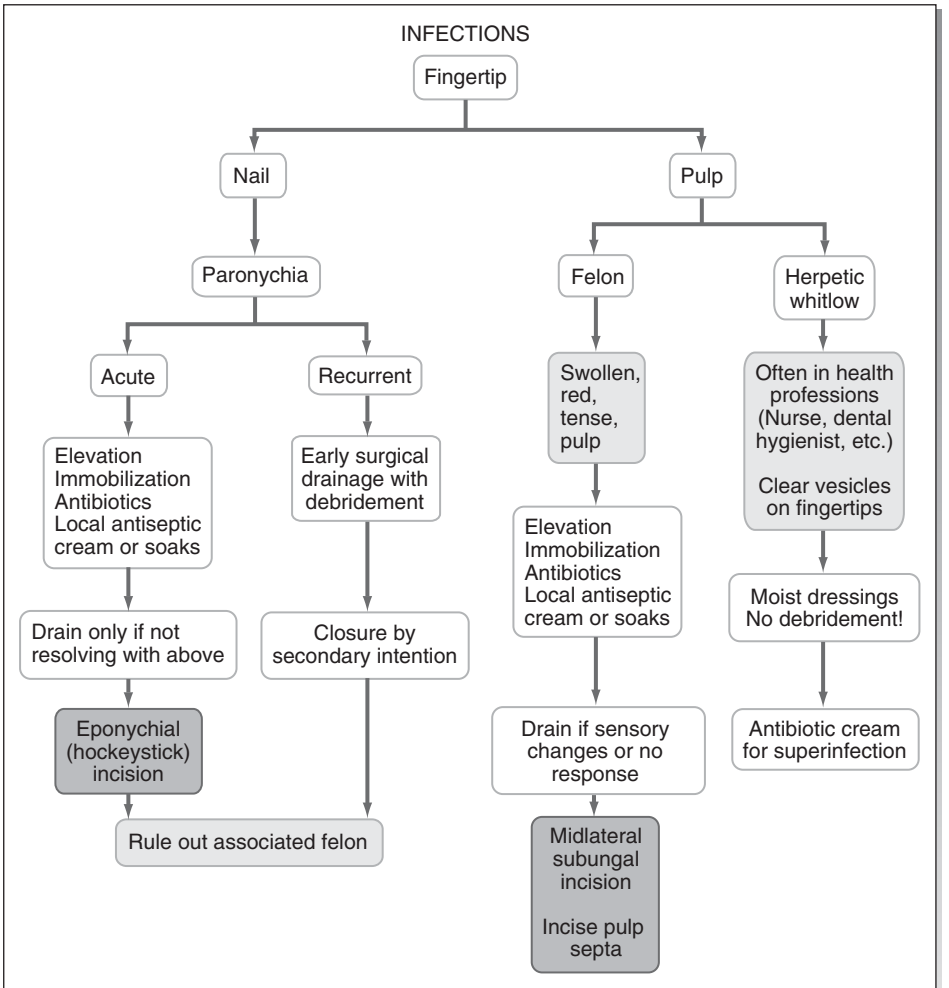

Figure 1 • Principles of treatment and management of fingertip infections. (Adapted from Germann G, Sherman R, Levin LS (eds): Decision Making in Reconstructive Surgery. New York, Springer-Verlag, 2000.)

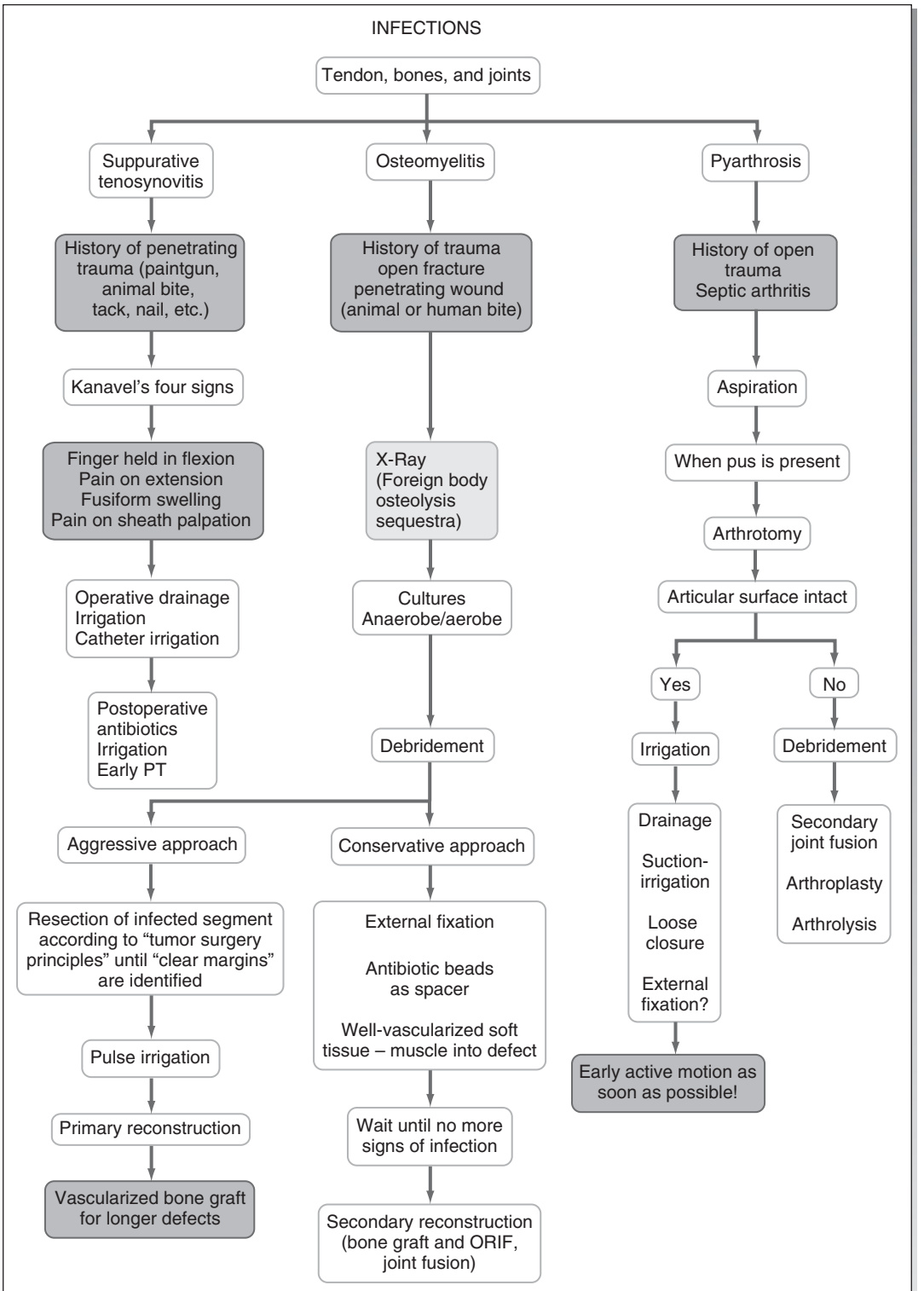

Figure 2 • General strategy for the treatment of infections of tendons, bone, and joints of the hand. (Adapted from Germann G, Sherman R, Levin LS (eds): Decision Making in Reconstructive Surgery. New York, Springer-Verlag, 2000.)

promptly. Most patients are hospitalized for strict immobilization, elevation, and parenteral antibiotic therapy (nafcillin or cephalexin). With appropriate antibiotic therapy, lymphangitis typically clears rapidly.

Infections of the Fingertip: Paronychia, Felon, Herpetic Whitlow

Paronychia and felon are the most commonly encountered hand infections. The nail conforms proximally and laterally to a depression, the nail fold. The thin membrane extending onto the dorsum of the nail is termed the paronychium laterally and the eponychium proximally; these areas are sensitive to the disruptive influences of daily living and cosmetic practices. Paronychia is a subcuticular abscess in the paronychial fold, with *Staphylococcus* commonly being the offending organism. The infection may also track deep to the nail plate and cause more serious infections, such as subungual abscess, felon, or osteomyelitis, that can eventually result in amputation. Therefore, in addition to surgical drainage, the authors recommend oral antibiotics (dicloxacillin), elevation, immobilization, packing, and local antiseptic creams or soaks. Drainage consists of incision over the area of greatest skin tension. It may also be necessary to remove the nail plate.

A felon is a subcutaneous infection of the pulp of a digit that is usually due to penetrating trauma and subsequent inoculation with *Staphylococcus*. The subcutaneous tissue of the pulp is compartmentalized by tangentially oriented fibrous septa. Because of these strong septa, there is no route for egress of the increased interstitial fluid, and therefore the infection leads to necrosis and abscess. If a patient is seen immediately after a puncture wound in the distal phalanx, antibiotics (dicloxacillin), elevation, and splinting may suffice. If there is no response to such a conservative treatment regimen, the authors prefer a straight midlateral incision. The incision is made dorsal to the neurovascular bundle, and care is taken to incise across all the involved septa. A fish mouth incision should be avoided because it is slow to heal, destabilizes the pulp, and can result in flap necrosis.

Herpetic whitlow is a viral infection, which manifests as intracutaneous vesicular bullae in the fingertip. It most commonly occurs in nurses, respiratory therapists, and dentists who have direct contact with active herpetic vesicles. Clinical symptoms include pain and pruritus before eruption of the vesicles. The diagnosis is made clinically by the history and physical examination. The mainstay of treatment is prevention of autoinoculation and transmission of the infection. Unroofing the vesicles is recommended, followed by the application of a dressing. Herpetic whitlow is self-limited; therefore, antiviral agents are not indicated. Incising the aseptic area of herpetic whitlow is contraindicated because secondary bacterial infection can ensue and cause prolonged, disabling fingertip problems.

Deep Space Infection

Infections of the potential subfascial or deep spaces of the hand are uncommon, but well recognized. They account for 5% to 15% of all hand infections. Kanavel has been credited with naming several potential subfascial or deep spaces, including the thenar, midpalmar, hypothenar, dorsal subaponeurotic, and interdigital web spaces. Aggressive surgical drainage of these deep space infections is advocated for such infections of the hand. Deep space infections are caused by mixed organisms such as staphylococci, anaerobes, and gram-negative rods. In addition to surgical intervention, antibiotic coverage (cefazolin or ampicillin-sulbactam) is essential.

The dorsal subaponeurotic space is a potential space on the dorsum of the hand, deep to the digital extensor tendons and superficial to the fascia overlying the interosseous muscles and the periosteum of the metacarpals. An abscess in that space leads to swelling and erythema of the dorsum of the hand. Fluctuation or purulence extruding from the wound directs treatment to the necessary surgical exploration and drainage. For diffuse infection, the authors prefer two longitudinal incisions, one over the second metacarpal and the other between the fourth and fifth metacarpals.

Thenar space infection should be referred to as either an anterior or posterior adductor space infection. The anterior border of the more commonly involved anterior adductor space is formed by the index finger flexor tendons and the midpalmar septum; the posterior border is the fascia of the adductor muscle itself. The princeps pollicis artery and parts of the deep palmar arch run within the posterior adductor space. The thenar region is usually dramatically swollen and exquisitely tender to palpation, and the thumb is held in abduction because this position increases the capacity of the deep spaces. Drainage of the anterior adductor space may be performed through a curvilinear palm incision parallel to the thenar crease on the ulnar side at the base of the thenar eminence. It may need to be combined with a dorsal, longitudinal incision between the thumb and index metacarpals to open the posterior adductor space.

The midpalmar space is a potential space between the flexor tendons of the long, ring, and little fingers and the fascia of the second and third interosseous muscles and periosteum of the third, fourth, and fifth metacarpals. The radial border is the midpalmar septum, and the ulnar border is the hypothenar septum. In midpalmar space infection, the normal palmar concavity of the hand is lost, and motion of the middle and ring fingers is usually limited. Drainage is performed via a transverse incision parallel and proximal to the distal palmar crease.

The hypothenar space is the potential space between the intermuscular septa of the isolated hypothenar musculature; it is bounded radially by the hypothenar septum and dorsally by the fifth metacarpal. Abscesses are drained through an incision at the point of maximal tenderness.

Infection of the web between any of the digits can be caused by puncture wounds or by an infected callus over the metacarpal head. Web space infections have a tendency to erode through the palmar fascia and form a secondary abscess on the dorsum of the hand. The fingers bordering the involved web space are splayed because tissue pressure holds them apart. This type of infection is also called a *collar button abscess*. The infections generally point in the looser tissue on the dorsal side, and recognition of the palmar component may require a high index of suspicion. For drainage purposes, a palmar zigzag incision and a dorsal longitudinal incision are performed. Incisions placed transversely across the web space should be avoided because of subsequent scar contraction and resulting loss of full finger abduction.

Pyogenic Flexor Tenosynovitis

Flexor tendon sheaths are poorly vascularized, are rich in synovial fluid, and offer a hospitable environment for infection, which can spread without obstruction from the proximal to the distal limits of the sheath. The most common microbial pathogens are *Staphylococcus*, *Streptococcus*, and *Pseudomonas*. The index, middle, and ring fingers are most frequently affected. Kanavel's four signs remain the key to diagnosing this condition: the presence of fusiform swelling, flexed attitude of the affected finger, tenderness along the course of the flexor tendon sheath, and pain with passive stretch of the finger.

The most sensitive of these signs is pain on passive finger extension. Suppurative flexor tenosynovitis requires prompt treatment to avoid adhesions, tendon necrosis, or amputation of the digit. Nonoperative treatment consisting of immobilization and intravenous antibiotic therapy is appropriate only if the infection is identified at a very early stage (within the first 48 hours). If a rapid clinical response does not occur, operative intervention is indicated. The goal is to drain the infection and allow continued drainage without compromising the function and anatomy of the flexor tendon and its canal. This goal is best accomplished through saline irrigation of the tendon sheath with a catheter. The authors' preferred method requires a transverse incision in the area of the A1 pulley and a midlateral incision along the middle and distal segments of the digit. Through the transverse incision, access is obtained to thread a catheter under the A1 pulley in the flexor tendon sheath for a distance of 2 cm. The catheter is sutured to the skin and the wound closed around the catheter. Through the midlateral incision, the sheath is resected distal to the A4 pulley. A small drain is brought from the tendon through the skin distally and sutured to the skin. The wound is closed around the drain. The system is flushed copiously with manual pressure every 2 hours until signs of infection have abated. Treatment also includes parenteral antibiotics such as cefazolin.

Necrotizing Fasciitis

Necrotizing fasciitis is a severe, fulminant infection of the superficial fascia and subcutaneous tissue; it is most commonly encountered in patients with diabetes mellitus, alcohol abuse, and intravenous drug abuse. The disease is caused by *Streptococcus pyogenes* or a synergistic infection of anaerobic and facultatively anaerobic bacteria.

The infection can spread unrecognized along fascial planes beneath seemingly normal skin. The relatively benign appearance of the extremity is misleading and often results in delay in diagnosis with increased morbidity and potential mortality. The classic signs and symptoms are erythema, advancing cellulitis, pain, crepitance, high fever, and other systemic signs of sepsis. Skin bullae and necrosis are signs of a later stage of the disease. Liquefactive necrosis of the fat and superficial fascia leads to the characteristic appearance of grayish, watery, and foul-smelling fluid often referred to as "dishwater pus." The necrotic fascia observed at the time of surgical exploration is pathognomonic. Immediate radical débridement is mandatory and must be extended until healthy muscle and fascia are observed. Serial débridement is warranted every 12 to 24 hours until the infection is suppressed. Early surgical intervention is the key to successful treatment of necrotizing fasciitis. A combination of intravenous broad-spectrum antibiotics is recommended, such as a cephalosporin (staphylococci and streptococci), penicillin (anaerobes), and gentamicin (gram-negative organisms). The patient may deteriorate rapidly, and sometimes early amputation may be necessary to control a particularly severe infection.

Gas Gangrene, Clostridial Myonecrosis

Gas gangrene is an anaerobic infection most frequently caused by *Clostridium perfringens* that develops in contaminated, devitalized wounds. It is a rare condition with an estimated 1000 to 3000 cases in the United States annually. Clostridia produce a number of toxins that are systemically toxic and can be lethal. Clostridial infections are seen in the hand after penetrating injury, and gangrene is manifested as swelling, pain, ecchymosis, and bullae. The exotoxins cause necrosis of muscle, subcutaneous tissue, and fat with palpable subcutaneous gas formation. Radiographs may confirm the presence of gas in soft tissue, a finding that demands immediate incision and aggressive surgical débridement of the necrotic tissue (Fig. 3).

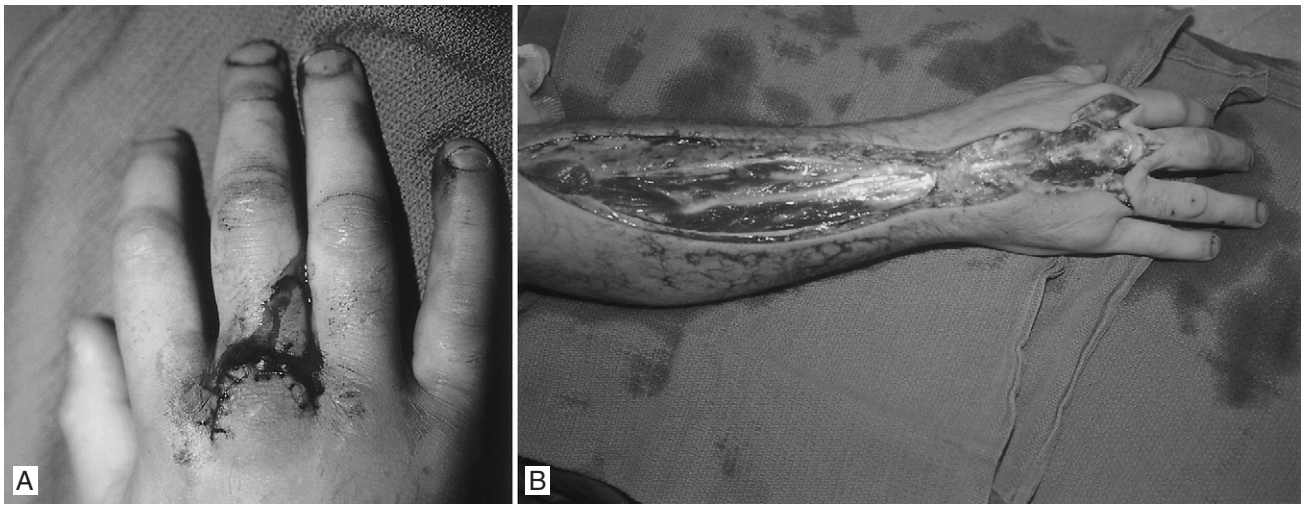

Figure 3 • Patient with gas gangrene after an open fracture in the proximal phalanx of the third right digit. **A,** Dorsal hand view showing the open fracture and marked swelling of the hand. **B,** The arm after débridement with loss of the extensor tendons to the long and ring finger.

Prompt surgical intervention is crucial in the event of gas gangrene because clostridial myonecrosis progresses rapidly and patients soon become septic and moribund secondary to irreversible hypotension and renal failure.

Animal Bites

More than 1 million animal bites requiring medical attention occur annually in the United States; approximately 1% to 2% require hospitalization. Dogs are responsible for 90% of all animal bites, but cat bites are more likely to become infected. Animal bites can cause significant infection despite a benign appearance initially. An in-and-out puncture wound may lead to inoculation of bacteria in a joint or one of the potential deep spaces of the hand, both of which represent an excellent culture medium. Animal bites cause mixed infections, but *Pasteurella multocida*, a gram-negative anaerobic bacterium, has been identified as an important pathogen in animal bite infections. The presence of cellulitis, lymphangitis, and serosanguineous or purulent drainage within 12 to 24 hours after a cat or dog bite strongly suggests *P. multocida* as the offending organism. Initial treatment consists of thorough irrigation and débridement of necrotic and damaged tissue. Antibiotic coverage should include cefazolin and penicillin.

Human Bites

Human bites can introduce infection via nail biting, traumatic amputation of digits, full-thickness bite wounds, or the so-called clenched fist injury. Human bite wounds are by far more virulent than a similar injury caused by an animal. Agents that most often cause infection are *Streptococcus, Staphylococcus*, and *Eikenella corrodens*. Far more serious conditions such as hepatitis, herpes, tuberculosis, actinomycosis, and possibly human immunodeficiency virus infection may also be passed through a human bite wound. In the case of a clenched fist injury or fight bite, a possible deep puncture into the joint must be suspected until proved otherwise. Note that the superficial laceration might be well proximal to the deep penetrating wound because of the clenched fist position of the hand when the injury occurred. Surgical exploration of such a wound with thorough irrigation and débridement is warranted. Antibiotics play a critical role in improving the clinical outcome. Cefazolin and penicillin are recommended. Complications of incompletely treated human bite infections include arthritis, joint stiffness, osteomyelitis, and toxic shock syndrome.

Pyogenic Arthritis

Joint infections are most likely secondary to direct penetration of the joint capsule, but hematogenous spread from tooth abscesses or from ear, gastrointestinal, and genitourinary infections occur as well. The joint is usually swollen, warm, and tender. The most likely pathogens are *Staphylococcus* and *Streptococcus*. Passive motion is restricted and painful. If there is any doubt that the infection has been detected early, drainage is imperative because of the deleterious effect of pus on cartilage; the joint

is also débrided and irrigated. Cefazolin is the antibiotic of choice.

Postoperative Care

Postoperative care includes constant elevation of the arm and the application of a plaster splint to ensure immobilization. Except for the cases that require closed irrigation, wounds should be left open and covered with wet saline gauze to promote drainage. Active hand motion is encouraged to prevent stiffness. When the infection resolves, the dressings can be changed to wet-to-dry three times a day until the wound is healed.

Pearls and Pitfalls

- An undrained area of infection is a common reason for failure to improve after initial treatment.
- Knowledge of the potential deep spaces of the hand helps to uncover extensions of the abscess.
- If an infection continues to smolder despite appropriate treatment, occult osteomyelitis should be suspected.
- Because nearly half of all digits with osteomyelitis ultimately require amputation and many others remain stiff or persistently symptomatic, it is important to recognize osteomyelitis of the hand and be familiar with the principles of treatment.
- The critical issue in osteomyelitis is bone necrosis because microorganisms residing in dead bone can cause flare-ups as late as 50 years after the initial injury. Surgical exploration is indicated in virtually all cases of osteomyelitis, and débridement of all infected and devitalized tissue is essential. Antibiotic therapy should include cefazolin and gentamicin, and it is oftentimes necessary to keep patients on that regimen for 4 to 6 weeks. Osteomyelitis secondary to septic arthritis has a particularly poor prognosis.
- After a period of brief immobilization, early active motion is critical in maintaining hand function.

SUGGESTED READINGS

Gonzalez MH: Necrotizing fasciitis and gangrene of the upper extremity. Hand Clin 14:635, 1998.

Kono M, Stern PJ: The history of hand infections. Hand Clin 14:511, 1998.

Neviaser RJ: Acute infections. In Green DP, Hotchkiss RN, Pederson WC (eds): Green's Operative Hand Surgery. Philadelphia, Churchill Livingstone, 1999, p 1033.

Patel MR: Chronic infections. In Green DP, Hotchkiss RN, Pederson WC (eds): Green's Operative Hand Surgery. Philadelphia, Churchill Livingstone, 1999, p 1048.

Szabo RM: Palumbo CF: Infections of the hand. In Chapman MW (ed): Chapman's Orthopaedic Surgery. Philadelphia, JB Lippincott, 2001, p 1989.

Tsai E, Failla JM: Hand infections in the trauma patient. Hand Clin 15:373, 1999.

The Mutilated Lower Extremity

LANCE EVERETT WYATT
JULIAN JOSEPH PRIBAZ

The initial assessment of a severely injured lower extremity must focus on whether the mutilated limb meets the criteria for amputation. Every effort should be made to prevent unsuccessful attempts at salvage that eventually result in amputation, a decision that can carry significant cost, morbidity, and at times, mortality. When amputation is inevitable, it should be performed early to minimize complications and shorten the length of hospitalization. Lower extremity salvage is the most desirable goal and should be attempted only if the criteria for amputation are not met.

Etiopathogenesis

Most severe, mutilating injuries are the result of high-energy blunt forces and occur in young, otherwise healthy patients. Equally devastating tissue injury can result from extensive osteomyelitis with osteonecrosis, tumor extirpation with or without radiation therapy, diabetic ulceration, peripheral vascular disease, failed joint replacement, or other causes. Children may present with challenging defects after severe burns, debulking procedures for vascular malformations, radical limb-sparing procedures for malignancy, congenital deficiencies, and vasculitis/infections (e.g., purpura fulminans). Power lawn mower accidents are a common cause of leg injuries in children younger than 16 years.

Pathologic Anatomy

The surgeon must consider the location of the wound and carefully appraise the extent of tissue injury. The thigh may be divided into the hip and proximal/lateral thigh, midthigh, and supracondylar knee. The leg is divided into thirds (proximal, middle, distal), reflecting the relationship of the tibia with surrounding soft tissue. The foot is divided into the proximal non–weight-bearing region (Achilles and malleolar area), the proximal weight-bearing region (heel and proximal plantar area), the dorsum, and the distal plantar region.

Tissue Assessment

COMPLETE AMPUTATION. The most severe injury, a complete amputation, is often associated with injuries at multiple sites. Severe mutilation can also result in incomplete amputation and other extensive segmented extremity injuries. Complete amputations are rarely of the sharp, clean, "guillotine" type, but rather are the result of extensive crush and avulsion that produce a wide "zone of injury" on each side of the amputated part. Within the "zone of injury" there is inflamed soft tissue that extends beyond the gross margin of the wound and is associated with increased perivascular friability and scar tissue. The inflammatory changes must be considered when assessing severely injured limbs because such changes contribute to a higher failure rate in lower limb microsurgical reconstruction. Proximal dissection of recipient vessels, the use of interpositional vein grafts, and the use of distal vessels are recommended to avoid the adverse effects of the zone of injury on vascular anastomoses.

Complete amputation injuries in children present additional challenges. The injured structures are small, patients are uncooperative, and growth potential must be considered, including late deformity secondary to abnormal growth. However, children are spared the adverse sequelae of ischemic

and neuropathic problems associated with diabetes and peripheral vascular disease that may be present in older patients. They may have a better blood supply, which augurs well for bone regeneration and repair, as well as superior regeneration of neural tissue.

Children frequently suffer from "run-over" injuries that may produce crushing and degloving of the lower extremity. Older children more frequently have distal injuries of the foot commonly produced by push mowers. Complete amputation from this mechanism may be associated with minimal crush and contamination and may be suitable for replantation.

INCOMPLETE AMPUTATION

Skin and Muscle. Muscle injury, deep vein injury, the extent of skin defects, and the presence of contamination should all be assessed. Muscle injury should be characterized as laceration, avulsion, or crush injury involving one or more tendons and compartments (or any combination of such injuries). The viability of contused or crushed skeletal muscle may be difficult to determine initially but should be assessed by standard objective criteria (color, consistency, bleeding, contractility). Deep vein injury may consist of contusion, partial or complete laceration, or avulsion. Superficial vein injury should also be noted. Skin defects may be described in terms of the circumference of the defect (one quarter, one half, three quarters, etc.) and the presence of avulsion or laceration, or both. Contamination may be mild, moderate, or massive, depending on the amount of dirt and other debris in the wound.

Bone and Joints. The degree of energy transfer and the wounding mechanism are important determinants of the extent of soft tissue and skeletal injury. Classifications of open tibial fractures reflect the prognostic significance of the mechanism of injury on the outcome of lower extremity injuries (Table 1). A plastic surgeon is usually requested to assist in the management of Gustilo type IIIb or IIIc injuries.

Neurovascular Status. The degree and duration of limb ischemia influence the ultimate outcome. Open tibial fractures with associated vascular injury (Gustilo type IIIc injury) significantly increase the probability of amputation. The injured extremity must be evaluated for the presence of normal perfusion, and initial pulse assessment may be characterized as absent, reduced, or normal. The status of perfusion with respect to general appearance, capillary refill, and temperature should be documented. Absence of perfusion may result from kinked vessels or vessels in spasm, and inflow may return after fracture reduction.

The presence or absence of plantar sensation is critical in the initial assessment of a mutilated extremity. Nerve integrity may be the deciding factor regarding whether to salvage or amputate. The cause of the deficit (neurapraxia versus complete transection) should be determined. Complete motor and sensory deficits suggest nerve division or irreversible injury related to traction. Progressive neurologic changes suggest ischemia related to arterial injury or compartment syndrome. Avulsion, crush, or complete transection of the posterior tibial nerve portends a poor functional outcome, and primary amputation should be considered. Other nerve injuries do not preclude salvage, but the injury should be distal enough to permit return of plantar sensation and function within a reasonable period.

TABLE 1 Classification of Open Tibial Fractures

From Byrd HS, Spicer TE, Cierney G: Management of open tibial fractures. Plast Reconstr Surg 76:159, 1989.

Diagnostic Studies

The initial management of a patient with severe lower extremity injury includes radiographic assessment proximal and distal to the level of injury. Duplex ultrasonographic imaging of both arteries and veins, in particular the dorsalis pedis–posterior tibial arterial system, should be performed. Patients may be evaluated by arterial duplex examination, magnetic resonance angiography, or conventional angiography. Angiographic definition of the limb vasculature is obtained for ischemic limbs and for patients undergoing free tissue transfers. Middle-aged amokers with long-standing diabetes or chronic peripheral vascular disease should be evaluated by arteriography. On-table angiography may be performed in the operating room if vascular injury is suspected. The irritant effects of the contrast agent used for arteriography may be minimized by performing arteriography 48 hours before free tissue transfer. A vasodilating agent may also be given during angiography before removing the catheter.

Predictive Index Scores

Numerous predictive indices were developed to assist in the decision between lower extremity salvage and primary amputation. The Mangled Lower Extremity Score Index (MESI), Limb Salvage Index (LSI), Predictive Salvage Index (PSI), and Mangled Extremity Severity Score (MESS) (Table 2) are designed to identify unsalvageable limbs based on objective criteria: skin and subcutaneous tissue, bone, nerve, vessel, and associated injury. The scoring systems have evident shortcomings. They are difficult to apply, define success as a viable rather than a functional extremity, and are unable to identify unsuccessful salvage efforts that eventually lead to delayed amputation. Regardless of the index used and the score obtained, the final decision on whether to attempt limb salvage relies on the clinical judgment of the surgeon.

TABLE 2 Mangled Extremity Severity Score Variables

VARIABLE	POINTS
Skeletal/Soft Tissue Injury	
Low energy (stab, simple fracture, "civilian" GSW)	1
Medium energy (open or multiple fracture, dislocation)	2
High energy (close-range shotgun or "military" GSW, crush injury)	3
Very high energy (above + gross contamination, soft tissue avulsion)	4
Limb Ischemia	
Pulse reduced or absent but perfusion normal	1*
Pulselessness, paresthesias, diminished capillary refill	2*
Cool, paralyzed, insensate, numb	3*
Shock	
Systolic blood pressure always >90 mm Hg	0
Hypotensive transiently	1
Persistent hypotension	2
Age (yr)	
<30	0
30–50	1
>50	2

*Score doubled for ischemia longer than 6 hours.
GSW, gunshot wound.
From Johansen K, Daines M, Howey T, et al: Objective criteria accurately predict amputation following lower extremity trauma. J Trauma 30:568, 1990, © 1990, The Williams & Wilkins Company, Baltimore.

Goals of Reconstruction

Severe lower extremities injuries are complex management problems best treated by a multidisciplinary team. The attending physician, surgical consultants (trauma, plastic, vascular, orthopedic), psychiatrists and psychologists, therapists, and social workers play important roles in providing comprehensive care to a patient with severe lower extremity injury.

A careful history to ascertain the cause and a general examination and assessment of what is injured or missing, as well as the surrounding vascularity, should be performed before a reconstructive plan is formulated. All management strategies aim to restore anatomy and function as best as possible to the preinjury state. If amputation is the treatment chosen, the goal of stump salvage is to preserve length with a maximum number of working joints and create a well-perfused stable stump that can be fitted with a functional prosthesis to minimize energy dissipation during ambulation.

Treatment

Replantation

Replantation of amputated lower limbs is technically feasible, but not uniformly practiced. Severe associated injuries usually preclude replantation, and prosthetic alternatives are widely acceptable. The results of replantation must be compared with those obtained with prosthetic devices; a marginally functional, replanted lower limb may be a greater liability when the risk of surgery, associated post-

operative pain, and extensive rehabilitation requirements are considered.

The goal of replantation is to provide a sensate, painless extremity with stable stance and functional gait. Careful patient selection helps to minimize replantation failure. The overall status of the traumatized patient with respect to coexisting injuries and baseline health is of primary importance. The amputated part should be assessed and the period of ischemia determined. Optimal care of the amputated segment is a critical prerequisite for any attempt at replantation. Amputated parts should be wrapped in saline-soaked gauze and placed in a plastic bag on ice. A 6-hour period for restoring perfusion to ischemic limbs is generally accepted, but no definite time limit can be established because many factors influence the outcome of a dismembered limb.

The surgeon must appraise the dismembered limb for the degree of crush injury. A distal, single-level amputation devoid of a degloving or crush component in an otherwise healthy, young nonsmoker may present the ideal situation for replantation. Multilevel injury, an extensive zone of injury, and unreconstructible joints in the proximal or dismembered segment mitigate against replantation. Special effort at aggressive salvage should always be made in children. Preservation of vascularized epiphyseal plates allows the amputated part to achieve normal bone length over time, and replantation may be preferable to serial prosthetic readjustment as bone growth proceeds.

Key operative elements for a successful outcome include a two-team approach with pneumatic tourniquet control on the proximal side. All devitalized tissue should be débrided, followed by bone shortening. Nerves, arteries, and veins should be identified. Bony stabilization and fixation are followed by definitive vascular repair. Interpositional vein grafts may be needed. Perfusion by shunt placement in the proximal and distal arteries truncates ischemia time and therefore minimizes additional tissue loss at the expense of blood loss. Tension-free nerve repair is critical. Immediate nerve grafting is controversial because of difficulty defining the proximal and distal zone of injury. Soft tissue repair is followed by prophylactic fasciotomy in the amputated segment. There is usually some bone shortening that can be corrected at a later date with distraction lengthening.

The risks of replantation, including disease transmission associated with transfusion, infection, hematoma, compartment syndrome, myoglobinuria, and acute renal insufficiency, must be weighed against revision amputation and prosthetic substitution.

Amputation versus Salvage

Successful salvage without restoration of function is not a true success. The management team must identify patients who would truly benefit from limb salvage with respect to functional outcome.

TABLE 3 Indications for Primary Lower Limb Amputation

Absolute
Total leg amputation in an adult
Sciatic nerve transection in an adult
Irretrievable devascularization
Relative
Life-threatening multisystem trauma
Insensate plantar surface of the foot from any source
Crushed foot or degloved plantar surface
Widespread leg crush injury with defunctionalization
Extensive bone loss
Multiple joint disruption
Multilevel injury
Elderly (age >55 years), especially with a history of diabetes or advanced peripheral vascular disease
Rehabilitation concerns

Modified from Hallock GG: Severe lower-extremity injury: The rationale for microsurgical reconstruction. Orthop Rev 15:465-470, 1986.

Factors that hold sway in the initial decision-making process include the location and extent of injury, the extent and severity of associated injuries, the physiologic reserve of the patient, and the experience of the surgeon. Decisions are made with the goal of maximizing functional results because these patients may have many working years ahead of them.

Primary amputation of a mutilated lower extremity can be achieved with low morbidity and satisfactory functional outcome and remains a valid reconstructive option. General guidelines for primary amputation are listed in Table 3. The characteristics of the patient (age, motivation to participate in the rehabilitation process) and the degree of family support may influence the decision-making process.

Amputation should create a mobile soft tissue envelope with proper bone contouring that absorbs shear and directs force to make the interface with the prosthesis comfortable. The ultimate aim is to return the patient to bipedal ambulation with a prosthetic device. Sufficient tissue must be débrided to allow for primary healing, with attention directed to the optimal site for prosthetic fitting (Fig. 1). The more distal the amputation, the greater the likelihood of effective weight bearing and ambulation without a significant increase in energy expenditure. Energy cost per distance increases above normal by the following approximate percentages: below knee, 25% (41% if bilateral); above knee, 65% (280% if bilateral); hip disarticulation, 82%; and hemipelvectomy, 125%. Walking speed decreases as the level of amputation increases.

Hip disarticulation and hemipelvectomy place significant demands on the patient. However, young and highly motivated patients may be able to return to a

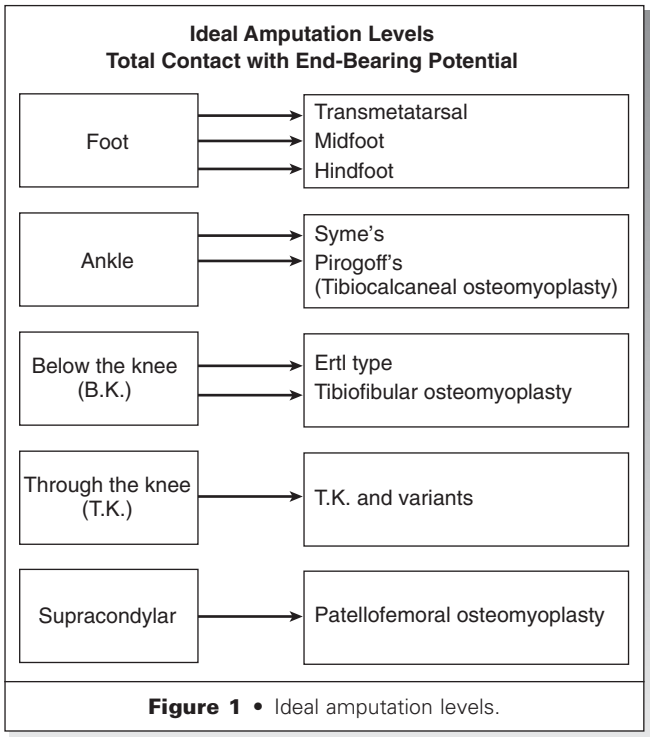

Figure 1 • Ideal amputation levels.

useful working life. Above-knee amputations are more difficulty to adapt to and require greater energy expenditure than below-knee amputations do. Therefore attempts should be made to preserve a functional knee joint. If an above-knee amputation must be performed, as long an above-knee stump as possible should be retained. Myocutaneous flap closure and modern prosthetics help to optimize function, but the large thigh muscle mass tends to atrophy with time, thus leaving a large amount of redundant skin that must be controlled by the prosthesis.

A knee amputation at the supracondylar level with patellofemoral fusion provides a stable tendon-to-bone anchor for excellent weight-bearing potential.

A through-knee amputation offers the advantage over above-knee amputation of full muscular control of the femur, and proprioception is preserved with an end weight-bearing prosthesis.

Below-knee amputations are the most common and the easiest to fit with a prosthetic device. Stumps measuring more than 5 cm from the medial joint line may be fitted with a prosthesis. Longer below-knee lengths increase mechanical efficiency and decrease energy expenditure. Osteomyoplasty improves end-bearing potential by producing a bony bridge between the tibia and fibula with the anterior and posterior muscle groups secured over the bridge.

Ankle-level amputations are functionally superior to amputations performed at a more proximal level. Syme's amputation (tibiotalar disarticulation) retains a proprioceptive heel pad with only a slight increase in energy expenditure, and it is indicated when one is unable to salvage the foot more distally. The sensate heel allows for accurate foot placement during ambulation. However, Syme's amputation is not frequently performed because the broad ankle may be bulky and prosthetic devices are frequently difficult to use. Although most prefer to perform the amputation below the knee, Syme's amputation may be performed in the pediatric population because through-bone amputations tend to be less satisfactory as a result of stump pressure points, discomfort, and shin breakdown caused by continued irregular bone growth at the amputation site.

Other hindfoot amputations include Boyd's amputation and Pirogoff's amputation (both with a calcaneotibial arthrodesis). These amputations may produce a less bulbous, more cosmetic stump with superior prosthetic fit and less heel pad migration.

Midtarsal (Chopart's) and tarsometatarsal (Lisfranc's) amputations may be performed when there is insufficient viable tissue for a short transmetatarsal amputation. Their major disadvantage is muscle imbalance, which may lead to an equinovarus position of the residual foot. Tendon lengthening or transfers along with arthrodesis can be performed to correct an equinovarus deformity.

Transmetatarsal amputation should be considered when most or all of the first metatarsal must be removed or when two or more medial rays (or more than one central ray) need to be resected. Preservation of as much length of the first metatarsal as possible may allow for effective restoration of the medial arch with a custom-molded inset and a rocker bottom sole.

Spare Parts

Preservation of viable parts of the mangled limb is a well-established practice, and severely traumatized limbs deemed unacceptable for salvage or replantation should be carefully evaluated for this purpose. Reconstructions that maximize available tissue should be considered in young healthy patients instead of the traditional options available for elderly patients. The amputated part may provide grafts of skin, nerves, veins, arteries, and tendons, as well as composite fillet flaps and toe transfers (for concomitant upper extremity injury). Composite free tissue transfer with tissue from the amputated part may provide additional bone and soft tissue to create longer and more functional stumps for those with inadequate bone length. If the knee joint cannot be salvaged, the surgeon may consider a free vascularized "Van Ness" transfer, a technique in which the amputated ankle joint is rotated 180 degrees and attached to the femur to create a neo–knee joint.

The surgeon should be aware of the value of "spare parts" and apply them accordingly. An amputated part that may be useful after adequate serial débridement of the stump may need to be replanted in an ectopic location and transferred later when local conditions allow (Fig. 2).

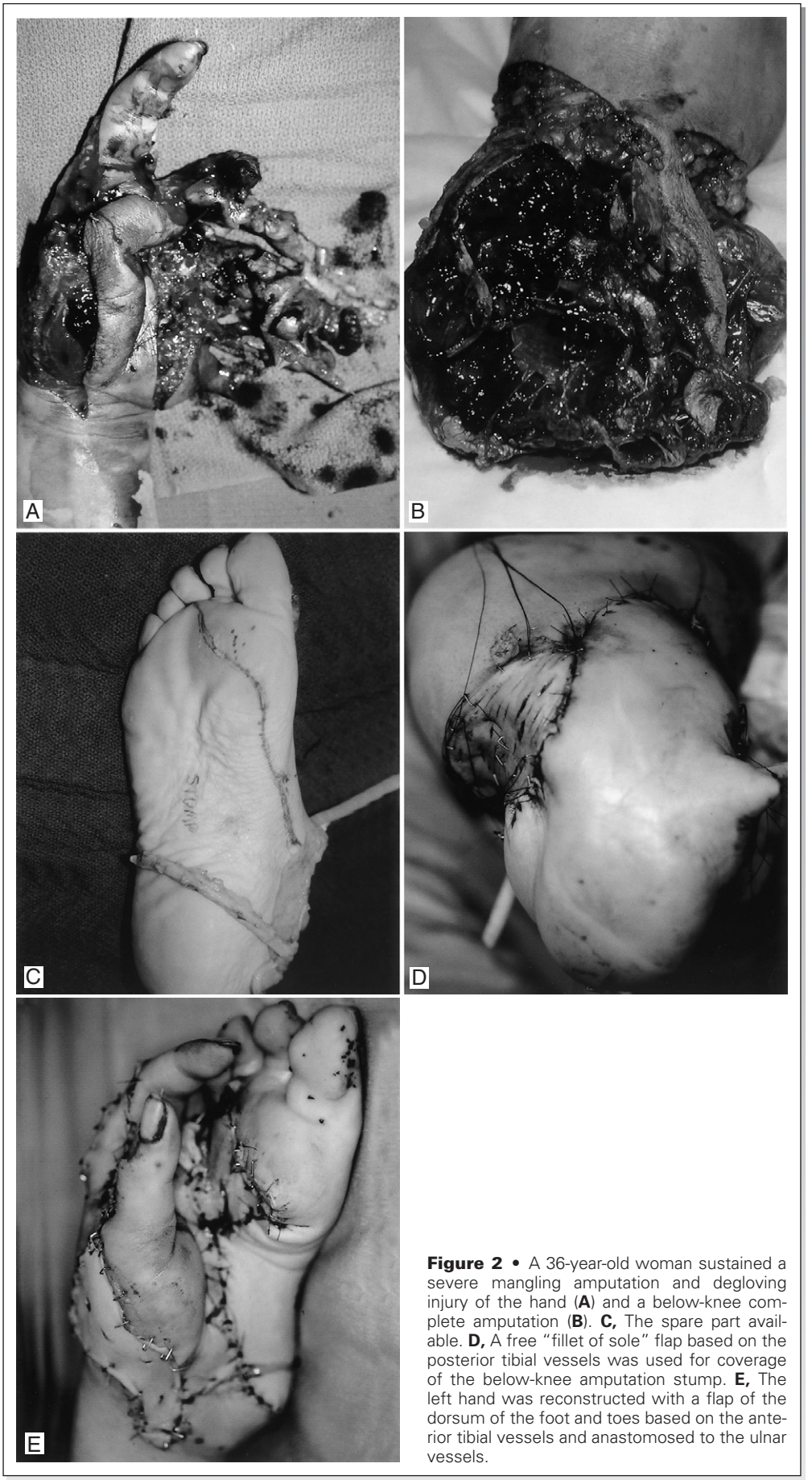

Figure 2 • A 36-year-old woman sustained a severe mangling amputation and degloving injury of the hand (**A**) and a below-knee complete amputation (**B**). **C,** The spare part available. **D,** A free "fillet of sole" flap based on the posterior tibial vessels was used for coverage of the below-knee amputation stump. **E,** The left hand was reconstructed with a flap of the dorsum of the foot and toes based on the anterior tibial vessels and anastomosed to the ulnar vessels.

Salvage

If salvage is to be successful, it is imperative that débridement be performed as early and as thoroughly as possible to prevent infection and prepare the mutilated lower extremity for reconstruction. Local and systemic factors conspire to predispose a severely injured lower extremity to infection. The lower extremity is also more prone to infection than other regions in the body are. Patients with mutilated lower limbs frequently have associated injuries and may have significant blood loss, thus setting the stage for an impaired delayed hypersensitivity response and decreased neutrophil chemotaxis as seen in a severely injured patient. Débridement is the basis for successful salvage and entails complete exposure of the wound with removal of all necrotic tissue. A pneumatic tourniquet is inflated on the proximal side. Vital structures in the wound are inspected and preserved. All devitalized soft and bony tissue should be removed. Large intermediate fracture fragments with attached soft tissue may be able to be preserved if wound coverage is provided in the acute phase of injury. Full- or split-thickness grafts may be procured from degloved tissue. Copious high-pressure irrigation cleanses the wound of bacteria and particulate matter. Four-compartment fasciotomy is performed on the distal segment in the setting of ischemia. Broad-spectrum antibiotic therapy should be administered but is of little value if débridement is incomplete. On-table angiography and immediate revascularization may be performed if necessary. Multiple débridement sessions at frequent intervals are often necessary to ensure that all devitalized or marginal tissue has been removed and the zone of injury is clearly defined. Interval moist saline gauze dressings or wound-healing adjuncts such as the vacuum-assisted closure (VAC) dressing may be used. The VAC dressing is especially helpful in immobilizing skin grafts over irregular surfaces.

Bone Fixation

Bone fixation is performed in close cooperation with the plastic surgeon, who may be asked to assist in wound coverage or vascularized bone transfer, or both. There is usually more soft tissue coverage for injuries above the knee, and external or internal fixation (plates or intramedullary nails) may be applied. Below the knee, an external fixator is generally applied for tibial injuries with extensive soft tissue injury. The use of antibiotic-impregnated beads has been shown to be a valuable adjunct in the prevention of infection in open fractures by producing high local levels of antibiotic in a moist environment. The Ilizarov distraction technique with an external fixator may be used for bone stabilization in cases in which adequate bone stock is present.

Soft Tissue Reconstruction

The time of definitive wound closure is controversial. Immediate emergency coverage within 24 hours after injury is rarely performed for high-energy–induced open fractures, but it may be necessary for open joint injuries or for coverage of exposed neurovascular structures. Acute, sharp, noncontaminated injuries may also be débrided and closed during the same operation. Soft tissue coverage is more commonly performed in the acute injury phase (within 5 to 7 days). A patient whose wound is closed within this period benefits from a shorter hospital stay, less pain with dressing changes, fewer operations, reduced incidence of flap failure, shortened interval from injury to full weight bearing, and decreased infection rate. Coverage within the acute injury phase may be necessary for wounds with exposed tendons or bone denuded of periosteum. However, in many patients with a high-energy injury, early soft tissue reconstruction may not be possible because of associated injuries and the poor general condition of the patient. In these circumstances, patients may be managed by serial débridement followed by radical redébridement and definitive wound coverage (ideally within 14 days of injury). This strategy allows marginal tissues to declare their viability and the zone of injury to be fully defined. Delayed bone reconstruction can be performed in 4 to 6 months. Each case must be individualized and the timing of closure determined by the condition of the patient and the certainty of proper wound débridement.

The type of reconstruction is determined by the size and location of the wound, the length of pedicle needed, and the presence and extent of dead space that needs to be filled. Primary closure and skin grafting may be appropriate in certain circumstances, but high-energy injuries usually call for vascularized flap options. Local or regional muscle flaps may be used to cover injuries in the hip, thigh, and knee region. Hip wounds with exposed orthopedic hardware may be covered by pedicled flaps (tensor fascia lata, vastus lateralis muscle, rectus femoris muscle) based on the lateral circumflex femoral artery. Bipedicle flaps may also be used to cover exposed bone.

The gastrocnemius muscles are frequently used for wounds of the knee and proximal third of the leg. Fasciocutaneous bipedicle flaps or small muscle flaps such as the tibialis anterior flap may also be used to cover small defects of exposed bone. The soleus muscle is classically used for wounds involving the midthird of the leg and occasionally for the upper aspect of the lower third of the leg. A hemisoleus flap may be used to preserve function. Wounds of the lower third of the leg are best managed by free tissue transfer.

Fasciocutaneous flaps may have a role in small to moderate wounds without significant dead space. They are indicated in patients in whom free tissue

transfer cannot be performed or in whom significant comorbidity mitigates against microsurgical transfer. Fasciocutaneous flaps are not as malleable as muscle flaps because of the rigidity of the fascia and subcutaneous fat and may not be an option in an obese patient, whose circulation to the skin may be compromised. These flaps may require a skin graft for coverage of the donor site.

Indications for free tissue transfer include high-energy injuries, some middle and most distal third tibial wounds, radiation wounds, areas of osteomyelitis, bony nonunion, and wounds after tumor extirpation. Free radial forearm, lateral arm, and anterior lateral thigh flaps are examples of fasciocutaneous flaps that may be used for coverage of wounds without significant dead space. The latissimus dorsi, serratus anterior, and rectus abdominis are muscle flaps that are frequently used because of large vessel diameter and long pedicle length. These flaps additionally provide sufficient bulk to fill the dead space present after sequestrectomy. A skin paddle may be incorporated, depending on the need for contouring or for monitoring perfusion of the flap.

Soft tissue coverage for chronic, infected wounds (osteomyelitis) usually follows débridement of infected bone and nonviable soft tissue and antibiotic therapy. Free tissue transfer using muscle flaps obliterates dead space, improves vascularity, and improves local immune function by enhancing leukocyte function.

Recipient vessel selection is determined by the vascular status of the lower extremity and the location of the wound. Both the anterior and posterior tibial arteries may be used proximal to the zone of injury. Vein grafts may be necessary. The anterior tibial artery distal to the zone of injury is easily accessible and may be suitable, provided that flow is pulsatile and sufficient for nourishing the flap. End-to-side anastomosis is preferred for the arterial anastomosis, especially if significant vessel size mismatch is present; it is also the only option in a single-vessel limb. The veins accompanying the arteries are generally used for end-to-end anastomosis with the deep venous system because veins in the superficial system (saphenous veins) have a greater propensity for spasm.

Bone Reconstruction

Bone reconstruction usually takes place 4 to 6 weeks after definitive wound coverage. Nonvascularized autogenous grafting is a well-established procedure with a high rate of success when used appropriately. This method is highly dependent on a healthy host vascular bed; extensive scarring or postradiation necrosis are examples for which this technique should not be used. Nonvascularized cancellous bone grafts may be used for small osseous defects (<6 cm), but they may also be used for larger defects if the fibula is intact and stable.

Vascularized bone grafts transfer their own vascular supply, as well as osteoinductive, osteoconductive, and osteoprogenitor elements. They generally do not undergo resorption, have satisfactory mechanical strength, and are more capable of resisting infection. Vascularized bone transfers are indicated for segmental defects larger than 6 cm or for situations in which the fibula is not stable. Patients treated for osteomyelitis may be candidates for vascularized bone transfers. Free or pedicled bone transfers may be performed. Pedicled transfers are limited by donor availability, as well as by pedicle length and position. The iliac crest and the fibula are donor sites commonly considered for free vascularized bone transfer. The fibula may be transferred alone or as composite transfer with skin and muscle; it can bridge defects up to 30 cm. The curvature of the iliac crest limits its effective length to approximately 10 cm. Other donor sites include the scapula, rib, metatarsal, radius, and skeletal tissue from dismembered parts.

Bone lengthening by gradual distraction (the Ilizarov procedure) may be used to correct malunion or nonunion. Acute shortening of bone to facilitate neurovascular repair along with use of the Ilizarov technique for bone lengthening at a distant site may be performed in certain circumstances.

Postoperative Care

Rehabilitation

High-energy lower extremity injuries require months to years of rehabilitation. The surgeon must establish communication with the patient and family early in the management process to explain and clarify the lengthy process, which may include secondary surgery. A protracted course of physical and occupational therapy is necessary to restore as much function as possible to the injured extremity.

Complications

Severe lower extremity trauma managed by either amputation or limb salvage results in a significant alteration in activities of daily living. The leg is subjected to hydrostatic pressures that increase the incidence of edema, deep venous thrombosis, and venous stasis. Compression garments or activity modification may be necessary. Many patients may have diminished function from a significant decrease in joint range of motion. Limitation of motion may also be due to muscle necrosis and fibrosis from both the injury and the muscle atrophy resulting from the long period of immobilization. The presence of stiff phalangeal joints can prevent not only shoe wearing but also normal gait. Patients may require the use of an assist device (cane or brace) for ambulation.

Tissue hypoxia and vascular injury may set the stage for length discrepancy, which is a common problem. Intentional foreshortening of the limb may be necessary to achieve nerve coaptation or reduce the soft tissue defect. Inequality is better tolerated and compensated for in the femoral component than in the tibia. Five centimeters or greater of inequality creates the need for additional procedures.

Lengthening and shortening of the involved extremity are the two options that must be discussed with the patient. Lengthening procedures mandate increased treatment time and additional hospitalizations. Patients must be carefully selected for this treatment modality because it is laborious and psychologically intensive. Scars, unsightly skin grafts, and bulky free flaps may be addressed by additional surgery to enhance aesthetic appearance.

Outcomes

Studies have shown that over a quarter of patients who were working before a severe lower extremity injury had not returned to work by 12 months after injury, with a fifth not returning to work after 30 months. Patient characteristics such as age, socioeconomic status, and preinjury psychosocial status obviously contribute to difficulties in household management, recreation, and social interaction. Outcomes after appropriate trauma care and rehabilitation continue to improve, but further research is needed to optimize reconstructive efforts.

Pearls and Pitfalls

- Many victims of high-energy trauma suffer a major disruption in their life activities, but those with a mutilated lower extremity represent a unique subgroup of trauma patients.
- The patients have greater expense at initial hospitalization, higher estimated loss of wages, a longer interval between injury and reemployment, and increased unemployment.
- The patients need to be made aware that the reconstructive process is expensive and entails a considerable time commitment.
- Many will have significant physical limitations, and some patients may not be able to return to their previous employment.
- In many situations, amputation may allow faster recovery and return to ambulation.

SUGGESTED READINGS

Bosse MJ, MacKenzie EJ, Kellem JF, et al: An analysis of outcomes of reconstruction or amputation after leg-threatening injuries. N Engl J Med 347:1924-1931, 2002.

Burgess AR: Emergency evaluation. In Yaremchuk MJ, Burgess AR, Brumback RJ (eds): Lower Extremity Salvage and Reconstruction. New York, Elsevier, 1989, pp 31-40.

Byrd HS, Spicer TE, Cierney G: Management of open tibial fractures. Plast Reconstr Surg 76:159, 1989.

Godina M: Early microsurgical reconstruction of complex trauma of the extremities. Plast Reconstr Surg 76:285, 1986.

May JW Jr, Gallico GG, Jupiter J, Savage RC: Free latissimus dorsi muscle flap with skin graft for treatment of chronic bony wounds. Plast Reconstr Surg 73:641, 1984.

Yaremchuk MJ, Brumback RJ, Manson PN, et al: Acute and definite management of traumatic osteocutaneous defects of the lower extremity. Plast Reconstr Surg 80:1, 1987.

The Diabetic and Ischemic Lower Extremity

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GREGORY A. DUMANIAN

Current estimates are that 5% to 10% of the U.S. adult population suffers from diabetes mellitus. Approximately 50% of the diabetic population has some evidence of lower extremity arterial insufficiency, and a nonhealing lower extremity ulcer will develop in 15% of diabetics within their lifetime. Of those in whom a foot ulcer develops, osteomyelitis will develop in 15%, and 15% will require some level of amputation secondary to osteomyelitis, tissue loss, or uncontrollable infection. Approximately 50,000 amputations are performed yearly in the United States, and fully 60% to 70% are in diabetic patients. Problems related to diabetic lower extremity ulcers account for billions of dollars in health care costs annually.

There is a wide spectrum of patients with lower extremity diabetic and ischemic ulcers. Many ulcers respond to behavior modification and routine local wound care. Others require débridement with or without some degree of vascular intervention. Finally, a group of patients have such significant tissue loss that revascularization alone does not suffice to close all wounds and provide a stable foot for ambulation. In these cases, the reconstructive surgeon can mean the difference between amputation and limb salvage. It is in this group of patients that free tissue transfers in combination with bypass surgery are used for extended limb salvage. Extended limb salvage is defined as procedures that combine arterial bypass surgery and free tissue transfer in patients with lower extremity wounds who would otherwise undergo below-knee amputations. In properly selected patients, extended limb salvage with free flaps and bypass grafts is an appropriate procedure to maintain bipedal ambulation and preserve quality of life. This chapter is designed to outline treatment options for patients with diabetic and ischemic lower extremity ulcers, with a focus on the use of free tissue transfer for salvage of the threatened lower extremity.

Etiopathogenesis/Causative Factors

The etiologic factor clearly involved in a patient with isolated lower extremity ischemia is atherosclerotic occlusive disease. However, the diabetic milieu is not one of atherosclerosis alone. Rather, an environment exists in which the pathologic mechanisms of neuropathy, ischemia, immunopathology, and infection work in concert. This pathophysiologic interplay typically results in greater tissue perfusion requirements to maintain or regain skin integrity in diabetic subjects than in patients with nondiabetic ischemic disease.

Neuropathy is linked to autoimmune mechanisms with resultant demyelination, nerve edema, and slowed nerve conduction. Sensory, motor, and autonomic neuropathies typically result. Ischemic disease in diabetic patients tends to be histologically similar to that in nondiabetic individuals, but it manifests in anatomically unique locations. Disease tends to be isolated to an infrapopliteal, but supratarsal region of the lower extremity. Polymorphonuclear cell dysfunction is a hallmark of diabetic immunopathology. Cell migration, phagocytosis, and opsonization are all dysfunctional. The magnitude and extent of physiologic and anatomic dysfunction in a diabetic lower extremity ultimately reflect the degree of neuropathy, ischemia, and immunopathology, which in turn reflect the severity and duration of hyperglycemia.

Charcot's Neuroarthropathy

Charcot's neuroarthropathy is present in the feet of 3% to 5% of all individuals with diabetes. Motor neuropathy leads to intrinsic muscle weakness, which results in dysfunction of the foot flexion-extension balance and an "intrinsic-minus" foot (analogous to patients with ulnar nerve dysfunction in the hand).

The toes extend at the metatarsophalangeal joints and flex at the interphalangeal joints because of absent function of the foot intrinsic muscles, thereby resulting in a "claw foot" deformity. Other gross findings include pes cavus deformity, digital extensor subluxation, and prominence of the metatarsal head.

Loss of proprioceptive protective sensory input in the setting of this deranged anatomy potentiates further destruction of articular surfaces and joint subluxation. The subsequent loss of normal pedal arches leads to the redistribution of peak plantar pressure to focal areas of the foot, including the metatarsal heads and calcaneus. Correlations between areas of high focal pressure and sites of ulceration support a mechanical contribution to the initiation of wound problems in the diabetic foot.

Mal Perforant Ulcers

Sensory neuropathy and autonomic neuropathy work in collusion in the diabetic foot to cause a mal perforant ulcer, often the inciting event in a cascade leading to skin breakdown, infection, osteomyelitis, and amputation. Autonomic dysfunction causes anhidrosis and hyperkeratosis with hard, dry callus formation on insensate, pressure-bearing areas of the anatomically deranged diabetic foot. Concomitant sensory neuropathy then facilitates unrestricted, pain-free weight bearing of hard, keratotic material on the underlying dermis, which can lead to fissure formation and full-thickness dermal necrosis and ulceration. The resultant mal perforant ulcer, typically located at the metatarsal heads, provides ready access for bacteria. The preexisting ischemic environment and the immunopathology of hyperglycemia facilitate the development of an invasive, polymicrobial infection. Often, even invasive infections go unnoticed because of sensory impairment. It is not until the development of tissue necrosis or systemic infection that medical attention is sought.

Microcirculation

A common misconception exists regarding the unique atherosclerotic changes in the microcirculation of diabetic patients. The misconception is that there is obstruction and occlusion of vessels at the capillary and tissue perfusion level in diabetic patients suffering from peripheral vascular disease. The clinical implications of this concept of "small vessel disease" would argue against the potential efficacy of any arterial reconstruction in the diabetic lower extremity. Both clinical experience and multiple studies have failed to demonstrate any evidence of microcirculatory occlusion in diabetic patients. This unproven concept lingers and thereby adds to a sense of futility in the treatment of diabetic foot wounds. There is evidence, however, that capillary basement membranes are thickened in diabetic patients, but no evidence of obstruction in the capillary lumen diameter has been correlated with the thickening. It is postulated, but remains unproved, that diabetic vessels are resistant to the diffusion of nutrients and waste products as a result of basement membrane thickening. No significant differences in transcutaneous Po_2 levels have been found between diabetic and nondiabetic patients with peripheral vascular disease, further debunking the myth of "small vessel disease" unique to diabetes.

Pathologic Anatomy

Patterns of Vascular Disease

Although histologic examination of lower extremity atherosclerotic lesions in diabetic and nondiabetic subjects reveals similar findings, the anatomic distribution of these lesions is unique to each group. A clear understanding of these patterns is critical to approaching wound management in diabetic patients. Diabetic patients manifest occlusive lesions in the distal popliteal and tibioperoneal arteries with relative sparing of vessels in the foot, particularly the dorsalis pedis. Profunda femoris occlusive lesions tend to be at least three times as common in diabetic subjects, whereas aortic disease tends to occur only half as frequently as in nondiabetic controls. The diseased vessels also tend to demonstrate a significant degree of calcification within the arterial media, an important technical issue when considering microvascular reconstruction in the lower extremity.

Pedal sparing provides the opportunity for distal revascularization in many patients. Patients with apparently ischemic feet and diseased tibioperoneal vessels may in fact have reconstituted dorsalis pedis and posterior tibial vessels, thus providing an array of treatment options beyond amputation. In addition to pedal sparing, nonsmoking diabetic patients typically demonstrate sparing of the superficial femoral and proximal popliteal vessels. This finding is in sharp contrast to patients with nondiabetic lower extremity occlusive disease, who most frequently have occlusion of the superficial femoral system, often at the adductor hiatus or Hunter's canal.

The clinical importance of these patterns is that successful limb salvage by arterial reconstruction alone often requires use of the superficial femoral artery or proximal popliteal vessels as inflow sources and the distal tibial or dorsalis pedis vessels as outflow targets. It is the refinement of such distal bypass procedures that has helped to improve outcomes in many diabetic patients.

Distribution of Wounds

Foot wounds can be grouped into one of several categories in the discussion of extended limb salvage. Pressure sores occur over the metatarsal heads

(the so-called mal perforant ulcer), on the midfoot between the sole and the metatarsal heads in patients with Charcot feet, and on the posterior aspect of the heel and calcaneus in bedridden patients. Dorsal foot wounds occur in patients with severe vascular disease in which the anterior tibial system is more severely affected than the posterior tibial system. Distal foot wounds are most typically due to infections that cause abscesses in the web spaces. The patient is late to recognize early signs of pain and swelling because of neuropathy, and the infection can cause significant tissue loss. These distal wounds can even occur in patients with strong ankle pulses.

Diagnostic Studies

Clinical evaluation of a diabetic patient with a lower extremity ulcer remains the most critical "diagnostic study" available. The ultimate questions to answer are "Will this wound heal with local wound care alone?" and "How extensive will surgical intervention need to be?" Serial examinations are critically important. Serial clinical evaluations often provide the best indication regarding whether a wound is vascularized sufficiently to heal spontaneously with good local wound care. After débridement, one can detect the formation of new granulation tissue or, conversely, the extension of tissue necrosis as the wound margins grow.

It is critical to decide whether a diabetic lower extremity wound is clean or infected and, if infected, whether it requires immediate débridement. The immunopathology and vascular inadequacy of many diabetic patients make the potential for overwhelming infection in the lower extremities much greater than in patients with atherosclerosis alone. Therefore, diligence is mandatory when evaluating these patients. Potential signs of systemic infection are noted, including fever, tachycardia, hypotension, or leukocytosis. The presence of any of these signs raises suspicion that an occult deep space infection exists. The extremity is evaluated for erythema, warmth, significant pain with palpation, or loss of soft tissue contours, which may indicate deep space infection or purulent fluid collections. The contralateral foot is often helpful in providing comparison. Although neuropathy is the rule in diabetic feet, most diabetic patients still demonstrate pain with a significant deep space infection or with undrained pus in the lower part of the leg or plantar compartments.

The wound is assessed for size, geometry, depth, and location. As these features are delineated, the requirements for a healed, stable wound become clear. Full-thickness wounds are carefully evaluated for exposed underlying structures, including tendons, bone, joint spaces, or vascular structures. Exposed bone is palpated to determine whether any soft areas (denoting osteomyelitis) exist. Necrotic tissue, soft bone, and desiccated tissue are noted and appropriate débridement planned. Wounds on the plantar arch typically require less durable coverage than those on the calcaneus. These factors help one to begin formulating a treatment plan.

Orthopedic abnormalities are noted and appropriate consultation obtained for defects that might be amenable to surgery. Tendon transfers, Achilles tendon lengthening, and joint fusions have been shown to stabilize and rehabilitate the diabetic foot, thereby decreasing reconstructive requirements by providing a more stable plantar platform. Nerve dysfunction is evaluated to look for lack of intrinsic muscle function and documented quantitatively with Semmes-Weinstein monofilaments to detect plantar sensory changes. Tinel's sign over the tarsal tunnel may indicate nerve compression. If found, tarsal tunnel release may improve plantar sensation. However, nerve conduction tests are not generally helpful.

Plain radiographs show the results of previous surgery, Charcot joints, osteomyelitis and bone destruction, occult foreign bodies, and calcification of blood vessels. Magnetic resonance imaging is also used to look for areas of undrained pus and to evaluate for osteomyelitis.

Vascular Evaluation

Wound healing requires adequate arterial inflow. Assessment of lower extremity pulses begins in the groin and ends at the foot, with the pulses being graded as present, weak, or absent. If pulses are not strong, noninvasive arterial Doppler studies with determination of arterial pressure are in order. Diabetic subjects, 30% of whom have calcifications significant enough to impede blood pressure cuff compression of lower extremity arteries, tend to have sparing of the digital vessels from calcifications. Toe pressure less than 30 mm Hg indicates ischemia. Pulse volume recordings also overcome some of the difficulty associated with lower extremity arterial calcifications; amplitudes at the transmetatarsal level greater than approximately 10 mm Hg indicate minimally adequate inflow. Biphasic or triphasic arterial waveforms are usually consistent with sufficient vascularity for wound healing, whereas monophasic waveforms are associated more with extremity ischemia.

If noninvasive testing is indicative of ischemia, an angiogram is obtained. Angiography remains the gold standard for vascular evaluation of the lower extremity. Angiography provides the information required to determine whether revascularization of an ischemic extremity is possible and, if so, whether angioplasty, stenting, arterial bypass grafting, or some combination of these procedures will be required to establish adequate inflow.

Angiograms demonstrate the availability of inflow and outflow vessels for arterial reconstruction

and aid in selecting the most appropriate distal arterial target for microvascular anastomosis. Lower extremity angiography is extremely helpful before undertaking free tissue reconstruction of the lower extremity in diabetic patients, even in the rare patients with pedal pulses who would not otherwise have undergone an angiogram. Magnetic resonance angiography, although routinely used in patients with renal insufficiency, is not as helpful in evaluating donor vessels for free tissue transfer at the ankle level as is traditional angiography.

Treatment: Nonsurgical

Medical optimization is critical to establish the most effective immunologic and wound healing physiology in the patient. Adequate caloric, vitamin, and mineral intake should be ensured. Vitamin C (ascorbic acid) and zinc have important roles in wound healing, as does vitamin A (retinoic acid) in patients taking steroids.

Nutritional optimization works in tandem with pharmacologic therapy to establish a euglycemic state with strict control of blood glucose levels. This goal often requires significant patient education in terms of nutrition, glucose-monitoring practices, and insulin or oral diabetic medication management. Many of the immunologic abnormalities of diabetes have been shown to be acutely reversible with the establishment of a euglycemic state.

Cardiac evaluation is key not only in cases in which surgery is anticipated but also in cases in which conservative therapy alone is planned. Optimal cardiac function ensures the best possible perfusion of the lower extremity, improves lymphatic function, and optimizes right heart pressures, thereby improving peripheral venous return and contributing to the management of lower extremity edema.

When surgery is planned, a thorough preoperative workup is mandatory. Neuropathy can mask chest pain, so stress testing is often indicated. The "anginal equivalent" of classic chest pain in a diabetic patient might be nausea or ear pain, whereas some patients have totally occult ischemia. When claudication or foot wounds limit the patient's ability to participate in exercise stress testing, dipyridamole thallium stress testing is performed to evaluate for myocardial perfusion deficits or ventricular wall motion abnormalities. Those with stress testing suggestive of ischemia typically undergo coronary angiography and are risk stratified depending on the results.

As more information is obtained regarding a given patient's medical status, the appropriate approach to lower extremity wound management often becomes obvious. Although a patient may not be considered capable of tolerating a lengthy extended limb salvage procedure, débridement followed by subsequent skin grafting may be a reasonably safe alternative. Additionally, the greater risk of a lengthier procedure may cause some patients to opt for the more expeditious, hemodynamically conservative approach of amputation. Ultimately, an open dialogue must exist between the patient, reconstructive surgeon, and internist involved in the patient's care.

Reconstructive Goals

The ultimate reconstructive goal for any patient with a diabetic foot ulcer is to establish a healed, viable extremity that maximizes function. The more simply this goal can be achieved, the better. Unfortunately, complex reconstructive procedures involving free tissue transfer are sometimes required when sizable tissue loss prevents more standard wound closure. The first priority in the care of a diabetic foot wound is to drain infected collections and remove necrotic tissue. Deep space infections typically do not elicit a large inflammatory response in patients with ischemic tissues. Infections often travel along tendon sheaths proximally. Soft bone should be excised. Patients are started on a regimen of broad-spectrum antibiotics to cover polymicrobial and anaerobic infections. However, antibiotics are not a substitute for surgical treatment of the diabetic wound or for improvement of blood flow.

Five broad categories of patients with diabetic lower extremity wounds can be considered for free flap surgery. The reconstructive goals and thought processes differ, and thus each group is considered separately. There is significant overlap among the groups because of the multifactorial nature of diabetic foot ulcers.

Ischemic Extremity with Tissue Loss

These patients have the most difficult feet to assess for tissue viability. Often, the necrosis is in a distribution of either the dorsalis pedis on the dorsal aspect of the foot or the heel and plantar surface of the foot, the latter suggestive of disease in the posterior tibial system. Tissue loss is not always at the most distal aspect of the foot. The added factors of pressure (affecting the heel commonly) or an abscess causing tissue destruction may result in more proximal wounds.

Patients with ischemic legs and tissue loss are first evaluated by the vascular surgeon to determine suitability for a bypass graft. The plastic surgeon's input is also appropriate at this stage because some patients have such significant wounds that no single free flap would adequately address all of the tissue loss. An example is simultaneous wounds of the Achilles tendon (a posterior free flap needed) and the dorsal surface of the foot (an anterior free flap required). If one large flap is inadequate to provide wound coverage, the foot is not considered salvageable.

Ischemic tissue loss of the foot is initially difficult to gauge in terms of extent and severity. Therefore, the best way to manage these wounds is to perform limited débridement of necrotic tissue at the time of the leg revascularization procedure. Delayed definitive débridement and wound closure are performed when the foot soft tissues have stabilized. A granulating wound with stable edges that will not undergo further necrosis facilitates adequate flap adherence to the wound bed. A delay between vascular bypass and free flap coverage has additional advantages. It allows some patients to avoid free flap surgery if other options such as transmetatarsal amputation or even a simple skin graft become possible as a result of the improvement in blood flow. Delayed flap surgery also allows assessment of the quality of the revascularization. Some wounds do not clinically improve despite surgical revascularization, and the wounds remain atrophic. For these patients, more time must be given to respond to the augmented inflow, the vascular team must perform a more distal bypass jump graft, or the attempt at extended limb salvage should be abandoned.

Pressure-Induced Tissue Loss

The approach to these patients is education and pressure relief, not a free flap procedure. No flap can withstand the relentless stress of the weight of the human body in an area that has already responded to such stress with tissue necrosis. Patients need relief of pressure, education, properly fitting pressure relief shoes, and local wound control to allow the wounds to heal without surgical intervention more complex than débridement. If the wound is slow to heal and there is no ankle pulse present, the vascular surgery team should consider a bypass even if by absolute criteria the leg is not frankly ischemic.

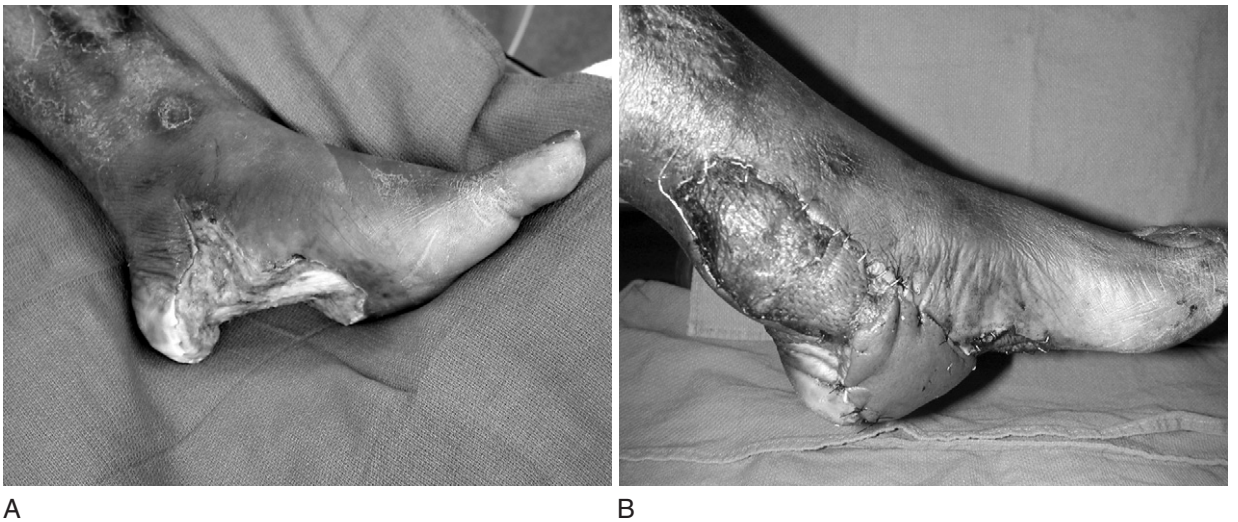
Some patients demonstrate "regional ischemia." Despite a pulse at the ankle, some foot wounds have ischemic features. One example is a patient with a pressure-induced calcaneal wound and a dorsalis pedis pulse. If the wound is slow to resolve despite adequate local wound management, an arteriogram should be considered to assess the status of blood vessels near the wound. Patients with regional ischemia may have a patent dorsalis pedis, but absent posterior tibial circulation. Such patients with pressure sores and regional ischemia may benefit from free flap reconstruction if local wound management does not succeed.

Infection-Induced Tissue Loss

These patients often have pulses, but they have had a necrotizing infection of such magnitude that local wound care or standard foot amputations, or both, are inadequate to achieve a stable foot. The approach to these patients is to drain all collections, débride necrotic tissue, and allow time to pass for the inflammation and edema to resolve. If the foot does not respond quickly and if no pulse is present at the ankle, a bypass should be considered even if by absolute criteria the leg is not frankly ischemic (as in patients with pressure-induced tissue loss). If vascular bypass results in a wound that is stable and improving and if no other options exist, a free flap can be considered for closure. This group tends to have the least complicated postoperative recovery (Fig. 1).

Tissue Loss over the Target Bypass Vessel

A small subset of patients have ischemic tissue or even frank tissue loss overlying a proposed



A B

Figure 1 • A, Diabetic foot wound. **B,** Healed wound after free flap coverage.

anastomotic site. These patients require a simultaneous bypass and free flap, with the free flap used to cover the macrovascular anastomosis and, in some cases, part of the length of the bypass graft. Gentle handling of tissues and precision are critical while tunneling the bypass graft because skin traumatized by surgery in an ischemic leg can become necrotic and thereby result in exposure of the bypass graft at a site distant from the newly placed free flap. When such secondary areas of tissue loss develop, this group of patients becomes very difficult to treat successfully.

Ischemic Leg with Tissue Loss and No Target Vessel

Patients in this category have ischemic feet, but no target vessel for a distal bypass or anastomosis. This category of patients requires indirect revascularization in which the interface between a newly placed free flap and the wound bed serves to transfer blood between the flap and the foot. The flap "footprint" should be larger than the wound to maximize the amount of neovascularization between the flap and the foot. The microvascular arterial anastomosis may require a vein graft from a more proximal site, depending on the length of the flap pedicle. The venous outflow tract is typically the posterior tibial system.

These five aforesaid groups certainly have areas of overlap, and the cause of wounds in many instances is multifactorial. In each case, a decision is made regarding whether vascular bypass is necessary. If noninvasive vascular testing implies satisfactory blood flow to the foot for wound healing, the wound is observed and local wound care performed. When a wound fails to progress toward healing, an arteriogram is performed to define the vascular anatomy and rule out cases of regional ischemia. An ankle pulse need not be present for successful skin grafts and local flaps. However, if a free flap is to be performed, it is preferable that an unambiguous pulse be present at the anastomotic site. If the pulse is subtle, an arteriogram is required to confirm the presence of a target vessel in the area with in-line flow from the groin.

Treatment: Surgical

Although the chapter focuses on salvage procedures for diabetic and ischemic lower extremities, local flaps and skin grafts remain the first line of treatment if they fulfill the goal of a stable ambulatory platform. Their full discussion is beyond the scope of this chapter.

Flap Selection

The choice of free flap is a matter of surgeon preference, defect size and shape, and patient characteristics. The concept of vein mismatch is important, and in accordance with this idea, flaps that are less likely to overload a venous drainage system are chosen (smaller and lower-flow flaps). The gracilis flap with a skin graft or the radial forearm flap are often used because of their consistently small vessels and relatively lower flow. For larger defects, flaps based on the subscapular system are useful. Vessels of the subscapular system tend to have less wall calcification than do flaps based near the groin, particularly in diabetic patients. Although vessel calcification is an occasional issue with flaps based near the groin, the vessels are usable in the vast majority of cases. Finally, omental flaps are reserved for indirect revascularization because of their high percentage of blood vessels and malleable shape. These flaps have the risks associated with an intraabdominal procedure, including hernia, bowel obstruction, and bowel injury.

Technique

The patient is gently hydrated the night before surgery, and several units of blood are typed and crossed. An epidural catheter can be helpful for postoperative analgesia and sympathectomy, but it is not routine.

Patient positioning is critical in facilitating simultaneous flap harvest and microsurgery (flap inset). When the posterior tibial system is used, the hip is abducted and externally rotated so that the medial aspect of the lower part of the leg provides a horizontal surface for microsurgery. A beanbag is useful to hold the leg in this position for the duration of the case. When the anterior tibial system is used, the patient is tilted in the opposite direction so that the lateral aspect of the leg is parallel with the floor and facing superiorly. Flap selection is based on the expected recipient vessel choice and the anticipated intraoperative patient position. An extra arm board placed at the end of the operating table makes the table several inches wider, thereby facilitating stabilization of the surgeon's forearms and elbows during microsurgery.

If foot débridement is required, it is performed at the start of the procedure to ascertain the wound dimensions. The foot is débrided sharply and the wound pulse-lavaged to reduce the bacterial load. Free flap harvest is begun after débridement. Hemostasis is crucial because of the need for postoperative anticoagulation. The flap is not made ischemic until the recipient vessels are fully dissected, the vascular bypass is functional, and the microscope is in position.

The dorsalis pedis artery is located between the extensor hallucis and the extensor communis tendons and underneath the extensor brevis muscle. The distal aspect of the extensor retinaculum may need to be divided to expose the vessel. At the level of the base of the first metatarsal, the artery divides with a large branch descending to communicate

with the plantar arterial system. The optimal place, therefore, for an arterial anastomosis is proximal to this descending branch and distal to the main portion of the extensor retinaculum. The venae comitantes of the dorsalis pedis are approximately 1 mm in diameter, and the presence of multiple side branches makes dissection difficult. Large 2- to 3-mm subcutaneous veins on the dorsum of the foot can be used for microvascular anastomoses if there has not been a significant amount of dorsal skin inflammation from infection. The dorsalis pedis artery is useful for free flaps resurfacing the dorsal aspect of the foot and for free flaps covering the metatarsals.

The posterior tibial artery is located posterior to the medial malleolus. Arterial mobilization is facilitated by opening the tarsal tunnel from the distal lower part of the leg to the level of the first toe abductor. The posterior tibial venae comitantes are usually of adequate caliber and quality and range in size from 1.5 to 3 mm. The posterior tibial artery is the easiest donor vessel to use for coverage of the heel and the plantar surface of the foot.

Another critical component of the procedure is selection of the flap venous outflow. Both the superficial veins and the deep venae comitantes can be used. Good communication between the vascular and plastic surgery teams is essential to preserve all possible outflow veins at the time of the bypass procedure. The deep venous system is preferred because the superficial veins are more easily compressed to the point of occlusion by dressings and tight skin closure. In patients with marginal venous outflow, early testing of venous patency should be performed by injecting heparinized saline into the vein. The solution should flow easily. Patients with venous hypertension in the leg as evidenced by serous transudate, persistent leg swelling, and skin trophic changes will be difficult to treat with free flaps because of problems with venous outflow. Compression, elevation, and local wound care should be used until the tendency toward venous hypertension improves.

Diabetic arteries are frequently calcified and difficult to manipulate. Obtaining temporary arterial occlusion to facilitate visualization during microvascular anastomosis can be problematic. Macrovascular vessel-occluding instruments must occasionally be used rather than microsurgical vessel clamps, and thigh and lower leg tourniquets are sometimes required. The recipient artery should be palpated to select the softest area for the microanastomosis, and an ellipse of arterial wall should be excised rather than simply making an arteriotomy. On calcified vessels, sutures should be placed from the "inside out" to keep the intima tacked to the adventitia. The majority of arterial anastomoses are performed in end-to-side fashion to preserve any existing arterial inflow to the foot. Finally, the calcified vessels are difficult to pierce for placement of the 9–0 and 10–0 nylon sutures commonly used in microsurgery. Prolene 8–0 sutures can be used in these situations. In certain cases, the flap artery can be sutured to a vein bypass graft. In these cases, the arterial anastomosis is usually straightforward because the vein graft is soft and without calcifications.

Critical maneuvers such as dissecting the recipient vessels before making the flap ischemic, having the proper arterial occluding clamps readied, and having the microscope draped and in position, reduce flap ischemia times.

Postoperative Care

Postoperative anticoagulation is decided by the clinical scenario. Factors such as the difficulty of the anastomoses, vein quality, arterial inflow, and the tendency of the débrided foot to bleed play a role in this decision. Typically, patients receive subcutaneous heparin at the start of the procedure. Ketorolac is given during the microanastomosis for its antiplatelet effect. If there are any intraoperative thromboses and an anastomosis is revised, postoperative heparin infusions at a relatively low dose of 600 to 1000 U/hr are begun.

Free flaps to the foot tend to bleed postoperatively as a result of postoperative anticoagulation and areas of newly débrided bone. With this in mind, flap insets are relatively simple because it is not uncommon to reelevate a flap and evacuate a small hematoma. Penrose drains are left under the flap because closed suction drains typically do not maintain their suction. In the intensive care unit, the hematocrit is determined frequently to ensure that patients do not become anemic as a result of continuous oozing from the foot. Flap checks are performed with an implantable venous Doppler device every 30 minutes for the first 48 hours. Legs are kept elevated in a posterior plaster mold for 1 week to reduce leg and foot motion and promote flap adherence.

The patient is sent to either a wound care facility for further leg elevation or home if sufficient family support is available. For the first week there is no leg dangling. For the second week, the leg is dangled up to 15 minutes at a time, three times a day, with an elastic bandage wrapped around the foot to prevent swelling. If healing progresses and flap edema is minimal, the patient can begin crutch walking during the third postoperative week with the use of continuous elastic bandage compression but no weight bearing on the involved extremity. Weight bearing is begun by 6 weeks, with full weight bearing on the flap by the eighth postoperative week.

Pearls and Pitfalls

- Critical to the successful outcome of these procedures is a close working relationship with the

vascular surgery team. Patients should be evaluated by all services at the initial assessment, at the time of the first débridement, during the bypass procedure, and at the time of the free flap procedure. If the limb salvage effort is judged to be unfeasible from the perspective of either team, the procedure is abandoned. Close cooperation with the vascular team allows for the most optimal placement of the distal arterial bypass graft and preservation of venous outflow.

- Adjunctive procedures are helpful at the time of the free flap transfer. While exposing the posterior tibial vessel at the ankle, tarsal tunnel release can be performed to improve plantar sensation. Orthopedic input is helpful to assess for possible Achilles tendon lengthening or for a tendon-balancing procedure.
- The proximal limit for forefoot amputations is the base of the first metatarsal because this is the site of insertion of the anterior tibial tendon. Disinsertion of the tendon leads to plantar flexion of the foot, altered foot mechanics, and early breakdown of the distal-most aspect of the stump. Therefore, when wound closure necessitates removal of the anterior tibial tendon, either it will need to be reconstructed under the new flap or an amputation will need to be performed.
- It is best to select a flap donor site that will not be problematic if the wound breaks down. For this reason, the rectus muscle flap is generally avoided because breakdown of the donor site can lead to a troublesome hernia. Given the wide size spectrum between the latissimus, gracilis, and radial forearm flaps, almost any size foot defect can be covered.
- More thought is often given to wound coverage than to coverage of the microanastomosis. If the flap is used to cover the anastomosis, part of the flap and pedicle length may be committed to an area outside the wound and thus an even larger flap will be required than initially anticipated. One solution is the flexor carpi radialis/radial forearm combination free flap. The forearm skin is used to cover the wound, and the flexor carpi radialis can be used to cover the more proximal microanastomosis.

SUGGESTED READINGS

Attinger CE, Ducic I, Neville RF, et al: The relative roles of aggressive wound care versus revascularization in salvage of the threatened lower extremity in the renal failure diabetic patient. Plast Reconstr Surg 109:1281-1290, discussion 1291-1292, 2002.

Godina M, Arnez ZM, Lister GD: Preferential use of the posterior approach to blood vessels of the lower leg in microvascular surgery. Plast Reconstr Surg 88:287-291, 1991.

McCarthy WJ 3rd, Matsumura JS, Fine NA, et al: Combined arterial reconstruction and free tissue transfer for limb salvage. J Vasc Surg 29:814-818, discussion 818-820, 1999.

Ramsey SD, Newton K, Blough D, et al: Incidence, outcomes, and cost of foot ulcers in patients with diabetes. Diabetes Care 22:382-387, 1999.

Sukkar SM, Saulis AS, Dumanian GA: Radial forearm skin with flexor carpi radialis muscle: A useful composite free flap. Ann Plast Surg 49:486-489, 2002.

Foot and Ankle Reconstruction

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The foot is subject to more repetitive trauma than any other part of the body. On average, an individual takes 10,000 steps a day. The foot tolerates such trauma by possessing specialized plantar tissue that can withstand shear stress forces. Blunt or penetrating trauma (or both) can cause immediate breakdown of soft tissue and bone. In addition, changes in blood supply, sensation, immune status, and biomechanics render the foot susceptible to breakdown. An inability to salvage an injured foot usually leads to major amputation and lifetime dependence on prosthetic devices.

Because the foot is such a complex body part, salvage requires a multidisciplinary approach, ideally consisting of a vascular surgeon, a foot and ankle surgeon, a plastic surgeon familiar with local and microsurgical free flap techniques, an infectious disease specialist, a podiatrist, and a pedorthetist skilled in orthotics and assisted foot orthosis. To address the patient's medical comorbid conditions, medical specialists are often consulted, such as an endocrinologist, hematologist, rheumatologist, and dermatologist.

For the plastic surgeon, the first task is to convert the existing wound into a healthy wound by aggressive débriding techniques and the application of modern wound-healing techniques. Most wounds can then be closed by simple soft tissue techniques such as delayed primary closure, skin grafts, or local flaps. However, many wounds require more sophisticated techniques that mandate intimate knowledge of the local angiosomes, arterial blood supply, and flap anatomy. All reconstructions must be biomechanically sound to avoid recurrent breakdown.

Etiopathogenesis

Assessment of a foot and ankle wound begins with determination of the cause: traumatic, infectious, ischemic, neuropathic, venous, lymphatic, immunologic, vasospastic, neoplastic, self-induced, or any combination of these causes. Usually, trauma is the precipitating event, and comorbid conditions make it difficult for the wound to heal. The most frequent of such conditions include diabetes, peripheral vascular disease, and connective tissue disorders.

Diabetes

Five percent of all Americans have documented diabetes mellitus, and a foot ulcer eventually develops in 15% of them. Almost 15% of the health care budget of the United States is allocated to the management of diabetes, with a large segment used for the treatment of diabetic foot ulcers, gangrene, and Charcot foot collapse. Over half of all major amputations performed in the United States are in diabetic patients. A diabetic patient arriving at the emergency department with an ulcer faces a 24% risk of immediate major amputation. There is an eightfold variation in whether the patient undergoes immediate major leg amputation or a salvage attempt. The variability depends on a physician's approach to limb salvage and on the location and type of hospital. With a team approach, more than 97% of diabetic limbs can be salvaged. Salvage not only allows the patient to maintain quality of life but it is also far less expensive for the health care system than a major amputation.

Peripheral polyneuropathy is the major cause of diabetic foot wounds. More than 80% of diabetic foot ulcers have some form of neuropathy, usually a consequence of vascular and metabolic abnormalities caused by chronically elevated blood sugar. Elevated intraneural concentrations of sorbitol, a glucose byproduct that is considered one of the principal mechanisms for nerve damage, cause the nerve to swell, with further damage resulting in anatomically tight spaces such as the tarsal tunnel. The combination of nerve swelling and a tight anatomic compartment leads to the "double-crush syndrome," which can be partially reversed by nerve release surgery. Unregulated glucose levels elevate advanced glycosylated end product levels, which may induce microvascular injury by cross-linking collagen molecules. Decreased insulin levels, along with altered levels of other neurotrophic peptides, may decrease maintenance or repair of nerve fibers. Other potential causative factors of peripheral polyneuropathy include altered fat metabolism, oxidative stress, and altered levels of vasoactive substances such as nitric oxide.

It is not completely understood why distal autonomic and sensory neurons are preferentially targeted in diabetic peripheral polyneuropathy. The decrease in protective sensation prevents patients from responding appropriately to minor trauma. Such decreased sensation can be quickly assessed by testing whether the patient can feel 10 g of pressure applied to the foot with a 5.07 Semmes-Weinstein filament. An inability to feel the filament indicates that the patient has lost protective sensation and is at high risk for future foot breakdown. Autonomic neuropathy has two effects: anhidrosis and opening of arteriovenous shunts. Anhidrosis leads to cracking of the skin and provides points of entry for bacteria. Opening of arteriovenous shunts enables the blood to partially bypass the higher-resistance skin capillary system and thereby decreases blood flow to the skin. Motor neuropathy also occurs and most often affects the intrinsic muscles of the foot by causing loss of plantar flexion of the toes at the metatarsophalangeal level. The toe extensors then overpower the toe flexors, and a hammer toe formation develops. This condition creates an additional retrograde downward force on the metatarsal heads. The combination of loss of toe flexion and increased downward pressure on the metatarsal head during gait renders the insensate soft tissue under the metatarsal heads more vulnerable to breakdown.

Diabetes also affects joints and tendons, as in the Achilles tendon where advanced glycosylated end products cross-link the collagen molecules of the tendon so that it loses its elasticity and eventually shortens. The patient loses the ability to dorsiflex the ankle normally, and strain is placed on the apex of the skeletal arch of the foot at Lisfranc's joint, as well as on the metatarsal heads during gait. The former is thought to contribute to the Charcot collapse of the midfoot that occurs in 1 in 800 diabetic patients with devastating sequelae, including eventual major amputation. An inability to dorsiflex the ankle means that the metatarsal heads experience increased pressure for a longer portion of the gait cycle. The risk for soft tissue ulceration can be very high when the increased pressure is considered in association with the estimated 10,000 daily steps on feet suffering from motor and sensorimotor neuropathy. Lengthening the Achilles tendon may relieve the excess pressure on the plantar surface of the forefoot during gait and allow healing within 5 weeks and a reduction in the ulcer recurrence rate from 25% to 3%.

Diabetic patients have a depressed immune response and hence have increased susceptibility to infection. High blood glucose levels diminish the ability of polymorphonuclear leukocytes, macrophages, and lymphocytes to destroy bacteria. In addition, the ability to coat bacteria with antibiotics is diminished, which further shields bacteria from phagocytosis. As a result of the depressed immune state, diabetic patients are especially prone to *Streptococcus* and *Staphylococcus* skin infections. Deeper infections tend to be polymicrobial, with gram-positive cocci, gram-negative rods, and anaerobes frequently being present on culture.

Up to 60% of diabetic patients with nonhealing ulcers have concurrent macrovascular disease, with the arteries below the popliteal trifurcation most commonly being involved and the smaller arteries of the foot and ankle often spared. For this reason, distal bypass in a diabetic patient is frequently possible. Noninvasive arterial studies and especially ankle-brachial indices are misleading in diabetic patients because vascular calcification decreases the compressibility of vessels. Therefore, digital toe pressures greater than 30 mm Hg or tissue oxygen levels (TCO_2) higher than 40 mm Hg are considered more reliable indicators of the quality of blood flow.

Peripheral Vascular Disease

Atherosclerotic disease is a common cause of nonhealing foot ulcers, especially when combined with diabetes. Hypercholesterolemia, hypertension, and tobacco use are major risk factors for atherosclerosis. Other causes of ischemia in the foot include thromboangiitis obliterans (Buerger's disease, generally seen in young smokers), vasculitis, and thromboembolic disease.

Noninvasive arterial testing should be performed with a hand-held Doppler device over the posterior tibial artery, the dorsalis pedis artery, and the anterior perforating branch of the peroneal artery. If the signal obtained is only monophasic, the patient should be evaluated by a vascular surgeon.

Connective Tissue Disorders

Connective tissue disorders (systemic lupus erythematosus, rheumatoid arthritis, and scleroderma)

are frequently associated with Raynaud's disease, a condition characterized by distal vasospasm and cutaneous ischemia. Treatment frequently requires immunosuppressive drugs such as steroids or chemotherapeutic agents, which can further inhibit healing. The anti–wound-healing effects of steroids can be partially reversed with oral and topical vitamin A. In addition, almost half of vasculitis patients have a coagulopathy that leads to a hypercoagulable state. Therefore, a coagulation blood panel should be obtained on these patients, and if abnormalities exist, they should be treated with anticoagulants by the hematologist. Treatment of these ulcers is usually medical. Once the abnormalities have been identified and corrected, wound-healing adjuncts can help in healing the wound.

Anatomy

Vascular Anatomy

The foot and ankle consist of six angiosomes, or composites of tissue supplied by a given source (named) artery. Neighboring angiosomes are connected to one another by "choke" arteries, which can open when there is sufficient pressure difference between two angiosomes. An artery feeding one angiosome can support an adjacent angiosome via choke vessels. For a source artery to support angiosomes beyond the immediately adjacent angiosome, surgical delay techniques must be applied.

The following source arteries feed the angiosomes of the foot and ankle: the distal anterior tibial artery supplies the anterior aspect of the ankle, and its continuation, the dorsalis pedis artery, supplies the dorsum of the foot; the calcaneal branch of the posterior tibial artery supplies the medial and plantar aspect of the heel; the calcaneal branch of the peroneal artery supplies the lateral and plantar region of the heel; the anterior perforating branch of the peroneal artery feeds the anterolateral aspect of the ankle; the medial plantar artery feeds the plantar instep; and the lateral plantar artery feeds the lateral midfoot and forefoot region (Fig. 1). Note that the plantar aspect of the heel enjoys a dual blood supply from both the calcaneal branches of the posterior tibial and peroneal arteries. When gangrene at the heel is present, one has to suspect severe vascular disease, probably involving both arteries.

Because the foot is an end-organ, there are many arterial-arterial anastomoses that allow a duplication of inflow to the main source arteries of the foot. The arterial-arterial anastomoses provide a margin of safety if one of the main arteries should occlude. At the ankle, the anterior perforating branch of the peroneal artery is connected to the anterior tibial artery via the lateral malleolar artery. At Lisfranc's joint, the dorsalis pedis artery courses into the first interspace to connect directly with the lateral plantar artery. This arch is critical in determining the direction of flow within the anterior or posterior tibial arteries because it can be antegrade, retrograde, or both. In addition, the plantar and dorsal metatarsal arteries are linked to one another at Lisfranc's joint by proximal perforators and at the web space by distal perforators. The posterior tibial and peroneal arteries are directly connected underneath the distal Achilles tendon by one to three connecting arteries. By using Doppler and selective occlusion, one can determine the patency of the connections, as well as the direction of flow—critical information in planning the harvest of local and pedicled flaps or amputations.

Motor and Sensory Anatomy

The tibial nerve travels in the deep posterior compartment, innervates muscles of the deep and superficial posterior compartments (except the gastrocnemius muscle), and divides into the medial plantar and lateral plantar nerves. These nerves supply the motor branches to the intrinsic muscles of the foot (except the extensor digitorum brevis muscle). The common peroneal nerve passes around the lateral aspect of the fibular head before splitting into the superficial and deep branches. The deep peroneal nerve innervates the extensor muscles in the anterior compartment before it exits the extensor retinaculum to innervate the extensor digitorum brevis muscle. The superficial peroneal branch innervates the everting muscles of the lateral compartment before it exits to provide sensation to the lateral lower aspect of the leg and dorsum of the foot.

The sensory nerves to the foot and ankle travel more superficially than the motor nerves do, and their degree of function is a useful index for localizing trauma in the lower extremity and assessing possible high intracompartmental pressure. The lateral aspect of the ankle and foot is supplied by the sural nerve (L5, S1) and the lower anterior aspect of the leg and dorsum of the foot by the superficial peroneal nerve (L4, L5, S1). The deep peroneal nerve (L4, L5, S1) supplies only the first web space. The posterior tibial nerve supplies the sole of the foot via its three branches: the calcaneal branch (S1, S2) supplies the plantar aspect of the heel pad, the lateral plantar nerve (S1, S2) supplies the lateral two thirds of the sole and lateral $1\frac{1}{2}$ toes, and the medial plantar nerve (L4, L5) supplies the medial third of the sole and the medial $3\frac{1}{2}$ toes.

Diagnostic Studies

Evaluation of a patient with a foot wound or ulcer begins with a complete history and physical examination. Important points in the history include the

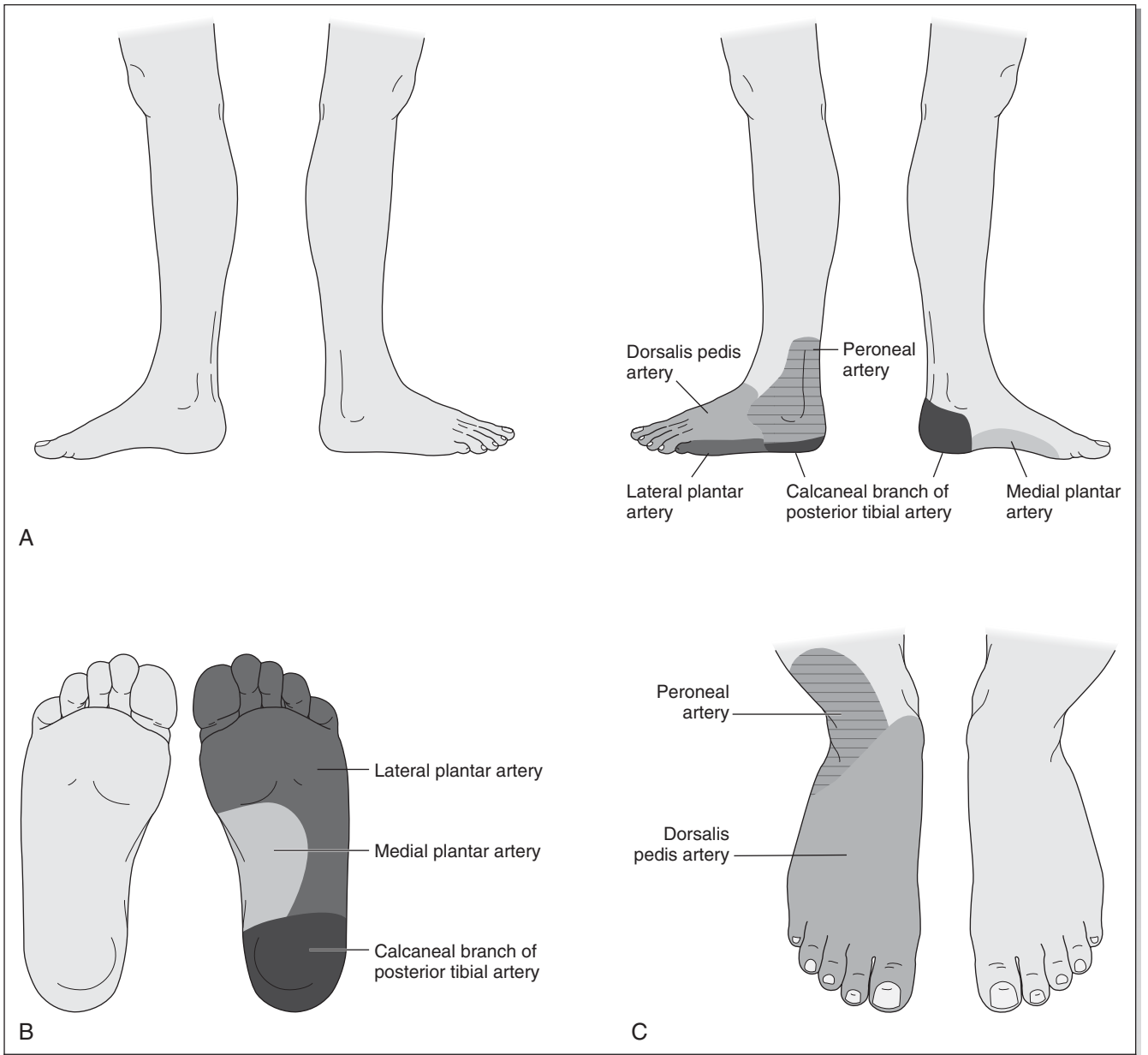

Figure 1 • The angiosomes of the foot and ankle include (**A**) the anterior aspect of the ankle supplied by the anterior tibial artery and the dorsum of the foot supplied by the dorsalis pedis artery; (**B**) the medial and plantar aspects of the heel fed by the calcaneal branch of the posterior tibial artery, the plantar instep supplied by the medial plantar artery, and the lateral plantar midfoot and plantar forefoot supplied by the lateral plantar artery; and (**C**) the anterolateral aspect of the ankle supplied by the anterior perforating branch of the peroneal artery and the lateral and plantar aspect of the heel supplied by the calcaneal branch of the peroneal artery.

cause, duration, and previous treatment of the wound or wounds; comorbid conditions; current medications; allergies; and nutritional status. A complete physical examination starts with careful wound measurement (length, width, and depth) and assessment of the types of tissue involved (epithelium, dermis, subcutaneous tissue, fascia, tendon, joint capsule, bone, or any combination of these tissues). Touching bone with a metal probe correlates 85% of the time with the existence of osteomyelitis. Diabetic ulcers with an area greater than 2 cm^2 have a 90% chance of underlying osteomyelitis regardless of whether bone is probed. The level of tissue necrosis and possible avenues of spread of infection via flexor or extensor tendons are also determined. If cellulitis is present, the border of the cellulitis is delineated with a marker and the date and time are noted. This approach permits the clinician, without the benefit of culture results, to monitor the progress of the initial treatment.

The vascular supply to the foot is examined. If pulses are palpable (dorsalis pedis or posterior tibial artery), the blood supply to the foot is usually sufficient. If one cannot palpate pulses, Doppler should

also be used to evaluate the anterior perforating branch and the calcaneal branch of the peroneal artery. This will help to determine the direction of flow in arteries that are important in flap design or amputation. A triphasic Doppler signal indicates excellent blood flow, a biphasic sound indicates adequate blood flow, and a monophasic sound warrants further investigation by a vascular surgeon. A monophasic tone does not necessarily reflect inadequate blood flow because it can be a result of lack of vessel tone and absent distal resistance. However, if only a monophasic signal is detected, the patient should be referred for formal vascular testing.

Sensory examination is performed with a 5.07 Semmes-Weinstein filament, which represents 10 g of pressure. If the patient cannot feel the filament, protective sensation is diminished with an increased risk of breakdown. Motor function is assessed by looking at the resting position of the foot and the strength and range of motion of the ankle, foot, and toes.

The bone is evaluated by determining whether the arch is stable, collapsed, or disjointed. A bony prominence can occur with collapsed midfoot bones (the cuboid in Charcot collapse), osteophyte formation, or abnormal biomechanical forces (hallux valgus, hammer toe, etc.) The Achilles tendon should also be evaluated. If the ankle cannot be dorsiflexed 10 to 15 degrees above neutral, the Achilles tendon is foreshortened and needs to be addressed because both the arch and plantar aspect of the forefoot are facing excessive pressure that could lead to Charcot collapse or forefoot plantar ulceration.

A radiograph of the foot is critical in further evaluating the foot. Lateral views of the foot should be weight bearing. Calcaneal, sesamoid, and metatarsal head views are used to examine these areas carefully. Radiographs permit evaluation of abnormal bone architecture, osteomyelitis, Charcot collapse, previous surgery, the presence of a foreign body, and other problems. The radiographic appearance of osteomyelitis lags behind the clinical appearance by up to 3 weeks. Magnetic resonance imaging can provide earlier detection of osteomyelitis and differentiation between osteomyelitis and Charcot collapse.

Reconstructive Goals

The goals of reconstruction are to maintain existing function and restore as much missing function as possible. If the wound is acute, attempts are made to restore normal anatomy as best as possible. If the wound is chronic, it must first be converted to an acute wound before it can heal in timely fashion. Both approaches include restoration of adequate blood supply, stabilization of the bony skeleton, rebalancing of the foot and ankle muscles, and finally, coverage of the wound with soft tissue.

Treatment

Reconstruction is guided by the principle that coverage of a wound should be accomplished as quickly as possible. After the wound is under control, the reconstructive ladder encompasses the following options: (1) allowing the soft tissue defect to heal by secondary intention, (2) closing the wound primarily, (3) applying a split- or full-thickness skin graft, (4) rotating or advancing a local random-pattern flap, (5) transferring a pedicled flap, and (6) applying a microvascular free flap. Useful guidelines suggest simple coverage if there is no tendon, joint, or bone involved. For more complex wounds with involvement of these structures, flaps (local, pedicled, or free) are usually necessary. The exception is that wounds over the Achilles tendon tolerate skin grafts if adequate granulation tissue first develops. Creative amputations that preserve length are often the most rapid way of obtaining adequate soft tissue with minimal sacrifice of function. With these criteria, more than 90% of all wounds can usually be closed by simple techniques and less than 10% require flap coverage.

Presurgical Treatment

The goal of treating any type of wound is to promote healing in timely fashion. The first step is to establish a clean and healthy wound base. An acute wound is defined as a recent wound that has yet to progress through the sequential stages of wound healing. If the wound is adequately vascularized, a clean base can be established with simple débridement and either immediate closure or coverage of the wound with a negative-pressure closure device (the vacuum-assisted closure [VAC] device) for subsequent closure. A chronic wound is a wound that is arrested in one of the wound-healing stages (usually the inflammatory stage). Converting a chronic wound to an acute one requires correcting medical abnormalities (elevated blood sugar levels, coagulation abnormalities, or improper drug regimens), restoring adequate blood flow, administering antibiotics if any infection is present, and débriding the wound aggressively. If the wound has responded to this type of therapy, healthy granulation tissue should appear, edema should decrease, and neo-epithelialization should appear at the wound's edge. The VAC device is a useful postdébridement dressing for an uninfected, well-vascularized wound because it will help to keep the wound sterile while promoting the formation of granulation tissue (Fig. 2). Healing should be characterized by a 10% to 15% decrease in wound area per week. If healthy granulation and neo-epithelialization are slow to appear, topical growth factors, cultured skin, or hyperbaric oxygen (or any combination) can be considered.

A useful approach to surgical débridement is to view a wound or ulcer as a tumor; the goal is to

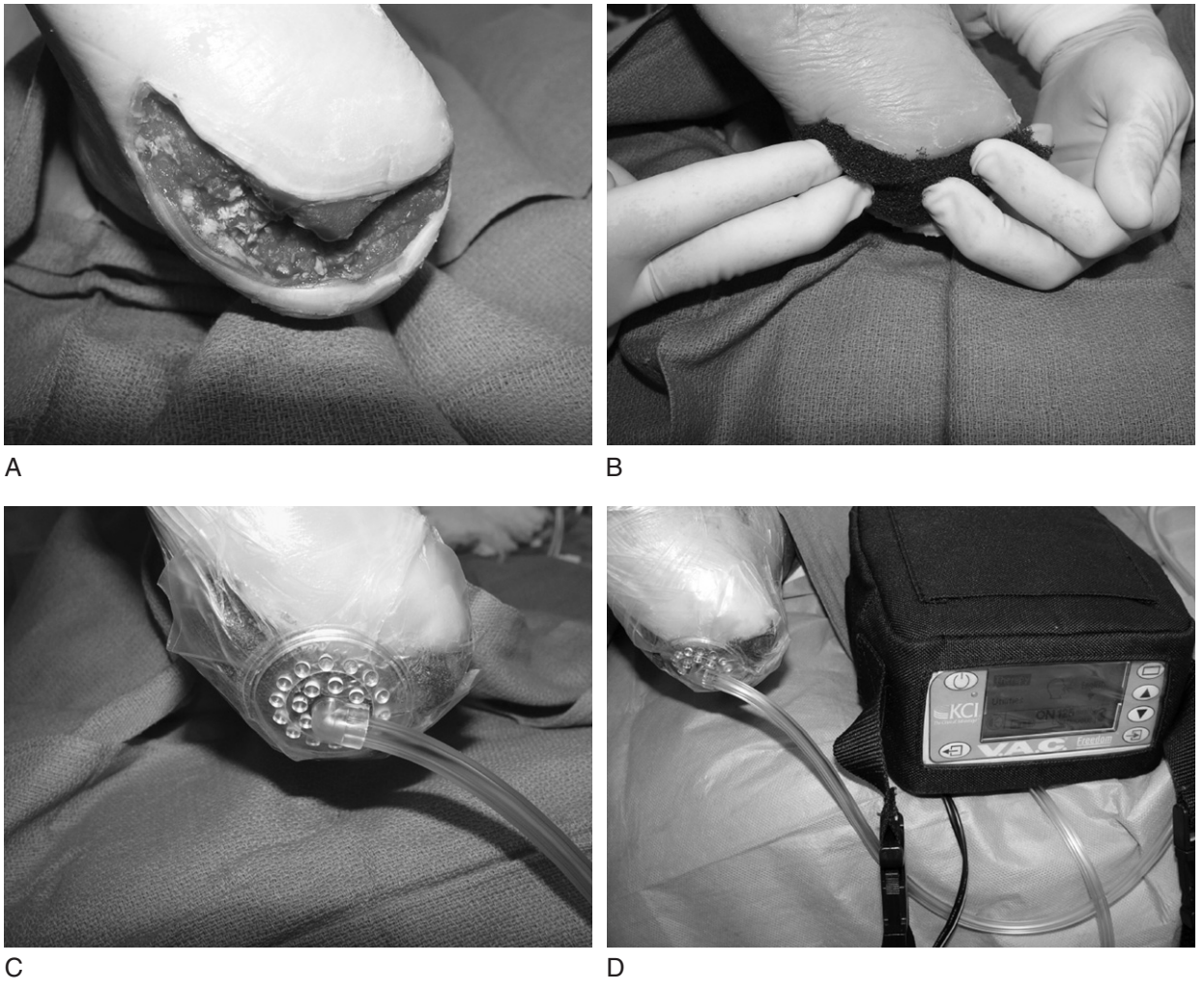

Figure 2 • A vacuum-assisted closure device is applied to a freshly débrided wound (**A**). A sponge is shaped to fit the dimensions of the wound and placed on it (**B**). It is covered with an occlusive adherent sheet in which a small hole is made. Tubing is placed over the hole (**C**) and attached to a portable suction machine (**D**). Up to 125 mm Hg of subatmospheric pressure can be applied in either intermittent or continuous fashion.

excise the wound to normal tissue margins. While performing adequate débridement, it is imperative to use atraumatic surgical technique to avoid damaging the surrounding healthy tissue that will serve as the subsequent base for normal healing. Special care, however, should be taken to preserve functional and exposed neurovascular bundles. Skeletal instability can generally be restored by the application of an external fixator. Deep uncontaminated tissue should be obtained for culture to guide subsequent antibiotic therapy. Concern about how to repair the soft tissue defect after débridement should *not* limit the extent of débridement. *Débridement is the single most underperformed stage in treating foot and ankle wounds.* For wounds that may still harbor significant bacteria, an appropriate topical antibiotic, a silver ion sheet, or antibiotic-impregnated methylmethacrylate beads covered with an occlusive dressing are effective dressings. For wounds that are clean and well vascularized, a moist dressing or VAC is preferred. Débridement should be rescheduled as frequently as necessary if there is progressive tissue necrosis or destruction.

Biologic débriding agents such as maggots (larvae of the green blowfly, *Phaenicia sericata*) are currently being reintroduced and are useful in very ill patients or those waiting for revascularization. They digest only necrotic tissue and bacteria by secreting enzymes that dissolve the necrotic tissue and biofilm that surrounds bacteria. Thirty larvae consume 1 g of necrotic tissue per day. The maggots are placed on the wound, covered with an air-permeable occlusive dressing, and replaced every 2 to 3 days. The maggots painlessly débride the wounds because they digest only necrotic tissue. Because maggots consume all bacteria, they sterilize wounds contaminated with antibiotic-resistant bacteria.

After débridement to clean tissue, it is important to prevent a buildup of metalloproteinases, which destroy naturally produced growth factors. The biofilm of proteinaceous débris that forms on the wound must be removed at regular intervals by scrubbing the wound daily or by using wet-to-dry dressings. The latter, however, also remove healthy new tissue. VAC, by applying negative pressure to the surface of the wound, prevents the buildup of proteases and bacteria that inhibit or break down growth factors. VAC additionally decreases peri-wound edema and increases local blood flow by rapid formation of well-vascularized granulation tissue. This, in turn, leads to rapid wound shrinkage and changes the reconstructive options from complex to simple closure. VAC also allows the surgeon to plan the timing of reconstruction without compromising the wound. VAC should be avoided in ischemic wounds because progressive necrosis can ensue.

An alternative to scrubbing, wet-to-dry dressing, or VAC is placement of skin equivalents on the freshly débrided, well-vascularized wound. Xenograft or cadaver skin provides an excellent biologic dressing that promotes wound healing. It should be used only if the wound base is relatively infection free ($<10^5$ bacteria per gram of tissue). If the underlying wound bed revascularizes the graft over the next 4 to 5 days, it is then ready to accept an autogenous skin graft.

If the wound fails to show signs of healing despite being clean and having adequate blood flow, it can often be converted to a healing wound by providing local wound-healing factors. One can apply either platelet-derived growth factor (Regranex [becaplermin]) daily to the wound or place a sheet of cultured skin that produces the entire range of growth factors every 1 to 6 weeks (Apligraf, Dermagraft). Systemic hyperbaric oxygen can also be used to convert a non-healing wound to a healthy wound. It stimulates local angiogenesis at the wound bed, promotes the formation of collagen, and potentiates the ability of macrophages and granulocytes to kill bacteria. When healthy granulation tissue appears, the wound can be skin grafted.

Surgical Reconstruction

After a clean and well-vascularized wound bed has been achieved, the simplest method to preserve a stable and functional foot should be selected. Biomechanics is a critical part of the reconstructive plan that involves bone rearrangement, partial joint removal/fusion, or tendon lengthening/transfer. The method of soft tissue reconstruction selected usually hinges on whether there is exposed viable tendon, joint, or bone (or any combination of these structures), as well as the type of access to the wound that is available (e.g., an Ilizarov frame limits access to the foot). If the wound cannot be closed primarily, the presence of exposed tendon, joint, or bone usually dictates flap coverage. Exceptions exist because of new healing strategies, including VAC or the application of a collagen framework such as Integra, followed by subsequent skin graft. The rest of the wound can usually be allowed to heal by secondary intention or application of a skin graft.

Wounds can be allowed to heal by *secondary intention* by daily changing of dressings, application of VAC, correction of the biomechanical abnormality, or any combination of these measures. A tight Achilles tendon is the principal cause of forefoot plantar ulceration in diabetic patients. By lengthening the tendon, the plantar wound heals without additional treatment over the next 5 weeks. Correcting the collapsed arch of a Charcot midfoot by reconstructing the arch and re-fusing the metatarsals to the hindfoot usually allows for loose approximation of the plantar soft tissue.

Delayed primary closure is easier to accomplish when the edema and induration of the wound edges have resolved. VAC can be helpful in this situation. After primary closure, one should always check that relevant pulses have not diminished because the closure was too tight. Delayed primary closure can be assisted by applying soft tissue–approximating clamps such as Proxiderm to both edges. Such clamps exert gentle, continuous pressure that approximates the edges over time and should be used only if there is a narrow defect. An adequate soft tissue envelope can also be created by removing bone or by converting a large wound to a partial foot amputation and then using the resultant soft tissue envelope for closure.

Skin grafting can be used to close most foot and ankle wounds. A healthy granulating bed is a necessary prerequisite and can be achieved by the following methods: VAC, cultured skin, growth factors, or hyperbaric oxygen. In addition, a healthy granulation tissue base can be built up over inhospitable wounds (e.g., bone) by applying a collagen lattice framework such as Integra and waiting for it to revascularize (Fig. 3). The skin graft is meshed to prevent buildup of seroma or hematoma, which could prevent graft revascularization. The use of VAC on low continuous suction as a temporary dressing for the first 3 to 5 postoperative days ensures the highest possible skin graft revascularization rate. It absorbs all excess fluid and keeps the wound relatively sterile. If the skin graft is placed over a moving muscle or joint, there is a risk of disruption by shear forces. In this instance, it is critical to immobilize the foot and ankle by splinting or placement of an external fixator until the skin graft has completely healed.

The ideal skin donor site for a plantar wound is the plantar instep. This glabrous skin graft holds up much better under the shear forces applied to the plantar surface of the foot during ambulation. It should be harvested at 30/1000th of an inch, meshed, and sutured with a bolster or covered with a VAC dressing. For plantar wounds in which the

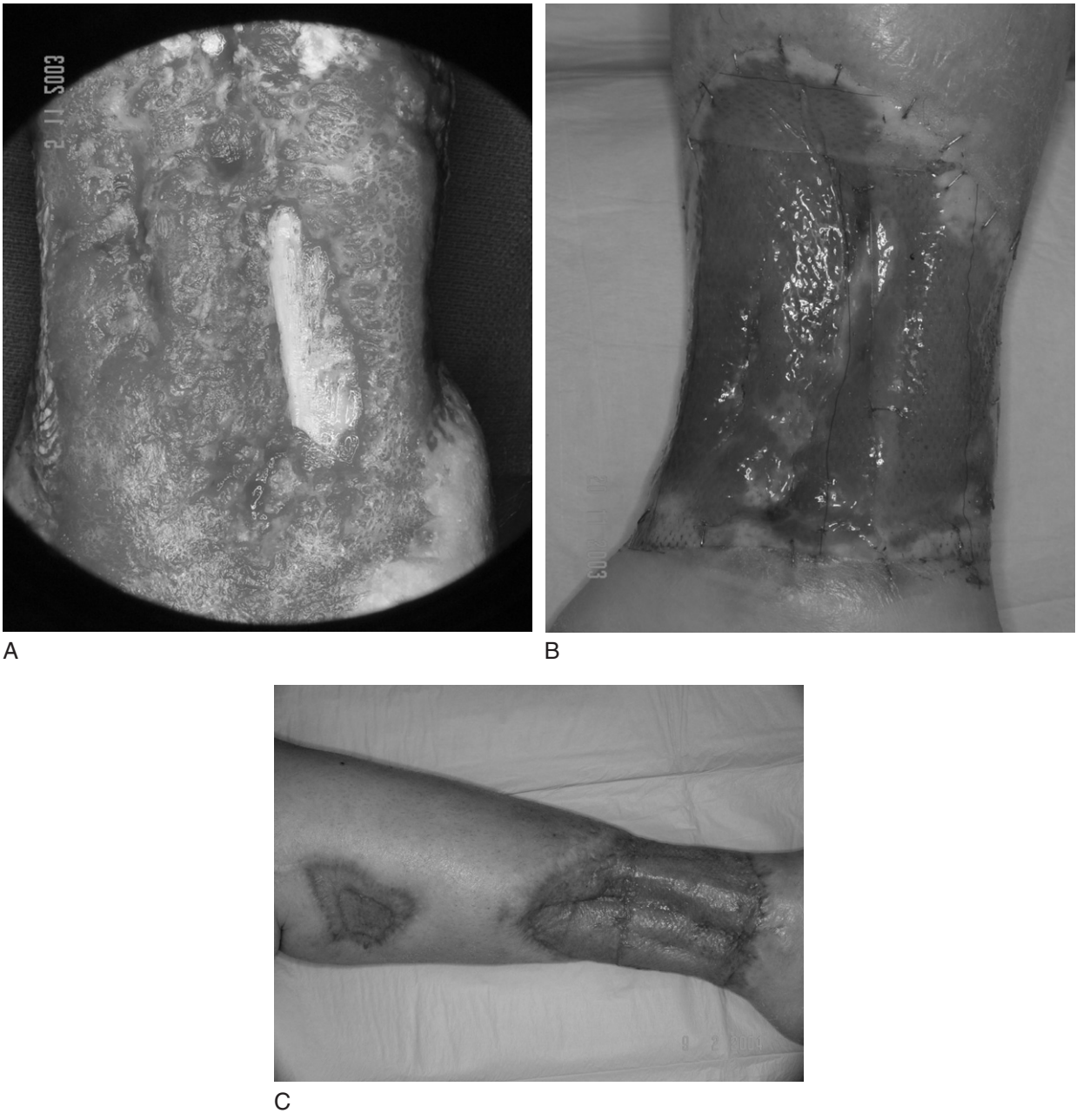

Figure 3 • A patient with gangrene of the entire lower part of the leg and exposed tibia requiring serial débridement (**A**). Integra was meshed, placed over the entire wound, and covered with a vacuum-assisted closure dressing. After 3 weeks, the collagen framework was revascularized and could be successfully skin grafted (**B**). The wound healed without problems (**C**).

patient is noncompliant either by choice or because of body habitus, consideration should be given to protecting the skin graft by placing an Ilizarov frame with a footplate until the graft has healed.

The use of any flap requires an accurate assessment of blood flow. For local flaps, there should be Doppler evidence of a perforator close to the base of the flap. For pedicled flaps, the dominant branch to the flap should be patent. For microvascular free flaps, there should be an adequate recipient artery and vein or veins. If any question arises, an angiogram, duplex scan, or magnetic resonance angiogram should be obtained.

Local flaps are useful for coverage of foot and ankle wounds because they can be designed in a small dimension to cover the exposed tendon, bone, or joint while the rest of the wound is skin grafted. Such flaps frequently obviate the need for larger pedicled or free flaps. In addition, a local flap is a useful mode of reconstruction when closing a wound around or through an Ilizarov external fixator. For plantar wounds, local flaps are helpful because the

wounds are resurfaced with healthy plantar tissue rather than by secondary intention with the attendant thick scar that later becomes a source of recurrent callus formation.

Knowledge of the various *pedicled flaps* in the foot and ankle area is critical for closing wounds that cannot be closed by simpler methods. These flaps are often more difficult to dissect and have a higher complication rate than a free flap. Harvesting a pedicled flap often leaves a donor deficit on the foot and ankle that has to be skin grafted. However, pedicled flaps allow a rapid operation with a shorter hospital stay and long-lasting satisfactory results.

The most useful *muscle flaps* in the foot and ankle include the abductor hallucis, abductor digiti minimi, flexor digitorum brevis, extensor digitorum brevis, reverse extensor hallucis longus, reverse extensor digitorum longus, and reverse anterior tibial muscle flap (provided that the ankle is fused).

The most useful *skin* or *fasciocutaneous flaps* in the foot and ankle include the supramalleolar flap, medial plantar flap, lateral calcaneal flap, reverse anterior tibial artery flap, reverse peroneal artery flap, and sural artery flap. When using reverse flaps based on the anterior tibial or peroneal artery, it is important that all other vessels be intact and the distal arterial-arterial connections be patent to prevent ischemia or tissue necrosis. The dissections are often tedious and difficult, and the distal reach of the flap often provides insufficient tissue. It is very important to understand the size limitations of each flap.

MICROVASCULAR FREE FLAP. *Microvascular free flaps* in the foot and ankle carry the highest failure rate in the microsurgical literature and should be planned carefully. One of the reasons for the high failure rate is that complications arise when the anastomosis is performed at or near the zone of injury. In addition, the arteries are often calcified and special hardened microneedles are required. The anastomoses should be performed away from the zone of injury, either proximal or distal to the zone of injury, provided that the neurovascular bundle is intact. An end-to-side anastomosis to the recipient artery should be performed whenever possible. Two veins should be used if possible to minimize postoperative swelling.

The choice of which free flap to use depends, in large part, on the length of pedicle needed. For long pedicles, the serratus, latissimus, vastus lateralis, or rectus femoris muscles are excellent choices. It is important to remember that a longer pedicle can be obtained when the pedicle dissection is extended into the muscle belly. For the dorsum of the foot and the ankle, thin fasciocutaneous or cutaneous flaps work best. For the plantar surface of the foot, muscle flaps covered by a skin graft hold up better over the long run.

FOREFOOT COVERAGE. Toe ulcers and gangrene are best treated by limited amputations that preserve viable tissue for wound closure. Attempts to preserve at least the proximal portion of the proximal phalanx should be made so that it can serve as a toe filler and prevent the toes on either side from drifting into the empty space. If the hallux is involved, an attempt should be made to preserve as much of the toe as possible because of its critical role in ambulation. A toe island flap from the second toe is an excellent technique for filling a defect on the hallux without having to resort to surgical shortening.

Ulcers under the metatarsal head or heads occur because biomechanical abnormalities place excessive pressure on the plantar surface of the forefoot during the gait cycle. Although hammer toes can be contributing factors, the abnormal biomechanical forces are usually due to a tight Achilles tendon that prevents ankle dorsiflexion beyond the neutral position. If the patient can dorsiflex the foot only when the knee is flexed, the gastrocnemius portion of the Achilles tendon is tight. Simple gastrocnemius recession should correct the problem. If the patient cannot dorsiflex the foot with the knee flexed or extended, both the gastrocnemius and soleus portions of the tendon are tight. In addition, the posterior capsule of the ankle joint may be tight. Percutaneous release of the Achilles tendon is performed, and if the foot still does not dorsiflex, posterior capsular release is performed. The patient is kept non–weight bearing for 1 week and in a cam walker for an additional 5 weeks. Complications associated with gastrocnemius recession include hematomas from premature tears in the soleus muscle. Overly aggressive release can lead to a calcaneal gait and, eventually, plantar heel ulcers, which are extremely difficult to heal. Tightening of the Achilles tendon or ankle fusion may be required. With release of the Achilles tendon, forefoot pressure drops dramatically, and the ulcers, if they do not involve bone, heal simply by secondary intention in less than 6 weeks. Lengthening of a tight Achilles tendon has decreased the ulcer recurrence rate in diabetic patients from 25% to 3%.

If the Achilles tendon allows for normal dorsiflexion, small ulcers under a metatarsophalangeal joint without bone involvement can be treated by simply elevating the metatarsal head with preplanned osteotomies and internal fixation. The metatarsal head is thus shifted 2 to 3 mm superiorly. Upward movement with attendant pressure relief is usually sufficient for the underlying ulcer to heal by secondary intention. There should not be any transfer lesions to the other metatarsal heads because the anatomic metatarsal head parabola is preserved. However, if the metatarsal head has evidence of osteomyelitis, it should be resected. The ulcer should heal by secondary intention if all weight is kept off the foot while it heals.

Small deep forefoot ulcers without an obvious bony prominence can also be closed with a local flap: filleted toe, toe island, bilobed, rotation, Limberg, or V-Y flap.

For larger ulcers in which the metatarsal head has been resected, consideration should be given to ray amputation. Resecting the more independent first or fifth metatarsal causes less biomechanical disruption than does resection of the second, third, or fourth metatarsal because the middle metatarsals tend to operate as a single unit.

All effort should be made to preserve as much of the metatarsals as possible if more than one is exposed because they are so important to normal ambulation. Local tissue is often insufficient for this purpose in the forefoot, and therefore a microvascular free flap should be considered. If ulcers are present under several metatarsal heads or if a transfer lesion from one of the resected metatarsal heads to a neighboring metatarsal has occurred, consideration should be given to panmetatarsal head resection. This technique is performed with two or three dorsal incisions, and care is taken to preserve the proportional lengths of each metatarsal so that the normal distal metatarsal parabola is preserved. Removing the metatarsal heads while leaving the toe flexors and extensors intact prevents the inevitable equinovarus deformity that accompanies loss of the distal extensors.

If more than two toes and the accompanying metatarsal heads have to be resected, a transmetatarsal amputation should be performed. The normal parabola with the second metatarsal being the longest is preserved. All bone cuts should be made so that the plantar aspect of the cut is shorter than the dorsal one. If the extensor and flexor tendons of the fourth and fifth toes are intact, they should undergo tenodesis with the ankle in neutral position. This maneuver prevents the equinovarus deformity that can result from the loss of extensor forces, which usually leads to breakdown under the distal fifth metatarsal. If the Achilles tendon is tight, it should be released. As much plantar tissue as possible should be preserved so that the anterior portion of the amputation consists of healthy plantar tissue. When medial and lateral defects are present, the plantar flap should be rotated to cover the entire forefoot.

The most proximal forefoot amputation is the Lisfranc amputation, in which all of the metatarsals are removed. To prevent an equinovarus deformity, the anterior tibial tendon should be split and its lateral aspect inserted into the cuboid bone. In addition, the Achilles tendon should be lengthened. The Lisfranc amputation can be closed with volar or dorsal flaps if sufficient tissue is available. If there is not adequate tissue for coverage, a microvascular free muscle flap with a skin graft should be used.

MIDFOOT COVERAGE. Defects on the medial aspect of the sole are non–weight bearing and are best treated with a skin graft. Ulcers on the medial and lateral plantar aspect of the midfoot are usually due to Charcot collapse of the midfoot plantar arch. If the underlying shattered bone has healed and is stable (Eichenholtz stage 3), the excess bone can be shaved via a medial or lateral approach and the ulcer can either be allowed to heal by secondary intention or be covered with a glabrous skin graft or a local flap. For small defects, useful local flaps include the V-Y, bilobed, rhomboid, or transposition flap. If a muscle flap is needed, a pedicled abductor hallucis flap medially or an abductor digiti minimi flap laterally works well. For slightly larger defects, medially based random-pattern rotation flaps or a pedicled medial plantar fasciocutaneous flap can be successful. Larger defects should be filled with microvascular free muscle flaps covered by skin grafts. If the midfoot bones are unstable (Eichenholtz stage 1 or 2), they can be excised via wedge excision to allow recreation of the arch by fusing the proximal metatarsals to the talus and calcaneus via an Ilizarov frame. Shortening of the skeletal midfoot usually leaves sufficient loose soft tissue to close the wound with a local flap.

HINDFOOT COVERAGE. Plantar heel defects or ulcers are among the most difficult of all wounds. If they are the result of the patient being in a prolonged decubitus position, they may also be a reflection of severe vascular disease. Partial calcanectomy may be required to develop an adequate local soft tissue envelope to cover the resulting defect. Although patients can ambulate with a partially resected calcaneus, they will require orthotics and possibly molded shoes. If there is an underlying collapsed bone or bone spur causing a hindfoot defect, the bone should be shaved down. These ulcers are usually closed with double V-Y flaps or larger medially based rotation flaps. Plantar heel defects can also be resurfaced with pedicled flaps, such as the medial plantar fasciocutaneous flap or the flexor digiti minimi muscle flap.

Posterior heel defects are better closed with an extended lateral calcaneal fasciocutaneous flap or a retrograde sural artery fasciocutaneous flap. If the defect is large, a muscle microvascular free flap with skin graft should be used. The flap should be carefully tailored so that there is no excess tissue and it blends well with the remainder of the heel.

Medial or lateral calcaneal defects usually occur after fracture and attempted repair. There is associated osteomyelitis of the calcaneus. After débridement of the infected bone and placement of antibiotic beads, the medial defect can generally be covered with an abductor hallucis muscle flap medially or an abductor digiti minimi flap laterally. The exposed muscle is skin grafted. After 6 or more weeks, the beads can be replaced with an autogenous bone graft.

The two hindfoot amputations are Chopart's and Syme's amputations. Chopart's amputation leaves an intact talus and calcaneus while removing the midfoot and forefoot bones of the foot. To avoid an equinovarus deformity, a minimum of 2 cm of the Achilles tendon is resected. A calcaneal-tibial inter-

medullary rod can occasionally be used to stabilize the amputation. Syme's amputation should be considered if the available tissue is insufficient to close Chopart's amputation primarily and the arterial blood supply is insufficient for a microvascular free flap or the talus and calcaneus are involved with osteomyelitis. The tibia and fibula are divided above the ankle mortise and the deboned heel pad transferred anteriorly. The heel pad has to be anchored to the anterior portion of the distal end of the tibia to prevent posterior migration. The amputation can be difficult to shape because large dog-ears occur when the heel pad is brought anteriorly. The resulting dog-ears contain part of the blood supply to the heel and cannot be easily removed. They can be carefully trimmed at the initial operation or 4 to 6 weeks later to allow for alternative blood flow patterns to be established. The ultimate goal is a thin, tailored stump that can fit well into a prosthesis. A poorly designed Syme's amputation is difficult for a prosthetist and can lead to repeated breakdown of the stump.

DORSUM OF THE FOOT. Defects on the dorsum of the foot are often treated with simple skin grafts. Local flaps for small defects include rotation, bilobed, rhomboid, or transposition flaps. Pedicled flaps include the extensor digitorum brevis muscle flap, the dorsalis pedis flap, the supramalleolar flap, and the sural artery flap. The extensor digitorum brevis muscle's reach can be increased by cutting the dorsalis pedis artery above or below the tarsal artery, depending on where the defect is and whether there is adequate antegrade and retrograde flow. The reach of the supramalleolar flap can be increased by dividing the anterior perforating branch of the peroneal artery before it anastomoses with the lateral malleolar artery. For defects at the sinus tarsus, the extensor digitorum brevis flap works well. The most appropriate microvascular free flap is a thin fasciocutaneous flap. The radial forearm flap, also thin, is an excellent choice because it is sensate and provides a vascularized tendon (palmaris tendon) to restore extensor function. Thin muscle flaps and skin grafts or fasciocutaneous flaps with skin grafts are effective options as well.

ANKLE DEFECTS. The soft tissue around the ankle is sparse and minimally flexible. With sufficient granulation tissue, a skin graft works well. The Achilles tendon, if allowed sufficient time to granulate, tolerates a skin graft that is durable over time. Local flaps include rotation, bilobed, and transposition flaps. Local flaps can also be individually designed with the posterior tibial and peroneal arterial perforators. Pedicled flaps include the supramalleolar, dorsalis pedis, retrograde sural artery, medial plantar, abductor hallucis, abductor digiti minimi, and extensor digitorum brevis muscle flaps. Microvascular free flaps can be either fasciocutaneous or muscle with a skin graft, but they should be kept thin. To promote healing, the ankle should be temporarily immobilized with an external fixator.

Postoperative Care

After foot and ankle surgery, the foot is frequently placed in a splint or is stabilized with an external fixator to prevent movement of the joints and compromise of the skin and local flaps. Patients are not generally allowed to bear weight on the operated foot for 6 weeks if the plantar surface of the foot is involved. For dorsal wounds, the patients are allowed to ambulate far sooner provided that they have a dressing that prevents damage to the reconstruction site. Because of these limitations, a patient will often need a course of physical and occupational rehabilitation to gain the strength and mobility to be independent at home. Patients should be monitored closely during the postoperative period and should be seen by an orthetist and perhaps a pedorthetist to have appropriate shoe wear when they bear weight. Patients with diabetes should return to the podiatrist for preventive foot care.

Pearls and Pitfalls

- Successful limb salvage requires a team approach that includes a vascular surgeon, a foot and ankle surgeon, a plastic surgeon, and an infectious disease specialist.
- One first has to ensure adequate blood flow because without it the wound will not heal.
- Débridement is the platform from which all reconstruction springs. It has to be aggressive and repeated as many times as necessary until the wound is clean.
- Most wounds can be closed with simple techniques, but a minority require more sophisticated anatomic knowledge to perform the necessary pedicled or free flaps.
- All amputations should be performed in two stages to avoid complications.
- The VAC dressing is a useful adjunct in preparing the wound for closure after débridement.
- Reconstruction should proceed only when all signs of inflammation are absent and signs of healing are present (healthy granulation tissue and neo-epithelialization).
- Biomechanical issues should be addressed to achieve a functional foot and avoid recurrent breakdown.

SUGGESTED READINGS

Attinger C, Cooper P, Blume P, Bulan E: The safest surgical incisions and amputations applying the angiosome principles and

using the Doppler to assess the arterial-arterial connections of the foot and ankle. Foot Ankle Clin 6:745-799, 2001.

Attinger CE, Venturi M, Kim K, Ribiero C: Maximizing length and optimizing biomechanics in foot amputations by avoiding cookbook recipes for amputation. Semin Vasc Surg 16:44-66, 2003.

Dellon L: Treatment of symptomatic diabetic neuropathy by surgical decompression of multiple peripheral nerves. Plast Reconstr Surg 89:689-697, 1992.

Masqualet AC, Gilbert A: An Atlas of Flaps in Limb Reconstruction. Philadelphia, JB Lippincott, 1995.

Taylor GI, Corlett RJ, Caddy CM, Zelt RG: An anatomic review of the delay phenomenon: II. Clinical applications. Plast Reconstr Surg 89:408-416, 1992.

Wrobel JS, Mayfield JA, Reibel GE: Geographic variation of lower-extremity major amputation in individuals with and without diabetes in the Medicare population. Diabetes Care 24:860-864, 2001.

Lower Extremity Reconstruction after Ablative Surgery

ARMEN KASABIAN

Ablative surgery and reconstruction are often necessary for the treatment of sarcomas, melanomas, skin and soft tissue tumors, and infections of the lower extremity. Amputation is occasionally the treatment of choice because it provides the most complete method of eradication. However, limb-sparing surgery accompanied by radiotherapy and occasionally chemotherapy can be effective amputation in many cases. The goals of reconstruction are to maximize the functional capacity of the extremity without compromising oncologic principles. The introduction of microsurgery and other reconstructive techniques has allowed limb-sparing surgery to become a viable alternative to amputation. The requirement for coverage of vital structures and protection from postoperative irradiation necessitates reconstruction with well-vascularized tissues and presents reconstructive challenges.

Reconstruction after ablative surgery requires a team approach, including the oncologic surgeon, plastic surgeon, oncologist, radiation oncologist, and physiatrist to ensure the best possible method of tumor eradication with maximal preservation of function.

Etiopathogenesis

Skin tumors and sarcomas are the most common lower extremity problems requiring ablative surgery. On occasion, massive infections such as necrotizing fasciitis may necessitate radical débridement of skin, soft tissue, and muscle. Defects resulting from trauma and posttraumatic osteomyelitis are not discussed in this chapter.

Basal cell carcinomas are locally invasive tumors. They are usually diagnosed at an early stage, and postablative soft tissue defects rarely require complex reconstruction.

Squamous cell carcinomas, also usually recognized at an early stage, can metastasize when they become larger or more invasive. Marjolin's ulcers may delay diagnosis, and extirpation of the tumor may result in a large defect.

Melanomas require wide and deep excision. The margins of resection depend on the depth of invasion of the tumor and may vary from 1 to 3 cm. When a 3-cm margin is used, the defect created may exceed 7 cm. With the paucity of available donor tissue in the lower extremity, resection may create a significant soft tissue defect and, depending on the location of the tumor, may expose tendon, bone, or other vital structures.

Soft tissue sarcomas account for approximately 1% of malignant neoplasms. Sarcomas (liposarcomas, leiomyosarcomas, malignant fibrous histiocytomas, synovial cell sarcomas, rhabdomyosarcomas, etc.) are usually invasive and diagnosed late. Most are locally invasive, but metastasis is possible. With delayed diagnosis, invasion of local soft tissue, muscle, nerve, and bone results in a complex composite defect and presents a reconstructive challenge.

Bone tumors, such as osteogenic sarcoma and Ewing's sarcoma, give rise to significant postablative bone and joint defects and problems with soft tissue coverage. The bone defects may be reconstructed with endoprostheses, bone allografts, vascularized bone transfers, and distraction techniques, but such treatment often requires additional soft tissue coverage. Invasion of nerves and major vascular structures may require additional reconstructive and revascularization techniques.

Pathologic Anatomy

Postablative defects of the lower extremity requiring reconstruction may vary from small skin defects to complex composite defects consisting of skin, soft tissue, nerve, muscle, blood vessel, bone, and joints. Large composite defects represent a reconstructive challenge. Even small skin defects of the lower extremity may be challenging because the lower extremity has a paucity of local tissue, which makes it difficult to achieve primary closure, and because the potential need for postoperative radiation requires a durable type of reconstruction.

Diagnostic Studies

Although restoration of function is an important aspect of ablative surgery, tumor eradication and survival are the most important considerations. Diagnostic studies, including radiography, total body computed tomography, magnetic resonance imaging, positron emission tomography, and angiography, depend on the pathology of the tumor. The studies must be performed accurately to detect the extent of the tumor and the presence of any metastasis. The extent of the tumor or the presence of metastasis can change the ablative and reconstructive goals. Absence of metastasis would dictate more aggressive local tumor eradication and more complicated reconstruction, whereas the presence of metastasis may prompt more conservative resection and reconstruction.

Reconstructive Goals

Treatment options for a major tumor of the lower extremity are ablative surgery followed by reconstruction or amputation. The goals of reconstruction after ablative surgery are to maximize functional restoration with minimal postoperative complications. The results must be equal to or better than the alternative: amputation.

Various studies have demonstrated that radical ablative limb-salvaging surgery combined with radiation therapy and chemotherapy produces survival rates comparable to those of amputation. More aggressive local surgical excision combined with adjuvant therapy has been successful in minimizing local recurrence and metastasis. Limb salvage and reconstruction are associated with a reduced incidence of local recurrence and a reduced need for subsequent amputation.

Functional outcome studies have shown fair to excellent results after limb salvage and reconstruction. The results vary depending on the extent of ablation (size of the defect), the structures requiring reconstruction, the administration of radiotherapy, the need for immediate or delayed reconstruction, and the level of the reconstruction on the limb. The alternative of amputation causes considerable functional impairment with a decrease in activities of daily living and increased energy of ambulation, as well as psychologic effects.

Both functional and aesthetic considerations are important. Tissue should be replaced with like tissue as much as possible. Skin defects should be replaced by skin and soft tissue by soft tissue. Tendon, bone, major vessels, and nerves should be covered with vascularized tissue, whether it be a local flap or free tissue transfer. Bone defects may be replaced with allograft, vascularized bone transfers, an endoprosthesis, or distraction techniques. When tumors involve major vascular structures, vascular reconstruction can be achieved either with autologous vein grafts or with polytetrafluoroethylene (PTFE; Gore-Tex) grafts.

Treatment

Treatment of a postablative surgical defect depends on several factors:

What is removed:
- Skin
- Soft tissue
- Muscle/tendon
- Major vascular structures
- Nerve
- Bone
- Joint

What is exposed:
- Soft tissue
- Muscle
- Tendon
- Major vascular structures
- Nerve
- Bone
- Joint

Whether postoperative radiation therapy will be administered

When planning reconstruction, the surgeon must rely on the following *reconstructive ladder* (Table 1):

1. Local wound care
2. Split-thickness skin graft
3. Full-thickness skin graft
4. Local random-pattern flap
5. Local pedicle flap
6. Muscle pedicle flap
7. Microvascular free tissue transfer

TABLE 1 Coverage of Defects

DEFECT	COVERAGE OPTIONS
Skin	Healing by secondary intention Split-thickness skin graft
Soft tissue	Healing by secondary intention Split-thickness skin graft Local flap
Muscle, tendon	Split-thickness skin graft Local flap Free tissue transfer Delayed tendon or muscle transfer
Vascular structure	No reconstruction if no vital need Autologous vein PTFE graft
Nerve	No reconstruction Nerve graft
Bone	Cancellous bone graft Distraction technique Vascularized fibula Endoprosthesis
Joint	Joint fusion Endoprosthesis

PTFE, polytetrafluoroethylene.

Bone reconstruction has specific options:

1. Allograft
2. Distraction
3. Microvascular free bone transfer
4. Endoprosthesis

When nerve is resected, unless the defect is very small, the only option for reconstruction is autologous nerve grafting. When major blood vessels are sacrificed, reconstruction requires either an autologous vein graft or a synthetic graft of PTFE (see Table 1).

Skin Defects

Skin defects are best treated with split-thickness skin grafting. Skin grafts usually do well on the lower extremity, and full-thickness skin grafts are rarely needed. Smaller wounds may be allowed to granulate and heal by secondary intention, but such management usually results in a contracted wound scar.

Soft Tissue Defects

Soft tissue defects may also be treated with split-thickness skin grafts, but these grafts usually result in a closed wound with a significant contour abnormality. To improve the aesthetic result, the wound may be allowed to fill with granulation tissue, followed by a delayed skin graft, which may lead to an improvement in contour. Vacuum-assisted closure of the wound may promote granulation and decrease the size of the wound. In patients with excess soft tissue, the wounds may be resurfaced with local advancement flaps. When possible, these flaps usually provide more durable coverage. However, local flaps result in a soft tissue depression with additional scarring. There is also a risk of flap necrosis if not designed properly.

Muscle Defects

Exposed muscle can easily be covered by skin grafts or local flaps. If muscle has been resected in the ablative procedure, functional muscle reconstruction, such as vascularized free muscle transfers, is rarely successful in restoring muscle function. However, there is usually redundant muscle function that can assume some of the lost function. Tendon transfers for other functional losses may be required in a delayed reconstruction.

Major Vascular Defects

If major vessels are sacrificed in the lower extremity, vascular reconstruction may be necessary. If the superficial femoral artery is sacrificed, reconstruction is necessary for lower limb viability, which can be achieved with either a saphenous vein graft or a Gore-Tex graft. If a vessel is sacrificed below the knee, the need for revascularization depends on the status of the other major vessels in the lower part of the leg. The leg can usually survive with a single major vessel to the foot. If a single major vessel in the lower part of the leg is sacrificed during ablative surgery, revascularization is not necessary unless the remaining two major vessels are inadequate.

Nerve Defects

If a major nerve is resected in the ablative procedure, repair of the nerve with nerve grafts may be a consideration, but the results after long nerve grafts in the lower extremity remain disappointing.

Bone and Joint Defects

Reconstruction of bone defects depends on the location and extent of the defect (Table 2). Smaller skeletal defects can usually be treated with non-

TABLE 2 Reconstruction of Bone Defects

DEFECT	RECONSTRUCTION
0-3 cm	Cancellous bone graft
3-7 cm	Distraction
5-20 cm	Vascularized fibula Endoprosthesis
Knee fusion	Vascularized fibula
Knee reconstruction	Endoprosthesis

vascularized cancellous bone grafts. Intermediate defects can be treated by either distraction, bone lengthening, or vascularized bone transfers. Although longer bone defects have been reconstructed by the distraction technique, it is better suited to intermediate-length defects. A vascularized free fibula transfer can theoretically bridge a gap up to 24 cm or more. It is most useful for longer bone defects. Joint fusion can be performed with nonvascularized bone and internal fixation. Vascularized fibula transfer for joint fusion has been used successfully and may be more reliable. Endoprostheses with knee joints spanning long distances have been used effectively for knee reconstruction after ablative surgery.

Coverage of Exposed Tendon, Bone, Arteries, and Nerves

If ablative surgery results in exposure of tendon, bone, arteries, or nerves in the lower extremity, these structures must be covered by well-vascularized tissue. Small areas of exposed tendon or bone may be treated with local wound care alone. The vacuum-assisted closure dressing is useful for promoting granulation tissue around areas of exposed bone and tendon. However, waiting for the wound to granulate may delay the institution of postoperative radiation therapy or chemotherapy. Small defects may be closed with a local random-pattern flap. Defects of the knee are best treated with a gastrocnemius muscle flap. Mid to lower leg defects are optimally treated with a soleus muscle flap. Larger defects, defects of the lower third of the lower leg, and defects in the upper part of the leg that cannot be covered by local techniques are best treated by microvascular free tissue transfer (Table 3).

TABLE 3 Coverage of Exposed Vital Structures

DEFECT OF EXPOSED VITAL STRUCTURES	TREATMENT
Upper leg	Local flap Free tissue transfer
Knee	Local flap Gastrocnemius flap Free tissue transfer
Middle third of lower leg	Local flap Soleus flap Free tissue transfer
Lower third of lower leg	Free tissue transfer

Postoperative Care

Postoperative complications after reconstruction of postablative defects include those associated with any major flap or free tissue transfer.

Careful monitoring of flap viability along with prevention of wound necrosis, infection, and exposure of implants or vital structures is crucial to a successful outcome. Flap monitoring by visual inspection, temperature probes, or Doppler flowmeters has been used with varying degrees of success. Early return to the operating room is indicated for any questionable microvascular complication or evidence of wound necrosis that may lead to implant exposure.

Gradual progression of the limb to dependent positions is necessary to avoid excessive edema, venous congestion, and flap necrosis. Deep venous thrombosis is a major complication of lower extremity reconstruction, and immobilization and anticoagulation must be instituted.

Pearls and Pitfalls

- The extent of tumor resection should not be compromised to avoid a large wound and the need for a free tissue transfer.
- Necrosis of wounds and exposure of vital structures and hardware will most likely lead to infection and subsequently to amputation.
- The principles of treatment are obliteration of local tumor, restoration of function, and coverage of vital structures and wounds with well-vascularized tissue to avoid the potential complications associated with radiation therapy and chemotherapy.

SUGGESTED READINGS

Cordeiro PG, Neves RI, Hidalgo DA: The role of free tissue transfer following oncologic resection in the lower extremity. Ann Plast Surg 33:9-16, 1994.

Ham SJ, Schraffordt Koops H, Veth RP: Limb salvage surgery for primary bone sarcoma of the lower extremities: Long-term consequences of endoprosthetic reconstructions. Ann Surg Oncol 5:423-436, 1998.

Imparato AM, Roses DF, Francis KC, Lewis MM: Major vascular reconstruction for limb salvage in patients with soft tissue and skeletal sarcomas of the extremities. Surg Gynecol Obstet 147:891-896, 1978.

Renard AJ, Veth RP, Schreuder HW, et al: Function and complications after ablative and limb-salvage therapy in lower extremity sarcoma of bone. J Surg Oncol 73:198-205, 2000.

Serletti JM, Carras AJ, O'Keefe RJ, Rosier RN: Functional outcome after soft-tissue reconstruction for limb salvage after sarcoma surgery. Plast Reconstr Surg 102:1576-1583, 1998.

Topham NS: Reconstruction for lower extremity limb salvage in soft tissue carcinoma. Curr Treat Options Oncol 4:465-475, 2003.

Zenn MR, Levin LS: Microvascular reconstruction of the lower extremity. Semin Surg Oncol 19:272-281, 2000.

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