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Maria Z. Siemionow and Marita Eisenmann-Klein (Eds.)

Plastic and Reconstructive Surgery

With 476 Illustrations

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Preface

It is a pleasure to introduce a new specialist surgery series textbook entitled *Plastic and Reconstructive Surgery*. When I was approached to edit this book, I realized that it would be a tremendous task and asked Dr. Marita Eisenmann-Klein, from Regensburg, Germany, to serve as coeditor. The reason for inviting Dr. Eisenmann-Klein was her important role as Deputy General Secretary of the International Confederation of Plastic, Reconstructive, and Aesthetic Surgery and the intention, to have an international approach and divide the book into chapters where the best experts from different continents and countries would present their contributions.

The 52 chapters in this book are divided into eight subcategories of topics and are outlined in the following order:

Part I – General Principles presents physiology and wound healing, immunology of tissue transplantation, anesthesia, and critical care.

Part II – General Surgical Techniques presents principles of wound repair, grafts, local and regional flaps, microsurgical techniques, minimally invasive techniques of plastic surgery, liposuction techniques, biomaterials in craniofacial surgery, and tissue engineering.

Part III – Skin and Adnexa presents skin anatomy and physiology, congenital malformations, burns and trauma, benign and malignant skin tumors, and aesthetic skin treatments.

Part IV – Head and Neck presents head and neck embryology and anatomy, craniofacial clefts and syndromes, benign and malignant tumors of the head and neck, craniofacial trauma and reconstruction, eyelid and periorbital aesthetic surgery, nasal reconstruction and aesthetic rhinoplasty, lip and cheek reconstruction, auricular reconstruction for microtia, aesthetic surgery of the aging face and neck, treatment of headaches with plastic surgery, and facial reanimation.

Part V – Breast presents congenital malformations of the breast, breast reduction and mastopexy, postmastectomy breast reconstruction, augmentation mammoplasty, and gynecomastia.

Part VI – Hand and Upper Extremity presents hand anatomy and examination, congenital deformities and reconstruction, hand trauma, dislocations and fractures, infections, peripheral nerve injuries, tendon repair and reconstruction, benign and malignant hand tumors, ischemic and vasospastic conditions, acquired diseases of the hand, toe-to-hand transfers, and brachial plexus injuries and repair.

Part VII – Trunk and Lower Extremity presents trunk reconstruction, lower extremity reconstruction following trauma and tumors, abdominoplasty, lymphedema of the extremities, postbariatric reconstruction, and reconstructive and aesthetic surgery of the genitalia.

Part VIII – Future Directions in Research presents anesthesia and pathophysiology of microcirculation, experimental composite tissue transplantation models, and clinical experience with hand transplantation.

The diverse group of 85 authors from three continents and 13 countries, including Austria, Belgium, Canada, France, Germany, Great Britain, Ireland, Italy, Poland, Switzerland, Taiwan, Turkey, and the US have presented, in this book, their most updated practical expertise and personal techniques, and I am confident that this book will be more comprehensive than the plastic surgery texts currently available.

Most importantly, the idea when designing this book, was to allow the established authors of the chapters to present their own techniques and innovations; consequently, it is not simply an update of existing plastic surgery texts., In fact, it is quite unique, in the sense that many of the techniques and approaches presented, have been described for the first time.

The process of editing this book was relatively smooth and, as expected, with many contributors and chapters, constant communication and diligence were required in responding to editorial queries. I would like to take this opportunity to thank my coeditor, Dr. Marita Eisenmann-Klein, and all the 85 contributors for their excellent collaboration and timely responses.

I would like to emphasize, once again, that the international and intercontinental contributions to this book should make it a unique plastic surgery text for the reader, and an important and invaluable addition to every plastic surgeon's library.

Maria Siemionow

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Part I

General Principles

1

Physiology and Wound Healing

Raymund E. Horch, Oliver Bleiziffer, and Ulrich Kneser

Summary

Wound Healing is a complex and tightly regulated process involving different cell types and a large number of growth factors and cytokines which have their specific roles in the wound healing phases which are referred to as inflammation, proliferation and regeneration. Dysregulation of the physiologic wound healing process may lead to disturbed wound healing such as scar formation or delayed healing. The aim of current wound therapy is to optimize conditions for the healing process and provide custom-tailored individual treatment for different wound healing problems by the use of adjunct therapies as well as innovative therapeutic strategies.

Abbreviations

bFGF	Basic fibroblast growth factor
GM-CSF	Granulocyte monocyte colony stimulating factor
IFNγ	Interferon-gamma
IL	Interleukin
MMP	Matrix metalloproteinase
PDGF	Platelet-derived growth factor
PF	Platelet factor
TGF	Transforming growth factor
TNF	Tumor necrosis factor
tPA	Tissue-type plasminogen activator
uPA	Urokinase-type plasminogen activator
VEGF	Vascular endothelial growth factor

Introduction

All surgical specialties rely on a detailed knowledge of the mechanisms of wound healing and frequently encounter the challenge of the treatment of chronic wounds. The healing of a wound requires a sequence of processes to occur in a characteristic manner, with distinct roles for a large number of different types of cells, growth factors, cytokines, and other agents. Although countless experimental as well as clinical studies have identified the key players and their role in wound repair, clinicians still face conditions in which the regular healing process is disturbed. Alterations of physiologic wound healing can occur in certain circumstances where dysregulation of the cellular processes can lead to excessive scarring, resulting in hypertrophic scars and keloids. On other occasions, abnormalities in wound repair result in deficient wound healing, as can be seen in chronic, nonhealing wounds. The aim of this review is to summarize the current understanding of the wound healing process, mainly focusing on skin wound healing. Furthermore, current wound therapy is briefly reviewed.

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Phases of Wound Healing

Wound healing occurs in three distinct but overlapping phases, which are referred to as inflammation, proliferation, and regeneration[5] (Figure 1.1).

Hemostasis and Inflammation (Immediately After Wounding Through Days 4–6)

Upon wounding, blood vessels are injured, resulting in activation of the endothelium and adjacent platelets followed by vasoconstriction and activation of the coagulation cascade, respectively. A fibrin clot is formed, which consists of fibronectin, thrombin, platelets, and collagen. The importance of the clot is twofold. First, it is a rich source of cytokines and growth factors, which are released as activated platelets degranulate.[14] Second, it serves as a scaffold for invading cells, such as neutrophils, monocytes, macrophages, and endothelial cells, which are chemotactically attracted via cellular signaling immediately after clot formation.[12]

Chemotaxis and Activation

The influx of neutrophils is enabled through vasodilation, which in turn is caused by prostaglandins activated through inflammatory mediators released through platelet degranulation and products of proteolysis of fibrin and other matrix components. Interleukin (IL)-1, tumor necrosis factor alpha (TNF-α), transforming growth factor (TGF-β), platelet factor-4 (PF4), and bacterial degradation products all attract neutrophils into the wound. Infiltration with neutrophils occurs within 24 to 48 h after injury. They do not appear to contribute to the normal healing process other than preventing infection and debriding the wound. Depletion of neutrophils does not result in significant alteration of the healing process. Subsequently, monocytes are attracted into the wounded area, where they transform into macrophages. The recruitment of macrophages and their activation are essential for effective wound healing; failure to recruit them will result in severely impaired wound healing. They represent the predominant cell type within the wound between 48 and 72 h after wounding. The tasks of macrophages include phagocytosis of expendable neutrophils, pathogenic organisms, cell and matrix debris; mediation of angiogenesis and fibroplasia; and synthesis of nitric oxide (NO). They also initiate the transition to the proliferative phase.[21]

Epithelialization, Angiogenesis, and Provisional Matrix Formation (Day 4 Through 14)

Once activated, macrophages release a multitude of agents at the wound site, thereby initiating the proliferative phase of wound healing: Collagenases debride the wound; interleukins and TNF stimulate fibroblasts, which initiate granulation, tissue formation, and collagen deposition, TNF-α and basic fibroblast growth factor (bFGF) promote angiogenesis; while TGF stimulates keratinocytes, which in turn leads to epithelialization. Macrophages also secrete IL-1 and keratinocyte growth factor 2 (KGF-2), which stimulates fibroblasts to secrete KGF-2 and IL-6, which in turn cause keratinocytes to proliferate and migrate.[5] Keratinocytes are then able to express IL-6 and NO themselves and thereby perpetuate the

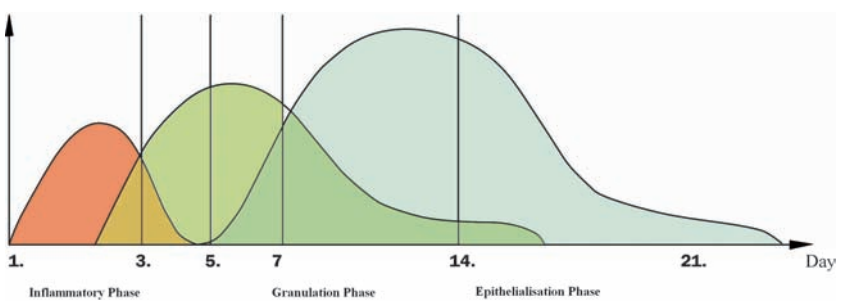

Figure 1.1. Time course and overlapping of the three distinct phases of wound healing. Time is indicated by days after wounding. Figure modified from: Arco G, Horch RE. Chirurgie der Narben. Grundlagen, Prävention und Behandlungsmethoden. CHAZ. 2009; 10:1 German.

process. If the basement membrane has been destroyed, epidermal regeneration occurs from proliferating epithelial cells located on the skin edge of the wound. In order to restore the integrity of the epidermal layer, keratinocytes must migrate over the wound margin and therefore cut a path through the fibrin clot or along the interface between the clot and the healthy dermis. For this purpose, leading edge keratinocytes express particularly high levels of tissue-type plasminogen activator (tPA) or urokinase-type plasminogen activator (uPA), both of which activate plasmin, the chief fibrinolytic enzyme. Various members of the matrix metalloproteinase (MMP) family are also preferentially generated by leading edge keratinocytes as well as fibroblasts, macrophages, and monocytes. In particular, MMPs-1, -9, and -10 facilitate migration of the above cells through the extracellular matrix.

The connective tissue in the wound is referred to as granulation tissue because of the granular appearance caused by the invading capillaries. Angiogenesis, that is, the process of forming new blood vessels, is ongoing throughout the previously mentioned phases of wound healing. bFGF and vascular endothelial growth factor (VEGF) are released at the wound site by endothelial cells, macrophages, and keratinocytes. Endothelial cells also generate NO in response to hypoxia, and this in turn stimulates more VEGF production. NO causes vasodilation of the endothelium and has a protective effect on newly formed tissue with regard to ischemia and reperfusion injury.

The formation of granulation tissue is the final part of the proliferative phase. Platelet-derived growth factor (PDGF) and EGF, which are generated by fibroblasts and macrophages, serve as the main signals for incoming fibroblasts to synthesize collagen. Fibroblasts themselves perpetuate the process with autocrine and paracrine stimulation with PDGF.

Fibroblasts that are located directly at the site of injury not only synthesize collagen for granulation tissue formation but can also be stimulated by macrophages (through TGF-β1 and PDGF) to transform into a myofibroblast and contribute to wound contraction. By about a week after wounding, fibroblasts are the predominant cell type in the wound. At the same time, the fibrin clot will have been remodeled toward a collagen-rich matrix, and wound contraction will subsequently take place under the influence of the myofibroblast.[5]

Maturation and Remodeling

Maturation and remodeling of wound healing begins at around a week after wounding and continues for over months for until a year after wounding. It encompasses collagen deposition in an organized manner toward a stable network. Clinically, maturation and remodeling is a particularly relevant stage in wound healing, since problems will occur through matrix deposition deficits (reducing wound strength) as well as through excessive matrix deposition (formation of hypertrophic scars and keloids). The collagen initially formed after wounding is thinner than that in unwounded skin. The increased rate of collagen deposition after wounding is the result of both a net increase in collagen production per fibroblast and an increase in the number of fibroblasts.[7] TGF-β directs the construction of the collagen matrix, with growth factor levels peaking in the wound between day 7 and 14 after wounding. Over time, the initial collagen is replaced by collagen strands that are thicker and therefore more stable. This can be verified by the increase in tensile strength of the wound over time. Nevertheless, the collagen in the resulting scar will never reach the stability of the collagen present in intact skin. Therefore, wound strength may reach up to only about 80% of that in uninjured skin, compared with 3% at 1 week and 30% at 3 weeks.[19]

Table 1.1 gives an overview of the several cell types involved in the different phases of wound healing as outlined here, the growth factors and cytokines they secrete, and their actions. A summary of the growth factors involved and their actions can be found in Table 1.2.

Abnormal Wound Healing

A multitude of local as well as systemic factors or conditions are associated with or will result in abnormal wound healing. Abnormal wound healing is often multifactorial.

Delayed Wound Healing

Numerous local and systemic factors can greatly influence wound healing. Adequate blood supply to provide glucose and oxygen during the healing process, which is characterized by increased metabolism and protein synthesis, is of paramount importance. Hypoxia results in a delay of the healing process. Prolonged times of

Table 1.1. Growth factors, cytokines, and other mediators and their role in wound healing.

Growth factor	Source cells	Functions
PDGF	Platelets, macrophages, monocytes, fibroblasts, smooth muscle cells, endothelial cells	Chemotaxis and activation of neutrophils and macrophages, fibroblast proliferation, chemotaxis and collagen metabolism, angiogenesis
VEGF	Receptors found on endothelial cells only Expressed by most skin cells	Does not act on macrophages, fibroblasts, and smooth muscle cells
EGF	Platelets, macrophages	Mitogenic for keratinocytes and fibroblasts, stimulation of keratinocyte migration
TNF-α	Macrophages, mast cells, T lymphocytes	Activation of macrophages and stimulation of angiogenesis, mitogenic for fibroblasts
KGF	Fibroblasts	Stimulation of keratinocyte proliferation, migration, and differentiation
TGF-α	Macrophages, T lymphocytes, keratinocytes	Mitogenic for keratinocytes and fibroblasts, stimulates keratinocyte migration
TGF-β	Platelets, T lymphocytes, macrophages, endothelial cells, keratinocytes	Chemotaxis of cells stimulating angiogenesis and fibroplasia
Interleukins	Macrophages, mast cells, keratinocytes, lymphocytes	IL-1: Induction of fever and adrenocorticotrophic hormone release, activation of granulocytes and endothelial cells, stimulation of hematopoiesis, enhances TNF-α and IFN-γ IL-2: activates macrophages, T cells, natural killer cells, and lymphokine-activated killer cells, stimulates differentiation of activated B cells, stimulates proliferation of activated B and T cells, induces fever IL-6: induces fever and enhances release of hepatic acute-phase proteins IL-8: enhances neutrophil adherence, chemotaxis, and granule release
FGF	Macrophages, mast cells, T lymphocytes, endothelial cells	Chemotaxis and mitogenesis for keratinocytes and fibroblasts, stimulation of angiogenesis

Table 1.2. Commercially available growth factor products.

Name	Growth factor	Comments
Regranex (Ortho-McNeil Pharmaceutical)	PDGF-BB	FDA-approved for diabetic foot ulcers but also appears to be efficient in treatment of other types of wounds, such as pressure ulcers, pyoderma gangrenosum, ulcers of vasculitis, and acute surgical defects. First recombinant human growth factor to be used in clinical practice.
Procuren (Curative Health Services)	PDGF	The first product to be commercially available. Platelet collected from patient's blood. Therefore, other growth factors may be part of the preparation.
Leukine (Immunex Corp.)	GM-CSF	Primarily treatment for patients with AML and bone marrow rescue, but also tried in some chronic wounds with promising results, injectable around ulcerated areas, topically as aqueous solution.
Repifermin (Human Genom Sciences)	KGF-2	Treatment for cancer therapy-induced mucositis, venous ulcers, and skin grafts. Shown to significantly increase healing and epithelialization over the wound bed. Initial trial in venous ulcers with very promising results. Currently phase two clinical development.

hypoxia will result in endothelial cell apoptosis induced by TNF-α,[13] while wound neutrophils show decreased activity. Their function is also impaired through low temperature, low pH, and elevated glucose concentrations.[1] Fibroblasts respond to hypoxia with a reduced formation of the extracellular matrix, resulting in delayed healing.[20]

Edema leads to increased interstitial pressure and thereby tissue ischemia. Clinically, tissue

edema after ischemia-reperfusion injury in skeletal muscle can lead to the compartment syndrome. Raised tissue pressure induces increased capillary closure, leading to severe hypoxia, which in turn results in cell death with necrosis of various tissues.[15]

Local wound infection exerts an inhibitory effect on wound healing, because bacteria prolong the inflammatory phase and inhibit epithelialization, contraction, and collagen deposition. Collagen degradation is increased due to increased collagenase levels. Bacterial infection is precipitated by foreign bodies. Moreover, they constitute a physical obstacle within the wound, preventing wound contraction and complete epithelialization.

Wound complications also occur when patients have characteristics on a systemic level that predispose them to wound healing problems. Conditions such as old age, smoking, obesity, burns, steroid therapy, and diabetes have been associated with delayed wound healing for a long time. The impairment of healing in diabetic patients is due to several etiologies.[5] So-called diabetic ulcers usually occur in diabetic patients suffering from neuropathy, leading to impaired sensibility and failure to release cutaneous pressure. The concomitant vasculopathy leads to ischemia and reduced supply of oxygen and other nutrients. The risk for infections is increased due to impaired function and chemotaxis of granulocytes. Diabetic ulcers are also characterized by prolonged inflammation, impairment of neovascularization, decreased collagens synthesis, increased levels of metalloproteinases, and defective macrophage function.

Increased serum glucose has a major effect on wound healing. Traditionally, diabetic complications were believed to be related mainly to microvascular occlusive disease, but recent research points to an additional direction.[6] Accumulation of the toxic byproduct of glucose metabolism, sorbitol, appears to account for vascular, renal, and ocular complications associated with diabetes. Dermal vascular permeability is increased and leads to pericapillary albumin deposition, resulting in impaired diffusion of oxygen and nutrients. The function of structural and enzymatic proteins is impaired due to hyperglycemia-associated nonenzymatic glycosylation, the latter increasing collagen's resistance to enzymatic degradation and rendering it less soluble.[9]

Experimental as well as clinical studies of diabetic wound healing show decreases in granulation tissue formation and decreased collagen levels in granulation tissue along with defects in collagen maturation. Wound maturation is delayed and the number of dermal fibroblasts is decreased. The levels of several different growth factors were shown to be reduced as well.[6]

Adjuncts to Wound Healing

Adjuncts to wound healing attempt to correct some of the described obstacles to wound healing on several different levels. Some of the most promising adjuncts are presented in the following sections.

Bioengineered Skin

Skin replacement products such as bioengineered skin can be differentiated based on their composition and classified based on their structure as either epidermal, dermal, or composite and as living or nonliving (Table 1.3). Their purpose is to supply the wound with ingredients favorable to healing, such as growth factors, cytokines, a collagen matrix, and – depending on the product – cells.[10] Apligraft, for example, is a composite consisting of neonatal fibroblasts, keratinocytes on a collagen allograft. The cells appear to act as a rich source of growth factors and collagens, which stimulate epithelialization, formation of granulation tissue, angiogenesis, and chemotaxis while they themselves proliferate, thereby contributing to wound coverage in an autocrine and paracrine fashion.[8]

Table 1.3. Bioengineered skin substitutes and their features.

Composition	Structure/living	Trade name
Cultured keratinocyte autografts	Epidermal/yes	Epicel
Treated cadaver skin allograft	Dermal/no	AlloDerm
Bovine collagen/glycosaminoglycan/Silastic	Dermal/no	Integra
Neonatal fibroblast/polyglactin mesh allograft	Dermal/yes	Dermagraft
Neonatal fibroblast/keratinocyte collagen allografts	Composite/yes	Apligraf

Growth Factors

Numerous experimental studies have demonstrated the beneficial effects of recombinant growth factors in different wound healing models in the past. This has prompted clinical studies in the course of which some of the initial hopes were disappointed but, nevertheless, resulted in clinical approval of several commercially available growth factor products[5] (Table 1.2).

Negative Pressure Therapy

Negative pressure therapy, also labeled vacuum-assisted closure, uses a subatmospheric pressure dressing and converts an open wound into a controlled, closed wound. Negative pressure provides wound conditioning and promotes healing through several different mechanisms. First, tissue oxygenation is improved by reduction of edema and interstitial fluid. Granulation tissue formation was shown to be enhanced compared with controls. Infected wounds benefit because wound bacterial count is reduced by ongoing negative pressure therapy (Figure 1.2). Inflammatory mediators that suppress the normal progression are removed more efficiently.[2] Hence, the indications for use of vacuum-assisted closure devices is manifold and includes soft tissue loss, exposed bone, hardware, or prostheses, and as a skin graft bolster. Contraindications are few and include malignancy present in the wound or when the foam would be placed directly over exposed arteries or veins. Caution should be exerted when there is active bleeding in the wound; hemostasis is difficult due to debridement or anticoagulant therapy.[11] Overall, the vacuum dressing has been a valuable addition to wound therapy, because it gives the patient and the surgeon time to improve wound conditions and thereby significantly improve the efficiency of other therapeutic measures, including reconstructive plastic defect coverage.

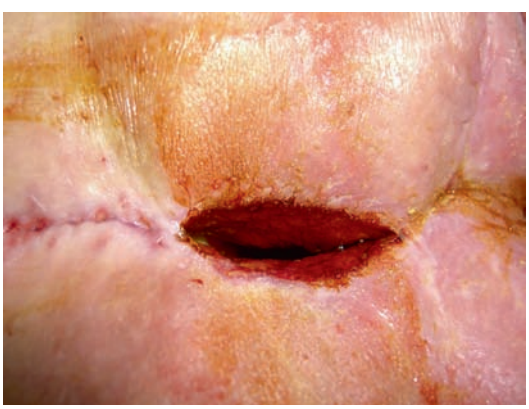

Figure 1.2. Negative pressure therapy can help to induce granulation tissue and eliminate bacterial burdens from an infected wound like in decubital ulcers, as shown here in a presacral ulcer after 1 week of continuous vacuum therapy.

Keloids and Hypertrophic Scars

Hypertrophic scars are raised but limited to the borders of the incision. Keloids, on the other hand, are scars that have overgrown the boundaries of the incision. Both are fibroproliferative disorders characterized by excess accumulation of collagen within the wound.[4] Abnormalities have been described in cell synthesis and migration, synthesis and secretion of extracellular matrix proteins, and remodeling of the wound matrix. Increased activity of fibrogenic cytokines and an exaggerated response to these cytokines have also been reported. Additionally, abnormal epidermal–mesenchymal interaction and mutation in regulatory genes have been proposed to help explain abnormal healing in this context.

Keloids occur during periods of physical growth in the majority of cases, with a peak between age 10 to 30 years.[17] All races are affected by keloids, but darkly pigmented skin is affected 15 times more often than light skin. The most common causes appear to be unspecific trauma, vaccinations, and tattoos, with the strongest predisposing factor being earlobe piercing.[5]

The etiology of keloid formation remains to be identified; possible causes include abnormal fibroblast activity, increased levels of certain growth factors such as TGF-β1 and -2, decreased apoptosis, increased levels of plasminogen activator inhibitor-1, abnormal immune reactions, and hypoxia.

Hypertrophic scars usually occur within 4 weeks after injury and may regress with time. Most keloids form within 1 year after wounding but may begin to grow years after the initial injury. A multitude of treatment options are available, but none of them has been shown to be completely effective. Keloids are particularly

notorious for high recurrence rates. Surgical excision or excision using a laser, steroid injections, radiation therapy, magnetic discs, cryosurgery, and application of silicone gel sheets have all been used and shown to be beneficial to a certain extent. However, no universally effective treatment has emerged so far. Corticosteroid injections using triamcinolone are commonly regarded as an efficacious first-line therapy. Silicone gel sheets are often a good recommendation in children and those who do not tolerate the pain associated with other therapies.[3]

Future Wound Healing Therapies

Skin is an easily accessible tissue, and its most superficial part, the epidermis, is characterized by a high turnover rate. During wound healing, a multitude of cytokines and growth factors undergo short-term up- and downregulation. All these facts render skin wound healing an ideal setting for gene therapy approaches, which are currently believed to be the most promising tool to enhance wound healing in the future. Short-term gene expression, which is often a drawback of many gene therapy vectors in other circumstances, is desirable when it comes to wound healing. Induction of the gene into the wound can be carried out either directly or indirectly by keratinocytes or fibroblasts, which can be harvested and cultured in vitro, followed by transduction with a gene of interest, for example, a gene encoding a growth factor, and finally transplanted into the wound. Many different protocols and vectors have been investigated,[18] and the most common and promising are presented in the following section.

Viral Techniques

The most common viral vectors in gene therapy for wound healing have been retroviruses, adenoviruses, and adeno-associated viruses. Recombinant viral vectors are generally created by deletion of certain parts of their genome, thereby disabling viral replication, while at the same time a gene of interest is inserted, most commonly encoding for a growth factor. The packaging capacity of the vector limits the size of the gene that can be inserted and is dependent on the type of virus. The gene of interest is usually cloned under the control of a particularly powerful promoter such as the cytomegalovirus promoter to optimize the expression of the desired gene. Retroviruses have a high efficiency in ex vivo transduction but carry the risk of insertional mutagenesis and subsequent tumorigenic transformation. Adenoviruses also attain good transfection efficiency in vivo but can induce an immune response and allow only small DNA inserts up to 8 kb. Adeno-associated viruses can provide particularly long-lasting gene expression, while they are difficult to grow to high titers and also carry the risk for insertional mutagenesis. Herpes simplex virus allows for particularly large DNA inserts but is difficult to manipulate due to its complex life cycle and carries the risk of potential wild-type breakthrough. Generally, nonviral gene transfer techniques are considered safer but often less efficient in terms of efficiency.[16]

Naked DNA

Transduction using naked DNA is probably the safest method for gene delivery. Simple injection using hypodermic needles was shown to deliver and express genes in clinically relevant concentrations, even though the transduction efficiency is very low. The "gene gun" approach where DNA-coated gold particles are employed or microseeding, which employs a set of oscillating needles to which DNA is delivered via an infusion pump, provided superior gene transfer to wounds by increase in the surface area and induction of microtrauma of the treated tissue, thereby improving DNA uptake.

In electroporation, brief electric impulses transiently create pores in the plasma membrane of the cell to allow for DNA diffusion into the cell.

Cationic Liposomes

These positively charged lipid vesicles form a complex with negatively charged DNA. Transfer of the DNA across the cell membrane appears to occur through an endocytosis-like process. Due to their lack of immunogenicity, repeated deliveries in vivo are possible. Another advantage is the potential to deliver large amounts of DNA and to incorporate large transgenes. The limiting factor, however, is their low transfection efficiency in vivo compared with that of viral vectors.

Conclusion

Adequate wound management relies on a multitude of factors and requires a profound knowledge of the mechanisms of wound healing and the factors that influence it.

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2

Immunology of Tissue Transplantation

Aleksandra Klimczak and Maria Siemionow

Summary

Composite tissue allograft (CTA) is one of the tissue reconstruction options in plastic and reconstructive surgery. Surgical techniques in CTA are well established; however, the immunologic characters of the components of CTA still require extensive research to protect allografts from rejection. CTA are histologically heterogeneous and composed of tissues such as skin, muscle, bone with bone marrow, lymph nodes, nerves, and vessels, which represent variable levels of immunogenicity and may generate different types of immunologic responses.

This chapter introduces the immunological status of CTA components, the role of immunomodulatory and inflammatory mediators for allograft acceptance, immune response to foreign antigens, and the hierarchy of antigenicity of individual components of CTA compared with the whole CTA transplant.

Abbreviations

APC	Antigen-presenting cells
CGRP	Calcitonin gene-related peptide
CLA	Cutaneous lymphocyte-associated antigen
CTA	Composite tissue allografts
DDC	Dermal dendritic cells
FDC	Follicular dendritic cells
ICAM-1	Intercellular adhesion molecule-1
LC	Langerhans cells
MAd-CAM-1	Mucosal addressin cellular adhesion molecule-1
MHC	Major histocompatibility complex
OPN	Osteopontin
SALT	Skin-associated lymphoid tissues
SIS	Skin immune system
Tc	T cytotoxic
Th	T-helper
VCAM-1	Vascular cell adhesion molecule-1

Introduction

Composite tissue allograft (CTA) is one of the tissue reconstructive options in plastic and reconstructive surgery and inclusion of CTA into the armamentarium of reconstructive surgery is encouraged. CTAs differ histologically and immunologically from solid organ transplants. CTA are histologically heterogeneous and composed of different tissue types, such as skin, muscle, bone with bone marrow, lymph nodes, nerves, and vessels, which exhibit differential antigenicity and susceptibility to rejection. After transplantation, host immunocompetent cells may elicit immune responses to the foreign antigens coming from the diffe-rent tissues of CTA. This may result in allograft rejection, which is an inflammatory process coordinated by a series of events, such as activation of leukocytes, adhesion to the vascular endothelium, and migration to the target tissues, leading to their destruction.

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The uniqueness and complexity of responses specific to different CTA components, such as skin, muscle, bone with bone marrow, lymph nodes, nerves, and vessels, are outlined here.

Inflammatory Mediators

Immune response is a multistep process involving cell adhesion molecules (CAM), cytokines, and chemokines, leading to T-cell activation. *Cytokines* are regulatory proteins secreted by a variety of cells; however, constitutive production and secretion of cytokines are usually low or absent. Cytokine production is regulated by various stimuli and is usually transient. They act locally by binding to the cell originating from the same line (autocrine) or to the target cell in the vicinity (paracrine).[1] According to their functional and biological role in inflammation, cytokines are subdivided into proinflammatory cytokines (interleukins: IL-1α, IL-1β, IL-6; tumor necrosis factors: TNF-α, TNF-β), cytokines involved in T-cell differentiation (IL-2, IL-4, IL-5, IL-10, IL-12, IL-13, and IFN-γ), and cytokines of immunoregulatory function belonging to the TGF-β family, which promotes wound healing and fibrosis.[1]

Chemokines are a subset of cytokines that are defined as small chemotactic cytokines and are produced by leukocytes and other cells. Chemokines, which are constitutively expressed, are involved in homeostatic lymphocyte trafficking to the lymphoid organs. The main role of proinflammatory chemokines (MIP-1α, MIP-1β, MCP-1, and RANTES) is to attract neutrophils to inflammatory sites and trigger T-lymphocytes to elicit inflammatory responses.[2]

Chemokines bind to the cellular component by a specific chemokine receptor. Chemokine receptor binding initiates a cascade of intracellular events, leading to the activation of leukocytes by upregulation of adhesion proteins.[1]

Cell adhesion molecules play a role in leukocyte migration from the circulation to tissues. Three types of CAM are involved in the transmigration process: selectins (L-, E-, and P-selectins) mediating the rolling of leukocytes along the vascular endothelium, integrins [intercellular adhesion molecule-1 (ICAM-1), vascular cell adhesion molecule-1 (VCAM-1), mucosal addressin cellular adhesion molecule-1 (MAdCAM-1)] leading to leukocyte adhesion to the endothelium, and finally immunoglobulin superfamily platelet endothelial cell adhesion molecule-1 (PECAM-1) responsible for transmigration of leukocytes.[3]

The complex specific migration of leukocytes to the target tissue requires a coordinated process of proinflammatory mediators. Proinflammatory cytokines, IL-1α and TNF-α, may induce expression of proinflammatory chemokines. Chemokines play a major role in the activation of integrins needed for adhesion of rolling leukocytes to the vessel endothelium, and this process leads to leukocyte transmigration to the surrounding tissue, initiating the inflammatory process.

Hierarchy of Immunogenicity of CTA

The hierarchy of immunogenicity of tissues and organs was introduced for the first time by Murray and skin was assessed to be the most immunogenic organ.[4] In composite tissue allograft (CTA) experimental models, the knowledge of the relative antigenic strengths of allograft is based on experimental data of allograft survival and split tolerance studies.[5–7] The highest degree of antigenicity was assessed to the skin, and muscles were determined of intermediate immunogenicity. Bone marrow and lymph nodes contain immunocompetent cells and may participate in the immunologic response. Lower immunogenicity was assessed in the nerve, bone, and vessels, and the least antigenic tissues were found to be cartilage and tendon.[6,7]

Skin

Immune System of the Skin

Skin is the largest organ in the human body with a specific immunological microenvironment formed by cells and humoral compounds with a precise organization and represents a natural barrier with the ability to respond to foreign antigen with innate (inflammatory) and adoptive (specific) immune responses. The active defense function of the skin is accomplished by a specific immune system known as skin-associated lymphoid tissue (SALT) and skin immune system (SIS). The cellular components of the SALT include (1) *antigen-presenting cells (APC)*, (2) *skin-seeking lymphocytes*, (3) *keratinocytes and fibroblasts*,

(4) *dermal microvascular unit*, (5) *neural immunologic network*, (6) *skin-draining lymph nodes*.[8] However, in many studies the skin-draining lymph nodes are not included in the immune system of the skin, and these immune components of the skin without skin-draining lymph nodes are known as SIS.[9]

Skin-Resident Cells – a Component of the Skin Immune System

Antigen-Presenting Cells

Within the skin there are different types of cells with antigen-presenting function. In the epidermis they are Langerhans cells (LC), in the dermis they are dermal dendritic cells (DDC),[10] and both belong to the network of cutaneous dendritic cells (DC). LC and DDC represent the main populations of professional antigen-presenting cells (APC) in the skin, and they are able to internalize and process antigen, migrate to the peripheral lymphoid organs, and stimulate naïve T cells. All express major histocompatibility complex (MHC) class I and class II molecules; costimulatory molecules CD80, CD86, CD45RO, CD13, and CD33[10]; adhesion molecules CD11a, CD44, and CD54; as well as cutaneous lymphocyte-associated antigen (CLA) and L-selectin (known as a lymph node homing molecule).[10,11] Moreover, LC and DDC produce and secrete a set of cytokines, such as IL-10, TGF-β1, and IL-23. Skin DC have the capacity to induce primary response to foreign antigens invading the skin, and in the presence of proinflammatory cytokines IL-1α, IL-1β, TNF, and prostaglandin E2, they migrate into draining lymph nodes to initiate immune responses[12] (Figure 2.1). However, immunogenic and tolerogenic functions of skin-resident DC to foreign stimuli constitute a major barrier to skin and

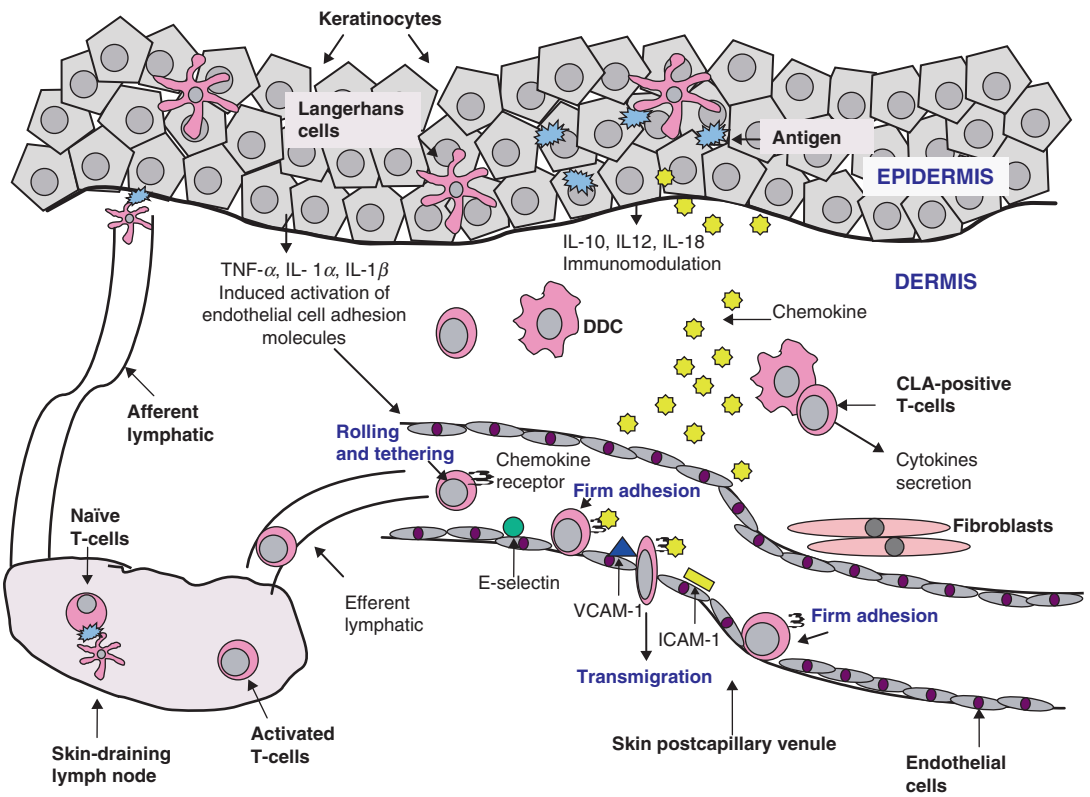

Figure 2.1. Schematic overview of the skin-associated immune system. Defense function of the skin is accomplished by interactions between antigen-presenting cells, dermal T–lymphocytes, and proinflammatory and immunomodulatory mediators, such as cytokines, chemokines, and cell adhesion molecules.

bone marrow allotransplantation, as skin DC are essential for initiation of immune response and allograft rejection.[13,14]

Skin-Seeking Lymphocytes

Lymphocytes present in normal skin are of T-cell type, and 90% of them are preferentially localized around the vessels. Skin perivascular T cells are composed of CD4 cells (T-helper subset (Th)), and in most cases they are activated as express MHC class II molecules. In contrast, intraepidermal T cells are mostly CD8 cells (suppressor-cytotoxic subset (Tc)).[15] Memory T cells present in the skin are responsible for initiation of immune responses. T-helper cells are divided into two subgroups: Th1 cells producing IL-2, IL-12, and IFN-γ cytokines are responsible for cell-mediated immune response, and Th2 cells secreting IL-4, IL-5, IL-6, IL-10, and IL-13 cytokines are responsible for humoral immune response.

B cells were not detected in the normal skin.[15]

Keratinocytes and Fibroblasts

Keratinocytes represent the principal cell population of the epidermis, and via secretion of cytokines and expression of adhesion molecules, they create a specific microenvironment and regulate the immunologic response to exogenous antigens. Many cytokines are produced by keratinocytes constitutively or on induction of various stimuli. A set of cytokines produced and secreted by keratinocytes includes (i) proinflammatory cytokines IL-1, IL-6, IL-8, and TNF-α; (ii) T-cell-tropic cytokines IL-7 and IL-15; and (iii) immunomodulatory cytokines IL-10, IL-12, and IL-18. Keratinocytes were also recognized as being a source or target of IL-10 family members such as IL-20 and IL-24.[16] Moreover, on stimulation by IFN-γ keratinocytes express MHC class II molecules and ICAM-1. Keratinocytes expressing MHC class II molecules have the ability to not only take up antigen but also efficiently process it and present to both the Th1 and Th2 types of CD4 + T cells. These findings demonstrate that activated keratinocytes may act as an APC and are able to induce functional responses.[17] Cytokine production by keratinocytes has systemic effects on the immune system and multiple implications for migration of inflammatory cells.

Skin fibroblasts belong to the nonimmune-response associated cells in the skin, and, as proposed by Postlethwaite, fibroblasts are included in the dermal immune system (DIS) mainly because they are intrinsically related to homeostasis of other skin components such as epidermis.[18] Fibroblasts produce a variety of cytokine types and growth factors including IL-1, IL-2, IL-8, IFN-β, G-CSF, M-CSF, GM-CSF, TGF-α, TGF-β, and SCF. Moreover, stimulated by proinflammatory cytokines, skin fibroblasts express ICAM-1 involved in adherence of leukocytes in the dermis. In addition, extracellular matrix proteins such as fibronectin, vitronectin, and collagen produced by skin fibroblasts may modulate immune responses.

Dermal Microvascular Unit

The dermal microvascular unit constitutes a specific microenvironment around the postcapillary venules and is composed of endothelial cells of the vessels, perivascular mast cells, DDC, macrophages, and T cells. Endothelial cells of the skin postcapillary venules constitutively express low levels of E-selectin and ICAM-1, adhesion molecules that are responsible for the initial step in emigration of T cells from the blood into the skin. After initiation of antigen stimuli perivascular mast cells secrete cytokines that activate endothelial cells and by this way regulate expression of adhesion molecules.[9]

Neural Immunologic Network

The dermis is replete with unmyelinated nerve axons that are located very close to the dermal vascular unit mainly in the vicinity of mast cells and endothelial cells. Nerve endings in the epidermis are located close to Langerhans cells. Neurogenic influence on the reaction of the immune system may be regulated by neurotransmitters such as calcitonin gene-related peptide (CGRP), vasoactive intestinal polypeptide (VIP), and substance P, which can activate mast cells.

Skin-Draining Lymph Nodes

In many studies skin-draining lymph nodes are not included in the SIS; however, regional lymph nodes are the first destination of the APC migrating from the skin. Skin APC stimulated by foreign antigens are capable of capturing antigen and migrating to the regional lymph nodes, where they present antigen to naïve and memory T cells. Activated and memory T cells then home to the skin and can recognize foreign antigens.

Skin-draining lymph nodes, in addition to T- and B-lymphocytes, contain several phenotypically distinguishable dendritic cell populations at different maturation stages capable of induction of primary or secondary immune responses against foreign antigens delivered to the lymph nodes.[19]

Hair Follicle

The hair follicle constitutes an integral part of hair-bearing skin. In adult skin the bulge of hair follicle contains a reservoir of stem cells, which can be mobilized to regenerate the new follicle with each hair cycle and to reepithelialize epidermis during wound healing. When active hair growth (anagen) ceases, the lower half of each follicle degenerates (catagen).After the rest period (telogen), the stimulus involving the dermal papilla signals follicle epithelial cells at the base to initiate the regeneration of the lower follicle to produce a new hair.[20] Hair follicle immune system is represented by cellular components such as Langerhans cells, T-lymphocytes, mast cells, and macrophages. Langerhans cells are restricted only to the distal part of the follicle where they usually display dendritic phenotype.

Distribution of intraepithelial T cells to the distal outer root sheath is similar to the localization of Langerhans cells. In the mouse, intraepithelial T-lymphocytes are represented mainly by γδT-cell subpopulation, whereas in humans αβT-cell subset predominates.

Perifollicular mast cells and macrophages, despite extraepithelial localization, constitute integral components of the hair follicle immune system, and mast cells are most numerous in hairy human skin. In addition to their role in antimicrobial defense, mast cells and macrophages participate as cellular components involved in hair biology, regulating hair growth, by secreting cytokines during hair follicle regression (catagen).[21]

The important finding in hair follicle is that the proximal epithelial hair bulb represents an immunologically privileged site in the mammalian body. The most distinctive difference between SIS and hair follicle immune system is the absence of expression of MHC class I and class II antigens in the proximal epithelial hair bulb of the normal hair follicle, making hair follicle an immune privileged site.[21] There is also a decreased number of Langerhans cells and lack of any type of T-lymphocytes. The immune privilege mechanism is also accomplished in the anagen phase of hair follicle by the production of immunosuppressive cytokines TGF-β and IGF-1.[21] The loss of immune privilege status of the hair follicle results in upregulation of MHC class I and class II molecules on follicular epithelium, which is usually the effect of increased IFN-γ production as presented in human autoimmune conditions in alopecia areata.[22] Moreover, IFN-γ may induce microvascular endothelial cells of hair follicle to express the adhesion molecules ELAM-1 and ICAM-1 responsible for the homing of lymphocytes to sites of inflammation.[22]

Muscle

Muscle constitutes one of the largest cellular compartments of CTA. Skeletal muscle represents a specific immunologic microenvironment and can actively participate in local immune responses. Under certain conditions muscle has important immunoregulatory capacities due to specific pathways of positive and negative muscle-derived regulators, which may initiate immune responses.[23] Under physiological conditions, neither MHC class I nor class II molecules are detectable on the mature muscle fibers. However, MHC class I molecules are upregulated on the muscle cells in various autoimmune and inflammatory diseases. Studies on expression of MHC class II molecules on the surface of muscle fibers in inflammatory myopathies are inconsistent.[24,25] However, it was proven recently that IFN-γ, TNF-α, and IL-1β induce expression of MHC class II molecules on the cultured human myoblasts.[26] If the muscle does express MHC class II molecules in vivo, they could hypothetically present not only viral or bacterial antigens but also muscle autoantigens or alloantigens to CD4 T cells.[23]

In addition to expression of classical MHC class I and MHC class II molecules, skeletal muscle can express HLA-G 'nonclassical' MHC class I molecule. HLA-G has been described as a molecule that mediates immunotolerizing function.[27]

Muscle fibers do not express the classical costimulatory molecules B7.1 and B7.2. Recently, it was reported that under inflammatory conditions (e.g., inflammatory myopathies) muscle fibers express muscle related costimulatory members of the B7-family (ICOS-L, B7-H, B7-H2), and ICOS-L is capable of interacting with the ICOS receptor present on activated T cells[28,29]

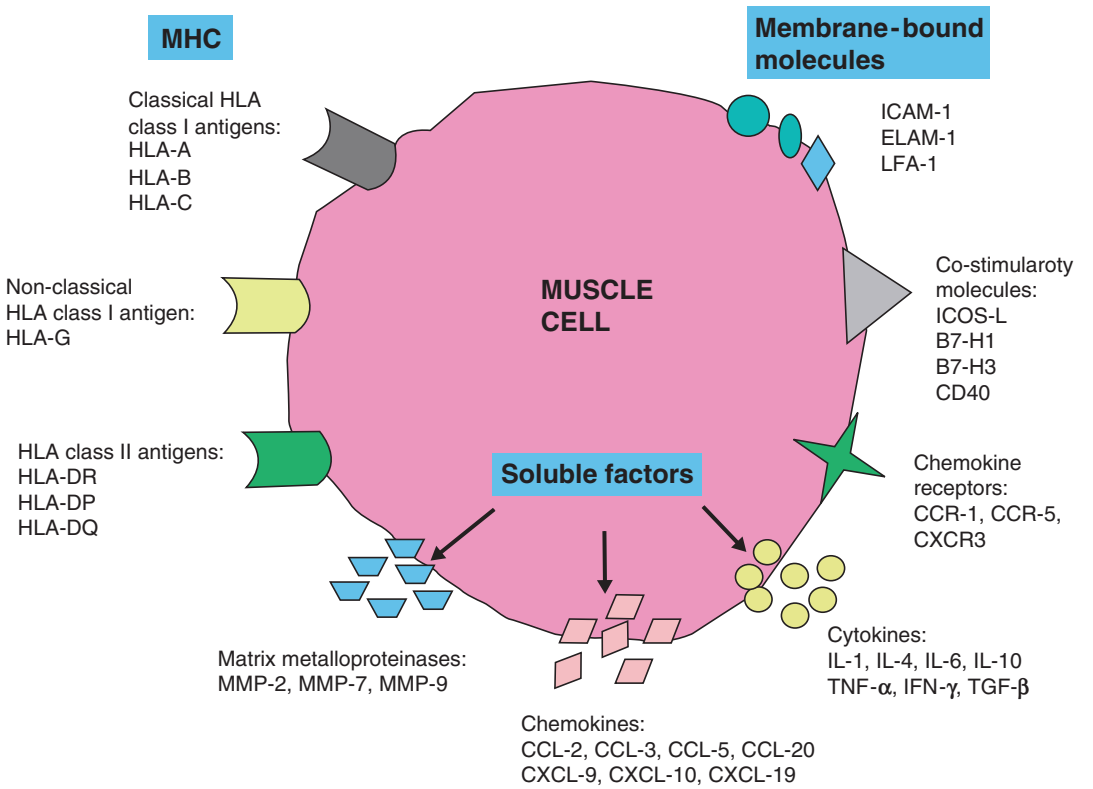

Figure 2.2. Immunoregulatory function of skeletal muscle cells. Immunocompetence of the muscle cells is accomplished by expression of classical and "nonclassical" HLA molecules; adhesion and costimulatory molecules; and secretion of cytokines, chemokines, and matrix metalloproteinases.

and triggers immune response (Figure 2.2). The presence of MHC class I and class II molecules and nonclassical costimulatory molecules on the muscle fibers supports the concept that muscle plays an active role in muscle–immune interactions and under certain conditions (IFN-γ, TNF-α) may act as a nonprofessional APC.[23]

Nerve

Nerves represent an important component of CTA responsible for proper motor function of transplanted allograft. Peripheral nerves comprise neural and nonneural elements such as (i) conducting axons, (ii) insulating Schwann cells, and (iii) surrounding connective tissue matrix. Schwann cells ensheath myelinated nerve fibers individually, whereas unmyelinated nerve fibers are surrounded by Schwann cells in groups. Myelinated and unmyelinated nerve fibers are embedded within a connective tissue called the endoneurium. The endoneurium is encircled by the perineurium, which is composed of concentrically arranged elongated perineural cells. The perineurium divides nerve fibers into fascicles. Nerve fascicles are embedded within a connective tissue compartment called the internal epineurium. Both the internal and external epineurium contain fibroblasts, macrophages, mast cells, blood vessels, and fat.[30]

Schwann cells represent the natural component of the nerve tissue and may act as immunomodulators by producing and secreting a variety of cytokines. Under certain conditions, Schwann cells are capable of regulating the production of proinflammatory cytokines IL-1, IL-6, and TNF-α and immunoregulatory cytokine TGF-β in a specific autocrine manner. Schwann cells may also synthesize other proinflammatory and immunoregulatory mediators such as prostaglandin E_2, thromboxane A_2, and leukotriene C_4, which may

regulate the immune cascade in the inflammatory conditions.[31] It was also reported that Schwann cells constructively express MHC class I but not MHC class II molecules.[32] However, after nerve injury in immune mediated disorders, in the presence of activated T-lymphocyte-released IFN-γ, MHC class II molecules were also detected on the Schwann cells, suggesting that these cells may act as an APC[33] and may contribute to local immune response.

The immunomodulatory function of Schwann cells is accomplished by the production of erythropoietin, which prevents axonal degeneration, reduces TNF-α production and Wallerian degeneration, and decreases pain-related behaviors after peripheral nerve injury.[34]

The peripheral nervous system is protected from the immune compartment by the blood–nerve barrier. However, this barrier is not complete at the root entry and exit zones, and soluble factors or immunocompetent cells may invade the nervous system (Figure 2.3). Activated T-lymphocytes and B-lymphocytes constantly patrol the peripheral nervous system, irrespective of their antigen specificity.[35] Macrophages represent APC in the peripheral nerve compartment and constitute a major population of nerve-resident cells. Macrophages are identified as a cellular source of proinflammatory cytokines IL-23 and TNF-α. Their role as an APC was confirmed by expression of MHC class II molecules and costimulatory molecules B7-1 and B7-2, which are essential for effective antigen presentation to T cells, thereby modulating the local immune response.[35]

Bone

Bone is a key component of hand transplant, and the antigenicity of the bone unit is considered to be low. Osteopontin (OPN) is a natural protein, which constitutes a bridge between bone and the immune system. OPN is a multifunctional protein

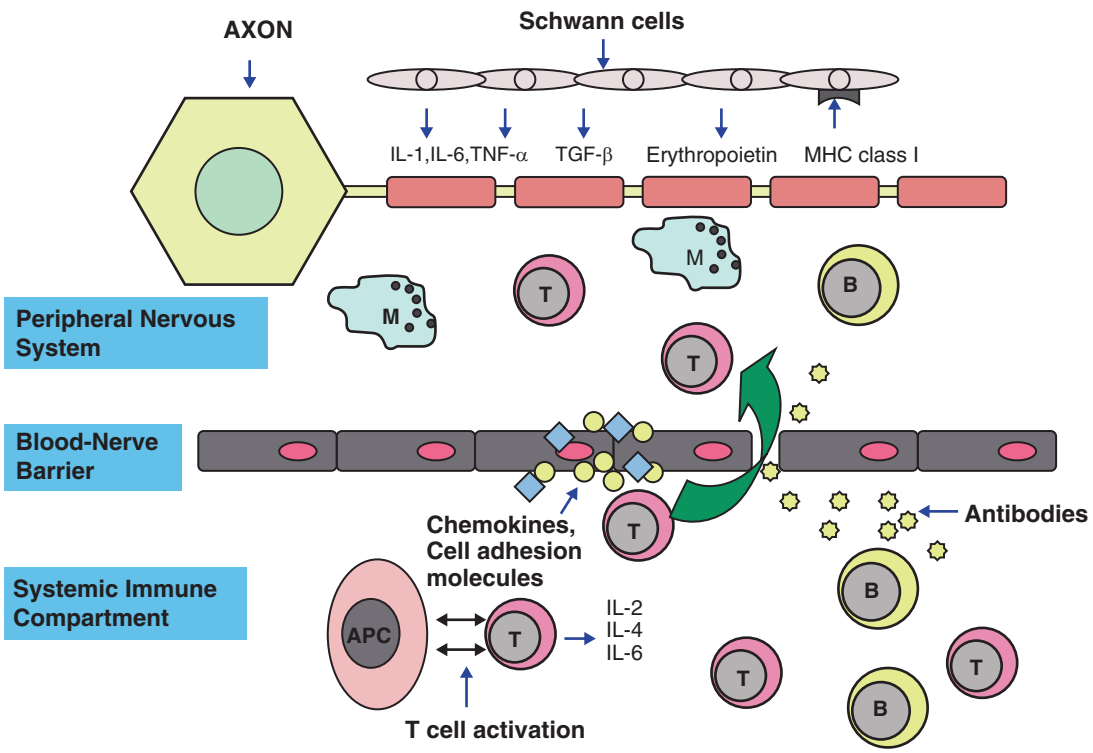

Figure 2.3. Schematic overview of local immune system in the peripheral nerve. Peripheral nerve immune system constitutes cellular (macrophages, T-lymphocytes, B-lymphocytes, Schwann cells) and extracellular components (cytokines, chemokines) modulating local immune response.

secreted by activated macrophages, leukocytes, and activated T-lymphocytes and is present in the extracellular matrix of mineralized tissue.[36] OPN is abundant in the bone, where it facilitates the attachment of osteoclasts to the bone matrix via interaction with cell surface integrins and CD44. Binding of OPN to these cell surface receptors stimulates cell adhesion, migration, and specific signaling function.[36] Upregulation of OPN leads to fibrosis, including cardiac fibrosis,[37] biliary atresia fibrosis,[38] interstitial kidney fibrosis[39] as well as acute kidney allograft rejection, which was confirmed in multiple tissues.[40]

The bone component of the hand transplant contains hematolymphoid tissue such as bone marrow and surrounding stromal elements. Bone marrow has the potential to attack the recipient immune system or under proper conditions (immunosuppression) may downregulate the host immune response against the graft and induce tolerance.[41]

Bone Marrow Cells

Transplanted vascularized bone, such as human hand, contains multilineage hematopoietic cells such as myeloid, lymphoid, and erythroid at various stages of differentiation and maturation. Within hematopoietic tissue, hematopoietic stem cells are present and are capable for lymphomyeloid reconstitution in the recipient body. The donor-origin hematopoietic cells may be involved in tolerance induction. After CTA transplantation donor bone marrow cells may migrate from transplanted tissues and colonize lymphoid and nonlymphoid organs of recipients, and engraftment of donor-origin cells into recipient lymphoid and nonlymphoid tissues is known as a chimerism.[42] Donor hematopoietic cells can develop two types of macrochimerism: (1) full chimerism when recipient immune system is destroyed by myeloablation and replaced fully (donor cells 100%) by donor hematopoietic cells or (2) mixed chimerism induced after nonmyeloablative host conditioning when donor and recipient hematopoietic cells coexist with the recipient (donor cells > 1% < 100%).[42] The third type of chimerism is defined as microchimerism, which usually occurs spontaneously after organ transplantation, and donor-origin cells represent less then 1%.[42]

The importance of the development of donor-specific chimerism in CTA is debatable. However, it is important to remember that over-representation of donor immunocompetent cells is associated with development of graft-vs-host disease, which may be fatal.[43]

The immunomodulatory function of bone marrow compartment may also be also accomplished by bone marrow-derived dendritic cells. Depending on the maturation status, bone marrow-derived dendritic cells may act either as an APC or may lead to tolerance induction.[44] After migration to T-cell areas of secondary lymphoid organs (e.g., draining lymph nodes), bone marrow-derived dendritic cells can both induce and regulate immune responses.[45]

Lymph Nodes

Lymph nodes represent the immunocompetent component of CTA and may participate in immunologic response. In contrast to other organs, lymph nodes comprise cellular elements (mostly lymphocytes) that are only temporary residents, since naïve lymphocytes continuously recirculate between different lymphoid organs. In the lymph nodes, T- and B cells are localized into separate zones. B cells are organized into follicles, located in the cortex of the lymph node; T cells are located in the paracortex in the T-zone. The B-cell follicle also contains follicular dendritic cells (FDC) and a small number of T cells. Continuous cellular migration facilitates interaction between different cellular components. Naïve T cells enter lymph nodes via postcapillary venules in the paracortex. Naïve T cells are primed by interaction with peptide/MHC on dendritic cells. When primed T cells ligate the same peptide/MHC on the surfaces of B cells and they can trigger activation of B cells. B-cell activation requires interaction between T cells and B cells that recognize the same antigen. Once activated, B cells proliferate and differentiate, leading to antibody formation. Moreover, proliferation of B cells in the germinal center resulted in the production of memory and plasma cells. This process is thought to involve B cells, T cells, and FDC.[46] Memory and effector cells and APC migrate to the lymph nodes via afferent lymphatics.

Lymph nodes within the transplanted tissues are important contributors of induction of the recipient alloimmune responses. As presented in the mice intestinal transplantation model, recipient T cells migrate to the lymph nodes of the

transplanted organ where they undergo extensive proliferation and develop effector function, leading to allograft rejection.[46]

Vessels

The vascular unit of CTA constitutes a specific component of the allograft that is responsible for graft revascularization and blood supply. The vascular system is lined by endothelial cells that play a multifunctional role, including regulation of thrombosis and thrombolysis, platelet adherence, modulation of vasomotor tone and blood flow, and regulation of immune and inflammatory responses. As a barrier, the vessel endothelium is semipermeable and controls the transfer of small and large molecules. An immune and inflammatory reaction is regulated by controlling leukocyte interaction with the blood vessels. Under inflammatory conditions, vessel endothelial cells may secrete proinflammatory cytokines IL-1, IL-6, and IL-8, and subsequently activated endothelial cells induce the expression of P-selectin and E-selectin and cell adhesion molecules (ICAM-1 and ICAM-2 and VCAM-1), facilitating leukocyte extravasation into surrounding tissue.[47] Moreover, vessel endothelial cells express APC-related MHC class II molecules and costimulatory molecule CD40, leading to proliferation and differentiation of activated or memory but not naive T cells.[48]

Immunogenicity of CTA Components vs Immunogenicity of Whole CTA

Skin is the most immunogenic component of the CTA, and in contrast to the other organs or tissues, true tolerance to the skin was not achieved in the clinic. However, in the experimental face transplant model, operational tolerance to the skin flap was introduced under a low nontoxic dose of CsA therapy.[49] Recently, in the experimental rat model, tolerance to limb allograft including skin was successfully reported under temporary T-cell depletion and short-term administration of calcineurin inhibitors, without chronic immunosuppression.[50,51] Long-term survival of limb allografts and tolerance was associated with the presence of stable donor-specific chimerism in the rat model.[50,51] However, in the miniature swine hind-limb allograft model, tolerance was achieved to the musculoskeletal part of the limb allograft but not to the skin.[5,52] These studies demonstrated split tolerance to the musculoskeletal part of the allograft but not to the skin component, and chimerism declined once 12-day CsA therapy was discontinued.

Interestingly, in the rodent experimental model immunogenicity of the whole CTA was found to be less antigenic than single components of the limb allograft.[7] This phenomenon is still unclear, but decrease in skin immunogenicity may be explained by antigen competition.[41]

The hierarchy of antigenicity of limb allograft tissues was also confirmed by the cytokine profile of individual limb allograft components and showed that the skin component produced the greatest Th1-type cytokines and appears to be the critical component of the overall antigenicity of the whole limb allograft, as evidenced by the shift to Th2 profile when skin was removed. Muscle induced the least Th1 type of response, whereas nerve presented with intermediate response.[53]

The peripheral nervous system is separated from the immune system by the blood–nerve barrier, and this is the first factor making nerve tissue less immunogenic compared with skin and muscle. Expression of many immunological mediators by the nerve tissue suggests that the secretion of proinflammatory mediators may be balanced by the immunomodulatory function of Schwann cells, due to secretion of erythropoietin, and this may be a second explanation why nerve tissue is less immunogenic compared with skin and muscle.[35]

Human hand transplants, which contain bone with bone marrow, represent a model of vascularized bone marrow transplant (BMT). However, in human hand allograft recipients, peripheral chimerism has not been detected. Only transient microchimerism was reported as a presence of donor APC in the epidermis at 77 days after hand transplantation, but it was not detected thereafter.[54] The bone marrow in human CTA is thought of as an immunoregulatory organ where mature immune cells of the graft without proper immunosuppression may induce immune response. On the other hand, donor hematopoietic cells may play an immunomodulatory role, facilitating solid organ transplant acceptance.[55] This was clinically applied recently as a supportive therapy in first partial human face transplant.[56]

The immunomodulatory effect of donor-origin hematopoietic cells for chimerism induction and allograft acceptance was proved in experimental models by the delivery of donor bone marrow cells in different forms, such as cellular BMT[57,58] and vascularized or an unprocessed (crude) form of BMT.[50,51,59]

The immune response may be altered when specialized immune organs are absent. In the absence of secondary lymphoid organs, normal immune responses to the viruses are significantly delayed.[60] Experimental studies demonstrated that the absence of spleen resulted in defective antibody response to vascularized organ transplants. Moreover, in the mouse experimental model, in the absence of both spleen and lymph nodes, mice accepted transplanted heart allografts indefinitely.[61]

Vascularized CTA may initiate alloimmune responses independent of secondary lymphoid organs, since vessel endothelial cells are effective as an APC. Discordant stimulation of vessel endothelial cells or uncontrolled immune response lead to endothelial injury, dysfunction, and activation and subsequently to allograft vasculopathy.[47,48]

Conclusion

Composite tissue allograft transplantation is accepted as an alternative reconstructive option in plastic and reconstructive surgery. However, immunological characteristics of transplanted CTA are complex and require further investigations. Further, the need for lifelong immunosuppression following CTA transplantation to prevent allograft rejection is still required, and this limits routine application of CTA in clinical practice. With better understanding of the mechanism of rejection and novel therapies targeted at different CTA components, the future is open for CTA in plastic surgery.

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3

Anesthesia and Critical Care

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Summary

The development of modern anesthesia has enabled safe conduction of the most extensive reconstructive surgical procedures, even in patients with multiple comorbidities. At the same time, continuous development and modification of anesthetic armamentarium have enabled a smooth shift from hospital-based procedures to office and ambulatory centers, without compromising patients' safety and satisfaction. The role of the anesthesiologist involves not only intraoperative patient care but also extends to preoperative evaluation as well as postoperative recovery. Careful preoperative assessment helps decide which anesthetic technique will suit the patient best and provide optimal surgical conditions. Although all anesthetic agents and techniques are very safe, there is a growing body of evidence that anesthetic choices during the perioperative period may affect long-term outcomes because of their influence on the immune system as well as perioperative inflammatory processes.

Abbreviations

ACC	American College of Cardiologists
AHA	American Heart Association
ASA	American Society of Anesthesiologists
BB	Beta blockers
BIS	Bispectral index
CEPOD	Confidential Enquiry into Perioperative Deaths
LMA	Laryngeal mask airway
MAC	Monitored anesthesia care
PONV	Postoperative nausea and vomiting
TEE	Transesophageal echocardiography
TIVA	Total intravenous anesthesia

Introduction

Development of modern anesthesia (since its introduction in 1846) and critical care has allowed for the safe conduction of extensive surgical procedures with acceptably low risk for patients. The role of the anesthesiologist has evolved over time from merely providing anesthetic care in the operating room to orchestrating preoperative evaluation and optimization, as well as postoperative critical care and acute and chronic pain management for surgical patients. Continuous improvements of anesthetic techniques, perioperative monitoring, and preoperative optimization processes have made anesthesia very safe, with quoted mortality related solely to anesthesia at 1 in 185,000, whereas in 7 of 10,000 cases anesthesia contributed to mortality as reported by the Confidential Enquiry into Perioperative Deaths (CEPOD).[16] Overall mortality related to anesthetic care is estimated at 1 per 13,000 anesthetics and has remained stable over the past two decades.[45]

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With a low rate of mortality and major morbidity related to anesthetic care, there is increasing interest in the effect of anesthetic care on more subtle, yet very important, outcomes such as postoperative cognitive dysfunction, immunomodulation, modulation of inflammatory processes and their effects on long-term outcomes, as well as overall patient satisfaction, quality of life, and economic efficiency.

This chapter outlines perioperative anesthetic care and the effects of anesthesia on selected outcomes.

Preoperative Assessment

Anesthetic care starts with preoperative evaluation to ensure that all comorbid conditions and anesthetic issues are optimized before a scheduled surgical procedure. The importance of preoperative evaluation and optimization (for instance, starting beta blockers (BB) or statins in patients with coronary artery disease, optimization of chronic medical conditions such as diabetes, hypertension, etc.) has been shown to reduce morbidity as well as surgery cancellation rate.[24,60,74] Review of past medical history and physical examination, as well as additional tests and past anesthetic history, will help the anesthesiologist formulate an anesthetic plan, assess patient risk, order additional workup, or implement perioperative interventions when appropriate. Out of the three components of preoperative assessment (past medical history, physical examination, and laboratory tests), past medical history is the most valuable. Studies have shown that almost 86% of diagnoses are dependent entirely on the information obtained from a patient's history.[37]

Preoperative evaluation may be conducted in different forms, such as telephone interview, medical records review, or formal interview and assessment at the preoperative clinic where a patient is examined and assessed by an anesthesiologist or nurse practitioner trained to perform preanesthetic evaluations. A preoperative interview focused on planned anesthesia has been shown to reduce a patient's preoperative anxiety level on the day of the surgery.[25] Routine preoperative testing should be minimized; extensive laboratory workup increases the cost of providing care without any apparent medical benefit.[29] The currently recommended routine tests for an otherwise healthy patient are summarized in Table 3.1. Preoperative tests should be ordered based on a patient's specific medical condition and the planned surgical procedure.[49,56]

Preoperative assessment also helps to determine a patient's candidacy for office or ambulatory-based surgery as well as how to tailor the anesthetic plan to the patient's medical and psychological condition. Although office- and ambulatory-based surgeries are cost-efficient alternatives to hospital-based surgery, proper patient selection is very important. Patients with significant comorbidities who may require extended recovery or are at high risk of developing postoperative complications may be better served in hospital settings,[64] where resources are readily available to deal with postoperative problems should they arise. Conditions that may preclude procedures requiring anesthesia care from being done in the outpatient setting include serious, potentially life-threatening diseases that are not optimally managed (e.g., brittle diabetes, unstable angina, symptomatic asthma), history of difficult airway, expected significant blood loss or postoperative pain, morbid obesity complicated by symptomatic cardiovascular or respiratory problems, history of substance abuse, ex-premature infants less than 60 weeks of

Table 3.1. Recommended preoperative tests for asymptomatic patients.

Age	Male	Female
< 6 Months	None	None
6 Months–40 years	Hemoglobin or hematocrit	Hemoglobin or hematocrit, ± pregnancy test for females in childbearing age
40–65 years	Hemoglobin or hematocrit and ECG	Hemoglobin or hematocrit and ECG, ± pregnancy test for females in childbearing age
65 years and older	Hemoglobin or hematocrit, ECG, BUN, glucose	Hemoglobin or hematocrit, ECG, BUN, glucose

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postconceptual age, who require general endotracheal anesthesia (increased incidence of postoperative apnea), lack of transportation from home or a responsible adult at home to care for the patient on the evening after surgery, and history of previous adverse reaction to anesthesia.[76] The American Society of Anesthesiologists (ASA) developed a simple classification system based on a patient's comorbid conditions, which helps to determine clinical risk (see Table 3.2). It provides an excellent measurement of the global well-being of a patient, based on the patient's physical status, medical comorbidities, and physiological stability.[4,45]

Since cardiovascular complications account for a significant portion of perioperative morbidity and mortality, an expert panel from the American College of Cardiologists (ACC) and the American Heart Association (AHA) has developed a rational, stepwise approach to cardiac workup before noncardiac surgery. A full description of the guidelines is beyond the scope of this chapter; however, it is important to remember that the need for testing and perioperative intervention depends on interaction between the patients' cardiac risk factors, risk of the surgical procedure, and the patients' exercise capacity.[30] It was shown in the CARP trial that coronary revascularization before noncardiac vascular surgery did not affect overall postoperative morbidity and mortality.[50] There is a uniform agreement that cardiovascular interventions (coronary artery bypass, percutaneous coronary intervention) are not indicated unless they need to be performed irrespective of perioperative context.[30]

All locations (office, ambulatory center, hospital) where anesthetic care is provided are held at the same high standard of patient safety.[7,8] Many states have strict regulations as to personnel, organization, and equipment requirements for ambulatory and office-based practices where sedation or anesthesia is administered. The ASA has also developed guidelines to assist its members who are considering the practice of ambulatory anesthesia in the office setting.[7]

Table 3.2. ASA physical status classification.

ASA physical status	Description
I	Healthy patient
II	Mild systemic disease
III	Severe systemic disease with functional limitation
IV	Severe systemic disease, constant threat to life
V	Moribund, unlikely to survive 24 h

Source: Adapted from ASA (1963) New classification of physical status. *Anesthesiology* 24:111.

Monitoring and Anesthetic Technique

In early 1986, ASA was the first medical specialty to adopt standards for patient care for its members. Today more than 30 standards, guidelines, and statements developed by the society address minimum requirements for the care of patients before, during, and after surgery. These guidelines were developed to improve patient safety and to provide the same high quality of patient care regardless of the type of anesthesia and the anesthetizing location.[50] Standards of basic monitoring during anesthetic care are described in published ASA guidelines[8] and require continuous monitoring of the patient's oxygenation, ventilation, circulation, and temperature by a qualified anesthesia provider. Use of additional invasive monitors beyond this standard, such as arterial line, central venous pressure, pulmonary artery catheter, and transesophageal echocardiography (TEE), are dictated by the patient's comorbid conditions and the invasiveness of the surgical procedure.

The anesthetic technique should be tailored to provide optimal surgical conditions and account for patient-specific requirements. Some of the procedures can be successfully done with a local anesthetic injection at the surgical site and minimal intravenous sedation (frequently described as conscious sedation). Other procedures, however, might require a deeper or changing level of sedation, nerve block, or a combination of both. It is important to understand that sedation is a continuum ranging from anxiolysis when all protective airway reflexes are preserved and the patient is able to cooperate, to a state of general anesthesia, where patients are completely unconscious, airway reflexes are absent, and airway and respiratory function require support.[6] Conscious sedation may be provided by a nonanesthesiologist in the presence of a person trained in establishing a patent airway and basic life support. Deeper levels of sedation (monitored anesthesia care [MAC], general anesthesia) and regional or central nerve blocks should be administered by a trained

anesthesia provider.[8] The choice of the anesthetic technique is based on the surgical requirements (type and length of the planned surgical procedure), the patient's comorbidities and preferences, as well as position during the surgery. Two patients undergoing the same procedure may require different types of anesthetic due to a unique combination of physical, medical, and psychological status.

Procedural sedation/analgesia is defined as the proper administration of drugs to obtund, dull, or reduce the intensity of pain or awareness; and where the administration of those drugs by any route carries the risk of loss of protective reflexes.

Definition of levels of procedural sedation/analgesia are shown in Table 3.3 (levels 1 and 2 are not covered in this chapter).[6]

Monitored Anesthesia Care

Monitored anesthesia care is provided by a trained anesthesia provider to achieve patient comfort and optimal operating conditions for the surgeon when the level of sedation is beyond simple anxiolysis. Since the level of sedation

Table 3.3. Continuum of depth of sedation definition of general anesthesia and levels of sedation/analgesia[a] (approved by ASA house of delegates on October 13, 1999, and amended on October 27, 2004).

	Minimal sedation (anxiolysis)	Moderate sedation/ analgesia ("conscious sedation")	Deep sedation/ analgesia	General anesthesia
Responsiveness	Normal response to verbal stimulation	Purposeful[b] response to verbal or tactile stimulation	Purposeful[b] response following repeated or painful stimulation	Unarousable even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular function	Unaffected	Usually maintained	Usually maintained	May be impaired

Minimal sedation (anxiolysis) is a drug-induced state during which patients respond normally to verbal commands. Although cognitive function and coordination may be impaired, ventilatory and cardiovascular functions are unaffected.

Moderate sedation/analgesia ("conscious sedation") is a drug-induced depression of consciousness during which patients respond purposefully[b] to verbal commands [note, reflex withdrawal from a painful stimulus is not considered a purposeful response], either alone or accompanied by light tactile stimulation. No interventions are required to maintain a patent airway, and spontaneous ventilation is adequate. Cardiovascular function is usually maintained.

Deep sedation/analgesia [monitored anesthesia care (MAC)] is a drug-induced depression of consciousness during which patients cannot be easily aroused but respond purposefully[b] following repeated or painful stimulation. The ability to independently maintain ventilatory function may be impaired. Patients may require assistance in maintaining a patent airway, and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained.

General anesthesia is a drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation. The ability to independently maintain ventilatory function is often impaired. Patients often require assistance in maintaining a patent airway, and positive pressure ventilation may be required because of depressed spontaneous ventilation or drug-induced depression of neuromuscular function. Cardiovascular function may be impaired.

Because sedation is a continuum, it is not always possible to predict how an individual patient will respond. Hence, practitioners intending to produce a given level of sedation should be able to rescue[c] patients whose level of sedation becomes deeper than initially intended. Individuals administering moderate sedation/analgesia ("conscious sedation") should be able to rescue[c] patients who enter a state of deep sedation/analgesia, while those administering deep sedation/analgesia should be able to rescue[c] patients who enter a state of general anesthesia.

[a] Monitored anesthesia care does not describe the continuum of depth of sedation, rather it describes "a specific anesthesia service in which an anesthesiologist has been requested to participate in the care of a patient undergoing a diagnostic or therapeutic procedure."

[b] Reflex withdrawal from a painful stimulus is NOT considered a purposeful response.

[c] Rescue of a patient from a deeper level of sedation than intended is an intervention by a practitioner proficient in airway management and advanced life support. The qualified practitioner corrects adverse physiologic consequences of the deeper-than-intended level of sedation (such as hypoventilation, hypoxia, and hypotension) and returns the patient to the originally intended level of sedation.

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during MAC may change depending on the procedure or the patient's needs, airway support may be necessary. The boundaries between MAC and general anesthesia are fluid and frequently crossed during the same procedure. Frequently, MAC is used to supplement local anesthesia achieved with generous infiltration of local anesthetic at the surgical site. Although different medications (or combinations) have been successfully used to provide MAC, the introduction of propofol to clinical practice has made it possible to precisely control the desired depth of MAC with minimal effect on the recovery time.

General Anesthesia

There are four components of general anesthesia: hypnosis, amnesia, analgesia, and areflexia. The concept of "balanced anesthesia" was developed to achieve each of the components with a specific agent rather than the use of a single drug to provide all aspects of general anesthesia. This approach enables better control over the desired depth of a particular component of general anesthesia and minimizes undesirable side effects. During general anesthesia, airway-supporting devices are frequently used to maintain the patency of airway passages; they range from simple oral and nasal airways to laryngeal mask airway (LMA) and endotracheal tubes. If there is a need for muscle relaxation and mechanical ventilation, the airway is secured preferably with an endotracheal tube. However, under certain circumstances, it is acceptable to deliver positive pressure ventilation via LMA. LMA can be inserted without the use of muscle relaxants, and patients report less sore throat symptoms postoperatively as compared with an endotracheal tube.[39] Individuals who are at risk for aspiration of the gastric content ("full stomach," incompetent gastroesophageal sphincter, morbidly obese patients) require endotracheal intubation when the anticipated level of sedation impairs the protective airway reflexes. For craniofacial (major jaw reconstruction, etc.) and oral (hard palate) surgery, it may be necessary to perform nasal intubation so that the endotracheal tube does not interfere with the operating field; for this purpose, preshaped endotracheal tubes are used.

Induction of general anesthesia frequently renders a patient unable to support airway patency and apneic; therefore, it is crucial to be prepared if problems with establishing the airway and effective oxygenation arise after induction. ASA developed a difficult airway algorithm to facilitate management of such instances, offering a stepwise approach ranging from the use of different airway devices to the surgical airway (tracheostomy) in order to establish effective oxygenation and ventilation.[5] Some patients can be predictably difficult to mask ventilate and/or intubate due to craniofacial abnormalities or coexisting conditions. Predictors of difficult intubation include relatively long upper incisors, prominent "overbite" (maxillary incisors anterior to mandibular incisors), mandibular incisors anterior to maxillary incisors, interincisor distance less than 3 cm, inability to visualize uvula when tongue is protruded with patient in sitting position (e.g., Mallampati class greater than II), highly arched or very narrow hard palate, stiff, indurated, occupied by mass, or nonresilient submandibular space, thyromental distance less than three ordinary finger breadths, short or thick neck, and limited neck and head range of motion.[5] In these instances, the safest option of securing the airway is awake fiberoptic intubation before induction of general anesthesia with the patient being minimally sedated and breathing spontaneously while upper airways are anesthetized for patient comfort with topical anesthetic or by means of nerve blocks. If efforts to place the endotracheal tube are unsuccessful, a surgically placed tracheostomy may be necessary. Patients with a potentially difficult airway are not well suited for procedures requiring deep sedation or general anesthesia in the ambulatory or office-based setting.

General anesthesia can be maintained using a combination of inhalation and intravenous agents or only intravenous drugs (TIVA – total intravenous anesthesia). There is no obvious advantage to using one technique over the other in all patients; however, specific clinical situations may be better served with the TIVA technique. Description of the pharmacodynamics and pharmacokinetics of the medications used in anesthetic practice is beyond the scope of this chapter.

Regional Anesthesia

Regional anesthesia includes local infiltration (usually done by the surgeon), peripheral nerve blocks, and neuroaxial blocks (epidural, spinal, caudal). Regional anesthesia is frequently supple-

mented by sedation, MAC, or general anesthesia depending on surgical and patient needs. Advantages of regional techniques include excellent surgical analgesia, blockade of the systemic stress response to the surgical stimulation, superior postoperative pain control, and avoidance of the side effects of general anesthesia.

Neuroaxial Blocks

Spinal anesthesia is probably the simplest and most reliable regional anesthetic technique; local anesthetic with or without any adjuvants is deposited in the lumbar subarachnoid space. However, the incidence of side effects may be quite high, especially when used in the ambulatory setting. The most troublesome complications of outpatient spinal anesthesia are prolonged residual block of motor, sensory, and sympathetic nervous system function, which can contribute to delayed ambulation, dizziness, urinary retention, impaired balance, and delayed discharge from the ambulatory facility. When compared with general anesthesia, the use of spinal anesthesia, even with small doses of short-acting local anesthetics, is associated with a higher incidence of backache, 35% vs. 14%, which may be difficult to accept in the outpatient surgical setting.[59]

Epidural anesthesia may be technically more difficult to perform when compared with spinal anesthesia. It also has a slower onset of action and is associated with a greater chance of an incomplete sensory block than spinal anesthesia. On the other hand, one advantage of having the catheter placed in the epidural space is the ability to extend the duration of anesthesia for procedures with variable surgical times. An epidural catheter can also be used during the postoperative period to provide excellent postoperative analgesia, in particular after extensive reconstructive surgeries. An epidural catheter can be placed at different levels of the spine (lumbar, thoracic, or cervical) to limit the neuroaxial block to the surgical site only; and by using a different concentration of a local anesthetic, selective blockade can be achieved (sympathetic, sensory, or motor) to meet the specific clinical goals.

The use of a combined spinal–epidural anesthesia technique allows for the reliability of spinal anesthesia with the flexibility of continuous epidural anesthesia, which can extend to the postoperative period.[41,72] A small initial dose of intrathecal local anesthetic (spinal) with a needle-through-needle technique is given, and then an epidural catheter is placed. If necessary, the epidural catheter could be used to extend the block beyond the duration of the spinal anesthetic.

Peripheral Nerve Blocks

Peripheral nerve blocks are well suited for surgical procedures on extremities. They may be combined with sedation or light general anesthesia. One of the advantages of a peripheral nerve block is that the duration of analgesia extends well beyond the surgical procedure, hence immediate postoperative recovery and time to discharge can be shortened. Local anesthetics can be also delivered via a catheter placed around the major peripheral nerves, providing excellent analgesia for the extended period of time. The technique of peripheral nerve blocks has evolved over time; and nowadays, utilization of nerve stimulators and ultrasound imaging helps an anesthesiologist perform peripheral nerve blocks with great precision, efficiency, and with low risk of complications. The use of peripheral regional analgesic techniques as a single injection or continuous infusion can provide superior postoperative analgesia compared with systemic opioids[2,14] and may even result in improvement in various outcomes.[17,78]

One has to remember that some blocks may be time consuming to perform, and when the patients are discharged before resolution of the block or with the peripheral nerve catheter, there must be a system in place to monitor for possible complications and follow-up with the patient's recovery.

Patient Positioning

Patient positioning should accommodate a surgeon's need for proper exposure of the operating site; however, if done improperly it may cause injury to the anesthetized patient. Ideally, the patient should position himself/herself in an anticipated position without any discomfort and then be anesthetized. Unfortunately, this solution is frequently impractical due to difficulties with induction of general anesthesia and/or securing airway in certain positions (prone, lateral decubitus). The patient is most commonly positioned for the surgery after being anesthetized.

Table 3.4. Possible complications related to surgical positioning.

Position	Complication	Prevention
Supine	Ulnar nerve injury (most common peripheral nerve injury)	Avoid pressure on the ulnar groove and the spiral groove of the humerus
Prone	Eyes, face injury Increased intra-abdominal pressure Female breasts	Proper head support (foam pillows, horse-shoe headrest, head pins) Use of chest rolls Proper positioning of chest rolls
Lateral	Brachial plexus injury	Bringing the arm into a more anterior plane with the body Use of axillary roll
Lithotomy	Peroneal nerve injury Saphenous nerve injury Lower extremity compartment syndrome	Proper padding between the head of the fibula and the lithotomy bar Avoid pressure over the medial tibial condyle Avoid extensive hip flexion
Trendelenburg	Increases central venous pressure, intracranial and intraocular pressure, myocardial work, and pulmonary venous pressure and decreases pulmonary compliance and functional residual capacity, swelling of the face, eyelids, conjunctiva, and tongue, venous stasis in the head and neck	Limit the head down angle
Sitting	Air embolism Spinal cord ischemia, obstruction of carotid and vertebral arteries, and embolic or thrombotic stroke	Meticulous surgical technique Prevent excessive flexion of the neck

Therefore, all possible efforts should be made to protect pressure points, eyes, ears, nerves, etc., against injury. Table 3.4 summarizes the most common injuries related to patient positioning. Peripheral nerve injury is the most serious complication related to improper patient positioning. There are five preventable causes of nerve injury: stretch, compression, ischemia, metabolic derangement, and surgical transection, of which the first three causes may be directly related to the surgical position. The ASA task force addressed the issue of prevention of perioperative peripheral neuropathies in a practice advisory document.[1]

Postoperative Recovery, Postoperative Pain Control, Nausea, and Vomiting

Recovery from anesthesia may vary depending on the patient and type of anesthetic used; however, the experience that follows surgery and anesthesia is what the patient is likely to remember. In the postanesthesia unit, as the patient goes through the recovery process, common postoperative problems are addressed (postoperative nausea and vomiting [PONV], pain, etc.) as well as less common, but potentially serious, respiratory (hypoxia, hypoventilation) and cardiovascular (hypotension, hypertension) complications. At the same time, patients are continuously assessed whether discharge criteria are met. There are numerous scoring systems assessing patient readiness to discharge; probably the most popular is the modified Aldrete score. It is a simple sum of numerical values assigned to activity, respiration, circulation, consciousness, and oxygen saturation. Importantly, it provides a simple and easy way to assess patient readiness for discharge (a score of at least 9 out of 10 indicates patient readiness for discharge) (Table 3.5). Frequently, the Aldrete score is modified by adding the assessment of pain, nausea/vomiting, and surgical bleeding to the evaluation of vital signs and activity.[21] There are multiple factors affecting duration of postanesthesia unit length of stay. Some of them are directly related to the intraoperative anesthetic care, but some may be related to the nature of the surgical procedure. Postoperative nausea and vomiting, inadequate pain control or prolonged residual neuroaxial block, as well as type of anesthetic used (GA vs. sedation) all can delay recovery and time to discharge and need to be taken into consideration when planning anesthetic care. In addition, the type and duration of the surgical procedure, the

patient's ASA physical status, and intraoperative blood loss can affect the duration of recovery and time to discharge as well.[21]

Optimal postoperative pain control is important not only for patient comfort but also because it has been demonstrated that adequate pain control decreases the serum levels of stress hormones and reduces postoperative morbidity.[47] Use of patient-controlled analgesia enables titration of the analgesic (intravenous or epidural) to the patient's comfort and is more effective than intermittent intramuscular or intravenous analgesic injections.[75] Continuous epidural analgesia and peripheral nerve catheters provide excellent postoperative analgesia. They reduce the concentration of circulating stress hormones and provide pharmacologic sympathectomy, which prevents peripheral vasoconstriction and provides better blood flow to the tissues within anesthetized dermatomes. This may be beneficial after reconstructive surgery, in particular when free flaps are used.[65] A multimodal approach to treatment of postoperative pain has gained a great deal of popularity, since it allows an anesthesiologist to take advantage of a synergistic analgesic effect of agents acting by different mechanisms (e.g., opioids and NSAIDs) thus reducing the dose of each drug. Adequate pain control during the perioperative period may improve postoperative functional recovery and prevent development of chronic pain syndromes as well as improve patient satisfaction.[35] PONV has a significant impact on patient satisfaction, overall cost of providing care, and surgical outcomes. There are recognized factors affecting the risk of PONV: female gender, nonsmoking status, history of motion sickness and/or prior PONV, and use of opioids.[3,66] An aggressive multimodal approach to PONV prevention (antiemetics-5-HT_3 receptor antagonists, dexamethasone) as well as possible modification of the anesthetic technique (avoidance of opioids, use of regional technique, local anesthetics) can substantially decrease the incidence of this postoperative complication.[3,66] Prevention is by far more effective than treatment of well-established PONV. Studies have shown that a patient's fear of PONV

Table 3.5. Two examples of discharge criteria systems.

Postanesthesia recovery score (modified Aldrete score)	Postanesthesia discharge scoring system
Activity	**Vital signs (BP and pulse)**
2 = Moves all extremities voluntarily/on command	2 = Within 20% of preoperative baseline
1 = Moves two extremities	1 = 20–40% of preoperative baseline
0 = Unable to move extremities	0 = >40% of preoperative baseline
Respiration	**Activity**
2 = Breathes deeply and coughs freely	2 = Steady gait, no dizziness
1 = Dyspneic, shallow or limited breathing	1 = Requires assistance
0 = Apneic	0 = Unable to ambulate
Circulation	**Nausea and vomiting**
2 = BP + 20 mm of preanesthetic level	2 = Minimal: treat with PO medications
1 = BP + 20–50 mm of preanesthetic level	1 = Moderate: treat with IM medications
0 = BP + 50 mm of preanesthetic level	0 = Continuous: repeated treatment
Consciousness	**Pain**
2 = Fully awake	Acceptable to patient; control with PO medications
1 = Arousable on calling	2 = Yes
0 = Not responding	1 = No
Oxygen saturation	**Surgical bleeding**
2 = Spo_2 > 92% on room air	2 = Minimal: no dressing change required
1 = Supplemental O_2 req. to maintain Spo_2 > 90%	1 = Moderate: up to two dressing changes
0 = Spo_2 < 92% with O_2 supplementation	0 = Severe: more than three dressing changes
10 = Total score	10 = Maximum score
Score > 9 required for discharge	Score > 9 required for discharge

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is greater than fear of postoperative pain and other side effects of anesthetic care.[46,48]

Regional vs. General Anesthesia and Outcomes

Regional anesthesia is known to prevent or at least attenuate a systemic stress response to surgery by blocking afferent noxious neural transmission to the central nervous system.[43] However, it has been difficult to translate this benefit into improvement of major patient outcomes.[62] Multiple studies have shown lower serum concentration of the stress hormones during and after surgical procedures when the neuroaxial blocks were used as compared with general anesthesia.[10,20] However, there is still an unsettled debate as to whether regional or general anesthesia is more beneficial for patients' hard outcomes (mortality and major morbidity). Initial enthusiasm favoring regional techniques[80] has been tempered over time by the results of better designed trials and meta-analyses showing minimal difference in outcomes between the two techniques.[55,62] Many investigators have hypothesized that an exaggerated stress response to the surgical procedure may increase morbidity and mortality and as such needs to be blocked before the surgical stimulation.[20,35,71] Although further investigation of this matter failed to show convincing evidence of earlier suspected advantages of regional anesthesia, there is a strong belief by some that under selected clinical circumstances regional anesthesia may be advantageous.[63] This assumption is supported by studies linking regional anesthesia with decreased incidence of thrombotic complications,[71] improvement in microcirculation blood flow,[65] and diminished inflammatory and hypercoagulable response.[65,77] For instance, continuous epidural anesthesia and postoperative analgesia may be beneficial in reconstructive surgery to improve microcirculation in free flaps by induced sympathectomy and potentially reduce the complication rate related to thrombogenic activity and vasospasm. It also provides superb postoperative pain control with minimal systemic side effects, which is important after extensive reconstructive surgeries, since inadequate pain control activates the systemic stress response (increased sympathetic tone and hypercoagulable state). The benefit of the regional technique may be difficult to prove if only major, but relatively rare, postoperative complications are considered (death, myocardial infarction, pulmonary embolism, etc.) as outcome measures. To achieve adequate power, a large number of patients need to be enrolled in the study, making it extremely difficult to conduct. However, there are data suggesting that regional anesthesia and local anesthetics have a profound beneficial effect on the inflammatory response during surgery and immunomodulation in the perioperative period where impact on the outcome has to be determined.[23]

Anesthetic Consideration in Cosmetic Surgery

Anesthesia for cosmetic surgery presents a unique challenge since the procedures themselves have no medical indications, and the patients expect smooth, side-effect-free recovery. These patients not only expect to look and feel better after the procedure, but they also would accept no additional discomfort during the process; therefore, cosmetic surgery patients tend to be more demanding and highly critical of all aspects of perioperative care. It is a purely consumer-driven medical care. Although many small cosmetic procedures can be performed under local anesthesia and without or only with minimal sedation, more extensive procedures require MAC, general anesthesia, or nerve block. The goal of anesthetic management for these mostly outpatient, office-based procedures is to provide excellent surgical conditions as well as to facilitate fast and complication-free recovery. Some of the side effects of anesthesia, such as PONV, may not only be very dissatisfactory for the patient, but also jeopardize the results of the surgical procedure (facelift, abdominoplasty). There is no single ("fits all") anesthetic technique for a cosmetic procedure and as with any other procedure, anesthetic care has to be tailored to the clinical situation. Use of a local anesthetic at the surgical site helps to minimize or even eliminate the need for intravenous opioids and significantly decreases the incidence of PONV.[33] It has to be stressed that the maximal safe dose of a local anesthetic should not be exceeded due to the risk of serious cardiovascular complications. In particular, tumescent lidocaine solutions used during liposuction, lipoplasty, or suction-assisted lipectomy accounted for a sig-

nificant part of morbidity and mortality related to cosmetic surgery; therefore, utmost attention should be paid when using a large volume (dose) of a local anesthetic. Propofol infusion supplemented with ketamine and generous local anesthetic instillation at the operative site by the surgeon has been reported to be a very successful anesthetic technique for the most common cosmetic surgeries (liposuction, breast augmentation, abdominoplasty, facelift) with minimal to almost no side effects, great patient satisfaction, and fast recovery.[33]

Perioperative Anesthetic Care and Outcomes

Choice of Anesthetic

There is a growing body of evidence bringing forth new information that perioperative events and anesthetic clinical care choices may affect patient morbidity and mortality for months or even years following surgery.[28,42] Better understanding of the role of inflammation and immunomodulation during the perioperative period has led to a realization that the impact of anesthetic management may have long-term consequences.[12,28,54] Anesthesia and surgery acutely alter the function of the immune system through a multifactorial process.[29] In the perioperative period, signals affecting the immune system include fear, tissue injury, hypothermia, pharmacologic agents, blood transfusions, pain, infection, and hyperglycemia, just to mention a few. Volatile anesthetic agents have been shown in vitro to have a dose-dependent inhibitory effect on neutrophil function; they suppress cytokine release in peripheral blood mononuclear cells, decrease lymphocyte proliferation, and induce lymphocyte apoptosis.[22,26,52,69] Although human studies are more difficult to interpret because changes in the immunologic system seem to be multifactorial and frequently difficult to separate, Schneemilch and colleagues[69] compared the immune effects of TIVA (propofol, sufentanil) with balanced inhalation anesthesia (sevoflurane, nitrous oxide, fentanyl) during minor surgery. They found that absolute numbers of T-lymphocytes, expression of histocompatibility locus antigen, and activation markers decreased more in response to balanced inhalation anesthesia.[69] The causality of exposure to volatile anesthetics and increased rate of infection or better graft survival is not definitively supported by clinical studies; however, one has to consider the possibility that exposure to inhaled anesthetics could be associated with long-term sequelae.[40] At the same time, volatile anesthetics (sevoflurane, desflurane) have been implicated to have anti-inflammatory properties and protective effects against ischemia reperfusion injury of the myocardium during coronary artery bypass surgery with quite measurable improvement in the outcomes.[22]

The pleiotropic effect (sodium channel-independent) of local anesthetics exerted by interacting with other molecular sites (e.g., M1 muscarinic receptors, G-protein-coupled receptors) at concentrations far below those required to achieve neuronal blockade may be an important factor affecting selected outcomes.[43,55,80] Local anesthetics reduce inflammation by interfering with the inflammatory cascade at multiple levels and have demonstrated beneficial effects in the clinical treatment of acute and chronic inflammatory diseases.[13] The results of studies have demonstrated clearly that local anesthetics attenuate important proinflammatory effector functions, such as the expression of pro-adhesive leukocyte integrins (e.g., CD11b–CD18), formation of reactive oxygen metabolites, and the release of leukotrienes interleukin-1a and histamine.[26,52,70] This evidence helps to explain the ability of local anesthetics to ameliorate leukocyte adherence, transmigration, edema formation, and tissue damage in different animal models of acute injury (e.g., acute respiratory distress syndrome, thermal injury, myocardial infarction) and chronic inflammatory diseases such as inflammatory bowel disease.[13] What is interesting is that local anesthetic-induced reductions in leukocyte activation do not seem to be offset by clinically relevant reductions in microbicidal capacity.[58,61] Clinical benefits of systemic local anesthetics have been demonstrated by Herroeder et al.[38] in a prospective, randomized trial, which showed that patients who received intravenous lidocaine perioperatively had faster recovery of bowel function and shorter hospital stay after colorectal surgery when compared with patients receiving a placebo.

Although all modern anesthetic agents are safe and direct serious toxicity is almost nonexistent in clinically relevant doses, they may have an impact on long-term outcomes. The discussion of the pharmacokinetic and pharmacodynamic properties of all anesthetic agents is beyond the

scope of this chapter; however it is important to mention that there is a growing body of evidence suggesting that intravenous opioids inhibit both cellular and humoral immune function.[67,79] This effect may be important in oncologic surgery as well as have an impact on the incidence of postoperative infectious complications. Exadaktylos et al.[27] showed in a retrospective review that the use of paravertebral block for breast surgery can affect (lower) the rate of breast cancer recurrence. Although it would be oversimplification to state that general anesthesia increases the recurrence of cancer after oncologic surgery, one has to recognize the effect of different types of anesthetics on the function of natural killer cells, which are thought to play a key role in preventing tumor dissemination and growth.[12] This finding is in concordance with previous animal (rat) studies that demonstrated that surgical stress is attenuated better by regional rather than general anesthesia and that, consequently, natural killer cell function is better preserved and metastatic load to the lungs is reduced while regional anesthesia is used.[9]

Temperature Control

It is well documented that postoperative hypothermia negatively affects outcome after a surgical procedure.[51] Major outcome studies have demonstrated that the risk of surgical wound infection is reduced threefold simply by keeping patients normothermic.[28] All surgical patients are at a risk for wound infection, and after surgery, this risk increases if tissue perfusion is poor. Melling et al. illustrated that a 14% postoperative infection rate was reduced to 5% by applying a 30-min period of preoperative warming.[51] Frank et al.[31] showed in a randomized, controlled trial comparing routine thermal care (hypothermic group) to additional supplemental warming care (normothermic group) that hypothermia was associated with a higher incidence of morbid cardiac events and ventricular tachycardia. Kurz et al.[44] found that hypothermic patients on average stayed 2.6 days longer in the hospital than a normothermic group. Mild hypothermia reduces platelet function and decreases the activation of the coagulation cascade. In vitro studies are consistent with the clinical experience; hypothermia significantly increased blood loss and the need for allogeneic transfusion, which was demonstrated during elective primary hip arthroplasty.[68] It is well recognized that even mild hypothermia (0.5°C to 1.2°C below normal core temperature) increases levels of circulating norepinephrine by 100% to 700%, causing generalized systemic vasoconstriction.[32] It also directly impairs immune function (especially oxidative killing by neutrophils), decreases the cutaneous blood flow, which reduces the delivery of oxygen to tissue, causes protein wasting, and decreases the synthesis of collagen, which may impair wound healing.[19,28]

Preemptive Analgesia

The concept of preemptive analgesia (providing analgesic intervention before surgical incision) has been recently scrutinized in a systematic review of published randomized trials evaluating the effect of administered analgesia before incision on the level of postoperative pain.[53] The overall conclusion from the review was that preemptive analgesia had no effect on postoperative pain in the first 24 h.[53] It may be argued that perhaps the beneficial effect was delayed beyond the first postoperative day, thus it was not shown in the review. On the other hand, it was demonstrated in the small study that patients who received a local anesthetic or fentanyl via epidural catheter before incision had not only better pain control during immediate postoperative period but also had an increased activity level as well as less pain weeks after hospital discharge[35]; hence, it might be that the benefits of preemptive analgesia are more apparent in long-term follow-up of the surgical procedure and improved patient's long-term quality of life.

Depth of Anesthesia

The potential effects of anesthesia on long-term survival were suggested by Monk and colleagues.[54] They demonstrated that maintenance of deeper levels of anesthesia, as assessed by a bispectral index (BIS) monitor, were associated with higher 1-year postoperative death rates for patients 40 years and older undergoing major, noncardiac surgery. Farag et al.,[28] on the other hand, suggested in a prospective trail that deeper levels of anesthesia (assessed by a bispectral index) were associated with better cognitive function 4–6 weeks postoperatively, particularly with respect to the ability to process information. Further work is required to determine whether these results reflect a true pathophysiologic link

between management of the anesthesia depth and long-term outcome or a simple statistical association. Before one can make any recommendation regarding the depth of anesthesia in surgical patients, further prospective studies are needed using more sensitive tools to assess the long-term effects of general anesthetics on cognitive function.

Inspired Oxygen Concentration

Perioperative factors, such as providing supplemental oxygen, may modulate postoperative infection risk even though infections are not detected clinically until days later.[11,36] Infection risk is reduced by an additional factor of two if supplemental oxygen is provided (80% vs. 30%) during surgery and for the initial hours after surgery.[36]

Perioperative Glycemia Control

Although the contribution, if any, of perioperative tight glucose control on postoperative outcomes in a noncardiac surgery setting have yet to be suitably tested, it was demonstrated in an intensive care unit as well as cardiac surgery patients that tight glucose control improves selected outcomes.[18,34,57,73] Hyperglycemia causes endothelial dysfunction, increased expression of adhesion molecules, O_2 radical production, defect in NO production, tissue acidosis, poor wound healing, and so forth. Hence, it is plausible to assume that tight glucose control during the perioperative period in major reconstructive surgery may be beneficial.

Perioperative Medications

Statins showed promising results in reducing the incidence of cardiovascular events during the perioperative period, and their benefit is beyond the lipid-lowering effect.[24] Their beneficial anti-inflammatory properties and effect on postoperative outcomes still need to be further explored; however, initial results are promising.[24] Perioperative beta-blockers have been shown in earlier studies to reduce the risk of short- and long-term cardiovascular events. Mangano and colleagues[49] performed a randomized clinical trial in which 7 days of perioperative β-blockade was compared with placebo in high-risk patients undergoing noncardiac surgery. They reported significantly improved survival at 6 months, which remained significant during the 2 years of follow-up. However, recent trials (DIPOM, POBBLE) have questioned the benefit of perioperative beta blockers.[15,42] Therefore, ACC and AHA recently revised indications for their use during the perioperative period.[30] Although there is a well-documented physiologic rationale for use of perioperative beta blockers, there are still questions to be answered by ongoing trials regarding the perioperative benefit of these drugs.

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4

Medical Liability in Plastic and Reconstructive Surgery

Mark Gorney

Summary

With the increasing popularity and interest in aesthetic surgery among the general population, the need for an adequate understanding of the complicated interplay of circumstances that leads to dissatisfied patients and potential legal action has great significance. This chapter aims to identify the salient legal principles related to patient care as well as potential pitfalls. In addition, this chapter examines how the summative contributions of patient selection, the type of procedure, and the associated psychological aspects of anatomy modification could increase the likelihood of a physician being faced with a dissatisfied patient and/or legal action. By understanding these basic legal principles as well as the aforementioned predisposing factors, one may be able to minimize the frequency and severity of legal claims.

Abbreviation

BDD Body dysmorphic disorder

Legal Principles Applied to Plastic Surgery

Standard of Care

Malpractice generally means treatment that is contrary to accepted medical standards and that produces injurious results in the patient. Most medical malpractice actions are based on laws governing negligence. Thus, the cause of action is usually the "failure" of the physician to exercise that reasonable degree of skill, learning, and care ordinarily possessed by others of the same profession in the community. Although in the past, the term "community" was accepted geographically, it is now based on the supposition that all doctors keep up with the latest developments in their field. The community today is generally interpreted as a "specialty community." The standards are now for those of the specialty as a whole irrespective of geographic location. This series of norms is commonly referred to as "standard of care."

Warranty

The law holds that by merely engaging to render treatment, a doctor warrants that he or she has the learning and skill of the average member of that specialty and that he or she will apply that learning and skill with ordinary and reasonable care. The warranty is for due care. It is legally implied. It need not be mentioned by the physician or the patient.

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However, the warranty is one of service, not cure. Thus, the doctor does not imply that the operation will be a success, that results will be favorable, or that he or she will not commit any medical errors not caused by lack of skill or care.

Disclosure

While attempting to define the yardstick of disclosure, the courts divide medical and surgical procedures into two categories:

1. Common procedures that incur minor or remote serious risk, for example, the administration of acetaminophen.
2. Procedures involving serious risks that the doctor has an affirmative duty to disclose. He or she is bound to explain in detail the complications that might possibly occur.

Affirmative duty means that the physician is obliged to disclose risks on his or her own, without waiting for the patient to ask. The courts have long held that it is the patient, not the physician, who has the prerogative of determining what is in his or her best interests. Thus, the surgeon is legally obligated to discuss with the patient therapeutic alternatives and their particular hazards in order to provide sufficient information to determine the individual's own best interest. How much explanation and in what detail are dictated by a balance between the surgeon's judgments about his or her patient and the legal requirements applicable. It is simply not possible to tell patients everything without unnecessarily dissuading them from appropriate treatment. Rather, the law holds that patients must be told the most probable of known dangers and the percentage likelihood. More remote risks may be disclosed in general terms, while placing them in a context of suffering from any unusual event.

Obviously, the most common complications should be volunteered frankly and openly, and their probability, based on the surgeon's personal experience, should also be discussed. Finally, any or all of this information is wasted unless it is documented in the patient's record. For legal purposes, if it is not in the record, it never happened!

Informing Your Patients Before They Consent

In the last five years, most medical liability carriers have experienced a significant increase in claims alleging failure to obtain a proper informed consent prior to treatment. This trend is particularly noticeable in claims against surgical specialties performing elective procedures.

Informed consent means that adult patients who are capable of rational communication must be provided with sufficient information about risks, benefits, and alternatives to make an informed decision regarding a proposed course of treatment. (The same is true for "emancipated" or "self-sufficient" minor patients.) In most states, physicians have an "affirmative duty' to disclose such information. This means that you must not wait for questions from your patients; you must volunteer the information.

Without informed consent, you risk legal liability for a complication or untoward result – even if it was not caused negligently.

The essence of this widely accepted legal doctrine is that patient must be given all information about risks that are relevant to a meaningful decision-making process. It is the prerogative of the patient, not the physician, to determine the direction in which it is believed his or her best interests lie. Thus, reasonable familiarity with therapeutic (and/or diagnostic) alternatives and their hazards is essential.

Do patients have the legal right to make bad judgments because they fear a possible complication? Increasingly, the courts answer affirmatively. Once the information has been fully disclosed, that aspect of the physician's obligation has been fulfilled. The final decision on therapy is usually the patient's.

"Prudent Patient" Test

In many states, the most important element in claims involving disputes over informed consent is the prudent patient test. The judge will inform the jury that there is no liability on the doctor's part if a prudent person in the patient's position would have accepted the treatment had he/she been adequately informed of all significant perils. Although this concept is subject to reevaluation in hindsight, the prudent patient test becomes most meaningful where treatment is lifesaving or urgent.

The concept also may apply to simple procedures where the danger is commonly appreciated to be remote. In such cases, disclosure need not be extensive, and the prudent patient test will usually prevail.

Refusals

As part of medical counseling, many state laws mandate that physicians warn patients of the consequences involved in failing to heed medical advice by refusing treatment or diagnostic tests. Obviously, patients have a right to refuse. In such circumstances, it is essential that you carefully document such refusals and their consequences and that you verify and note that the patient understood the consequences.

Documentation is particularly important in cases involving malignancy, where rejection of tests may impair diagnosis and refusal of treatment may lead to a fatal outcome. Remember to date all such entries in the patient record.

If the information you present includes percentages or other specific figures that allow the patient to compare risks, be certain that your figures conform to the latest reliable data.

Consent-in-Fact and Implied Consent

What is the distinction between ordinary consent to treatment (consent-in-fact) and informed consent? Simply stated, the latter verifies that the patient is aware of anticipated benefits as well as risks and alternatives to a given procedure, treatment, or test. On the other hand, proceeding with treatment of any kind without actual consent is "unlawful touching" and may therefore be considered "battery."

When the patient is unable to communicate rationally, as in many emergency cases, there may be a legally implied consent to treat. The implied consent in an emergency is assumed only for the duration of that emergency.

Minors

Except in urgent situations, treating minors without consent from a parent, legal guardian, appropriate government agency, or court carries a high risk of civil or even criminal charges. There are statutory exceptions, such as for an emancipated adolescent or a married minor. If you regularly treat young people, you should familiarize yourself with the existing statutory provisions in your state and keep up to date.

Religious and Other Obstacles

Occasionally, you may be placed in the difficult position of being refused permission to treat or conduct diagnostic tests on the basis of a patient's religious or other beliefs. Although grave consequences may ensue, there is little that you can do in most states beyond making an intense effort to convince the patient; in some states, court intervention may be obtained. Here too, knowing the law of the state in which you practice is advisable. In all cases, the informed refusal must be carefully documented.

If a patient is either a minor or incompetent (and the parent or guardian refuses treatment), and you know serious consequences will ensue if appropriate tests and/or treatments are not undertaken, your legal and moral obligations change. You must then resort to a court order or another appropriate governmental process in an attempt to secure surrogate consent. The participation of personal or hospital legal counsel is advisable to ensure that the legal requirements applicable in your locale are met.

The Six Elements of Informed Consent

Where treatment is urgent (e.g., in a case of severe trauma), it may be needless and cruel to engage in extensive disclosure that could augment existing anxieties. However, you should inform the patient of the treatment's risks and consequences and record such discussions.

In general, it is important to discuss the following six elements of a valid informed consent with your patients and/or their families:

1. The diagnosis or suspected diagnosis
2. The nature and purpose of the proposed treatment or procedure and its anticipated benefits
3. The risks, complications, or side effects
4. The probability of success, based on the patient's condition
5. Reasonable available alternatives
6. Possible consequences if advice is not followed

In situations where the nature of the tests or treatment is purely elective, as with cosmetic surgery, the disclosure of risks and consequences may need to be expanded. Office literature can provide additional details about the procedure. In addition, an expanded discussion should take place regarding the foreseeable risks, possible untoward consequences, or unpleasant side effects associated with the procedure. This expansion is particularly necessary if the procedure is new,

experimental, especially hazardous, purely for cosmetic purposes, or capable of altering sexual capacity or fertility.

Documentation

Written verification of consent to diagnostic or therapeutic procedures is crucial. Also, remember, however, that in an increasing number of circumstances laws now require the completion of specifically designed consent forms.

Studies indicate that physicians sometimes underestimate the patient's ability to understand. If your records disclose no discussion or consent, the burden will be on you to demonstrate legally sufficient reasons for such absence.

It is a test of your good judgment of what to say to your patient and of how to say it to obtain meaningful consent without frightening the patient.

No permit or form will absolve you from responsibility if there is negligence, nor can a form guarantee that you will not be sued. Permits may vary from simple to incomprehensibly detailed. Most medical–legal authorities agree that a middle ground exists.

A well-drafted informed-consent document is proof that you tried to give the patient sufficient information on which to base an intelligent decision. Such a document, supported by a handwritten note and entered in the patient's medical record, is often the key to a successful malpractice defense when the issue of consent to treatment arises.

The Therapeutic Alliance

Obtaining informed consent need not be an impersonal legal requirement. When properly conducted, the process of obtaining informed consent can help establish a "therapeutic alliance" and launch or reinforce a positive doctor–patient relationship. If an unfavorable outcome occurs, that relationship can be crucial to maintaining patient trust.

A common patient's defense mechanism against uncertainty is to endow his or her doctor with omniscience in the science of medicine, an aura of omnipotence. By weighing how you say something as heavily as what you say, you can turn an anxiety-ridden ritual into an effective therapeutic alliance. Psychiatric literature refers to this as the "sharing of uncertainty." Rather than shattering a patient's inherent trust in you by presenting an insensitive approach, your dialogue should be sympathetic to the patient's particular concerns or tensions and should project believable reactions to an anxious and difficult situation.

Consider, for example, the different effects that the following two statements would have:

1. "Here is a list of complications that could occur during your treatment (operation). Please read the list and sign it."
2. "I wish I could guarantee you that there will be no problems during your treatment (operation), but that wouldn't be realistic. Sometimes there are problems that cannot be foreseen, and I want you to know about them. Please read about the possible problems, and let's talk about them."

By using the second statement, you can reduce the patient's omnipotent image of you to that of a more realistic and imperfect human being, who is facing, and thus sharing, the same uncertainty. The implication is clear: we – you and I – are going to cooperate in doing something to your body that we hope will make you better, but you must assume some of the responsibility.

To allay anxiety, you may seek to reassure your patients. However, in so doing, be wary of creating unwarranted expectations or implying a guarantee. Consider the different implications of these two statements:

1. "Don't worry about a thing. I've taken care of hundreds of cases like yours. You'll do just fine."
2. "Barring any unforeseen problems, I see no reason why you shouldn't do very well. I'll certainly do everything I can to help you."

If you make the first statement and the patient does not do "fine," he or she is likely to be angry with you. The second statement gently deflates the patient's fantasies to realistic proportions. This statement simultaneously reassures the patient and helps him or her to accept reality.

The therapeutic objective of informed consent should be to replace some of the patient's anxiety with a sense of his or her participation with you in the procedure. Such a sense of participation strengthens the therapeutic alliance between you and your patients. Instead of seeing each other as potential adversaries if an unfavorable or less than perfect outcome results, you and your patients are

drawn closer by sharing acceptance and understanding the uncertainty of clinical practice.

Patient Selection Criteria

Contemporary plastic and reconstructive surgeons practicing in the United States will find it virtually impossible to end their careers unblemished by a claim of malpractice.

However, well over half of this is preventable. Most are based either on failures of communication and patient selection criteria, not on technical fault. Patient selection is an inexact science. It requires a mixture of surgical judgment and gut reaction. Regardless of technical ability, a surgeon who appears cold, arrogant, or insensitive is more likely to be sued than one who relates at a "personal" level. A surgeon who is warm, sensitive, naturally caring, with a well-developed sense of humor and cordial attitude, is less likely to be the target of a malpractice claim.

Communication is the sine qua non of building a doctor–patient relationship. Unfortunately, the ability to communicate well is a skill that cannot be easily learned in adulthood. It is an integral part of the surgeon's personality. There are, however, a number of helpful guidelines.

- Great expectations. There are certain patients who have an unrealistic and idealized, but vague, conception of what elective aesthetic surgery is going to do for them. They anticipate a major change in life style, with immediate recognition of their newly acquired attractiveness. These patients have an unrealistic concept of where their surgical journey is taking them and have great difficulty in accepting the fact that any major surgical procedure carries inherent risk.
- Excessively demanding patients. In general, the patient who brings with him or her photographs, drawings, and exact architectural specifications, should be managed with great caution. Such a patient has little comprehension that the surgeon is dealing with human flesh and blood, not wood or clay. This patient must be made to understand the realities of surgery, the vagaries of the healing process, and the margin of error that is a natural part of any elective procedure. Such patients show very little flexibility in accepting any failure on the part of the surgeon to deliver what was anticipated.
- The indecisive patient. To the question "Doctor, do you think I ought to have this done?," the prudent surgeon should respond, "This is a decision which I cannot make for you. It is one you have to make yourself. I can tell you what I think we can achieve, but if you have any doubt whatsoever, I recommend strongly that you think about it carefully before deciding whether or not to accept the risks which I have discussed with you." The more the decision to undergo surgery is motivated from within and not "sold," the less likely recrimination will follow an unfavorable result.
- The immature patient. The experienced surgeon should assess not only the physical but also the emotional maturity of the patient. Youthful or immature patients (age has no relationship to maturity) may have excessively romantic expectations and an unrealistic concept of what the surgery will achieve. When confronted with the mirror postoperatively, they may react in disconcerting or even violent fashion if the degree of change achieved does not coincide with their preconceived notions.
- The secretive patient. Certain patients wish to convert their surgery into a "secret" and request elaborate precautions to prevent anyone from knowing they are undergoing cosmetic surgery. Aside from the fact that such arrangements are difficult to achieve, this tendency is a strong indication that the patient has a degree of guilt about the procedure. Thus, there is a higher likelihood of subsequent dissatisfaction.
- Familial disapproval. It is far more comfortable, although not essential, if the immediate family approves of the surgery being sought. If there is disapproval, less than optimal results may produce a "See, I told you so!" reaction, which deepens the guilt and dissatisfaction of the patient.
- Patients you do not like (or who do not like you). Regardless of the surgeon's personality, in life there are people whom you simply "do not like" or who do not

like you. Accepting a patient whom you basically dislike is a serious mistake. A clash of personalities for whatever reason is bound to affect the outcome of the case, regardless of the actual quality of the postoperative result. No matter how "interesting" such a case may appear, it is far better to decline the patient.

- The "surgiholic." A patient who has had a variety of plastic surgery procedures performed, and who is a "surgiholic," often attempts to compensate for a poor self-image with repeated surgeries. In addition to the implications of such a personality pattern, the surgeon is also confronted with a more difficult anatomical situation due to the previous surgeries. He or she also risks unfavorable comparison with previous surgeons. Often the percentage of achievable improvement is not worth the risk of the procedure.

Generally, there is a clear risk/benefit ratio to every surgical procedure. If the risk/benefit ratio is favorable, the surgery should probably be encouraged and has a reasonable probability of success. If the risk/benefit ratio is unfavorable, the reverse not only applies but also the unintended consequences of the unfavorable outcome may turn out to be disproportionate to the surgical result. The only way to avoid this debacle is to learn how to distinguish those patients whose body image and personality characteristics make them unsuitable for the surgery that they seek.

The Wheel of Misfortune: Exposures Most Likely to Generate Claims

It should come as no surprise that the overwhelming majority of all malpractice claims lodged against plastic and reconstructive surgeons are concentrated in a handful of aesthetic surgery operations. Unlike other surgical specialists, the plastic surgeon attending a patient who seeks aesthetic improvement is not trying to make a sick patient well, but rather a well patient better. This not only places a heavier burden of responsibility on the operating surgeon but also subjects him or her to a broader range of possible reasons for unhappiness. Sources of dissatisfaction can range from a poor result to something as unpredictable as a patient's hidden emotional agenda or a simple communications failure.

Competitive pressures in the last few years have also blurred strict criteria for patient selection. As a result it is not surprising to see a steadily upward trending in the frequency of claims against plastic and reconstructive surgeons. We have surveyed the genesis of patient complaints in a universe of plastic and reconstructive surgeons numbering roughly 700 across 15 years of experience. The loss experience in plastic surgery is notable for its frequency, rather than its severity (the large number of claims alleging relatively minor damages). The average plastic surgeon reports a claim every 254 years. Although severity has not characterized plastic surgery's loss experience in the past, the trend is toward larger awards, particularly in those cases where an elective procedure has resulted in a fatal outcome. An important example is the claims arising out of "large-volume," suction-assisted lipectomy. This category of claims is more carefully examined toward the conclusion of this chapter.

- Scarring in General

Most surgeons assume the patient understands that healing entails the formation of scars. Unfortunately, it is seldom discussed in the preoperative consultation. In plastic and reconstructive surgery, the appearance of the resulting scar can be the major genesis of dissatisfaction. It is imperative that the plastic surgeon obtains from the patients clear evidence of their comprehension that without a scar, there is no healing. The patients must be made to understand that their healing qualities are as individual to them as the texture of their hair or the color of their eyes; it is built into their genetic program. Documentation of such conversations in the preoperative chart is most important.

- Breast Reduction

The genesis of dissatisfaction most often involves the following:

- Unsatisfactory scar
- Loss of nipple or breast skin cover requiring revision
- Asymmetry or "disfigurement"
- Breast Augmentation

Litigation involving breast augmentation is even more common than breast reduction.

Approximately 44% of all elective aesthetic surgery claims involve augmentation. Setting aside for the moment breast implants and autoimmune disease, the most frequent causes of dissatisfaction are the following:

- Encapsulation with distortion and firmness
- Wrong size (too little/too much)
- Infection
- Repetitive surgeries and attendant costs
- Nerve damage with sensory loss
- Face-Lift/Blepharoplasty

Face-lift and blepharoplasty account for approximately 11% of claims. The commonest allegations are the following:

- Excessive skin removal resulting in a "starry" look
- Dry eyes/inability to close
- Nerve damage resulting in distorted expression
- Skin slough resulting in excessive scarring and additional surgery

The trend toward treating the vast majority of these patients on an outpatient basis deserves some comment. In a survey of blindness after blepharoplasty carried out by the author at The Doctors Company in 1999, it was discovered that the only trait all cases had in common was the fact that they were discharged very shortly after the termination of the outpatient surgery. Upon arrival at home, each did something to generate a sudden rise in blood pressure at the time of maximal reactive hyperemia, as the epinephrine in the local anesthetic wore off (constipated bowel movement, sudden coughing fit, bending over, and reaching down to tie shoes, etc.) It is imperative that all patients undergoing outpatient surgery involving undermining of heavily vascularized tissues be strictly warned not to undertake any maneuvers that will generate sudden elevations in blood pressure. Additionally, it is strongly recommended that no patient be discharged from an out-patient surgical facility until at least after 3 h have elapsed and there is evidence that all the local anesthetic effects have worn off.

- Rhino-Septoplasty

This category of cases constitutes approximately 8% of the claims. Among the commonest allegations are the following:

- Unsatisfactory result: improper performance allegations
- Continued breathing difficulties
- Asymmetry

The one most commonly seen (by far) is the first. Of all the operations performed by plastic and reconstructive surgeons, regrettably this is the one with the highest degree of unpredictability. The problem is greatly aggravated by inappropriate patient selection criteria. In these claims, there is almost universally a gap between the patient's expectations and results obtained, even when the surgical outcome appears excellent. The inappropriate use of imaging devices or the showing of "brag books" containing only excellent results often causes patients to have unrealistic expectations. The clear implication is "this is the kind of work that I do, and this is what you can expect." Unfortunately, in many cases the actual result falls short of the promise, and the usual cycle is put into motion: surprise – disappointment – anger – perceived arrogance – increased avoidance – rising hostility – visit to the lawyer.

- Abdominoplasty

Abdominoplasty with or without suction-assisted lipectomy represents approximately 3% of claims. The most common allegations are the following:

- Skin loss with poor scars
- Nerve damage
- Inappropriate operation
- Infection with preoperative mismanagement

There is little question that the combination of suction-assisted lipoplasty prior to the actual abdominoplasty has significantly increased the morbidity of this operation and increased the number of claims in this category. There is a higher percentage of skin sloughs in those procedures when preceded by suction-assisted lipectomy.

- Suction-Assisted Lipectomy

Suction-assisted lipectomy procedures, whether conventional or ultrasonic, have now become the single most requested elective aesthetic procedure in the United States. Approximately 145,000 of these procedures were performed in the year 1997, according to ASPRS statistics.[1] However, the rising popularity of this procedure has brought with it a host of problems. To begin with, since

[1] ASPRS statistic, 1997.

this is not a surgical procedure in the "traditional" sense, it is being performed by a wide variety of practitioners, some of them with no surgical background or clear understanding of the surgical anatomy involved. Secondly, it is a procedure most commonly done on an outpatient basis outside of the control of any regulatory authorities.[2] Additionally, with the advent of "tumescent" techniques, an unseemly race has developed to see who can suction out the most fat. The net result has been a dramatic rise in severe morbidity and fatal outcomes from "high-volume" liposuction. What is high volume? It is generally agreed that anything above 5000 cc of extracted fat constitutes "high volume." The extraction of this amount of fat causes profound physiological changes, which in turn can lead to severe complications and/or fatal outcomes. The infusion of large amounts of fluid with even a weak concentration of lidocaine has also resulted in a number of fatal outcomes as a result of anesthetic overdose.

To make matters even worse, these procedures are often combined with other prolonged operations. Our experience clearly indicates that when a patient has been under anesthesia for more than 6 h, undergoing multiple procedures, the percentage of complications and/or fatal outcome rises dramatically.

Overall, there are two categories of liability from conventional assisted lipectomy procedures.

- Minor Allegations
  - Disfigurement and contour irregularities
  - Numbness
  - Disappointment/dissatisfaction
- Major Allegations
  - Unrecognized abdominal perforation resulting in disabling secondary
  - Surgery or death
  - Lidocaine overdose with fatal outcome
  - Pulmonary edema from overhydration
  - Pulmonary embolism and death

The cavalier way in which this operation is sometimes performed requires rethinking, particularly when the amounts of fat extracted are major. In a number of venues in the United States, state medical regulatory authorities are beginning to take notice, and unless there is a significant downturn in the morbidity of this procedure, there will undoubtedly be some regulatory intervention to control the rising tide of misfortune.

- Skin Resurfacing

Chemical peels and laser resurfacing constitute the next category of claims, constituting roughly 3%. The principal allegations here are the following:

- Blistering/burns with significant scarring
- Infection/preoperative mismanagement
- Permanent discoloration postoperatively

Because of the unpredictability of individual healing characteristics, it is probably a good idea to do a "test patch" in an area that can be hidden (e.g., the back of the neck). Certainly, the documentation preceding this operation should contain clear warnings that the quality of healing is linked to the individual's genetic makeup and cannot be predicted.

The operator must make it clear to the patient that final color and texture determination is not in the hands of the surgeon and heavy makeup may be needed for an indeterminate period of time.

- Miscellaneous

Approximately 5% of all complaints against plastic and reconstructive surgeons have to do with miscellaneous allegations such as the following:

- Untoward reaction to medications or anesthesia
- Improper use of pre- or postoperative photos
- Sexual misconduct (doctor or employee)

There are certain common issues among all procedures performed by plastic and reconstructive surgeons that are commonly not brought to the attention of the patient in the preoperative consultations and often represent the triggering mechanism for a claim. They are as follows:

- Unexpected scarring
- Lack of adequate disclosure (tailored to the patient's level of understanding)
- General dissatisfaction: the patient's expectations were not met

Psychological and Psychiatric Aspects of Modifying Anatomy

The growing popularity of elective aesthetic surgery makes it imperative to establish clear criteria of patient selection.

[2] TDC Guidelines for SAL, 2001/ASPS Standing Guidelines.

Who is the "ideal" candidate for aesthetic surgery? There is no such thing, but the surgeon should note any personality factors that will tend to enhance or detract from the physical improvements sought. The surgeon must differentiate between healthy and unhealthy reasons for seeking aesthetic improvement.

There are basically two categories that make the patient a poor candidate for elective aesthetic surgery. The first is anatomic unsuitability, but the second is equally important, though more subtle – psychological inadequacy.

Strength of motivation is critical. It has a startlingly close relationship with the patient's satisfaction postoperatively. Furthermore, a strongly motivated patient will tend to have less pain, a better postoperative course, and a significantly higher index of satisfaction. Although these characteristics are impossible to predict with absolute accuracy, it is possible to establish some objective criteria for patient selection. These are illustrated in Figure 4.1.

Figure 4.1 depicts a patient's objective deformity along the horizontal axis (as judged by the surgeon) versus the patient's degree of concern over that deformity (vertical axis) as perceived by the patient. Two opposite extremes emerge:

1. The patient with major deformity but minimal concern (lower right-hand corner). This is a patient with an obvious major deformity in whom it is clear that any degree of improvement will be regarded with satisfaction.

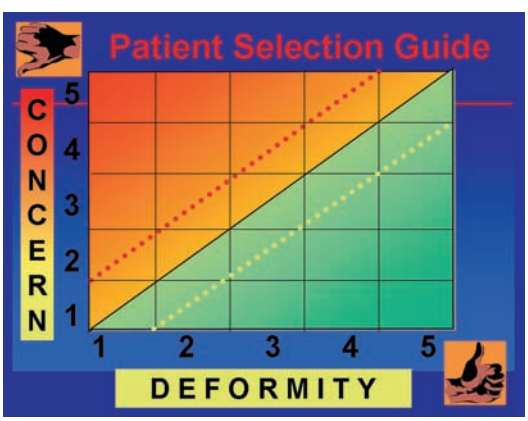

Figure 4.1. A patient's objective deformity, as judged by the surgeon, versus the patient's degree of concern over that deformity, as perceived by the patient.

2. The patient with the minor deformity but extreme concern (upper left-hand comer). This, in contrast, is a patient with a deformity which the surgeon perceives to be minor, but who demonstrates an inordinate degree of concern and emotional turmoil. Such patients are most likely to be dissatisfied with any outcome. The anxiety expressed over the "deformity" is merely a manifestation of inner turmoil, which is better served by a psychiatrist's couch than a surgeon's operating table.

Most who seek aesthetic surgery fit somewhere on a diagonal between the two contralateral corners. The closer the patient comes to the upper left-hand corner, the more likely is an unfavorably perceived outcome, as is a visit to an attorney.

- Effective Communication

Most litigation in plastic surgery has the common denominator of poor communication. This doctor–patient relationship can be shattered by the surgeon's arrogance, hostility, coldness (real or imagined), or simply by the fact that "he or she didn't care." There are only two ways to avoid such a debacle: (1) make sure that the patient has no reason to feel that way, and (2) avoid a patient who is going to feel that way no matter what is done.

Although the doctor's skill, reputation, and other intangible factors contribute to a patient's sense of confidence, rapport between patient and doctor is based on forthright and accurate communication. This will normally prevent the vicious cycle of disappointment, anger, and frustration by the patient and reactive hostility, defensiveness, and arrogance from the doctor, which deepens the patient's anger and ultimately may provoke a lawsuit.

- Anger: A Root Cause of Malpractice Claims

Patients feel both anxious and bewildered when elective surgery does not go smoothly. The borderline between anxiety and anger is tenuous, and the conversion factor is uncertainty – fear of the unknown. A patient frightened by a postoperative complication or uncertain about the future may surmise: "If it is the doctor's fault, then the responsibility for correction falls on the doctor."

The patient's perceptions may clash with the physician's anxieties, insecurities, and wounded

pride. The patient blames the physician, who in turn becomes defensive. At this delicate juncture, the physician's reaction can set in motion or prevent a chain reaction. The physician must put aside feelings of disappointment, anxiety, defensiveness, and hostility to understand that he or she is probably dealing with a frightened patient who is using anger to gain control.

The patient's perception that the physician understands that uncertainty, and will join with him/her to help to overcome it, may be the deciding factor in preserving the therapeutic relationship.

One of the worst errors in dealing with angry or dissatisfied patients is to try to avoid them. It is necessary to actively participate in the process rather than attempting to avoid the issue.

- Body Dysmorphic Disorder

As the popularity of aesthetic surgery increases, one is reminded of the fairy tale that asks the question: "Mirror, mirror on the wall, who's the fairest of them all?" The number of patients finding comfort and solace in repetitive elective surgical procedures is growing. Beyond the unrealistic expectations of aesthetic correction, many patients are seeking surgery when the need for it is dubious at best. The physical change sought through surgery is usually more a manifestation of flawed body image than a measurable deviation from physical normality. Body dysmorphic disorder (BDD) represents a pathological preoccupation by the patient about a physical trait that may be within normal limits or so insignificant as to be hardly noticeable. However, to the patient it has become a consuming obsession.

As the trend of advertising and "marketing" cosmetic surgery grows worldwide, there is greater probability that those living in the shadow of this diagnosis will eventually decide on the surgeon's scalpel, rather than the psychiatrist's consultation, as an answer to their problem.

Increasingly, we see traditional surgical judgment replaced either by financial consideration or plain ego on the part of the surgeon. Since patients with BDD never carry that diagnosis openly into the consultation with the plastic surgeon, medical disputes about the surgical outcome depend entirely on what was said versus what was understood.

In the best of all possible worlds, the prospective patient would project from the mind onto a screen exactly the changes he or she conceives for the surgeon to decide whether or not he or she can translate that image into reality. Lamentably, we are still many decades short of achieving such imaginary technology. It is easy for the well-meaning surgeon to be deceived about the patient's pathological motivation. It is also conceivable that the physical deformity really is at the center of the patient's psychological fragility. There are many examples of beneficial change wrought through successful aesthetic corrective surgery. Nonetheless, statistically the odds for an unfavorable result and a claim are much greater when the disproportion between the objective deformity and the distress it creates in the patient is out of proportion. The surgeon is cautioned to search for appropriate psychological balance and lean strongly against surgery in those in whom there is doubt.

At a time of convulsive change in the history of health care delivery in the United States, certain socioeconomic factors also come into play. With the rising number of practitioners in many specialties, competitive pressures have begun to affect patient selection criteria. There is a trend toward substitution of economic considerations for surgical judgment. Because of recent constrictions on medical incomes, some practitioners see elective aesthetic surgery as the last area of practice unencumbered by either insurance or governmental restrictions. This has attracted individuals with inadequate qualifications. Even within the ranks of board-certified plastic surgeons, the rising trend toward "marketing" and the need to "sell surgery (which should always be motivated by the patient: not the surgeon) have further blurred patient selection criteria.

As the popularity of aesthetic surgery grows, the trend to solve emotional or psychological problems with a scalpel grows with it – and so does the trailing liability. It is critically important that you remain wary. Always protect yourself with quality pre- and postoperative sequential photographs plus complete, clear medical records. Quite simply, these precautions make the difference between winning or losing your case. They are really all you have to verify your work.

Finally, all plastic surgeons soon learn that although it is impossible to eliminate every possibility of dissatisfaction or conflict arising out of elective surgery, it is certainly possible to reduce such thoroughly unpleasant experiences by adhering to some very basic principles: Be a complete physician in the full dimension of the term, not just a clever technician; avoid hyping

your unique talent; always strive to maintain good communication and rapport with your patients; restrict your procedures to the ones that you feel comfortable performing; and resist the temptations to rush into new procedures until you're ready.

There is a somber reminder in the ultimate manifestation of the consequence of ignoring those "shades of gray" and their silent signals. During the past four decades, the lives of a number of our colleagues, five of them in the United States, were lost when they were shot to death by aesthetic surgery patients terminally unhappy with their surgical result…quality not withstanding.

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5

Sociopsychological Issues and Research on Attractiveness

Marita Eisenmann-Klein

Summary

The success of a plastic surgeon depends on his or her understanding of the psychological and social aspects of physical attractiveness. Studies demonstrate that physical attractiveness has a dramatic effect on the life of an individual. Many parameters of physical attractiveness apply to men and women; within the past 20 years transcultural studies and observations indicate that there seems to be a development towards a global consent in the perception of physical attractiveness. Yet there is a whole variety of personality disorders, psychiatric diseases and neurotic abnormalities, which might be a contraindication for aesthetic surgery. It is essential that a plastic surgeon knows how to evaluate the state of mental health in a patient.

Introduction

Plastic surgery is frequently misunderstood as the speciality, which deals with beauty. The artist Ugo Dossi states about beauty: "I don't want to reduce beauty to a phenomenon of formal aesthetics. The concept of beauty, perfection and maturity has to be present inside of us like a kind of inner North Pole. The whole of evolution seems to pursue this goal which isn't discernible as such yet at the same time and out of the invisible attracts everything towards it."[3]

This statement explains why we should not use the term "beauty" for aesthetic purposes. Physical attractiveness, however, can be created or improved by plastic surgery. Interestingly and in contradiction to the enormous importance of physical attractiveness, there was little research in this field until the end of the twentieth century. A better understanding of psychological and social aspects, however, is essential for the success of a plastic surgeon. John M. Goin and Marcia Kraft-Goin deserve the credit for being the first plastic surgery research group in this field. Their book *Changing the Body: Psychological Effects of Plastic Surgery* was published in 1981.[8]

How to Define Physical Attractiveness?

Langlois and Roggman concluded in their study: "Attractive faces are only average". They found out that by mixing faces in the so-called "morphing process" the resulting images were considered to be more attractive than the natural images of women. This phenomenon might be evolution based (best selection of criteria) or occurs because major asymmetries get eliminated during the morphing process.[3] Characteristics from attractive female faces are sun tanned skin, oval-shaped face, full lips, wider distance between eyes, narrow dark eyebrows, long and dark eye lashes, high cheek bones, narrow nose and well-shaped lids.[1]

M.Z. Siemionow and M. Eisenmann-Klein (eds.), *Plastic and Reconstructive Surgery*, Springer Specialist Surgery Series,

The criteria for attractiveness in male faces are sun tanned skin, oval-shaped face, full lips, dark eyebrows, dark eye lashes, upper half of the face broader than lower half, high cheek bones, prominent lower jaws, prominent chin, no receding hairline, well-shaped lids and no visible nasolabial fold.[1]

Martin Gruendl demonstrated that by adding childlike features to a female face, attractiveness could be increased considerably. In male faces attractiveness could be improved by adding signs of dominance[1] (Figure 5.1).

Study results about the importance of symmetry are inconsistent. Studies by Gruendl et al.[1] have shown that the correlation between attractiveness of the face and symmetry is less important than estimated in the past.

Our study group also looked for an answer to the question whether high curved eyebrows are considered to be attractive in females. The results were striking: unanimously young test subjects considered a lower eyebrow position to be more attractive. Old female test subjects were the only group who preferred the high and curved eyebrow position. Old male test subjects tended to prefer the lower position.[6]

We also found a difference between male and female test subjects in an online experiment, in which more than 90,000 participants evaluated the attractiveness of a female body. There were 168 options in this virtual body contest (Figure 5.2a–d).

In summary, above average length of legs and hip/waist ratio were the main criteria of attractiveness

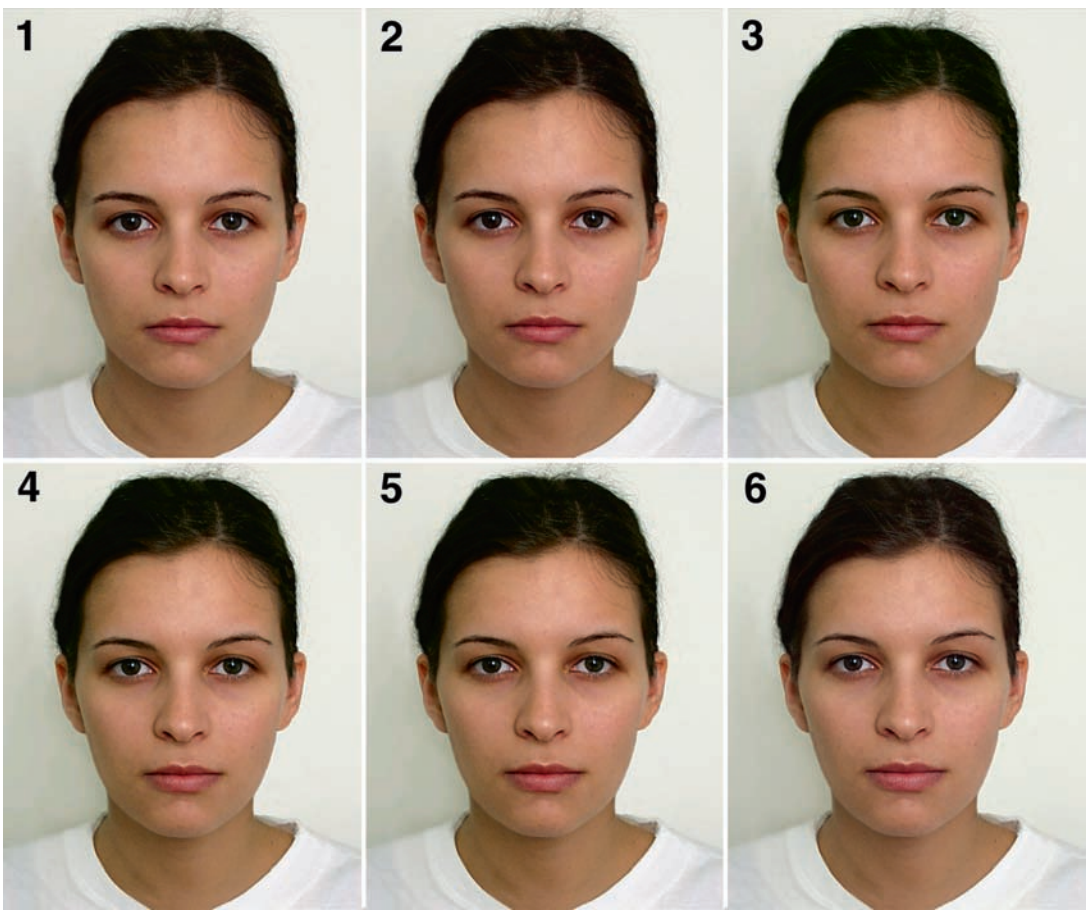

Figure 5.1. By using the morphing technique, the facial shape of the female face (No. 6) was changed gradually into the shape of a facial scheme of childlike characteristics. Only the proportions of the face were manipulated, whereas the color values remained unchanged. Face 1: 50% child, 50% adult woman. Face 2: 40% child, 60% adult woman. Face 3: 30% child, 70% adultwoman. Face 4: 20% child, 80% adult woman. Face 5: 10% child, 90% adult woman. Face 6: 0% child, 100% adult woman.

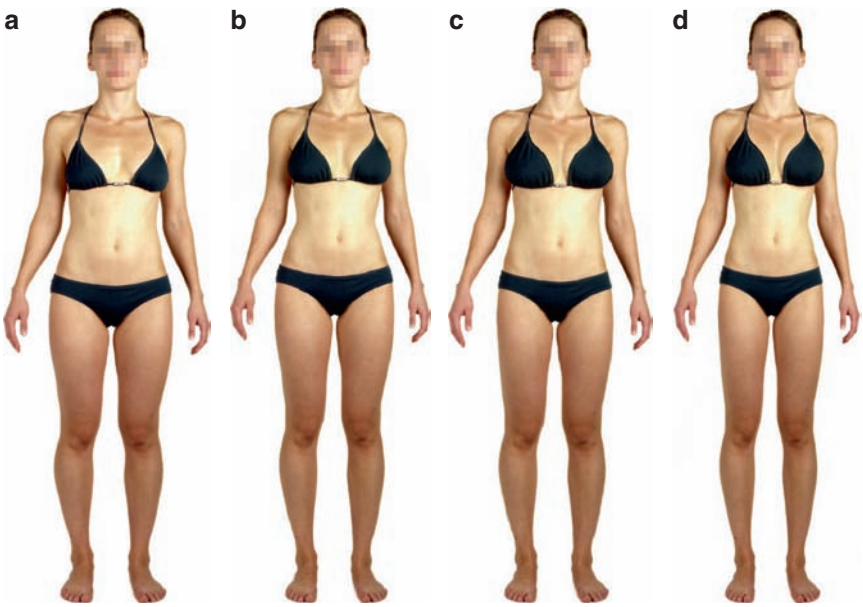

Figure 5.2. Four types of female bodies used as examples for the stimulus material (from left to right). (**a**) The average female figure with "normal measurements." (**b**) The classic 90–60–90 type with hourglass figure. (**c**) The athletic type: masculine, narrow pelvis, but large breasts. (**d**) The "Barbie-type": slim, large breasts, narrow pelvis, long legs.

in a female body. Male test subjects preferred larger breasts than female test subjects did.[9]

Attractiveness and Its Effect on Daily Life

All studies on social perception show that individuals with more attractive faces were assessed to be more successful, contended, pleasant, intelligent, sociable, exciting, creative and diligent. This is a confirmation of the so-called "Halo Effect". It means that attractive individuals are considered to be better human beings.[2,4] It is striking that new borns, only 14-h old, look at attractive faces longer than at non-attractive ones.[16] Attractive looking children get more attention from their parents and teachers. Their intelligence is estimated higher than that of non-attractive children. In the judgement of their classmates, they are considered to succeed in an academic career in 75% of cases, whereas the chances of non-attractive children were estimated to be in the range of 25%. These results were evaluated in a meta-analysis from 21 studies.[11] The influence of physical attractiveness in the choice of a partner is more important in women than in men. This seems to be a subject of change within the past 20 years. One of the reasons might be the fact that the media now frequently show perfect male bodies. An example of the silent manipulation is the popular toy figure GI Joe, which originally resembled the average American male individual 25 years ago. It has now changed to a perfect image of Arnold Schwarzenegger (Figure 5.3).

Figure 5.3. The toy figure GI Joe in 1982 and in 2004.

In summary, the results of all meta-analyses show that the effect of attractiveness is dramatic and universal.

Is There a Global Consent About Physical Attractiveness?

Transcultural studies have shown a high level of agreement among people of different races and different cultures about the attractiveness of a face.[1] Personal studies revealed that a slightly ascending angle of the eye to the lateral side increases the attractiveness of a face. These results might be interpreted as a sign of global consent in the perception of physical attractiveness.[6]

When the implant crises started in the United States in 1992, a breast that was considered to be normal sized in Europe was too small for the taste of Northern Americans and far too big for South Americans. However, the same implant sizes are used around the world. Obviously, we are approaching a global ideal of beauty of the female breast.

Psychological Features and Psychiatric Disorders

There is no doubt that the success of a plastic surgeon correlates with the right patient selection. It is essential to find out whether the patient is in a good state of mental health during the first consultation. Therefore, it is extremely important to understand the motives for aesthetic surgery. In our study we focused on three core psychological issues (self-esteem, body image, and psychopathology), which we tested in our patients preoperatively. In consistency with the results of other studies, conducted in Canada, we found that our patients had an above average level of self-esteem. However, they were more critical with their body features. Their body awareness was higher than that in control groups.[5,7,12,15]

Body Dysmorphophobic Disorders and Thersites Syndrome

The group of patients that in most cases fail to benefit from aesthetic surgery are patients with dysmorphophobia. According to Sarwer, 7% of candidates for aesthetic surgery belong to this group. Only a small minority benefits from surgery: about 10%. In 90%, patients decompensate and an exacerbation of symptoms occurs.

So far, there are no criteria to identify the minority group of patients who experience an improvement with plastic surgery.[14] The diagnosis of dysmorphophobia is not always easy. One should be cautious if the patient feels "ugly" although there are no objective signs of ugliness. The diagnosis is much more difficult in a patient with Thersites syndrome in which the dysmorphophobic disorder meets objective criteria of ugliness. Thersites was the ugliest warrior in the army of Alexander the Great.[13]

All these patients have in common the trait of overestimating the degree of deficiency. They are convinced that their deficits affect all aspects of their life. A high percentage of them admit that they thought about committing suicide.

Borderline Personality Disorders with Self-Mutilation

Borderline personality disorder is a common mental illness. Two percent of adults suffer from it. It is characterized by instability in mood, behavior and personal relationships.[17]

Self-mutilation in borderline disorders is not a rare problem. Most of these patients are young women with a history of being abused in childhood. Self-infliction might be interpreted as an attempt to gain control over the infringing act against their body integrity. There are multiple types of self-infliction: Most common are multiple parallel knife cuts on both arms, mostly forearms. We have also seen patients who scratched their faces. One of our patients injected toilet cleaner, mixed with soil, into her subcutaneous tissue. This patient had already undergone more than 300 surgical interventions. Another patient was admitted repeatedly with stab wounds in her abdomen.

The chances of curing this disorder are minimal. Frequently, but not always, the tendency to perform self-inflictions subsides when the patients reach the age of 30 years. A higher risk of committing suicide or dying from an accident remains throughout the life of these patients.[10]

Psychotic Disorders: Schizophrenia and Bipolar Disorders

Patients suffering from depressive disorders as well as from schizophrenia sometimes injure themselves by attempting to commit suicide (e.g., pressure sores after being unconscious). During a schizophrenic episode, some patients cut themselves or burn themselves. We treated a patient who lost eight fingers by holding her hands in boiling water. Injuries in psychotic patients are frequently very severe.

Neurotic Disorders

Neurosis is characterized as a mental disorder which affects only part of the personality (like anxieties, phobia, etc.). Patients with inadequate motives, unrealistic expectations or severe anxieties are quite common in plastic surgery. Neurotic patients frequently attribute their lack of success in life to other persons (parents, partners, etc.) or to unfavorable circumstances (e.g. ugly features). Neurotic disorders may be a relative contraindication for plastic surgery.

Conclusion

The term "beauty" should not be used as a synonym for physical attractiveness.

Criteria of physical attractiveness have been defined by researchers.

Physical attractiveness means an enormous competitive advantage in private, social and professional life. There is a global consent about attractiveness in different ethnic groups.

Patients asking for aesthetic surgery have a high level of self-esteem but are more critical about their body features. Dysmorphophobia and Thersites syndrome are relative contraindications for aesthetic surgery. Self-infliction is frequently associated with a history of abuse in childhood. Treatment options for this disorder are minimal. Self-infliction during psychosis may happen actively or indirectly by attempting to commit suicide. Neurotic disorders are common among patients opting for aesthetic surgery. They might cause contraindications for aesthetic procedures. Referring the patient to a psychiatrist frequently does not work out. Psychiatrists are powerless if patients do not see the necessity for treatment. In order to avoid postoperative disasters, it is essential that plastic surgeons have a basic knowledge of psychiatric disorders.

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Part II

General Surgical Techniques

6

Principles of Wound Repair

Oliver Bleiziffer, Ulrich Kneser, and Raymund E. Horch

Summary

Basic principles as reviewed in this chapter can be applied to any reconstructive problem, from the most basic to the most complex, and help improve the overall aesthetic outcome of wound closure and scar formation. A thorough assessment of the reconstructive problem, careful consideration of the affected anatomic region and proper patient selection are essential in choosing the optimal therapeutic approach to achieve the best reconstructive outcome.

Abbreviation

RSTLs Relaxed skin tension lines

Introduction

Application of certain basic principles allow the plastic surgeon to solve reconstructive problems in all areas of the body, to apply known procedures to other body parts, or to solve unusual problems altogether. These broad basic principles can be applied to simple skin excisions or to complex free tissue transfers.

Regardless of the area, size and shape of the defect, the objective is always to formulate the correct plan for closure or reconstruction. The so-called "reconstructive ladder" (Figure 6.1) implies consideration of the simplest alternative first and then progressing to more and more complex treatment strategies. In detail, progressive advancement from primary closure to skin grafting, local flaps to distant flaps and finally to microvascular free tissue transfer can be applied to any reconstructive situation.

In this chapter, skin excision and simple closure, Z- and W-plasties and simple local skin flaps are covered. More advanced techniques of reconstruction are covered in detail in the following chapters.

Closure of Skin Wounds

Surgical wound closure is the surgeon's contribution to wound repair that is propagated by the body itself through epithelialization, wound contraction and host defense mechanisms.

To attain an optimal scar, the following basic principles should be followed when suturing skin wounds: Skin edges should be debrided whenever necessary and everted and approximated without tension. The wound is closed in layers, with the dermal sutures providing the strength to the closure and relieving tension on the wound edges. On the other hand, subcutaneous fat does not necessarily require suturing, which may even lead to ischemia and necrosis.

In the early stages of wound healing, the suture (Figure 6.2) is mainly responsible for keeping the wound together. Wounds never attain the

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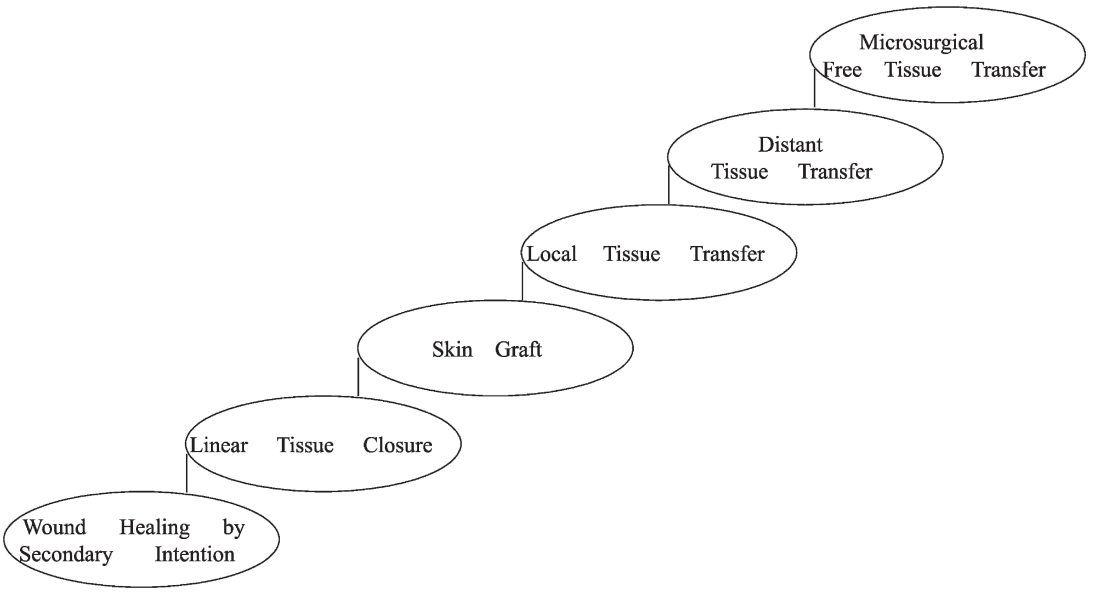

Figure 6.1. Reconstructive ladder demonstrating the range of options for defect closure, starting with the simplest and gradually progressing to the most sophisticated.

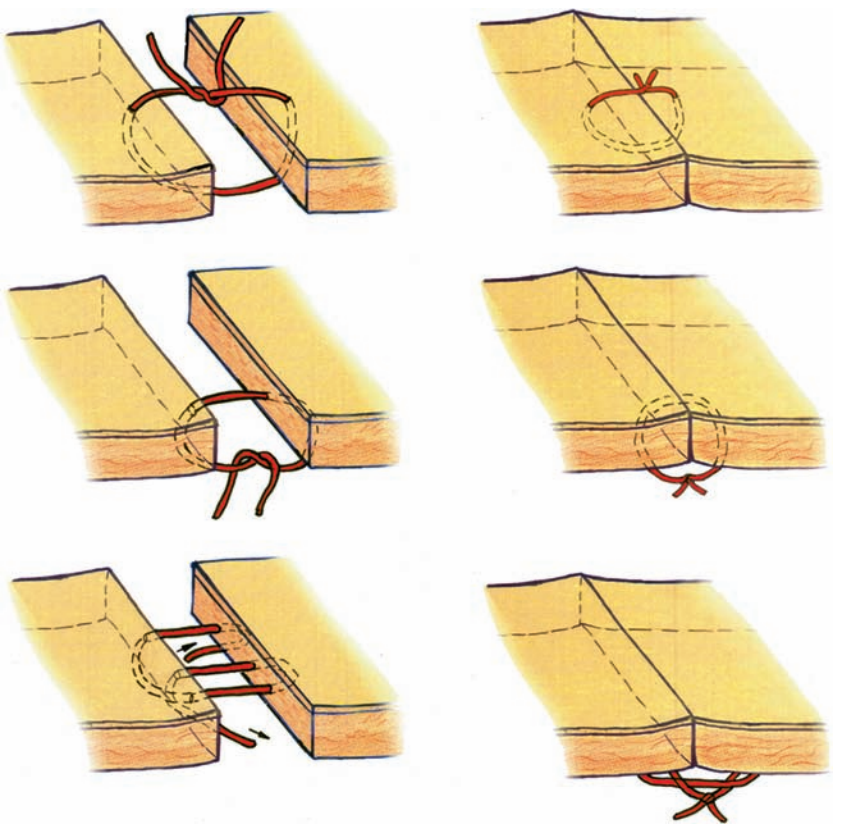

Figure 6.2. Suture techniques. Simple interrupted suture (*top row*). Interrupted inverted intradermal suture (*middle row*).Near-far pulley suture (*bottom row*).

tensile strength of normal unwounded skin, reaching a maximum of 80% of normal unwounded tensile strength. At 1 week after wounding, tensile strength is as low as 5% of unwounded skin, 10% at two weeks, 25% at 4 weeks, 40% at 6 weeks and 80% at 8–10 weeks.

Suturing Techniques

Simple interrupted sutures are the most common suturing techniques. The needle enters and exits the tissue at 90°, grasping equal amounts of tissue while passing into the deep dermis at a point of furthest distance from needle entry. After tying the knot, the suture will appear pear shaped in cross-section, everting the skin edges. To approximate edges when one side of the wound is higher than the other the tissues are grasped "high in the high", that is closer to the epidermis, and "low in the low", that is farther from the epidermis.

Vertical and horizontal mattress sutures are especially useful when wound edges are hard to evert with a simple interrupted suture. However, horizontal mattress sutures cause more ischemia, and vertical mattress sutures leave obvious cross-hatching.

Subcuticular or intradermal continuous sutures avoid suture marks and result in the most favorable scar as the needle is passed through the superficial dermis parallel to the skin surface. Placing of the suture at the same level is important. These statements, however, need to be taken with the caveat that at moments when wound situations are challenging, for example when the risk of infection is high, other suture techniques may be more appropriate. Moreover, when early suture removal can be anticipated, such as in the face, intradermal sutures are not necessarily mandatory to achieve an aesthetically favorable result.

Half buried mattress sutures, also referred to as McGregor stitch or three-corner stitch, are particularly useful when skin edges of different thickness or texture are to be adapted or a V-shaped wound needs to be closed. Moreover, all the knots will lie on one side of the suture line with no suture marks on the other side. This is an advantage when insetting the areola, for example, leaving the suture marks on the dark areola where they are less conspicuous.

Running sutures can be placed rapidly and provide hemostasis by compression. It is particularly popular in the scalp area.

Suture materials include both absorbable and nonabsorbable monofilaments that can function as foreign bodies and thereby evoke an inflammatory tissue reaction. This may lead to impairment of healing, infection or wound dehiscence. Therefore, selection of the appropriate suture material for a given situation is crucial and depends on the healing properties and requirements of the involved tissue, biologic and physical properties of the suture material, location of the wound on the body and individual patient considerations.

Skin Excision

Although wound closure is of high importance to achieve an optimal result in terms of scar formation, skin excision methods predetermine the final orientation and position of the scar. A number of different skin excision methods exist depending on the clinical problem at hand, and their proper execution is a prerequisite to achieve a satisfactory result in terms of scar formation. To predict the final appearance of scars following a skin incision to a certain extent, the knowledge of two theories describing inherent features of the skin are crucial: Langer's lines and relaxed skin tension lines (RSTLs).

When the skin of cadavers is punctured with a rounded sharp object, the resulting holes show an elliptical shape due to the tension of the skin. Langer was the first to describe this phenomenon and stated that human skin was less distensible in the direction of the lines than across them. Given that some tension lines were found to run across areas of natural creases, wrinkles and flexion lines and they do not correlate with the direction of dermal collagen fiber orientation, Langer's lines are nowadays mostly of historic interest. Instead, RSTLs are recommended as guidelines for the placement of skin incisions.

RSTLs are also known as wrinkle lines and are the lines of facial expression. They are accentuated by contraction of facial muscles, lie perpendicular to their long axis and become more discernible with increasing age.

According to these observations, the optimal scar is a fine, flat and concealed scar lying within a skin wrinkle or RSTL. Distortion of adjacent anatomic and aesthetic units or landmarks should be carefully avoided.

For the excision of skin lesions, elliptical, wedge or circular excisions may be used.

Most skin lesions can be removed by *simple elliptical excision*, with the long axis in, or paralleling a wrinkle, contour line or RSTL. The edges may be rounded or angular. As a general rule, it is recommended that the long axis be four times longer than the short axis. When the ellipse is made too short or one side of the ellipse is longer than the other, so-called "dog ears" are the result. They may be flattened over time but are best taken care of immediately. If the elliptical excision is too short, it can be lengthened to include the excessive tissue. Alternatively, the redundant tissue can be excised as two small triangles. If one side of the ellipse is longer than the other a short triangle or 45° incision at the end of the ellipse can be performed, and the redundant tissue can be removed.

Wedge excisions have their primary indications for lesions on the free margins of the ear, lip, eyelid or nostril.

Closure of circular defects are usually performed either by a local flap or a skin graft.

Under certain circumstances, *serial excisions* may become necessary when particularly large lesions such as giant naevi or large areas of scarring are excised. In these cases, serial excisions may be used in combination with tissue expanders.

The viscoelastic properties of the skin and creep and stress relaxation phenomena enable successful application of serial excision techniques. One indication has been the treatment of male-pattern baldness by serial excision of non-hair bearing areas of the scalp.

Surgical Treatment of Scars

Scar formation is an inherent part of every wound healing process regardless of whether it is caused by trauma or surgery. Scarring can be reduced by correct placement of incisions, minimization of trauma during surgery and use of appropriate suturing and dressing material and techniques. Younger patients are more prone to scarring than older patients, and African and Asian populations usually exhibit worse scarring compared with Caucasians. Scars usually become more conspicuous for around 3 months after surgery, followed by regression over the following months. Surgical correction should be performed once the scar has matured. This point is usually reached between 9 months and 2 years after surgery. Scar revision aims for making it level with adjacent tissue, dividing it into smaller pieces and reorienting it. Nonsurgical means to achieve the most favorable scars possible include protection from light, compression therapy and application of silicon sheets.

Z-Plasty

The Z-plasty is an ingenious technique in which two triangular flaps undergo transposition without tension[3,4] (Figures 6.3 and 6.4). The results are a gain in length along the direction of the common limb of the Z, which is useful in the management of scar contractures, and a change in direction of the common limb of the Z, which can be useful in the management of facial scars. The central limb is usually placed along the line of contracture or scar, whereas the two remaining limbs, which must be equal in length to permit the skin flaps to fit together after transposition, are positioned to resemble a Z or reversed Z. They can extend at varying angles from 30 to 90°, depending on the desired gain in length. The wider the angles of the triangular flaps, the greater the difference between the long and short diagonals and thus the greater the lengthening. The classic Z-plasty has an angle of 60° and provides a 75% theoretical gain in length of the central limb. A prerequisite for successful execution of a Z-plasty is sufficient laxity laterally to achieve the appropriate lengthening perpendicular to it. Angles significantly less than 60° do not achieve sufficient lengthening and result in flap narrowing, increasing the risk for vascular compromise and flap tip necrosis. On the other hand, angles that are too wide produce undue tension in adjacent tissue, thereby preventing flap transposition. A 30° angle produces a 25% increase in length; a 45° angle, a 50% increase; a 75° angle, a 100% increase and a 90° angle, a 120% increase. These theoretical values provide a good approximation of the actual final lengthening, which will be slightly less based on mechanical properties of the skin.

In scar revision, the final central limb will lie perpendicular to the original central limb after flap transposition and should be selected first. Since the total length of the limb of multiple Z-plasties can equal the length of the central limb

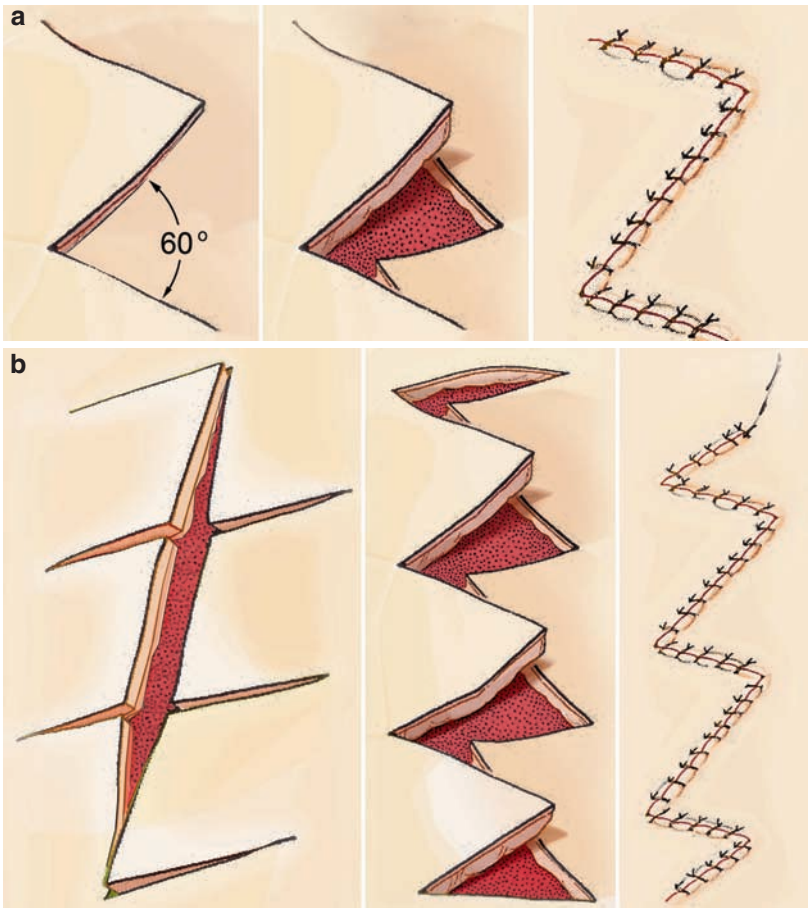

Figure 6.3. (**a**) Design and planning of Z-plasty. *Left panel*: The limbs of the Z must be equal in length to the central member. The angle between the limbs is 60° in this example. *Central panel*: The flaps are raised. *Right panel*: The flaps are transposed to their final position, altering the original direction of the scar. (**b**) Multiple serial Z-plasties. Transverse shortening is reduced and lateral tension is distributed more evenly compared to a single Z-plasty. Reprinted from [5] with friendly permission from Dr. R. Kaden Publishing.

of a single Z-plasty, they both can produce a similar degree of lengthening. Multiple small Z-plasties, however, tend to produce results superior to one large Z-plasty. Moreover, they produce less transverse shortening and provide a more equal distribution of lateral tension over the entire length of the central limbs. Applications include facial scars as well as U-shaped and circumferential scars. Caution should be executed in burn contracture, where the necessary lateral skin excess on either side of the contracture is not available.

An example of efficient application of multiple Z-plasties is the double opposing Z-plasty, also referred to as jumping man plasty. It is particularly useful for release of contractures of concave body regions, for example the medial canthal region or interdigital Web spaces. The two Z-plasties on each side of the central flap are transposed, whereas the central flap is advanced in a Y-V fashion.

W-Plasty

This technique is another method of changing the direction of a linear scar (Figure 6.5). The scar is excised using multiple small triangles on

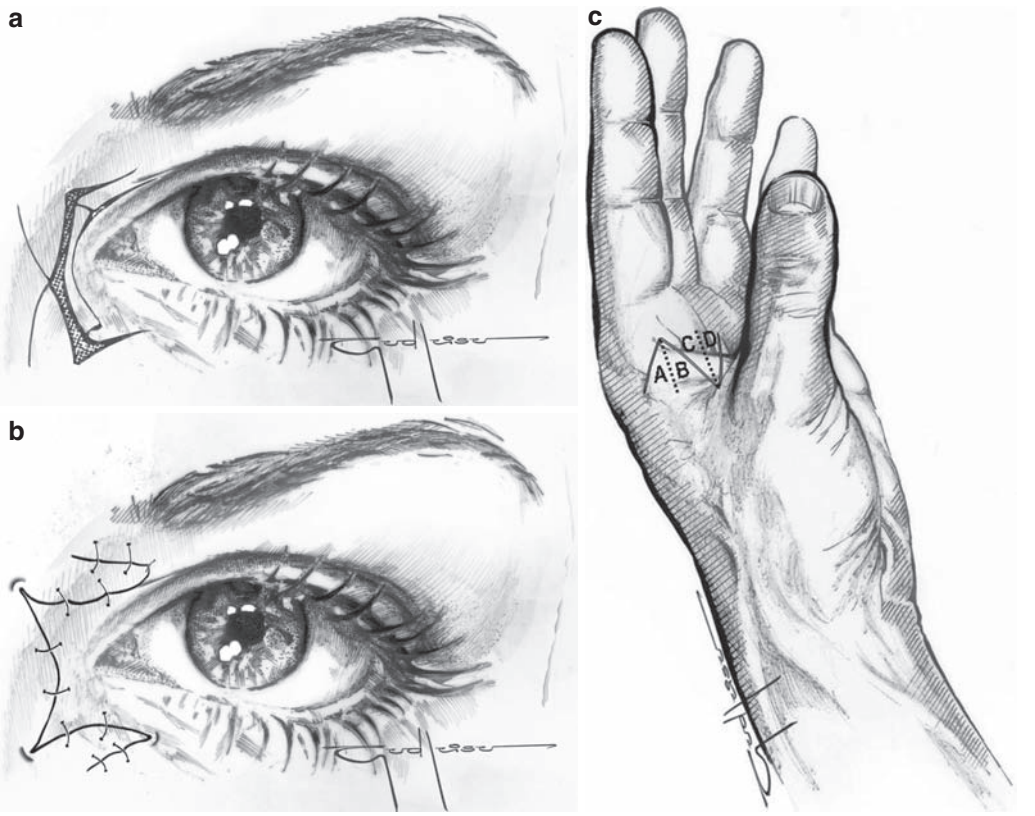

Figure 6.4. Clinical applications of Z-plasty. (**a**) and (**b**) Opposing Z-plasty involving the medial eyelid. (**a**) Incision and raise of the lateral flap which is to be advanced. (**b**) Flap advancement, gaining length in the vertical direction. (**c**) Four-flap Z-plasty for correction of a thumb adduction contracture. Reprinted from [5] with friendly permission from Dr. R. Kaden Publishing.

either side of the scar, with the opposite sides of the triangles interdigitating with each other.[1,2] Towards the ends of the scar, the size of the excised triangles should gradually decrease, resulting in flattening of the W's limbs. The W-plasty does not decrease tension in a scarred area but rather increases it due to the inherent sacrifice of tissue. It should also be avoided when the goal is to lengthen a contracted scar, in which case a Z-plasty is preferable.

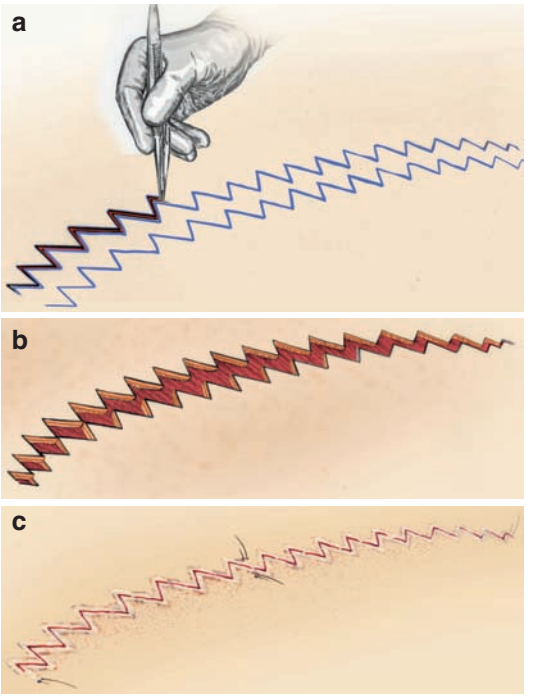

Figure 6.5. W-plasty. (**a**) Zigzag pattern incision along both sides of the scar. (**b**) The scar has been excised, resulting in tips and corresponding bases of the incisions lying opposite each other. (**c**) Result after wound closure where corresponding tips and bases interdigitate with each other, resulting in the typical W-shaped scar. Reprinted from [5] with friendly permission from Dr. R. Kaden Publishing.

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7

Grafts, Local and Regional Flaps

Jay W. Granzow and J. Brian Boyd

Summary

Over the last century, numerous solutions have been devised for the closure of defects where tissue is missing or which cannot be closed with the simple approximation of the wound edges. Grafts and flaps both represent tissue transfer from one location to another. Grafts differ from flaps in that they do not have their own blood supply, whereas flaps bring their own blood supply when transferred to a new location. This chapter provides a basic overview of flaps and grafts and lists several examples of each.

Abbreviations

TRAM	Transverse rectus abdominis musculocutaneous
VAC	Vacuum assisted closure

Introduction

Surgeons have faced the dilemma of wound closure for thousands of years. Indeed, the first account of skin flaps, attributed to Sushruta, was recorded at least 400 years before Christ. This ingenious surgeon devised a method, very similar to the modern-day forehead flap, for the repair of nasal defects that were often sustained in battle or inflicted as punishment for adultery in ancient India. In 1597, Gasparo Tagliacozzi (Figure 7.1) wrote a landmark treatise discussing the closure of wounds with tissue from adjacent or distant areas of the body.

Over the last century, numerous solutions have been devised for the closure of defects where tissue is missing or which cannot be closed with the simple approximation of the wound edges.

Grafts and flaps both represent tissue transfer from one location to another. Grafts differ from flaps in that they do not have their own blood supply, whereas flaps bring their own blood supply when transferred to a new location. Grafts survive on a blood supply acquired from the recipient bed, and typically, the tissue of a graft must lie within 1–2 mm of the recipient blood supply to survive. This limits the type and amount of tissue that can survive as a graft. In contrast, properly designed flaps can be used to transfer much larger amounts of tissue in a safe, predictable, and reliable way.

There exists a simple paradigm often known as a "reconstructive ladder" (Figure 7.2). It states that when reviewing options for reconstruction, simple methods are considered first and progressively more complex and difficult ones are thought of next. Of course, specific instances and indications may call for a surgeon to "skip" basic steps and immediately proceed with more

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Figure 7.1. Plate from Gasparo Tagliacozzi's De Curtorum Chirurgra perinsitionem, Libri Duo (Venice, 1597).

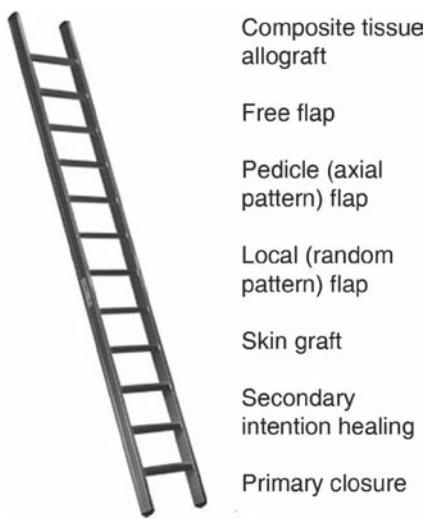


Figure 7.2. Reconstructive ladder.

complex procedures, but in general, the exception proves the rule.

Principle of Aesthetic Units

Certain areas of the body require specific attention to maximize the functional and aesthetic result. One such important area is the face (Figure 7.3), which consists of different facial subunits. These subunits describe natural patterns that the eye and the brain recognize as fitting together. It has been found that reconstruction of an entire subunit is preferable to reconstruction of only a partial subunit. Incisions should fall within the borders between the subunits to minimize their prominence and allow for an improved aesthetic result. Similarly, other portions of the body, such as the breasts (Figure 7.4), are also said to have subunits, and aesthetic reconstruction achieves better results when these subunits are respected.

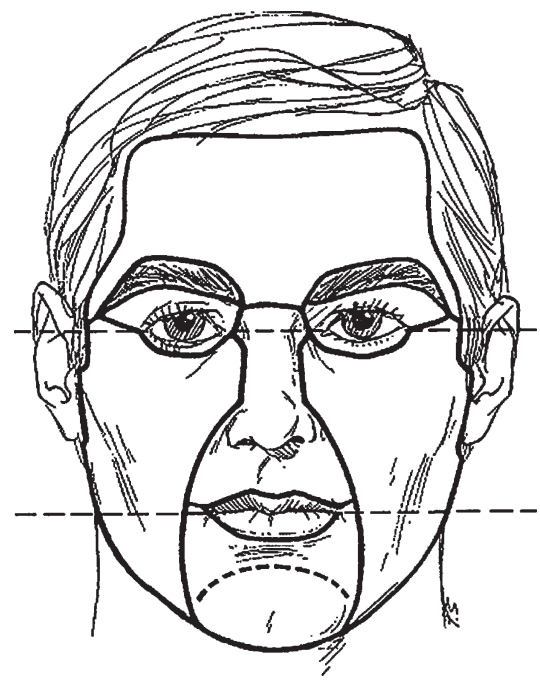


Figure 7.3. Aesthetic units of the face. (Reprinted from Gonzalez-Ulloa M. Restoration of the face covering by means of selected skin in regional aesthetic units. *Br J Plast Surg*. 2005;9:212. Copyright (1956), with permission from Elsevier.)

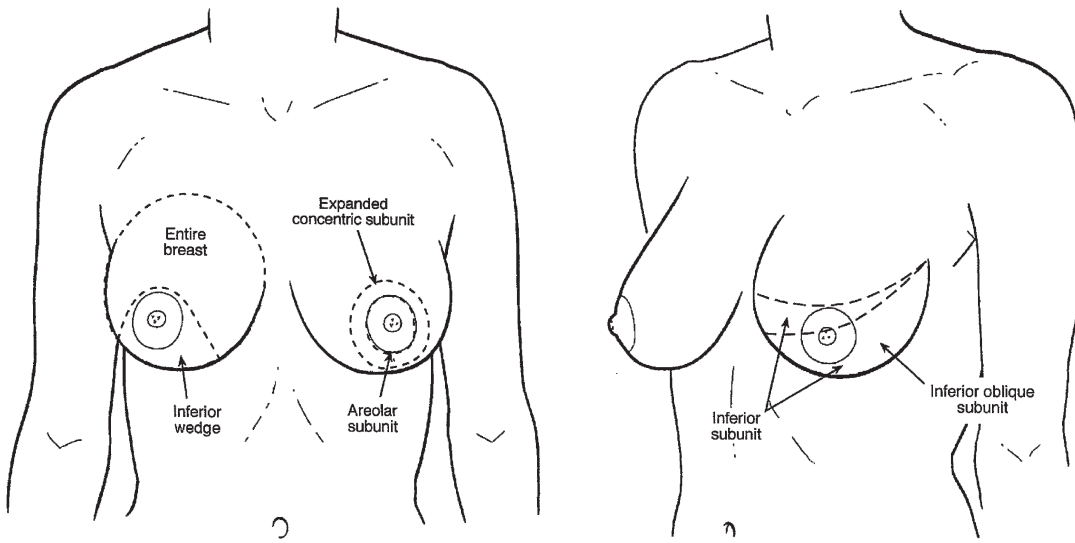


Figure 7.4. Examples of subunits of the breast. (Reprinted with permission from Scott and Davison.[6])

Grafts

A graft is a tissue that is transferred but does not maintain its own blood supply. Instead, the graft relies on the recipient bed for nutritional support while it becomes incorporated at its new location. The graft can consist of virtually any native tissue such as skin, fat, nerve, blood vessel, fascia, tendon, or bone.

Cells within a graft typically need to lie within 1–2 mm of the blood supply in the recipient bed to survive and be effective. Split- or full-thickness skin grafts may have a large surface area, but they must be extremely thin to maintain cellular viability while circulation is reestablished.

Certain tissues with very low metabolic activity or which contain a large amount of minimally vascularized framework, such as bone, tendon, or cartilage, can be transferred in larger units and still remain viable.

Since grafts depend on picking up a blood supply from the recipient bed on which they are placed, that bed must have good vascularity. There are times when the bed has insufficient blood supply to support a graft. The following structures will not support a graft:

Bone
Cartilage
Tendon
Hardware, implants, or other foreign material

However, some of the supporting tissues associated with bone (periosteum), cartilage (perichondrium), and tendon (paratenon) have a vigorous blood supply and may form healthy recipient beds for grafted cells.

Wound VAC

A relatively recent innovation, called the wound VAC (vacuum-assisted closure), has provided a novel way to encourage the growth of granulation tissue over a previously ungraftable wound (Figure 7.5).

VAC therapy consists of a controlled application of continuous or intermittent subatmospheric pressure to a sponge-like wound dressing to promote healing. Tissue edema and discharge are suctioned away from the wound, while cell migration and proliferation are promoted. The tissue that is encouraged to proliferate typically takes the form of granulation tissue, allowing improvement of the vascular supply in a given recipient site. This granulation tissue can spread across an avascular area and can also fill in defects and smooth out the contour of the recipient site. However, the VAC has its limitations and cannot be used in all instances, particularly when the wound is infected.

The dressing is changed much less frequently than routine wet-to-dry dressings: every 2–4

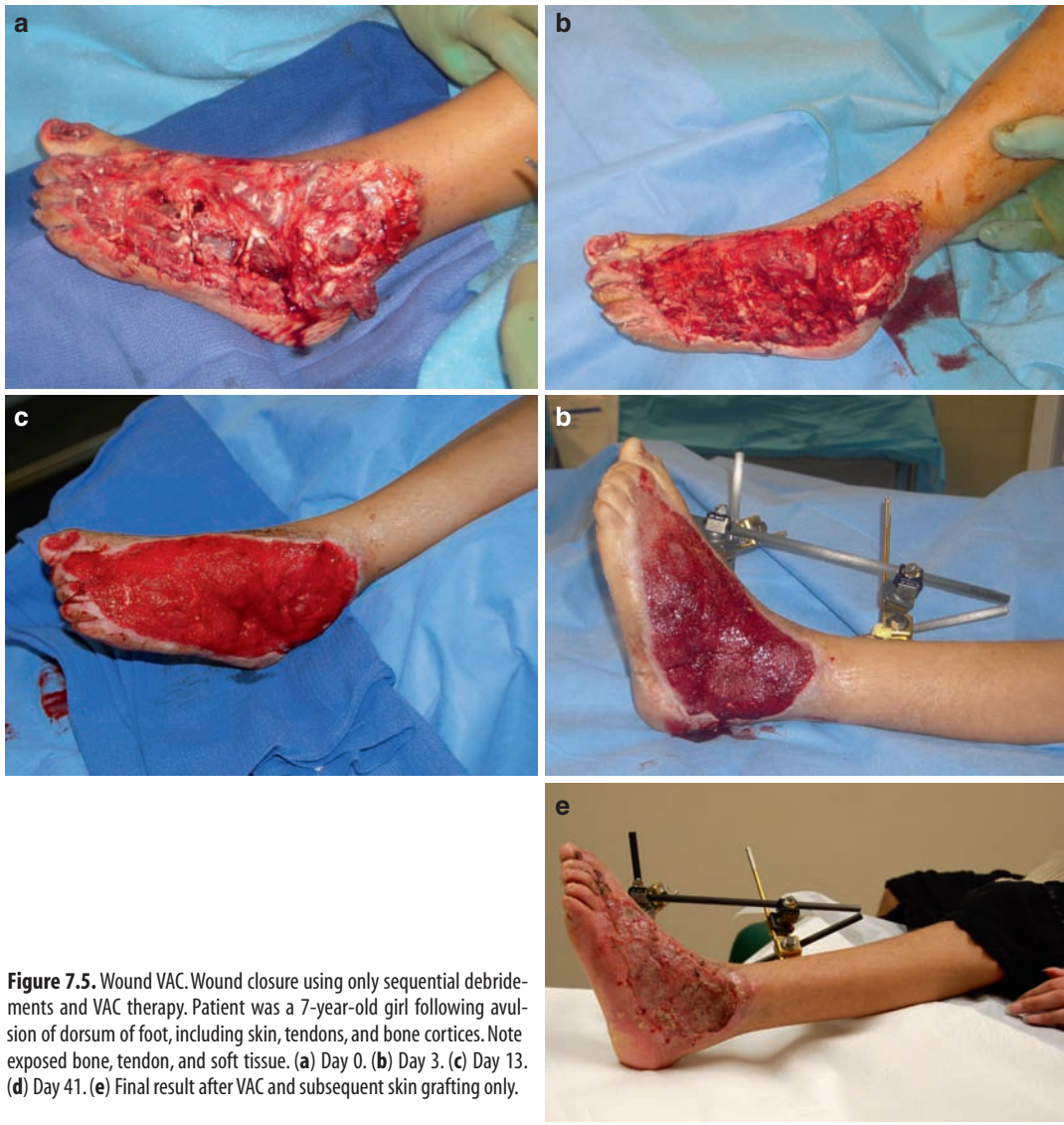


Figure 7.5. Wound VAC. Wound closure using only sequential debridements and VAC therapy. Patient was a 7-year-old girl following avulsion of dorsum of foot, including skin, tendons, and bone cortices. Note exposed bone, tendon, and soft tissue. (**a**) Day 0. (**b**) Day 3. (**c**) Day 13. (**d**) Day 41. (**e**) Final result after VAC and subsequent skin grafting only.

days rather than every 6–8 h. The appearance of a thickened bed of fine granulation tissue sets the stage for wound closure with simple grafting rather than more complex flap coverage.

Skin Grafts

Skin is a multilayered organ, which varies greatly in its characteristics throughout the body. Skin always consists of an epidermis with a basement membrane overlying a layer of dermis (Figure 7.6). The dermis is variable in thickness and ranges from several millimeters on the back to less than 1 mm in areas such as the eyelid. Skin contains multiple epithelial appendages, such as hair follicles, sebaceous glands and sweat glands, which extend into the dermis below the basement membrane. (The hair follicles may even protrude into the subcutaneous fat beneath the dermis.)

Epithelial cells from these structures that are embedded in the dermis form the basis for the re-epithelialization of a split-thickness skin graft donor site.

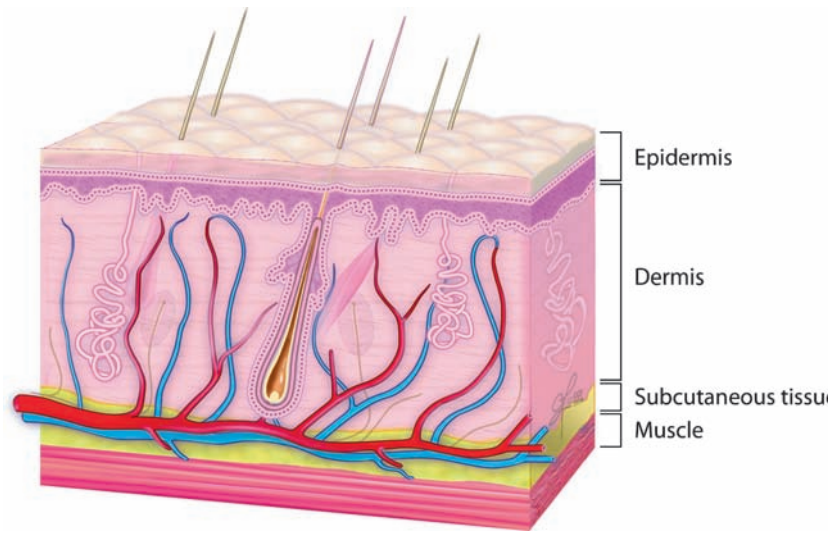


Figure 7.6. Elements of the skin.

Skin grafts allow the transfer of epithelial cells and can provide coverage for large open wounds. They have the advantage that they are easy to harvest, can close large open wounds quickly, and completely regenerate themselves in 2–4 weeks. Drawbacks include a recipient site less resistant to surface trauma and shear than normal skin, a color mismatch with the surrounding tissues, and a contour deformity due to the lack of subcutaneous fat.

These grafts may be taken as either split- or full-thickness grafts. A full-thickness skin graft consists of epidermis plus the entire dermis. A split-thickness skin graft consists of epidermis and only a fraction of the dermis. It should be noted that both split- and full-thickness skin grafts contain the epidermis plus more or less of the underlying dermis. The thicker the graft, the more durable the result and the less wound contracture seen as the graft heals. This is thought to be due to the ability of fresh dermis to inhibit the action of myofibroblasts in the wound bed.

Split-thickness skin grafts initially contract less than their thicker, full-thickness counterparts, because of a smaller amount of elastic tissue contained within them. However, as healing occurs, these grafts are much more susceptible to contracture and shrinkage than full-thickness skin grafts.

Split-thickness skin grafts are easy to harvest and are typically taken directly with a knife or with an instrument such as a dermatome. They may be expanded in size using a meshing device that evenly perforates the graft and allows both its expansion as well as the egress of fluids, which if trapped beneath the graft would impair its incorporation (Figure 7.7).

Skin Graft Failure

Causes of skin graft failure include the following:
Infection – Infection results in the destruction of the skin graft by the invading microorganisms or interference with its adherence to the bed.
Shear stress – It is the mechanical breakdown of the connections between the skin graft and its recipient bed after initial adherence. It is caused by movement of the graft over the recipient site.
Accumulation of fluid under the graft – This is typically prevented by meticulous hemostasis, an even pressure dressing, and by either meshing or "pie crusting" the skin graft.

Skin Graft Staging

Skin graft occurs in three stages.

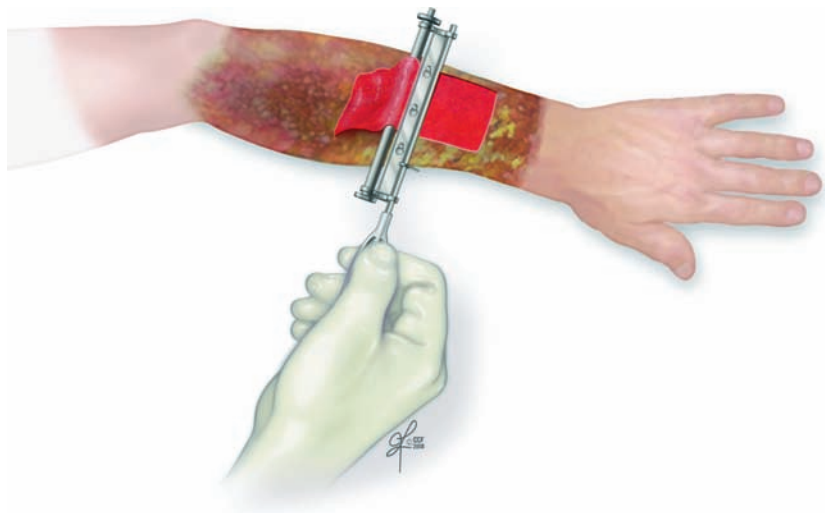


Figure 7.7. Split-thickness skin graft.

Plasmatic Imbibition

Plasma derived from the recipient capillary bed enters the graft's open vessels by capillary action. Nutrients and oxygen are thereby exchanged between the cells of the graft and those of the recipient bed. This allows survival of the immediate postoperative ischemia after skin graft harvest but before circulation is established.

Inosculation and Capillary Ingrowth

At approximately 24–48 h after placement of a graft, a very fine network of vessels begins to be established. Capillary buds from the recipient bed grow into the open vessels on the undersurface of the skin graft. This is the precursor to the establishment of circulation within the graft.

Revascularization

Revascularization describes the process in which a series of circulatory loops are established within the graft. Soon a proper circulation is created, and this is capable of nourishing the graft on a permanent basis.

Graft Classifications

Grafts can also be classified according to the origin of their tissues (Table 7.1):
Autografts – Grafts are from the same individual.

Table 7.1. Types of grafts.

Examples of types of grafts	
Graft type	**Application**
Split-thickness skin grafts	Open wounds, burns
Full-thickness skin grafts	Wounds, facial resurfacing
Bone grafts	Bone defect from trauma, cancer resection
Vessel grafts	Vessel bypass or vascular repair
Nerve grafts	Nerve defect repair
Hair transplants	Hair loss (hereditary, traumatic)
Cultured autografts	Extremely large defects with few donor sites

Allografts – Grafts are from the same species.
Xenografts – Grafts are from different species. Xenografts are not incorporated into the host. They can provide some temporary cover and homeostasis, but they undergo rejection and will have to be removed.
Isografts – Grafts are between identical twins (same genotype).

Human Cadaveric Dermis (Alloderm and Others)

Allografts of human cadaveric dermis are commonly used to replace tissue lost to trauma or surgery or to supply strength to otherwise weak

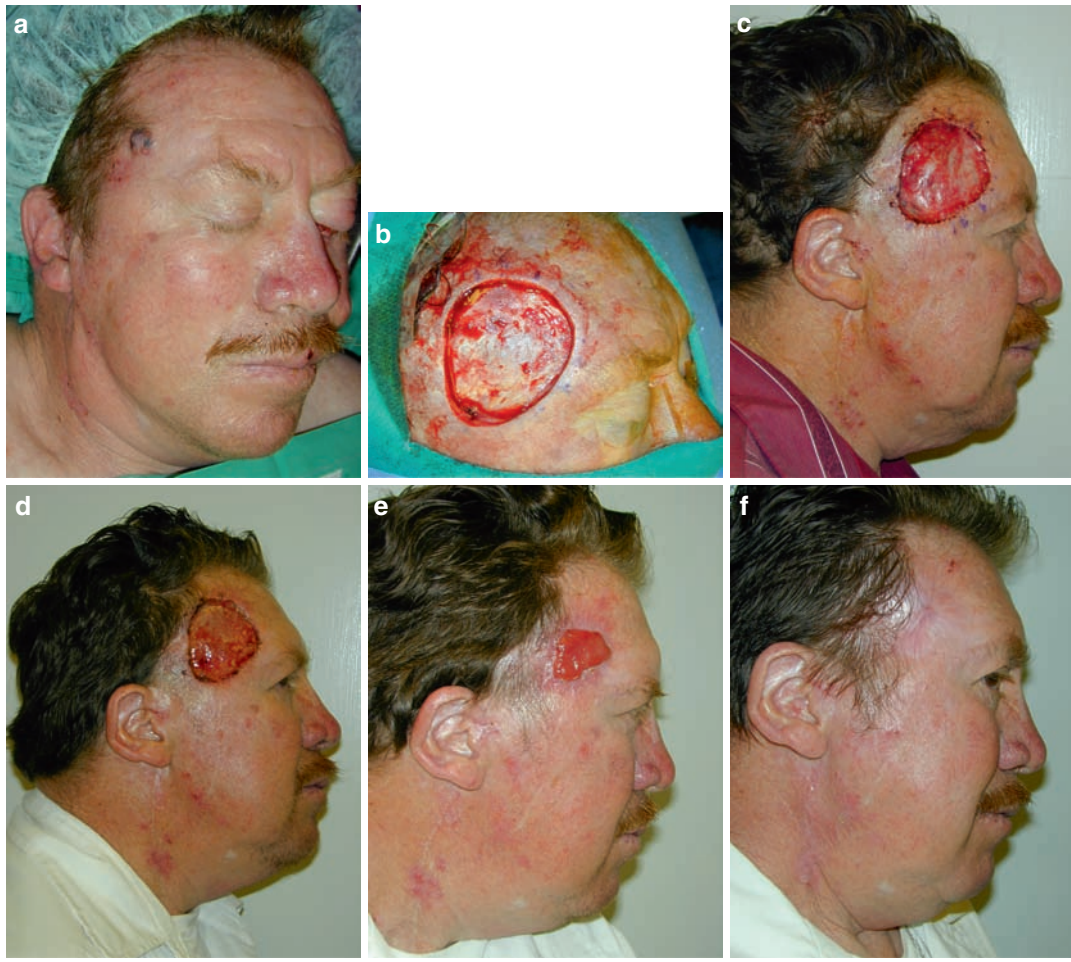


Figure 7.8. Closure of a temporal defect with human cadaveric dermis. The allograft provided a scaffold for secondary healing to provide good contour and color match without significant wound contracture or brow elevation. (**a**) Initial malignant melanoma. (**b**) Result of wide local excision. (**c**) One day after allograft application. (**d**) Seven days postop. (**e**) Thirty-five days postop. (**f**) Sixty days postop.

or deficient facial layers, such as in the abdomen. The material typically integrates very well with the recipient's tissues and has a low incidence of infection or extrusion (Figure 7.8).

Tendon grafts – Autologous or cadaveric tendon may be used as a tendon graft in the hand and extremities (Figure 7.9). These grafts require healthy vascularized soft tissue coverage, as they do not carry their own blood supply.

Bone grafts – Like tendon grafts, these grafts may be transferred to fill structural defects in a recipient area (Figure 7.10).

Cultured autografts – Cultured autografts may be used to graft large open wounds in a patient with little available autologous donor tissue. Such a situation would occur in an extensive burn. A small sample of the patient's own cells is harvested and cultured in vitro to produce a large sheet of epithelial cells. These may then be grafted on to the patient. Unfortunately, the lack of dermis makes the graft rather fragile and susceptible to shearing forces. Attempts are being made to use cultured epithelial cells together with dermal scaffolds such as Alloderm or Integra to make them more durable.

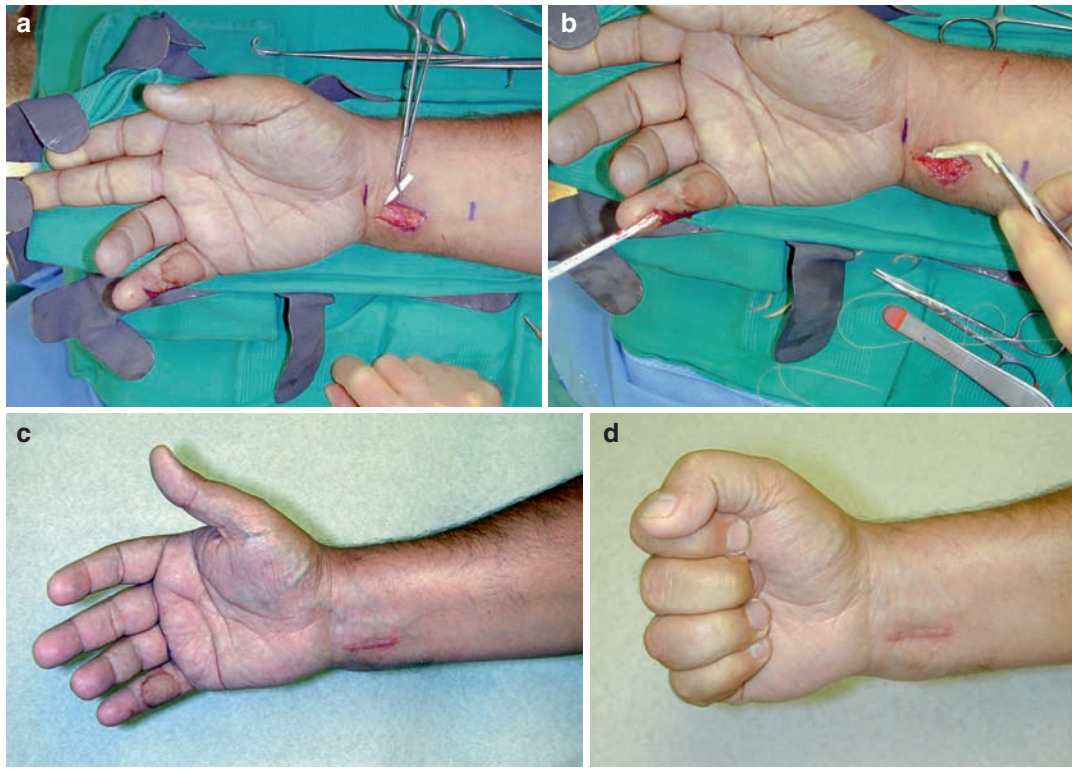


Figure 7.9. Tendon graft. Patient with previous zone two flexor tendon avulsion injury, cross finger flap, and Hunter rod placement. (**a**) Initial operative view with Hunter rod in place. (**b**) Substitution of Hunter rod with tendon graft. (**c**), (**d**) Postoperative flexion and extension of digit.

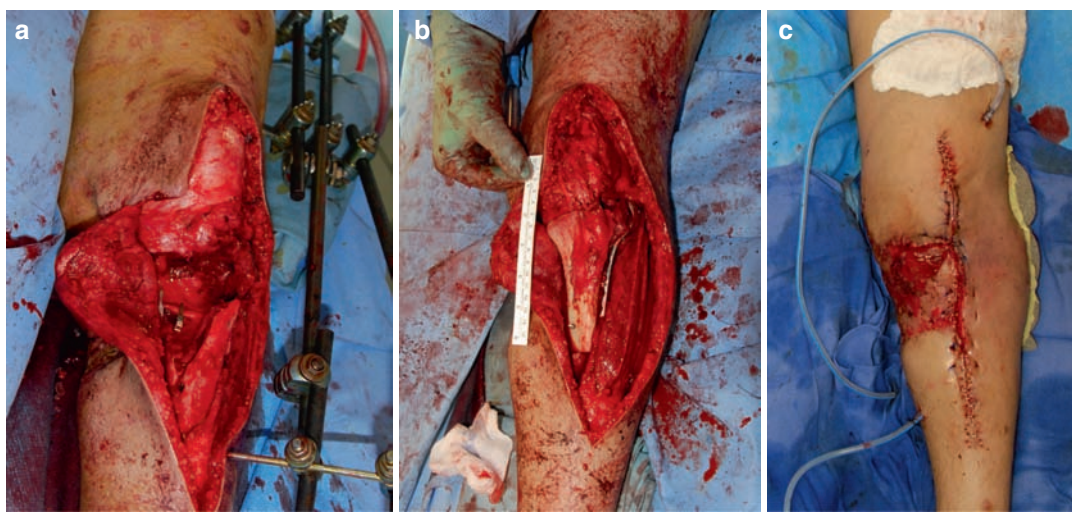


Figure 7.10. Bone graft. Patient with proximal tibial bone defect sustained from a shotgun wound. (**a**) Tibial bony defect. (**b**) Allograft and tibial fixation plate. (**c**) Postoperative appearance after additional lateral bipedicled advancement flap closure.

Flaps

A flap is a tongue of tissue designed for reconstruction and having its own intrinsic blood supply. Flaps are not new and date back to well before the time of Christ. In ancient India, the first forehead flaps were performed for nasal reconstruction. Flaps may be divided into two groups: axial and random. The former have a known vessel traversing their length, but the latter are based on a random, nonspecific blood supply.

Axial flaps may be pedicled at their base (pedicled flaps) or on their skeletonized blood vessels (island flaps). The vessels may be severed and the flap transplanted to some other part of the body using microvascular anastomosis (free flaps). Flaps may be further classified according to their constituent parts: muscle, bone, musculocutaneous, fascial, fasciocutaneous, osteofasciocutaneous, and so on. Perforator flaps refer to flaps in which the main feeding vessels pass through muscle and require meticulous dissection to isolate them for free tissue transfer.

Random flaps are generally classified according to their geometry (see Table 7.2). The blood flow to a random pattern flap may be improved by dividing a portion of the blood supply to the flap that is not incorporated into the flap pedicle approximately 7–10 days prior to complete elevation of the flap itself. This causes the blood supply to the remaining soft tissue pedicle to become more robust, increasing the viability of the flap tissue itself.

In summary, pedicled flaps remain attached to their intrinsic blood supply and are discussed further below. They may be axial or random. Free flaps take their blood supply from specific, axial vessels, which are carefully prepared and divided during the course of the flap harvest. These vessels then require to be joined to recipient vessels in their new location. The vessels are quite small, measuring a few millimeters in diameter. Commonly, the operating microscope is employed, thus giving rise to the term "microvascular surgery." Free flaps are addressed in another chapter of this text.

Table 7.2. Examples of types of flaps.

Examples of types of flaps	
Flap type	**Application**
Pedicled flaps	
Random pattern	Repair of small facial defects
Axial pattern (fasciocutaneous, muscle, and musculocutaneous	Repair of large adjacent defects, breast reconstruction, extremity reconstruction
Free flaps	
Fasciocutaneous flaps	Head and neck, extremity reconstruction
Muscle/musculocutaneous flaps	Breast, extremity reconstruction
Perforator (muscle sparing flaps, e.g., DIEP flap)	Breast, extremity reconstruction

Pedicled Flaps

Pedicled flaps remain attached at their site of origin. That attachment forms a vascular "leash," which can limit the distance a flap can be rotated to reach its recipient site. Such flaps tend to be relatively fast and easy to raise and can reliably transfer tissue a short distance from a donor to a recipient site. A flap that includes a named vessel is called an "axial" flap and can consist of large amounts of tissue.

Local Random Pattern Flaps

Local random pattern flaps are skin flaps that do not contain a named or axial vessel and are supported by the subdermal plexus. Classically, the maximum length-to-width ratio to permit complete survival of these flaps is said to be 3:1 or 4:1. Longer flaps may undergo necrosis at their tips – the portion of the flap of most clinical value. However, clinical observation of flap survival and controlled experiments in animals indicate that a certain finite length can be reached in most random pattern flaps regardless of a 3:1 classic length-to-width ratio.

Types of local random pattern flaps are listed below.

Monopedicle Advancement Flap

Monopedicle flaps are created from results from two parallel incisions that extend from the defect to adjacent tissue. Tissue of the flap is lifted and advanced into the area of the defect

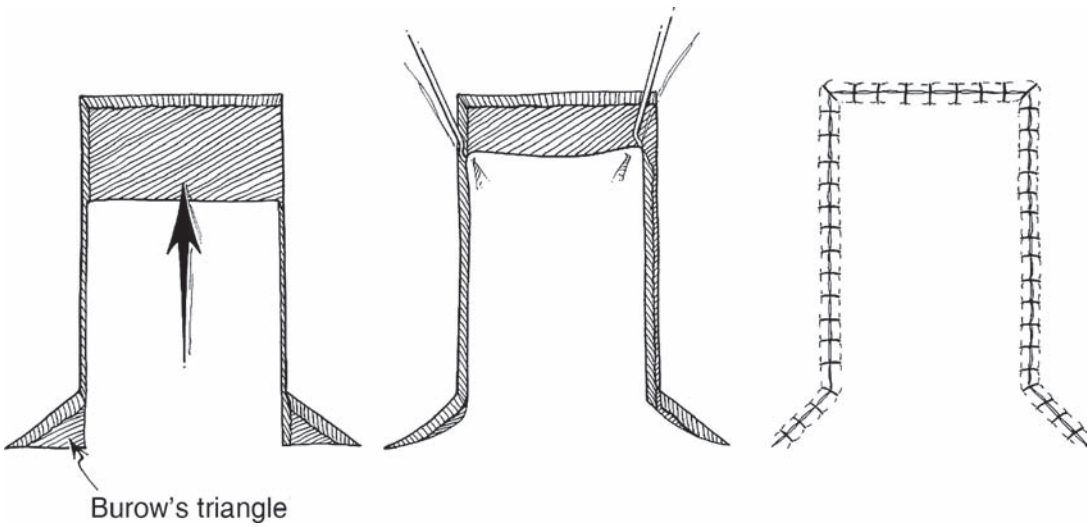


Figure 7.11. Monopedicle advancement flap. (From Jackson.[2])

(Figure 7.11). This creates areas of increased and decreased tension and also localized bunching of tissue, which is referred to as "standing cones." These standing cones may be removed at the time of flap surgery or at a later time by the excision of so-called "Burrow's triangles."

V-to-Y Advancement Flap

This is a triangular-shaped flap that allows advancement of tissue to an adjacent area and the primary closure of the trailing tissue (Figure 7.12). The initial defect appears similar to the letter V, and the resulting defect appears closer in shape to the letter Y. This triangular flap will lengthen tissue in line with its backward motion.

A-T Flap

An A-T flap allows bilateral advancement of tissues and is well suited for closure of a triangular defect, or defects, which are adjacent to a facial subunit or a linear junctional area such as the hairline or near the lip or eyebrow (Figure 7.13).

Rotation Flap

Rotation flaps allow the rotation of tissue along a semicircular arc from one area to another. The length of the perimeter of the flap is typically at least seven times the width of the defect, which allows easy closure of the donor site and appropriate tension distribution along the suture line. Sometimes a relaxing incision, or "back cut," is required to allow proper rotation of tissue. The line of maximum tension typically follows between the angles of 90° and 135° from the defect (Figure 7.14).

Transposition Flap

Transposition flaps are based at the edge of a defect and are moved over intervening tissue to close the defect. This allows skin to be taken from an adjacent loose area, where it can be spared, and used for reconstruction where skin is deficient (Figure 7.15).

Bilobed Flap

A bilobed flap is a double transposition flap. It involves a single pedicle carrying two lobes. The first fills the defect, and the second closes the donor site of the first. The lead flap is the size of the defect, whereas a secondary flap is approximately half the size of the defect (Figure 7.16). The second donor site is closed primarily. Its main advantage is that when loose skin is not immediately adjacent to a defect, it can be recruited from further afield where there is more

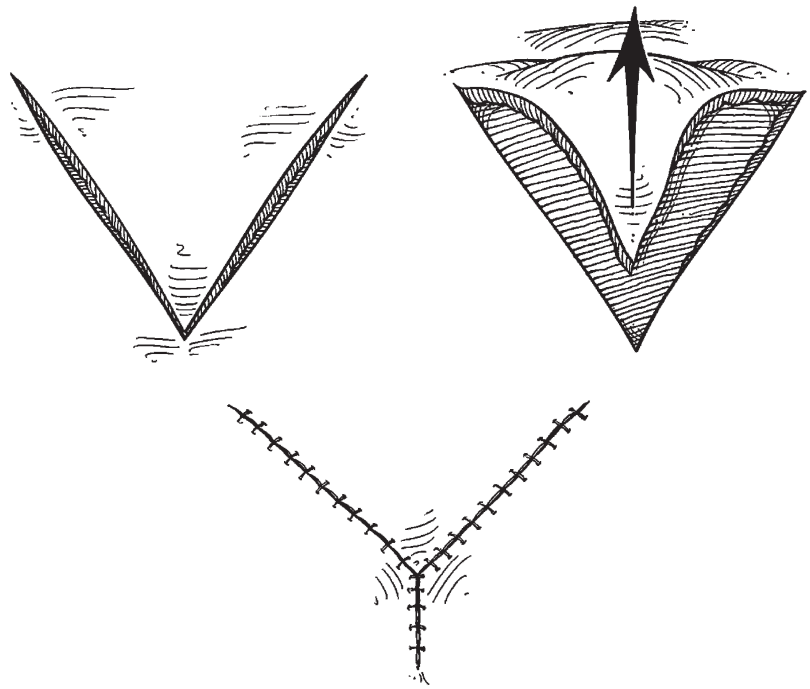


Figure 7.12. V-Y Advancement flap. (From Jackson.[2])

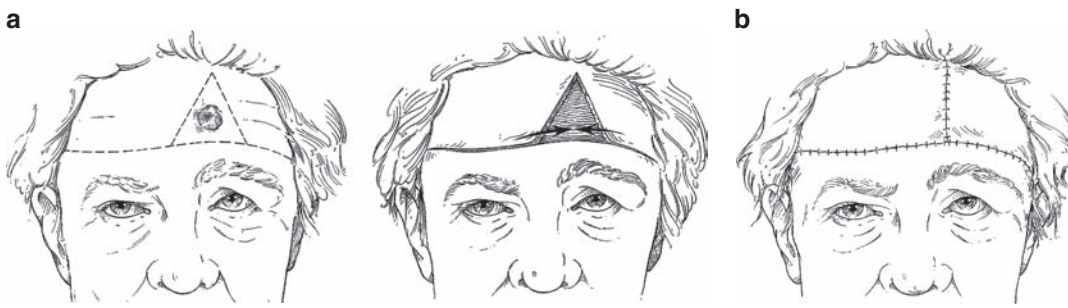


Figure 7.13. A-T flap. (From Jackson.[2])

laxity. The bilobed flap is commonly used on the nose. The disadvantages of a bilobed flap are that multiple scars are created and each lobe may contract, resulting in a pincushion appearance.

Rhomboid (Limberg) Flap

Originally attributed to Limberg, the rhomboid flap is a specially designed transposition flap involving closure of a recipient defect, which is made to take the shape of an equilateral rhomboid. Typically, the defect has two pairs of opposing angles of 60° and 120° (Figures 7.17 and 7.18). The design is versatile and allows for the creation of four possible rhomboid flaps with different orientations around a recipient defect. All the lines are equal in length and all the angles are either 60° or 120°. The leading edge of each flap is based on the lateral extension of a line joining the two 120° angles. Considerations of skin tension and scar position determine which one is selected.

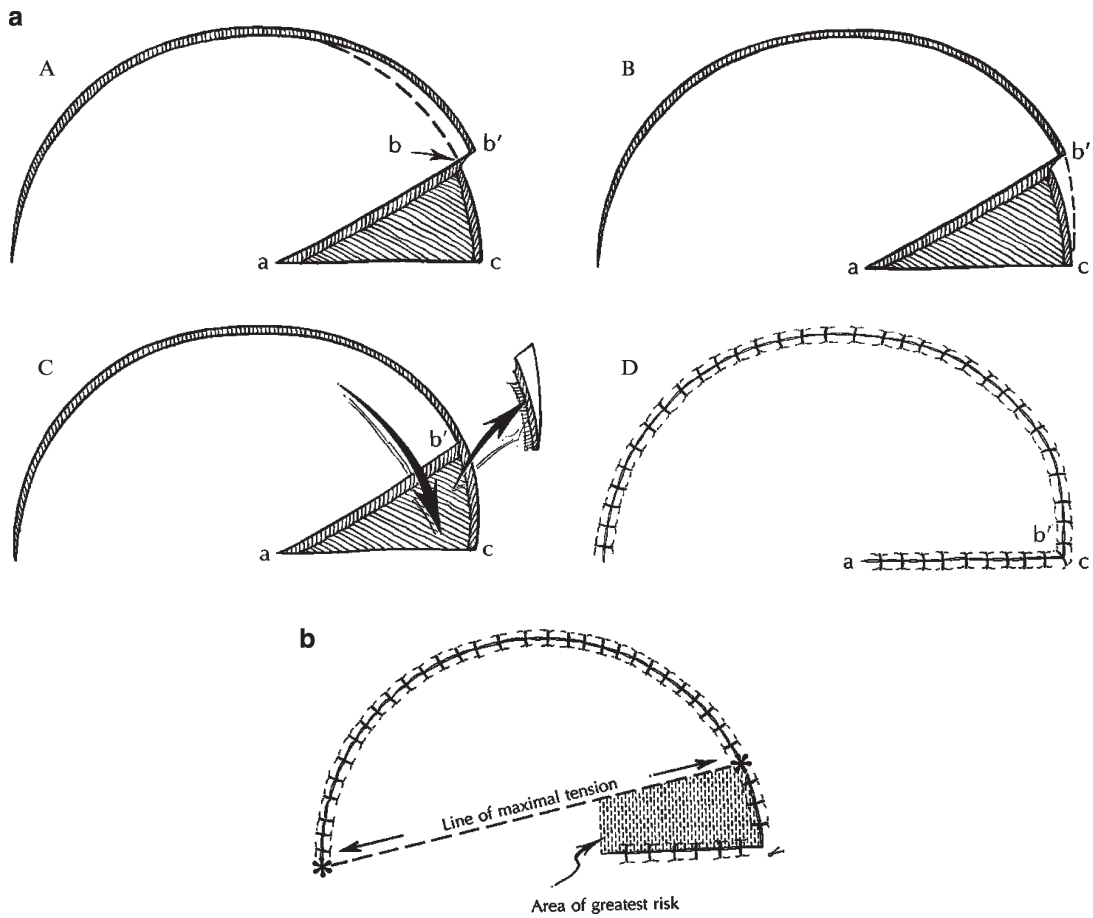


Figure 7.14. Rotation flap. (From Jackson.[2])

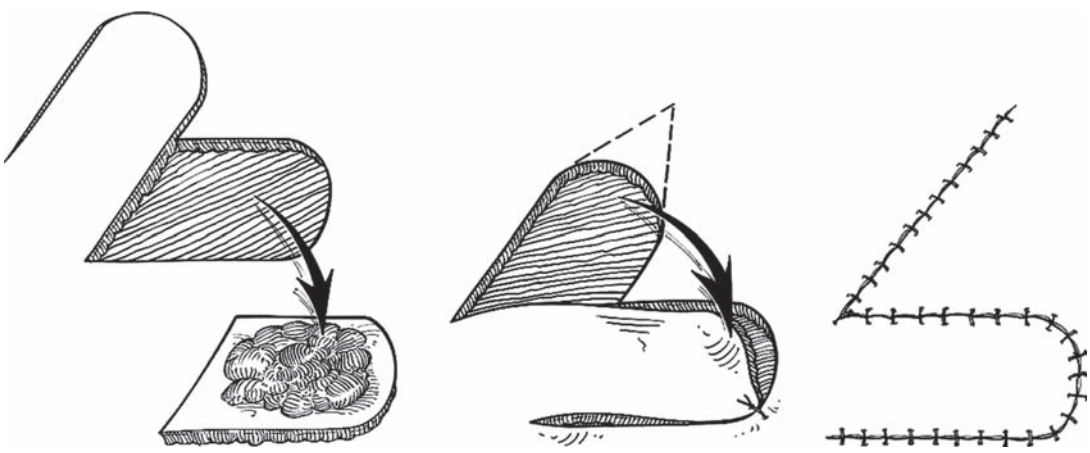


Figure 7.15. Transposition flap. (From Jackson.[2])

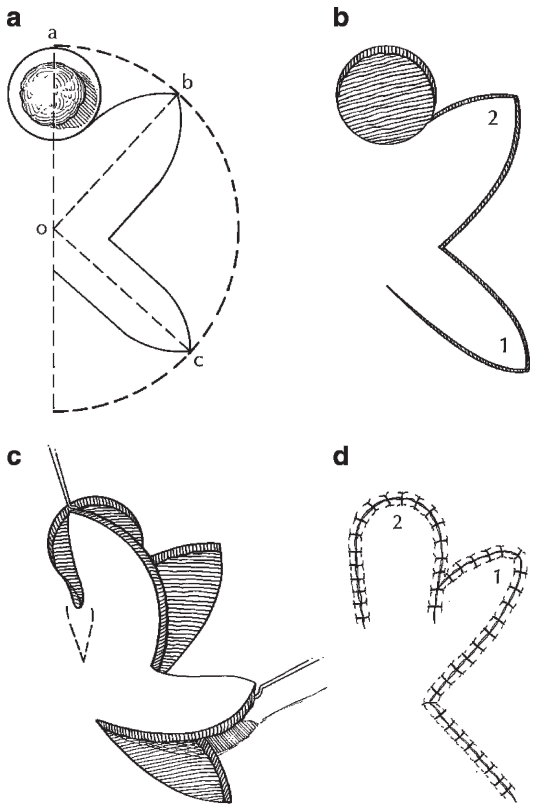


Figure 7.16. Bilobed flap. (From Jackson.[2])

Island Flap

This flap derives its blood supply from a subcutaneous connective tissue pedicle that is tunneled under intervening skin (Figure. 7.19). This is most safely performed with a named (axial) vessel in the supporting tissue.

Interpolation Flap

An interpolation flap is similar to an island flap, the difference being that the supporting pedicle crosses over the top of intervening tissue. Typically, the pedicle is divided when the flap is inset a few weeks later. By the time, neovascularization from the recipient site will sustain it.

Z-Plasty

Z-plasties involve the creation of two transposition flaps that are interdigitated with each other (Figure 7.20). The flaps are cut with identical line length and angles to allow precise closure. Z-plasties are typically used when increased length is required along a line, such as in a scar contracture. The center line of the Z-plasty is placed along the scar, since it is this component that will be lengthened. The angles of the Z-plasty limbs relate to a theoretical gain in length according to Table 7.3.

Table 7.3. Theoretical gain in Length from Z-plasty.

Z-plasty: Theoretical gain in length resulting from various limb angles	
Angle of each lateral limb (degrees)	**Theoretical gain in central limb length (as a % increase in wound length)**
30	25
45	50
60	75
75	100
90	120

Axial Pattern Flaps

Manchot[4] is usually credited with the first detailed description and modern understanding of the blood supply of the skin. Taylor[7] has shown that the entire skin is divided into territories, each of which is supplied by specific named vessels or groups of vessels. Each of these areas is termed an angiosome.

Axial pattern flaps are based on a single named vessel or vessels and provide a relatively predictable blood supply. The transfer of large amounts of tissues is possible, as the tissue transferred belongs to one specific angiosome and is supplied directly by a single vascular network. Taylor states that a flap elevated on a single vascular pedicle can recruit, at the most, an adjacent angiosome as well.

Fasciocutaneous Flaps

Fasciocutaneous flaps contain skin, fascia, and a named blood vessel. They allow the transfer of a thin flap of tissue for coverage of defects such as those of the nasal tip (e.g., forehead flap) or the extremity (e.g., reverse radial forearm flap, reverse sural artery flap).

Cormack and Lamberty devised a system (Figure 7.21) for classifying fasciocutaneous flaps.

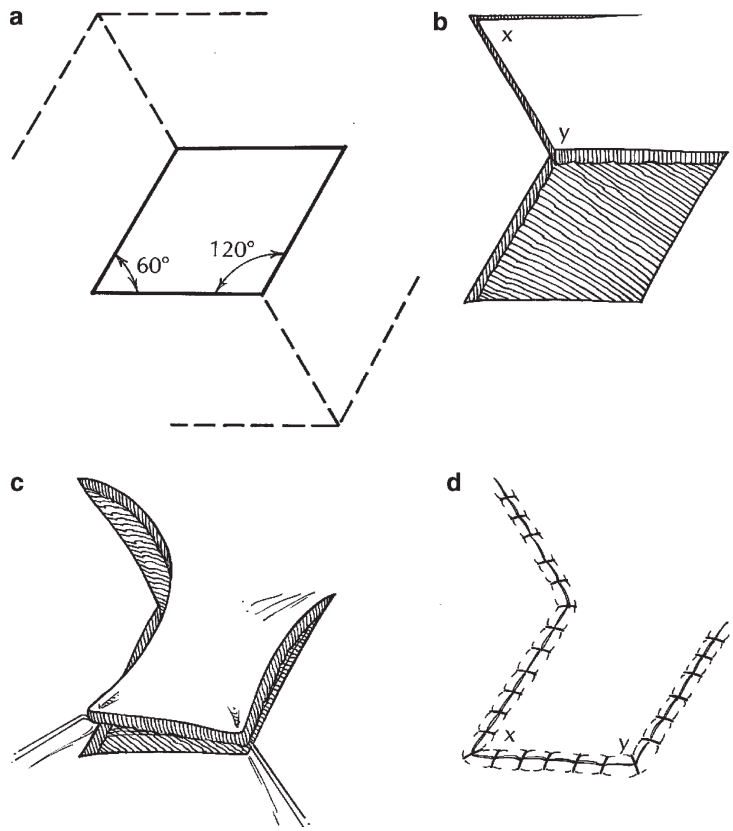


Figure 7.17. Rhomboid (Limberg) flap. (From Jackson.[2])

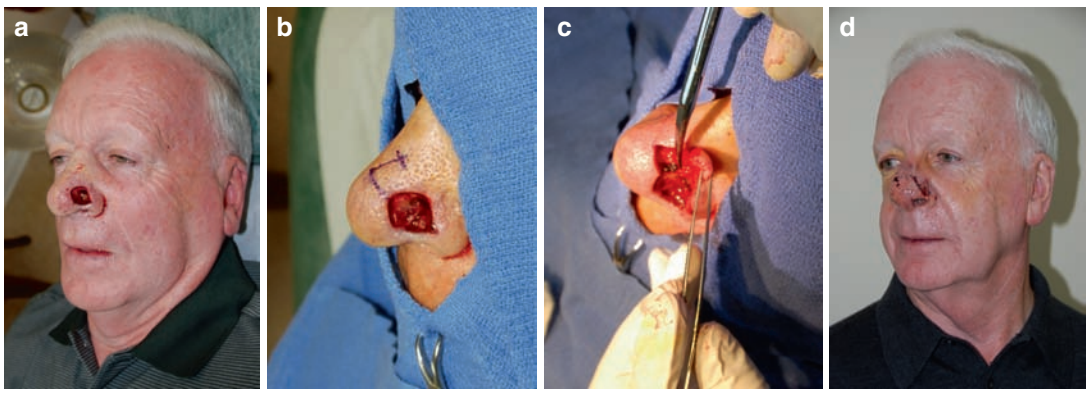


Figure 7.18. Rhomboid (Limberg) flap. (**a**), (**b**) Defect from excision of basal cell carcinoma. (**c**) Flap elevation. (**d**) Flap in position.

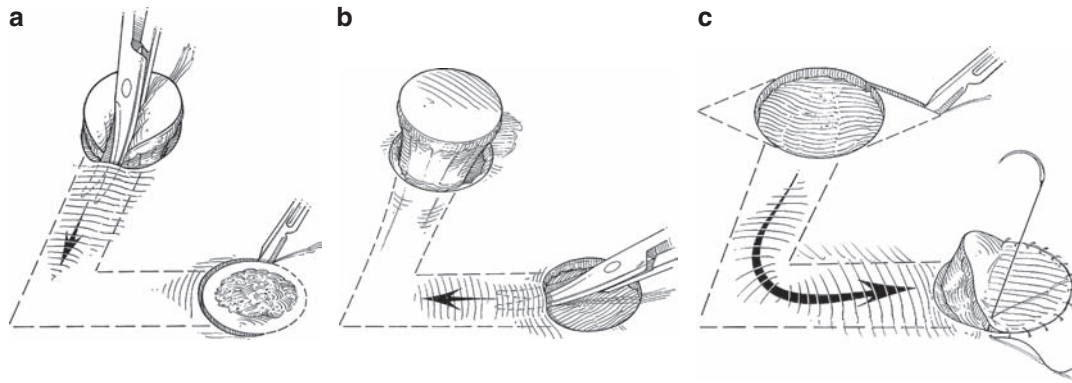


Figure 7.19. Island flap. (From Jackson.[2])

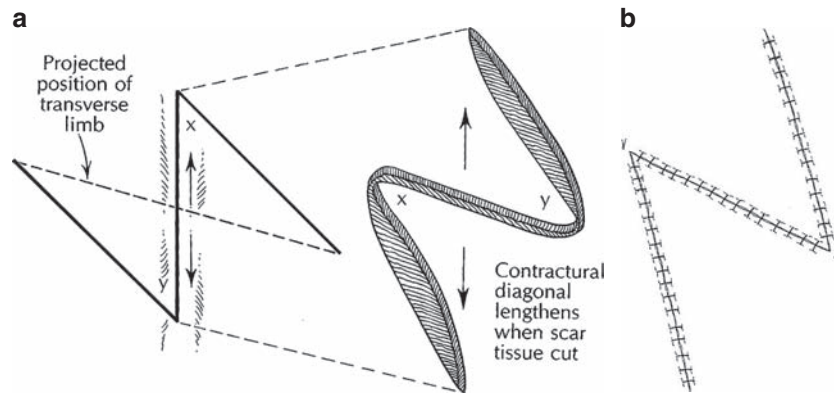


Figure 7.20. Z-plasty. (From Jackson.[2])

Paramedian Forehead Flap

The Paramedian forehead flap is a fasciocutaneous flap useful for covering nasal defects. It involves the use of skin, underlying subcutaneous fat, and the frontalis muscle and is based on the supratrochlear vessels.

Nasolabial Flap

Pedicled flaps taken from the nasolabial crease are often used to cover defects of the nose and nasal ala (Figure 7.22).

Reverse Sural Artery Flap

A distally pedicled flap based on reverse flow through the sural artery; the reverse sural flap offers a local flap alternative to a microvascular free flap for coverage of the distal third of the lower extremity. Advantages include ease of harvest and disadvantages include possible limitations of flap inset due to tethering of the supporting pedicle and increased likelihood of venous congestion (Figure 7.23).

(Reverse) Radial Forearm Pedicled Flap

Skin and fascia of the volar forearm, based on direct perforators from the radial artery, may be used to resurface portions of the forearm and elbow (Figure 7.24). This flap is more commonly taken as a free flap for defects such as those found in the head and neck after tumor resection.

Groin Flap

Skin and fascia from the groin area based on the superficial circumflex iliac artery can be used to cover difficult wounds of the hand and distal

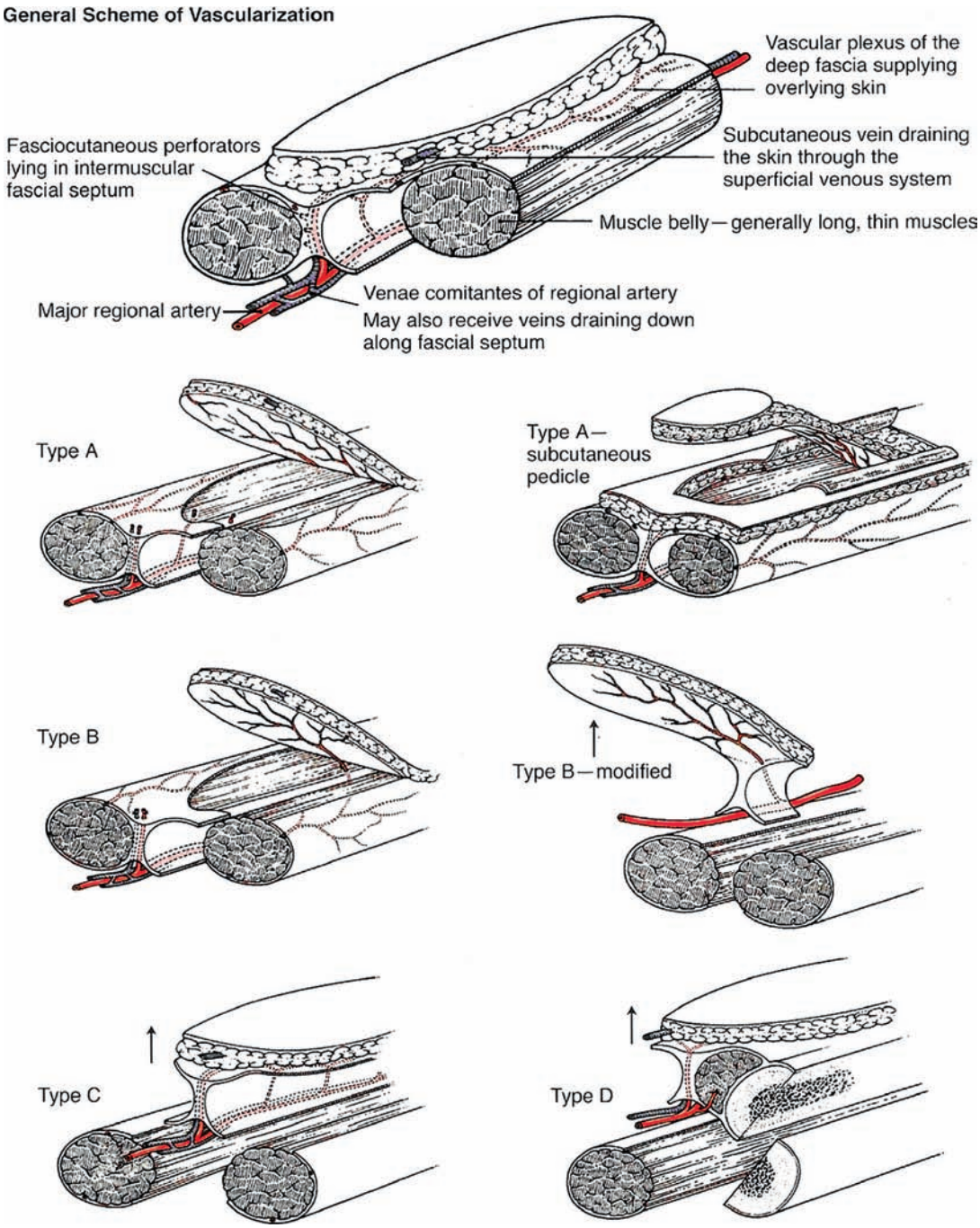


Figure 7.21. Cormack and Lamberty classification system for fasciocutaneous flaps. (From Cormack and Lamberty.[1])

Figure 7.22. Nasolabial flap. (**a**), (**b**) Basal cell carcinoma of left nasal ala. (**c**) Defect after tumor resection. (**d**) Placement of auricular cartilage scaffold. (**e**) Nasolabial flap. (**f**), (**g**) Defect 3 months after surgery.

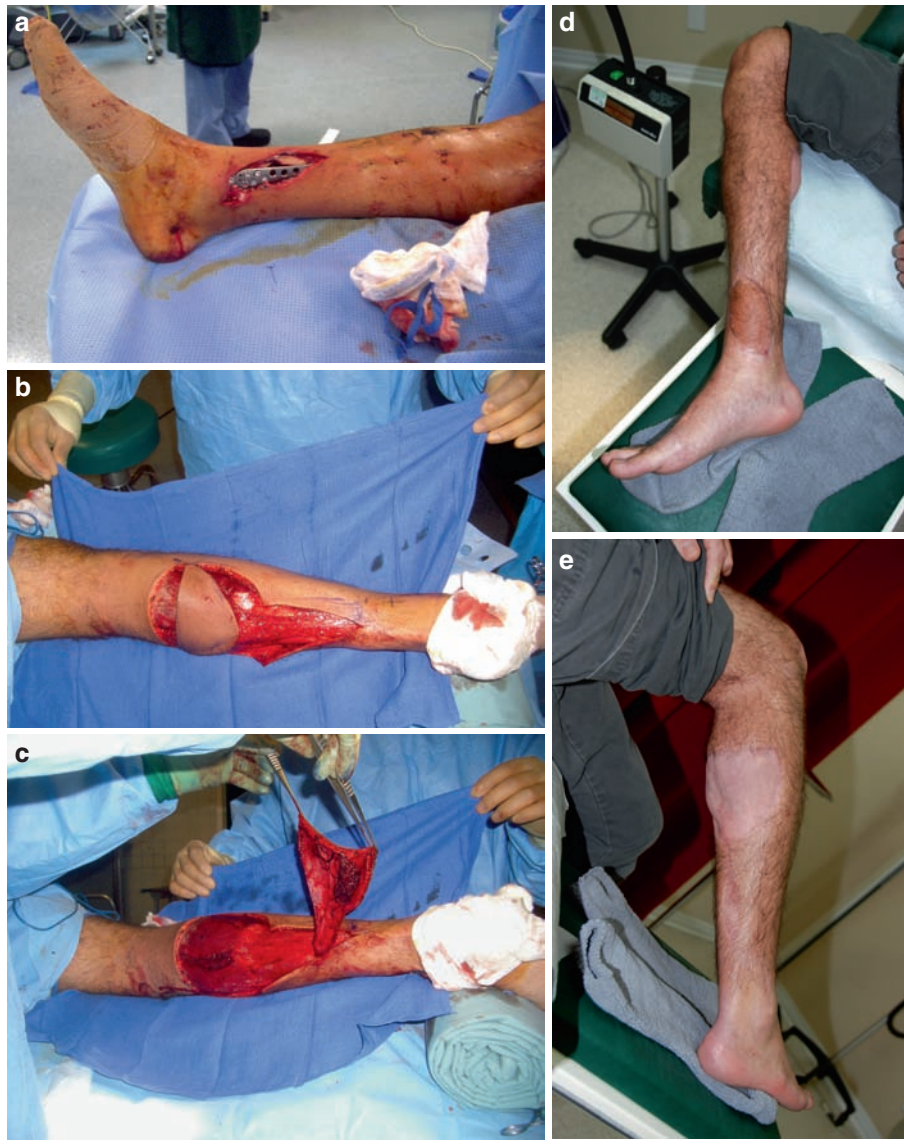


Figure 7.23. Reverse sural artery flap. The reverse sural artery flap for coverage of an open ankle fracture with exposed orthopedic hardware. (**a**) Initial presentation. (**b**), (**c**) Reverse sural artery flap. (**d**), (**e**) Flap 5 months after surgery.

upper extremity (Figure 7.25). This flap is often a "lifeboat" flap to be used when other options are not available.

Cross Finger Flap

Finger flap commonly used to cover defects on an adjacent finger with exposed bone or tendon. Tissue from an adjacent finger dorsum is pedicled to the volar defect and the pedicle divided after 12–15 days (Figure 7.26).

Moberg Flap

Flap of volar tissue advanced distally used to cover defects of the volar thumb tip (Figure 7.26).

Bipedicled Flap

A fasciocutaneous flap elevated over an easily covered area, such as muscle, with pedicled attachments at both ends and translated sideways to cover a defect. A skin graft used to cover the donor

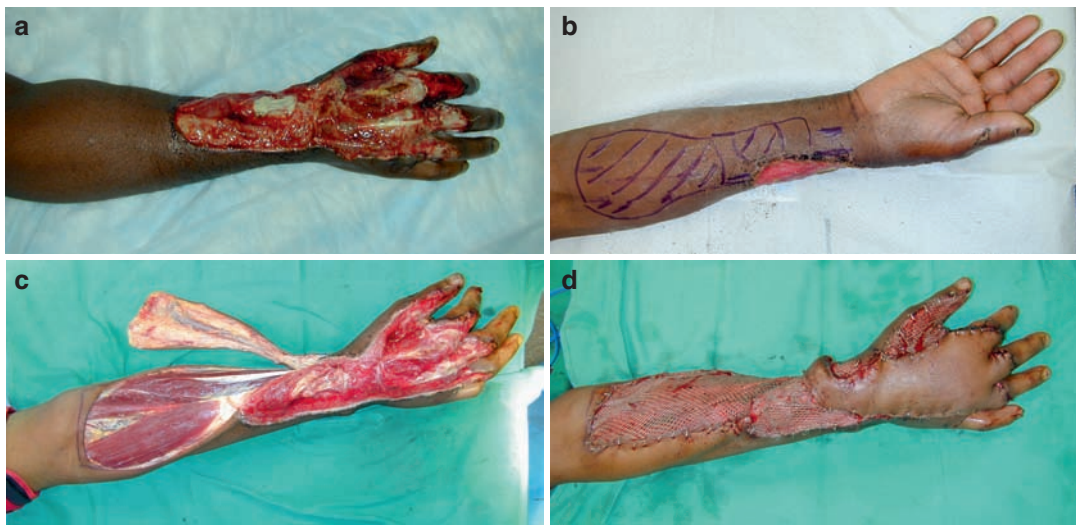


Figure 7.24. Reverse radial forearm flap: (**a**) Exposed tendons after MVA. (**b**) Flap marking. (**c**) Flap elevated. (**d**) Flap in place with split-thickness skin graft on donor area and graftable areas.

site. This is sometimes used in low-velocity lower-extremity wound coverage (Figure 7.27).

Cross-Leg Flap

An older type of flap once commonly used for coverage of distal lower-extremity defects before the advent of microsurgery and free flaps. A fasciocutaneous flap from the opposite leg is raised on a pedicle and used to cover the defect. The pedicle is divided approximately 2 weeks later.

Deltopectoral Flap

A now rarely used fasciocutaneous flap taken from the area overlying the pectoralis major and deltoid and based on the second and third intercostal perforators of the internal mammary artery. The flap is based medially and its distal end is used for head and neck reconstruction. It is an interpolated flap; its pedicle is divided 2–3 weeks after initial transfer.

Muscle and Musculocutaneous Flaps

Muscle and surrounding tissue may also be transferred as an axial pattern soft tissue flap. Musculocutaneous flaps still make up the majority of the pedicled, axial pattern flaps used today. They are typically employed to provide robust vascular coverage to a defect often lacking adequate blood supply as well as tissue. Examples include the transverse rectus abdominis musculocutaneous (TRAM) flap, the latissimus dorsi flap, and the pectoralis major flap. Musculocutaneous and muscle flaps may be classified according to the pattern of blood supply of the muscle concerned (Figure 7.28).

The advantage of such flaps is the speed and ease of flap harvest and their generally high reliability. The obvious limitation of these flaps is their tethered vascular pedicle, which limits their reach, and loss of function due to muscle sacrifice. In addition, the vascular pedicle may be subject to compression or kinking, resulting in interruption of the blood supply if compressed under adjacent tissue or if twisted during flap inset at the recipient site.

Pectoralis Major Flap

Pectoralis major flap is used rarely but is based on the thoracoacromial pedicle and can provide coverage for sternal, truncal, and head and neck defects. A skin paddle, supported by cutaneous perforators from the main pedicle, may also be harvested to provide epithelial coverage as needed (Figure 7.29).

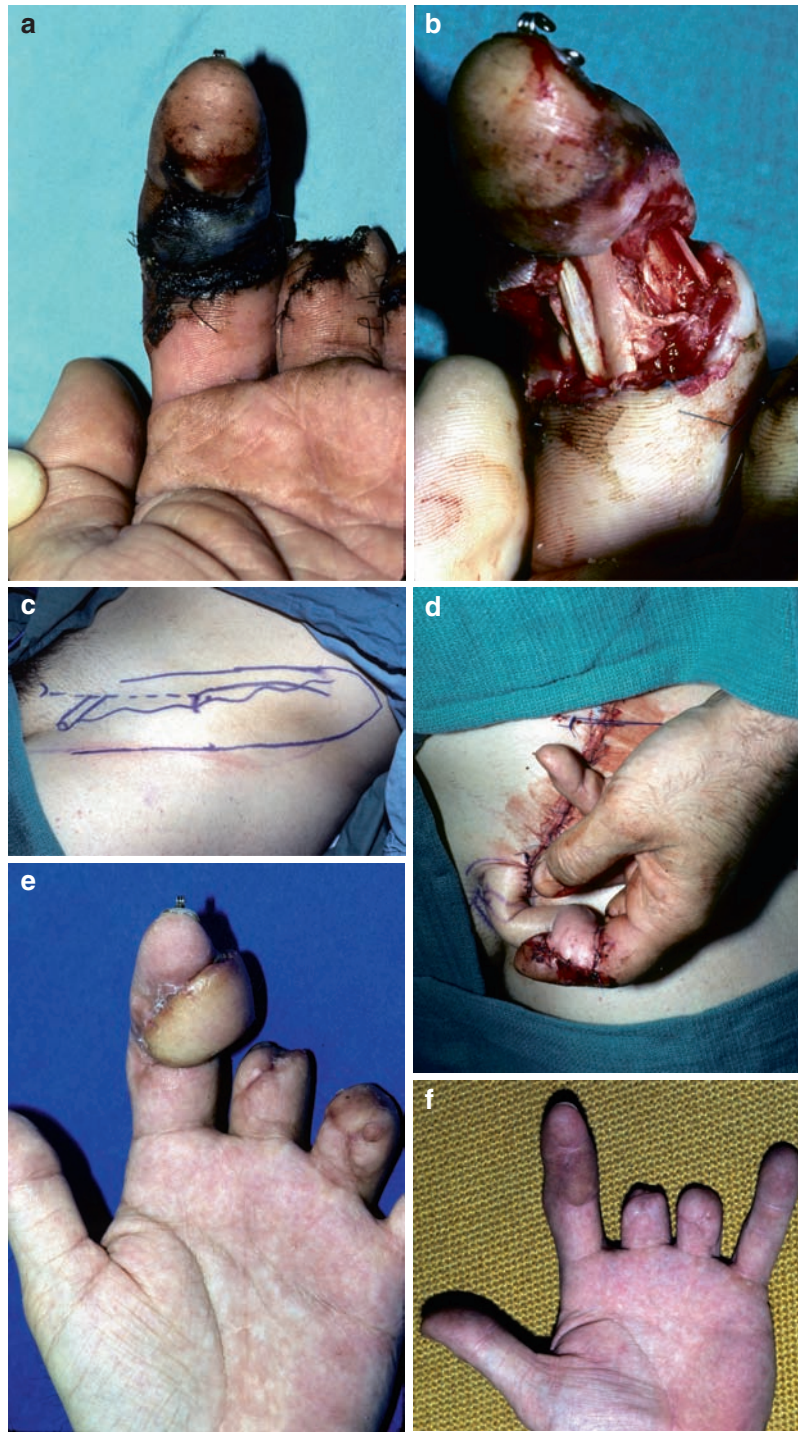


Figure 7.25. Groin flap. (**a**) Complete skin necrosis leaving exposed nerve and tendon. (**b**) Groin flap markings. (**c**) Tubed groin flap. (**d**) Groin flap after pedicle division. (**e**) Flap after revision.

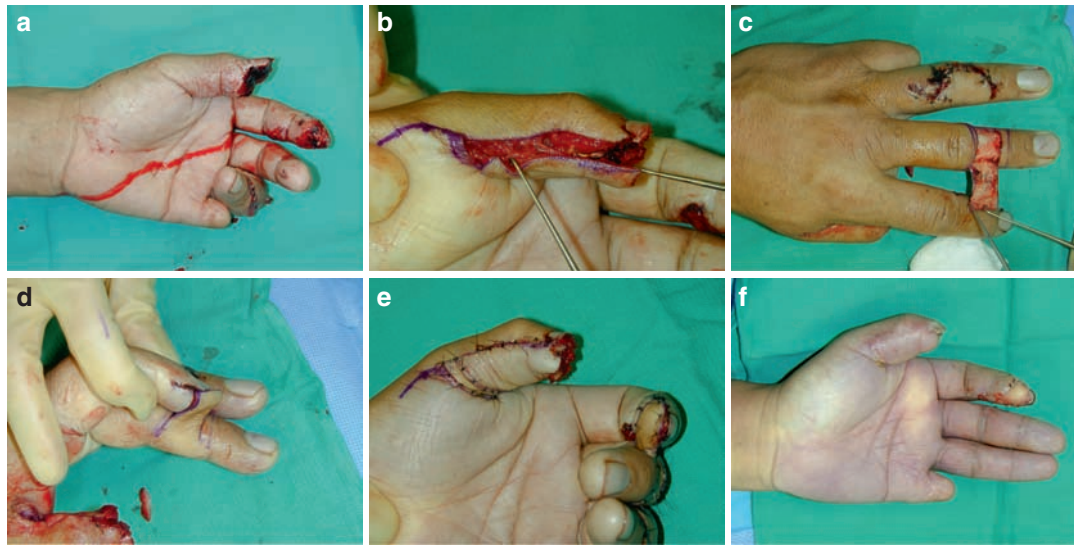


Figure 7.26. Fingertip defects covered with Moberg and cross finger flaps. (**a**) Initial defects of left thumb tip and index finger tip. (**b**) Elevation of cross finger flap. (**c**) Elevation of Moberg flap. (**d**) Inset of cross finger flap. (**e**) Inset of Moberg flap. (**f**) Result after division of cross finger flap and healing of wounds.

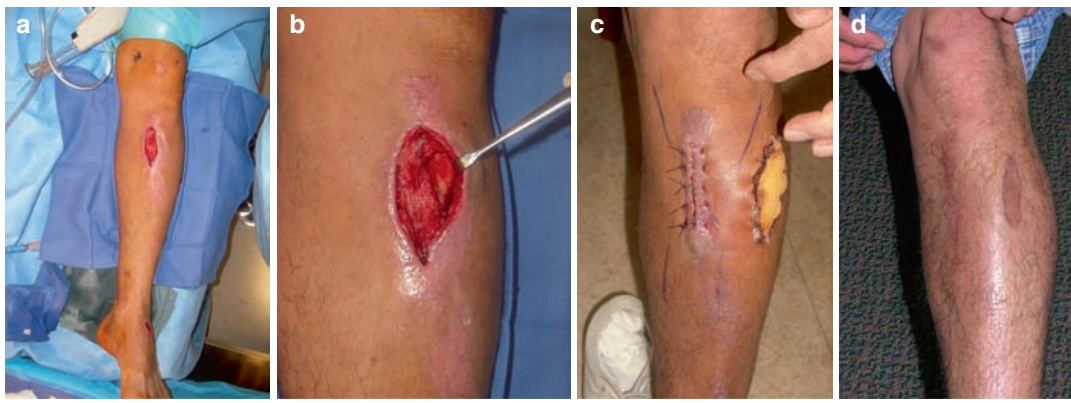


Figure 7.27. Bipedicled flap used to cover pretibial defect. (**a**), (**b**) Open pretibial wound. (**c**) Initial appearance 1 week after surgery. (**d**) Appearance 4 months after surgery.

Pedicled TRAM Flap

First described by Hartrampf, the pedicled TRAM flap is chiefly used for breast reconstruction by many community physicians. The rectus abdominis muscle is used as a carrier for the deep superior epigastric vessels. These vessels provide the blood supply to the abdominal skin and fat that are used to shape a new breast mound. This is an example of a single vessel carrying not only its own vascular territory but also the next one along. It provides a simple and reliable option for breast reconstruction but has a risk of fat necrosis and healing problems within the flap and a significant incidence of abdominal weakness and hernia (Figure 7.30).

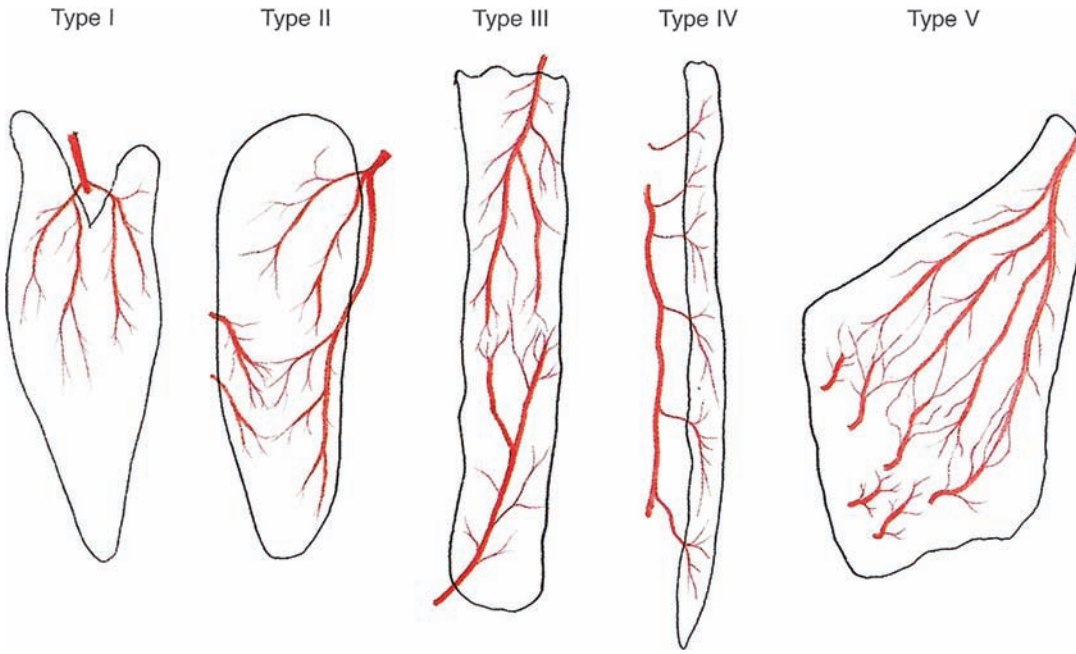


Figure 7.28. Mathes and Nahai classification of blood supply to muscle flaps. This figure was published in Mathes and Nahai[5(p. 41)]. (Copyright Elsevier, 1979. Reprinted with permission.)

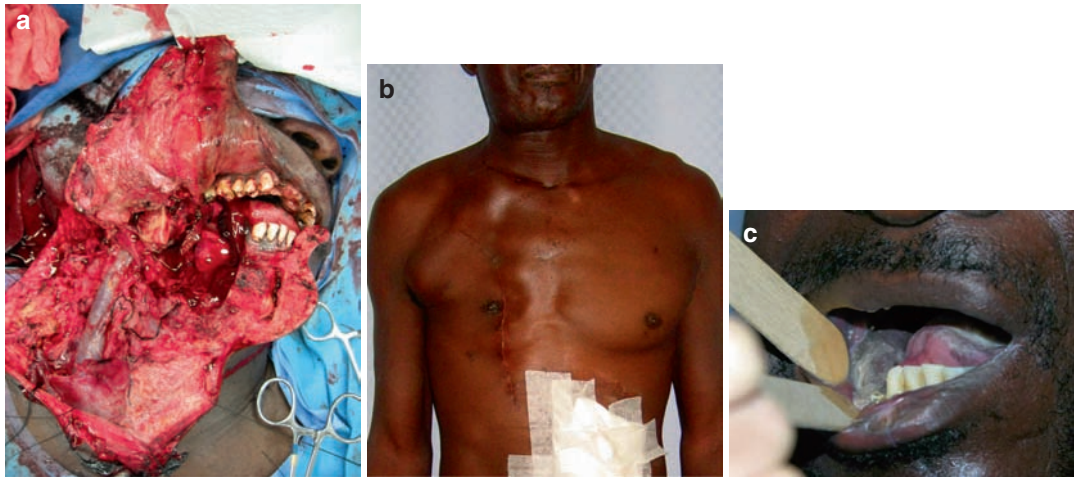
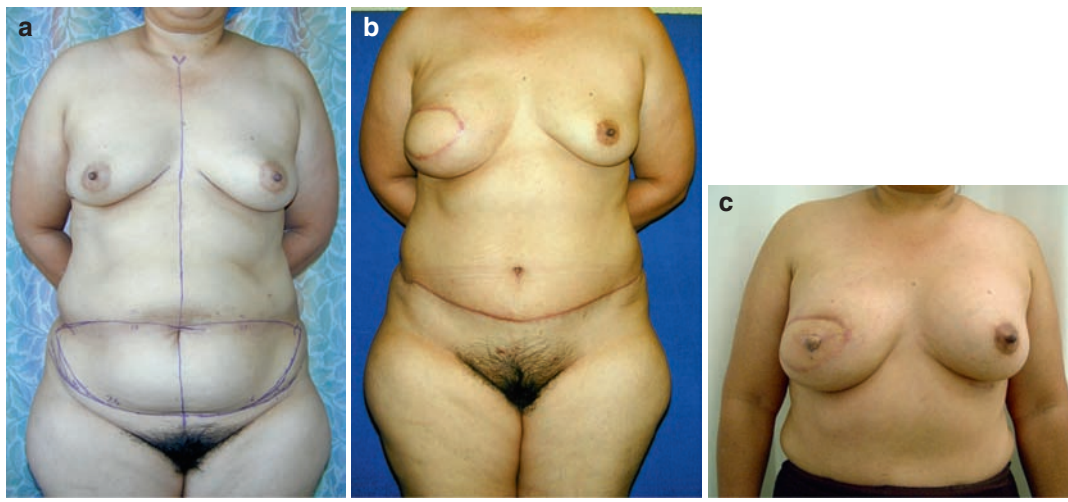


Figure 7.29. Pectoralis major musculocutaneous flap. (**a**) Defect following primary oral cancer resection including segmental mandibulectomy and neck dissection. (**b**) Postoperative appearance of chest wall donor site. (**c**) Postoperative appearance of oral skin paddle.

Figure 7.30. Pedicled TRAM flap. (**a**) Preoperative markings. (**b**) Postoperative appearance after initial surgery. (**c**) Appearance after nipple



reconstruction.

VRAM Flap

A variation of the pedicled TRAM flap, the VRAM flap uses the rectus abdominis muscle based on either the superior epigastric or deep inferior epigastric vessels – with or without an overlying skin paddle – for coverage of defects of the sternum, chest wall, or of the pelvic and perineal areas.

Latissimus Dorsi Flap

The Latissimus dorsi flap is often used for breast reconstruction. This reliable flap can be harvested from its native location on the back of the chest wall, pedicled on the thoracodorsal vessels entering the muscle in the axilla and tunneled to the recipient anteriorly. When used from breast reconstruction, an implant is often needed to supply adequate volume to reconstruct the appearance of the native breast mound.

Conclusion

In conclusion, both grafts and flaps are time-honored, safe, and reliable methods for soft tissue transfer and are integral to the armamentarium of any plastic surgeon. They are relatively simple and quick to perform and are used in the overwhelming majority of cases requiring wound coverage. Modern methods of microvascular surgery and free tissue transfer have supplanted pedicled flaps and grafts mostly in the most difficult wounds such as the areas of head and neck cancer reconstruction, distal extremity reconstruction, and increasingly in advanced autologous breast reconstruction.

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8

Microsurgical Techniques

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Summary

Although relatively young as a surgical discipline, microsurgery has become a critical part of the plastic and reconstructive surgeon's armamentarium over the last century. Familiarity with basic microsurgical instrumentation and technique, a sound understanding of indications for free tissue transfer, and judicious flap selection are paramount. Thoughtful operative planning and meticulous execution are essential to successful microsurgery. Careful postoperative flap monitoring as well as selective use of pharmacologic adjuncts, such as thrombolytics, heparin, aspirin, and dextran helps to ensure good outcomes. When flap viability is uncertain, reoperation and flap revision are required. Now routinely performed in most major medical centers, microsurgery has an exciting future due to the increasing demand and growing list of indications for these complex interventions.

Introduction

No chapter on microsurgery can begin without considering its relatively brief but dense history. Although microsurgery dates back to early 1921, when Nylen used a microscope for ear surgery, microvascular surgery did not begin in earnest until technological advances in equipment allowed small vessel re-approximation, first performed by Jacobsen and Suarez[1,2] in early 1960. Multiple reports of microsurgical replantation of severed body parts followed. With the success of these procedures, microscopes, suture material, instruments, and techniques all improved dramatically. This era of research and development culminated in the first free tissue transfer being performed in 1972 by McClean and Buncke[3] who transferred omentum for the coverage of a scalp defect.

The 1970s saw the expansion of donor site choices to meet the many different needs of reconstruction: toe to hand transfers, skin flap transfers, muscle transfers, bone transfers, as well as a combination of all the above. By the 1980s, microvascular surgery became a routine part of the plastic surgery armamentarium in many major medical centers. In fact, reconstructive microsurgery has now become a requisite and integral part of plastic surgery training.

Basic Instrumentation and Techniques

Optimal training requires the use of a microvascular laboratory in which techniques of microvascular surgery can be mastered using animals before performing these procedures on patients. The skills that must be mastered include becoming familiar with the operating microscope as well as

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the basic instrumentations, including micro needle holders, jeweler's forceps, and micro dissecting scissors to perform the microvascular anastomosis (Figure 8.1). Another important instrument, which aids in performing the microvascular anastomosis is a single or double microvascular clamp. The double clamp can be used to align and approximate the vessels to each other for easier placement of sutures while maintaining hemostasis, stability, and a tension-free, even vessel orientation. Clamps used for microvascular surgery should not exceed 30 g/mm^2 of pressure to avoid tissue damage of the intima and media.[4]

When using the operating microscope, choosing the appropriate level of magnification is crucial to efficient maneuvering and performance of the anastomosis. Magnification can be broken down into three basic levels. Low magnification (6× to 12×) can be used for vessel preparation and suture tying, middle magnification (10× to 15×) can be used for placement of the suture, and higher magnifications are used for performing small caliber vessel anastomosis and inspection of the anastomosis at the completion of the procedure. In certain cases, high-powered (5× to 6×) loupe magnification has also been used with great success for microvascular anastomosis of a vessel greater than 1 mm in diameter.[5] Loupe magnification allows efficiency, portability, freedom of movement and cost confinement in comparison with the use of a large operating microscope.

Understanding the use of appropriate suture material and needle for different vessel sizes is crucial to obtain a perfect anastomosis. Most commonly, free flaps have vessel diameters ranging in size from 1 to 3 mm. Those vessels of approximately 1 mm are best closed with 9–0 to 10–0 sutures, whereas those closer to the 3 mm range can be re-approximated with either an 8–0 or 9–0 suture. Appropriate needle choice is important to avoid unnecessary vessel injury and suture holes during the anastomosis.

Another method used to perform an anastomosis involves a coupling device (Figure 8.2). When using this technique, each of the vessel ends is brought through a polyethylene ring-pin

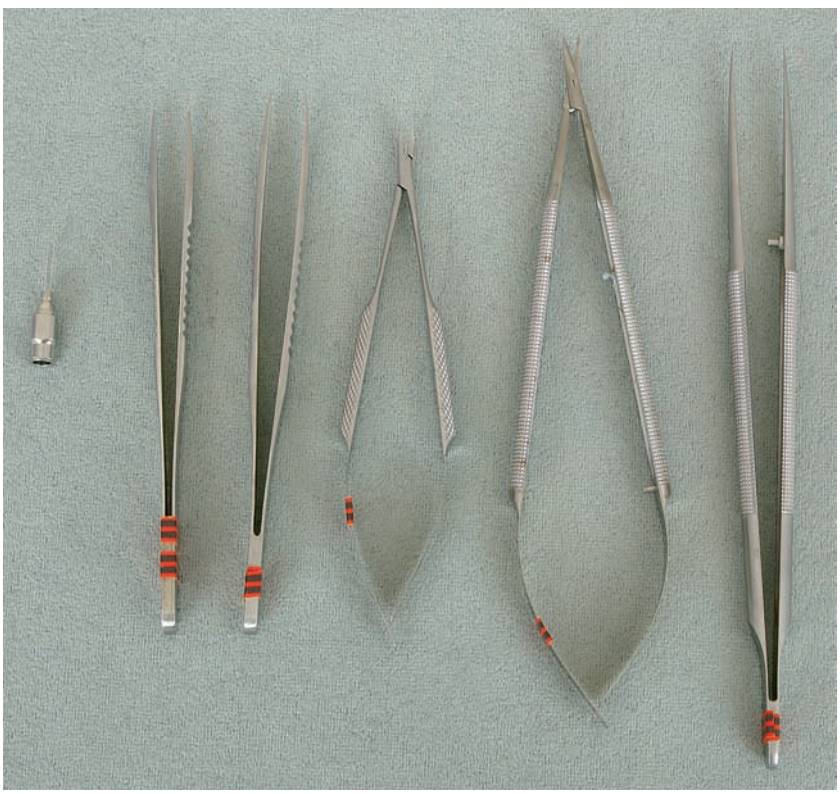

Figure 8.1. Basic microsurgical instrumentation, including irrigating catheter, jeweler forceps, micro needle driver, and micro scissors.

Figure 8.2. A microvascular coupling device, such as the one shown here, may be used to facilitate creation of the vascular anastomosis.

device, and the edges of the vessels are evenly secured through the pins. Once both sides of the vessel ends are secured, the two rings are then brought together with the aid of the coupling approximator device. Vessels suitable for this coupling technique are between 1.5 and 3.0 mm in diameter. Although the coupler has been used most frequently for venous anastomosis with great success, there have been recent reports of use of this device for arterial anastomosis in ideal and specific circumstances.[6,7]

In preparing to perform a microsurgical anastomosis, both the operating surgeon and the assistant need to position themselves comfortably, either sitting down or standing up. Most importantly, the hands and wrists of the operating surgeons have to rest adjacent to the operative field. If necessary, they may be supported with surgical towels. This avoids early fatigue of the operating surgeon's hands and wrists and ensures minimization of tremors.

Before placement of sutures, the surgical field should be arranged to optimize the conditions for successful anastomosis. If the vessels are located deep in the wound, placing a surgical sponge or instrument wipe will bring the vessels closer to the surface. Placing a background (e.g. plastic sheet) in addition to the sponge will keep the vessels clear from the surrounding debris and easily visualized during the placement of sutures. To keep the operative field relatively dry from excessive irrigating fluid or other tissue fluid, a small suction catheter can be placed under the surgical sponge mentioned above. This suction catheter can be fashioned from a small pediatric feeding tube connected to a small Frazier tip suction (Figure 8.3).

Before performing the anastomosis, the end of the vessels should be cleanly cut and excess adventitia should be removed using sharp scissors and fine forceps. Vessel ends can also be dilated using a specialized blunt tip, vessel dilating forceps to assist in size matching the vessel ends to be anastomosed. Over-dilation of the vessels is not recommended as it can damage the intima. Heparin irrigation at a concentration of 5,000 U in 100 ml of LR or saline is then used to wash out both vessel lumens and their edges. Prior to the anastomosis, a final inspection of the luminal intima has to be performed to identify any luminal irregularities or intimal damage that might cause turbulent flow and thrombogenicity. To stabilize the vessels for anastomosis, a double clamp is applied as mentioned above. Prior to the application of the clamps, administration of a single bolus of heparin between 2,000 and 5,000 U has been recommended to improve patency.

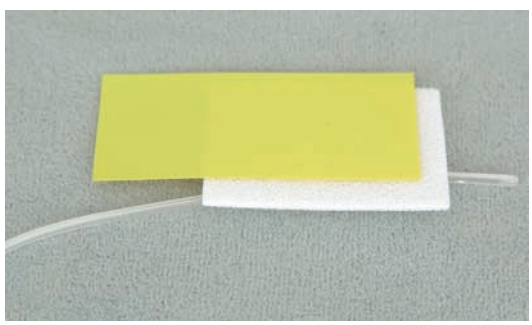

Figure 8.3. Preparation of the surgical field may include the use of a sponge and background material to facilitate vessel sewing and small catheter suction tubing to keep the field free of fluid.

In placing the sutures, one can grasp the adventitial tissue near the anastomosis site with forceps or support the inner lumen of the vessel with the forceps. The needle is then passed perpendicular to the surface of the vessel wall (Figure 8.4). It is important to follow the curvature of the needle while driving it through the vessel wall in order to avoid inadvertent tears and unnecessary injury to the vessel itself, especially if the vessel is previously irradiated, fragile, or calcified. For better accuracy, it is wise to pull through the needle and suture almost completely prior to the placement of the needle into the inner lining of the second vessel wall. Bites should be taken approximately 0.2–0.3 mm from the vessel ends and should be equal on both sides of the anastomosis. Before tying the knot, pull the suture through until there is only a short segment of the loose suture end, just enough to make a knot; too long of a suture end makes tying a knot cumbersome and difficult. In making the first knot, grasp the long end of the suture with the forceps and make a double loop over the needle holder (Figure 8.5). Then grasp

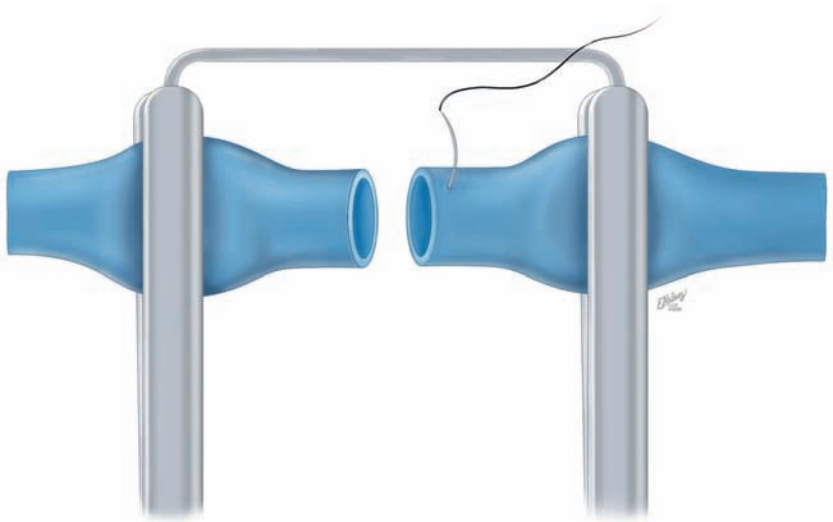

Figure 8.4. The needle is passed perpendicular to the vessel wall to minimize trauma to the vessel.

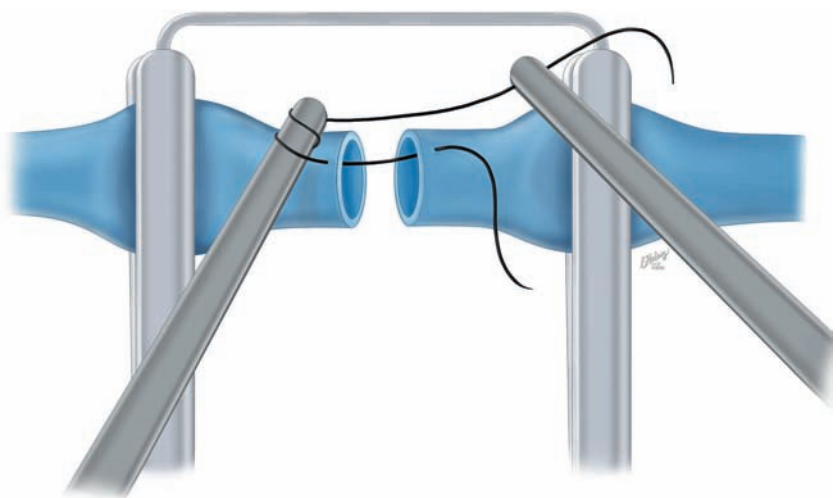

Figure 8.5. The first throw of the knot is performed by looping the long end of the suture twice around the forceps, then grasping the short end, and gently laying down the knot.

the short loose end of the suture and pull it through to make a knot. This double knot is also known as a surgeon's knot and is very useful if slight tension exists between the two approximating vessels. After this first knot, subsequent knots can be placed by using a single-loop technique (Figure 8.6). It is important that each knot be squared in opposite directions for secure tying. Usually, three to four knots are recommended to complete the tie.

The second suture is placed approximately 180° from the first one (Figure 8.7). This maneuver equally distributes tension on the vessel ends and brings the anterior half of the anastomosis into position for sewing. Subsequent sutures are placed by sequentially bisecting the remaining distance between two sutures, paying attention to precise suture placement and maintaining equal distance between each stitch. Once one side of the vessel wall is completed, then the other side of the vessel is completed in similar manner by flipping the double clamp that holds the vessels.

Before the completion of the anastomosis, it is important to irrigate the lumen of the vessel with heparin solution to remove any

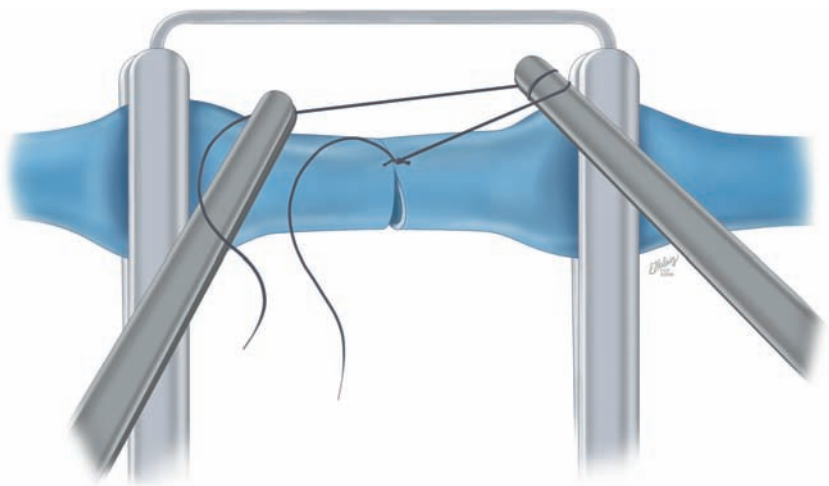

Figure 8.6. Subsequent knots are tied using single throws around the forceps.

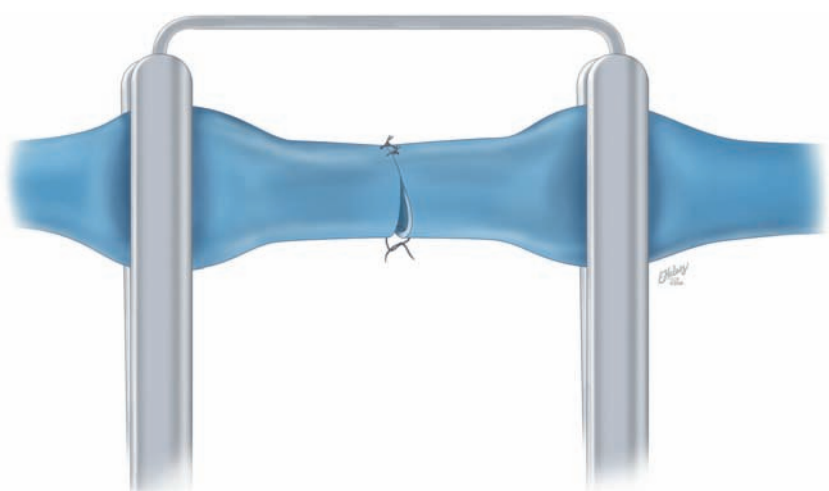

Figure 8.7. Placement of the second suture should be approximately 180° from the first one.

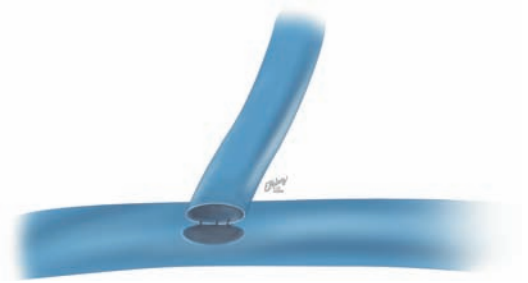

Figure 8.8. An end-to-side anastomosis.

debris left in the lumen and to inspect for any "back-walling" during the anastomosis. Once the anastomosis is completed, the clamps are gently removed to establish flow through the patent anastomosis.

The most common type of microsurgical anastomosis is the end-to-end anastomosis. This is usually performed when the vessels are equal in caliber. For vessel discrepancies greater than 2:1 ratio, an end-to-side anastomosis is preferable to avoid excessive turbulent flow and thrombogenicity. An end-to-side anastomosis is also performed when there is a significant size discrepancy between vessels (greater than 2:1) involving single, vital vessels that cannot be sacrificed.

In performing an end-to-side anastomosis, a small area from the side of a larger vessel is removed. For arterial anastomoses, first remove the adventitial layer with forceps and scissors by a gentle pulling of the tissue. Then carefully create an oval-shaped opening that is similar in size to that of the other vessel to be approximated (Figure 8.8). If the donor vessel size is small, the luminal size diameter can be enlarged by cutting its end obliquely or forming a funnel (Carrel's patch) at a branching point.

Indications for Free Tissue Transfer

The decision to use microvascular tissue transfer to solve a reconstructive problem is based on numerous considerations. Its best indication, however, is when no other type of reconstruction can solve the problem at hand. One example of this would be a complex wound of the distal third of the tibia where no local flaps can adequately cover the defect.

Although the principle of "reconstructive ladder" has been well-regarded and respected for many years, the time has come to use a "reconstructive elevator" (L. Gottlieb, 2002, personal communication).

Performing a microvascular procedure, which used to be regarded as complex surgery, has become the less complex alternative to many of the surgeries done in the past. As an example, consider the reconstructive problem of providing soft tissue coverage to the dorsum of the hand. One popular and long-accepted technique for addressing this problem involves use of the pedicled groin flap. This procedure requires sewing the flap into the defect and prolonged immobilization of the arm while the flap is vascularized by the recipient site. A subsequent procedure is then required to divide the flap once new blood vessels at the recipient site have been established. By choosing this flap, the patient is committed to at least two surgeries as well as prolonged and inconvenient arm immobilization. Alternatively, free tissue transfer using a lateral arm flap or other fasciocutaneous flap can safely accomplish wound coverage in a single operation with minimal donor-site morbidity.

Patient Considerations

In considering free tissue transfer, it is essential to take into account the patient's health condition. The patient has to be fit and appropriate to undergo a free flap procedure. Their chronological age is not necessarily the limiting factor as long as their health condition permits surgery. Free tissue transfers have been performed in children as well in elderly patients with great success.[8,9] However, basic criteria and principles of preoperative evaluations have to be respected for safety.

Although several studies have shown that smoking showed no significant difference in vessel patency, flap survival and re-operation rate in comparison to patients who do not smoke,[10] smoking remains a main contributor to wound complications in the donor site as well as the wound interface between the flap and recipient wound.[11]

As a result, for most surgeons, smoking continues to be a relative contraindication for

microsurgery reconstruction.[10,11] Its risk and potential complications have to be clearly explained to the patients who are to undergo such operations.

Evaluation of Defect and Flap Choice

There are almost as many flap choices as there are different types of tissue in the human body. Deciding which type of tissue to use to achieve a reconstructive goal is based on several factors. First and foremost the surgeon must identify the missing components of the defect. Is it only skin and fat? Does the deficit include missing function, bone or skeletal support? Will reconstruction require bulk or rather a thin and pliable coverage? What are the dimensions and how much volume is required? Once these questions have been considered, the specific decision regarding which tissue to transfer can be made. Mature planning of the reconstructive effort has to be thoroughly premeditated before the real execution. Important questions to consider include the following: will the flap pedicle length and caliber of vessels be sufficient? What is the likelihood of size matching between flap and recipient vessels? Will there be adequate length between the donor pedicle and the recipient vessels to reach the defect? Might an interpositional vein graft be necessary? Ultimately, the reconstruction must allow for a tension-free anastomosis.

The various tissue types used for microvascular transfer include muscle, skin and fat, fat alone, omentum, bone, jejunum, nerve, as well as composite toes to hands. Each of these tissue types has a specific advantage relating to the reconstructive goals.

The most commonly transferred tissues include skin and subcutaneous tissues. These can be further described based on their blood supply. An axial skin flap has a vessel traveling along the length of the flap that goes directly into the skin. The groin flap is an example of an axial skin flap. A fasciocutaneous flap is supplied by subcutaneous perforators that course between the muscles, with the radial forearm and lateral arm flaps being examples. Musculocutaneous flaps include the vessels that traverse the muscle coming from the blood supply to the muscle itself. Examples of this type of flap include latissimus dorsi including skin as well as the traditional TRAM free flaps. The final group is made up of a refinement of the musculocutaneous flap in which the perforators are dissected free of the muscle and only the skin, subcutaneous tissue and vessel is taken while leaving the muscle behind with preservation of its function. This has been categorized as perforator free flaps, which have been described more recently. The potential benefit of this type of free flap would be the minimization of the morbidity of the donor site. These types of flap can also include a nerve and be sensate, therefore possibly eliminating or decreasing a chance for injury to areas of the reconstructed site.

Another major group of free flaps are muscle only flaps. These flaps typically have a robust blood supply and conform well to irregular defects. In addition, muscle can be transferred as a functional flap in which the motor nerve is connected to a recipient nerve allowing restoration of function, such as using gracilis flap in facial reanimation.

Bone transferred as a free flap is another major category that is routinely used in microvascular reconstructions. These flaps can be used to replace deficits in support and form. Most commonly these flaps are needed after tumor resections, which often leave deficiencies in form and support. The harvest of these bone flaps can be combined with the adjacent tissue using the same or different types of pedicle as a composite tissue transfer.

Less commonly transferred tissues include jejunum, which has been used for laryngeal esophageal reconstructions of vascularized nerves to reconstruct brachial plexus for long nerve defects and omentum for numerous tissue deficits.

For a detailed description of the specific types of flaps including flap vascular supply and innervation, average vessel caliber and technique for flap elevation, the reader is encouraged to consult *Microvascular Surgery*: *Anatomy and Operative Techniques* by Strauch and Liang.[12]

Preoperative Planning

Once a specific flap is chosen for the reconstruction, there is extensive preoperative planning required before the surgery itself. On occasion, it

is necessary to obtain an arteriogram to define the vascular anatomy of the flap. This can be done with magnetic resonance arteriogram, computerized tomographic angiogram, Doppler sonography or traditional arteriogram.[13,14] These technologies have also been employed in evaluating the recipient vessels, particularly if there are known or suspected diseased or injured vessels.

In cases where reconstructive microsurgery is needed for an infected or traumatic open wound, preparation of the recipient site includes adequate debridement of the wound itself. If necessary, the main microsurgical reconstruction should be postponed until adequate control of the wound is achieved. Extensive knowledge of the vascular anatomy of the recipient site is necessary before reconstruction in a traumatic wound. The zone of injury needs to be taken into consideration with the anastomosis placed out of this zone.

Technical Considerations – Flap Elevation

Harvesting a free flap requires knowledge of the course of the vessels supplying the transferred tissue. Often this will require specific identification of the perforators, anatomic landmarks and the course of pertinent vessels. If available, preoperative mapping may be extremely beneficial. Such mapping, in many instances, will speed up the dissection and elevation of the tissue to be transferred. It is also important that in taking donor tissue the overall morbidity to the patient is taken into consideration. The benefits of using the tissue have to be weighed against the morbidity of taking that tissue. When possible, a two-team approach should also be considered to make efficient use of operating room (OR) time and minimize the length of general anesthetic time for the patient. A two-team approach allows for one surgical team to prepare the recipient site while the other team conducts the harvest. Donor- and recipient-site dissections can generally be performed under loop magnification. During the vascular anastomosis, a decision needs to be made as to whether it is appropriate to give anticoagulation therapy such as heparin, which is sometimes carried into the postoperative time period. Another commonly used agent in the postoperative period is an antiplatelet therapy such as aspirin.

Postoperative Care and Monitoring

The final essential aspect of microsurgery is monitoring of the flap during the initial perioperative period. Early microsurgical literature reported the risk of flap vascular thrombosis in microsurgery to be between 0.9% and 16.7%. When thrombosis occurs, this may culminate in flap loss. More recent reports indicate improvements in success rates in microsurgery, which now approach 95–98%. Maintaining this high success rate requires meticulous microsurgical technique and planning as well as vigilant postoperative monitoring. If there is an anastomotic compromise of either arterial or venous supply to the flap, it is crucial to recognize the problem promptly to salvage it.[15,16]

Clinical observation of the free flap provides information including color, turgor, capillary refill, and surface temperature. This clinical assessment has been the gold standard for monitoring free flaps and ideally should be performed hourly in the immediate postoperative period. However, with limited availability of trained personnel able to perform this duty, it is important to have other methods to objectively identify postoperative vascular compromise. The available adjunctive monitoring techniques include Doppler technology, surface temperature monitoring, tissue oxygen tension measurement, tissue pH levels, fluorescein dye mapping, near-infrared spectroscopy, thermodilution technology, photoplethysmography and nuclear medicine studies.[17] Ultimately, which adjunctive measures are chosen to monitor the transferred tissue is a matter of surgeon and institutional preferences. Whichever modality is chosen, close flap monitoring should be a standard part of the postoperative care of these patients during the first 72 h after surgery at a minimum.

Management of the Ailing Free Flaps

Although the success rate of free tissue transfer has been very high, in the range of 96–99%, there are times when flaps fail to thrive. As many of these flaps may be salvaged, it is important to accurately detect the failing flap and intervene

without delay. This is accomplished through attentive flap surveillance, especially in the first 72 h following surgery. When a marginally viable or failing flap is identified, one must first identify the most likely causes of impending flap failure. Although the most common causes are technical deficiencies in the vascular anastomosis, at least two other conditions exist that must be recognized as potential causes for flap compromise. External compression could be easily missed if unrecognized. The surgical dressing itself may compress the flap if too tight or if the dressing maintains the flap pedicle in a position that causes some degree of kinking. For example, a bulky dressing over the lower-extremity free flap, commonly placed for immobilization and protection of the newly reconstructed area, may actually compromise the microcirculation itself. Hematoma is another cause of vascular compression. A slow accumulation of hematoma can compromise the flow of the small caliber vessels. When these problems are detected, a simple decompression of hematoma by releasing some of the sutures or loosening the dressings oftentimes is a helpful measure in temporizing an immediate threat to the flap before the salvage procedure in the OR.

Another important consideration in dealing with a failing flap is the condition of the patient. Is the patient hemodynamically stable? Was there any recent administration of vasopressor agents to augment the patient's hemodynamic status? Has there been adequate urine output? Are the patient and the room where the patient is located cold? How is the temperature of the flap compared to the surrounding tissue in the body and room temperature? In general, flaps do best when the patient remains warm, well perfused and hemodynamically stable, and these are generally easy measures to address before any surgical intervention, which may improve flap salvage rates.

The most common cause of the failing flap, however, is technical error. Such errors include imprecise suture placement or unrecognized damage to the vessels due to rough tissue handling as previously mentioned. There should be neither hesitation nor delay in opting to examine the flap in the OR and check the anastomositic patency under the microscope. If there is some imperfection in the microcirculation, one should never hesitate to take down the anastomosis and do it again. During the take down of the anastomosis, one would look for any thrombus formation and for its correctible causes, including intimal/endothelial damage, significant vessel size mismatch, twists or kinks with turbulent flow.

If there is significant clot formation in the vessels, the affected vessels must be resected. If, after resection, there is insufficient vessel length to re-perform the anastomosis, an interpositional vein graft might be needed. This should be anticipated before the second-look operation so that a potential donor site can be prepared.

Even after a new anastomosis has been performed with perfection, it is still necessary to observe the flap microcirculation for some time in the OR under well-controlled, optimized conditions (hemodynamic stability, warm room and body temperature).

Thrombolytics in Flap Salvage

Thromblytic agents (streptokinase, urokinase, tissue plasminogen activator, and other similar agents) have been used by many surgeons in attempting to salvage an ailing flap. It is very important to avoid systemic fibrinolyzation when performing this maneuver; otherwise, a life-threatening bleeding complication can occur. The use of the thrombolytic agents is usually confined to the circulation within the flap itself with the use of microclamps or by venting the venous effluent with care to flush it out with a heparinized solution before restoring systemic circulation.[18–20] Moreover, there is some evidence that addition of systemic heparin following thrombolysis may further increase patency and salvage rates.[21]

Other Pharmacologic Adjuncts

Aspirin is a cyclooxygenase inhibitor that exerts an antiplatelet effect by means of inhibiting the products of arachidonic acid metabolism. This leads, in turn, to decreases in thromboxane-mediated vasoconstriction and platelet aggregation. Lower-dose aspirin regimens (50–100 mg) have been advocated based on an apparent selectivity for platelet-derived cyclooxygenase inhibition at these lower doses.[19,22,23] Platelet-derived cyclooxygenase is thought to be the primary source of thromboxane, the potent

vasoconstrictor and platelet aggregator. In contrast, endothelial-derived cyclooxygenase produces other prostaglandins. Selective inhibition of platelet-derived cyclooxygenase, then, may provide the antiplatelet benefits while permitting lower dosing and perhaps minimizing the risk of other adverse effects of therapy, such as gastritis or salicylate toxicity.

Heparin exerts an antithrombin effect by inhibition of antithrombin III.[24] In the setting of microsurgery, heparin is commonly used as an irrigant in preparing the vessels for anastomosis and just prior to completion of the anastomosis. In both of these instances, its use is meant to irrigate away any luminal debris and to rid the vessel of any adherent thrombus. There also seems to be a preventative effect of heparin irrigant on thrombus formation, which further justifies its topical use.[19,25–27] The generally agreed upon concentration of the heparin–saline irrigant is 100 U heparin/ml.[27]

Heparin may also be used systemically at the time of microvascular surgery prior to clamp release. Although this use finds support in the microsurgery literature,[19,28,29] the data remain mixed in the animal literature,[30,31] and systemic heparin use remains controversial. Its use must be balanced against the risk of bleeding complications and postoperative hematomas, which, in the extreme scenario, may lead to pedicle kinking and flap compromise.

Dextran is a polysaccharide synthesized from sucrose as a low molecular weight (dextran 40) or high molecular weight (dextran 70) polymer.[19] According to Conrad, "Its five mechanisms of action include 1) increasing the electronegativity on platelets and endothelium, thus preventing platelet aggregation; 2)modifying the structure of fibrin, making fibrin more susceptible to degradation; 3) inhibiting alpha-2 antiplasmin and subsequently activating plasminogen; 4) decreasing factor VIII and von Willebrand factor leading to decreased platelet function; and 5) altering the rheologic properties of blood and acting as a volume expander.[19]" Its use in microsurgery remains controversial although there is some support in the literature for its use in maintaining early patency of microvascular anastomoses.[32,33] Furthermore, there is accumulating evidence in the microsurgical and plastic surgical literature that dextran use is not as innocuous as once thought.[34] A measured approach in deciding to use dextran is advocated by many surgeons given the increasingly appreciated complications associated with its use, including pulmonary edema, bleeding, and anaphylaxis.[34–38]

In summary, there are many antithrombotic pharmacologic regimens that may be used in microsurgery. Some of these have been clearly shown to be beneficial, whereas others remain controversial. Whichever regimen or adjuncts are chosen, it is clear that no pharmacologic intervention can compensate for poor surgical technique. Meticulous technique is paramount in ensuring good outcomes.

The No-Reflow Phenomenon

The no-reflow phenomenon as it pertains to microsurgery describes the inability to maintain perfusion to the transferred tissue despite restoration of blood flow through a technically acceptable anastomosis. The no-reflow phenomenon was originally described by Ames et al.[39] in a rabbit cerebral ischemia model. Noting that ischemic areas at times failed to reperfuse once blood flow was reestablished, he suggested that another independent factor in determining viability after reperfusion may relate to early ischemic changes in the blood vessels. Since his 1968 article, the no-reflow phenomenon has come to be understood in terms of ischemia-induced endothelial injury.

It is believed that ischemic insult to vascular endothelium leads to cellular swelling, leakage of fluid into the interstitial spaces, exposure of subendothelial collagen, platelet aggregation, and ultimately slowed vascular flow through the injured vessels. If the slow flow state persists, thrombus formation and flap failure occur. The histologic changes associated with the no-reflow phenomenon may be reversible up to 12 h after reperfusion but are likely irreversible beyond that time.[40] Factors that may reverse or limit ischemic changes include thrombolytics,[41–44] nonsteroidal anti-inflammatory drugs,[45] and other pharmacologic substances that affect prostaglandin levels in the ischemic tissue.[46,47] However, the most important factor in minimizing ischemic changes in microsurgery is good surgical technique, including meticulous preoperative planning, careful donor-site preparation, and diligent efforts to keep ischemic times as brief as possible.

The Future of Microsurgery

As noted in this chapter many of the techniques have been refined to a point that microvascular surgery has become routine at large medical centers. There, however, is still room for growth in microvascular surgery, and on the horizon the next major milestones will likely come from composite, cadaveric tissue transfer. This work has already started with exciting results in cadaveric hand and partial face transplantation.

Overall microvascular surgery requires a significant degree of technical expertise, but given the advances made during the last 50 years, we can look forward to greater use of our techniques.

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9

Minimally Invasive Techniques in Plastic Surgery

Shashidhar Kusuma, Mohammad Alghoul, and James E. Zins

Summary

Is minimally invasive surgery a myth or is it based on reality? Can you achieve results of the quality similar to that of traditional open procedures while minimizing surgical scars, pain, decreasing recovery time, and decreasing morbidity? Or do minimally invasive techniques yield minimal results? Although the demand for these procedures increases, plastic surgeons may need to adapt and change their approaches to commonly performed procedures. In addition, training programs may need to take into account the latest trends in plastic surgery to appropriately prepare the young plastic surgeons for successful careers. Although it is impossible to account for all the available minimally invasive techniques, this chapter attempts to review the most commonly performed techniques.

Abbreviations

MACS	Minimal access cranial suspension
SMAS	Superficial muscular aponeurotic system
TUBA	Transumbilical endoscopic breast augmentation

Introduction

Can you get more with less and is the new wave of minimally invasive surgery a myth or reality? Or is it merely a marketing ploy? Although these concepts have great appeal, these questions will be answered only after review of evidence-based literature and long-term results. There is a constant desire among patients and surgeons to achieve more with less and to accomplish more with smaller incisions. Although there is a time and place to undertake and perform lesser procedures to achieve desired outcomes, such decisions should be made after thoughtful planning and sound judgment. It is of critical importance for plastic surgeons to be aware of all of the available innovations in the specialty. Although it is beyond the scope of this chapter to cover each of these minimally invasive procedures in detail, the purpose of this chapter is to introduce these concepts and techniques to the reader. References have been liberally cited to assist the reader who would like to explore these areas in greater detail.

Technological and conceptual innovations have led to a significant growth of newer plastic surgical procedures. Such techniques include, but are not limited to, injectables and fillers, endoscopic surgical techniques, laser and light-based

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modalities, limited incision procedures, and other less common procedures. Many products have come to the marketplace with claims of optimal results with minimal surgery and recovery time.

A number of endoscopic procedures have been added to the plastic surgeon's armamentarium with the benefit of limited postoperative morbidity, pain, and recovery time. Such procedures include the browlift, facelift, endoscopic nasal surgery, breast augmentation, and a variety of reconstructive procedures. Recently, endoscopic techniques have been used to repair facial fractures and perform craniofacial surgical procedures.

Endoscopic Techniques and Applications

Endoscopic techniques have changed the plastic surgeon's approach to a number of procedures since their introduction to plastic surgery in the late 1980s and early 1990s. In the early 1990s, Vasconez et al.[31] popularized the endoscopic browlift. This became possible when it was realized that if the soft tissue of the forehead was elevated from the periosteum, an "optical cavity" could be created. This became the first of many endoscopic techniques to be introduced to plastic surgery. Videoscopic equipment with magnification allowing visualization with great detail and new endoscopic instruments have been developed to aid the surgeon in performing operations with precision and accuracy.

Endoscopic Facial Surgery

The browlift is the most commonly performed endoscopic procedure in plastic surgery. It avoids the long scar associated with the more traditional coronal browlift. Since its introduction in 1992 by Vasconez et al.,[31] and its later popularization by others such as Ramirez and Robertson,[23] it has gained wide acceptance. This procedure has undergone many alterations and is one among several methods available to rejuvenate the ptotic brow and rhytids of the forehead. This procedure involves the creation of small (approximately 2 cm) access ports in the hair-bearing scalp through which an endoscope and various dissection tools are introduced. The number of access ports vary based on surgeons' preference. Under video guidance, the skin, soft tissues, and ligamentous attachments of the forehead are dissected and released. The dissection plane is either subperiosteal or subgaleal. The subperiosteal dissection allows for greater illumination when compared with the subgaleal release because of the lighter color of the bone when the subperiosteal dissection is performed. However, some feel that release is more effective in the subgaleal plane.[3] A critical part of this procedure is the adequate release of the attachments at the temporal fusion line and along the lateral orbital rim including the lateral orbital retaining ligaments. Some surgeons advocate wide release down to the zygomatic arch.[1] The plane of dissection is in the subperiosteal plane in the forehead and on the temporalis fascia proper (deep temporal fascia) in the temporal region. This is merged around the lateral orbital rim and the temporal fusion line to the subperiosteal plane in the forehead. Care is taken to avoid injury to the frontal branch of the facial nerve, which travels within the superficial temporoparietal fascia and lateral branch of the supraorbital nerve lying between the periosteum and galea just medial to the temporal fusion line.[17] Once adequate release is performed, the skin and soft tissues are anchored in a higher and lateral position to obtain the desired brow position. Many methods are used for anchoring the soft tissues including bone tunnels, resorbable screws, sutures, resorbable tines, and surgical adhesives. The optimum time needed for adequate fixation to occur is controversial.[19] In some cases no fixation is done with the belief that adequate release is the most critical maneuver in obtaining adequate brow elevation. Through the aid of video guidance, the medial brow depressors are also dissected, transected, or resected to eliminate the glabellar rhytids. Care is taken to avoid injury to the supratrochlear and supraorbital neurovascular bundles. The supratrochlear nerve bundle travels within the corrugator muscle mass. Some also score the frontalis to improve the horizontal forehead rhytids. Limitations or pitfalls of the procedure include inadequate soft tissue release, inadequate or improper fixation, and recurrent frown lines because of inadequate muscle resection. In addition, it is easy to overelevate the medial brow and harder to adequately elevate the brow laterally (Figure 9.1).

Using the same temporal access ports, the midface can be accessed. The midface lift can be done through this endoscopic temporal approach only[4,25] or through a combined temporal and intraoral approach.[20] Again, subperiosteal dissection is performed along the lateral and infraorbital rims and along the anterior surface of the maxilla. An intraoral incision may be used for additional access to the midface. This simplifies the dissection and shortens the procedure. However, it does, at least theoretically, increase the possibility of intraoral contamination of the temporal dissection. Once wide undermining is performed, resorbable or nonresorbable sutures are used to anchor the ptotic midface soft tissues, which are then elevated superolaterally and anchored to the deep temporal fascia. Alternatively, an endotine midface device can be used to maintain elevation of the midface tissues (Figure 9.2).

Figure 9.1. (**a**) A 49-year-old woman with eyebrow ptosis, preoperative view. (**b**) Same patient 2 years following endoscopic subperiosteal browlift.

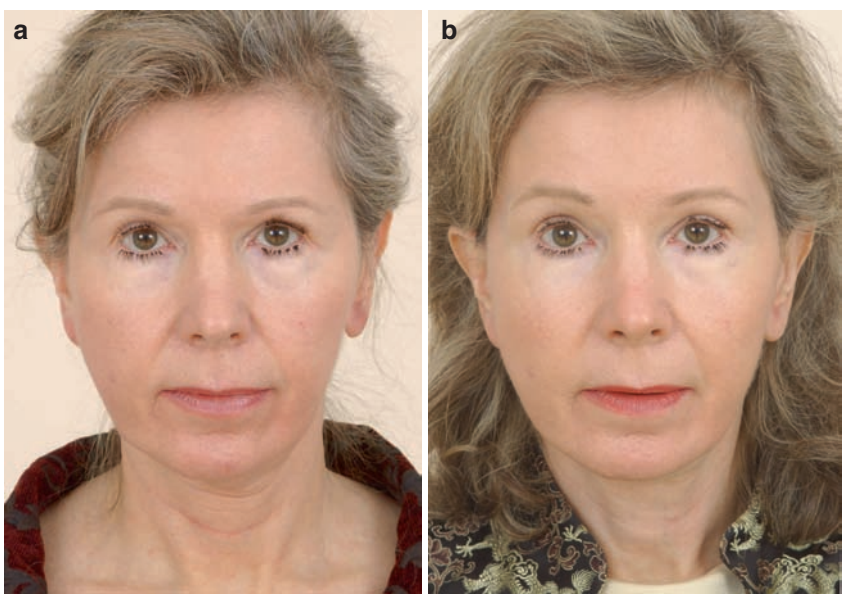

Figure 9.2. (**a**) Preoperative view of a 57-year-old woman with brow ptosis and midface descent. (**b**) Postoperative view following endoscopically assisted midface lift through a temporal approach and intraoral incision.

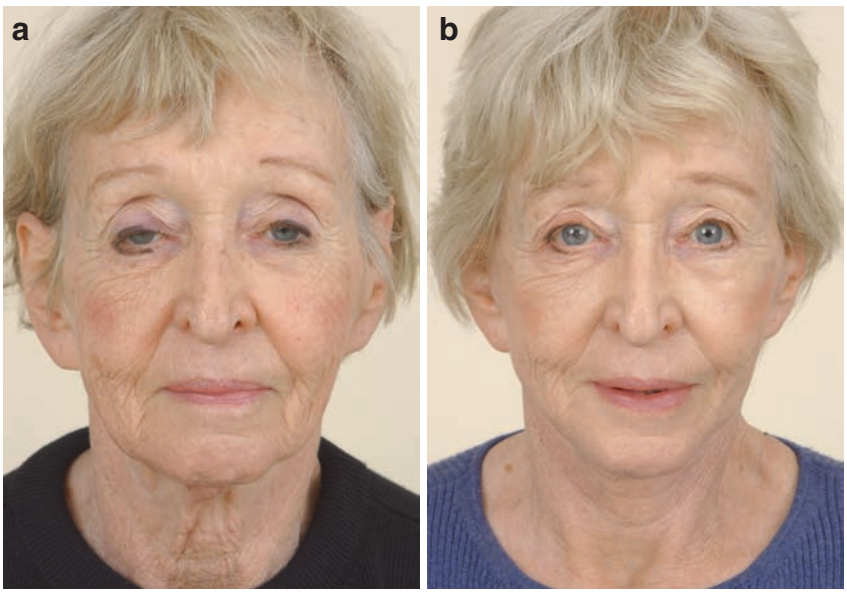

Figure 9.3. (**a**) Preoperative view of a 76-year-old woman with cheek laxity, deep nasolabial folds, jowling, and neck laxity. (**b**) Postoperative view of the patient following MACs facelift for cheek correction, necklift, and platymaplasty.

Although the endoscopic subperiosteal facelift was popularized by Ramirez,[24,25] it has not gained widespread acceptance in facial cosmetic surgery. The traditional route of skin, superficial muscular aponeurotic system (SMAS) surgery along with the newer small scar techniques has gained popularity.[29,30] Of the short-scar techniques, Tonnard's minimal access cranial suspension (MAC)'s lift and Baker's lateral SMASectomy are perhaps the most popular (Figure 9.3).

Endoscopic Nasal Surgery

A variety of endoscopic nasal surgical procedures are well described in the otolaryngology literature. Although these techniques have not gained popularity in plastic surgery, their application to aesthetic plastic surgery is clear, and they most likely will ultimately find a place in our armamentarium. The areas currently being addressed include inferior turbinates, middle turbinates, and the septum. In many situations, cosmetic rhinoplasty can be combined with functional endoscopic nasal surgery to address inferior turbinate hypertrophy and other anatomical obstructions in the nasal cavity. The same endoscopic equipment used for other endoscopic techniques can be used. The nose is first decongested with oxymetazoline or other decongesting agents. A 0° endoscope is introduced into the nasal cavity to visualize the anatomic problems with the inferior turbinates, septum, and the middle turbinates. With the use of powered instruments that can remove and reduce the size of the hypertrophied inferior turbinate submucosa, stroma and the bone can be removed with limited incisions in the mucosa (Figure 9.4). This is done without thermal injury to the mucosa, preventing injury to the vital physiologic function of the mucosa. With the use of these techniques, the morbidity and blood loss can be minimized. Recovery is rapid as there is less chance of complications such as synechiae formation, nasal crusting, and bleeding. Many publications in the ENT literature have demonstrated that the endoscopic techniques for inferior turbinate reduction are superior to the traditional techniques used.

Recently, Joniau et al.[15] completed a long-term comparison between submucosal cauterization and power reduction of the inferior turbinate. Their study concluded that endoscopic powered turbinoplasty leads to decreased patient morbidity during postoperative healing and to a better control of long-term results when compared with submucosal cautery. They demonstrated that endoscopic powered turbinoplasty was superior to cautery in all measured aspects, including

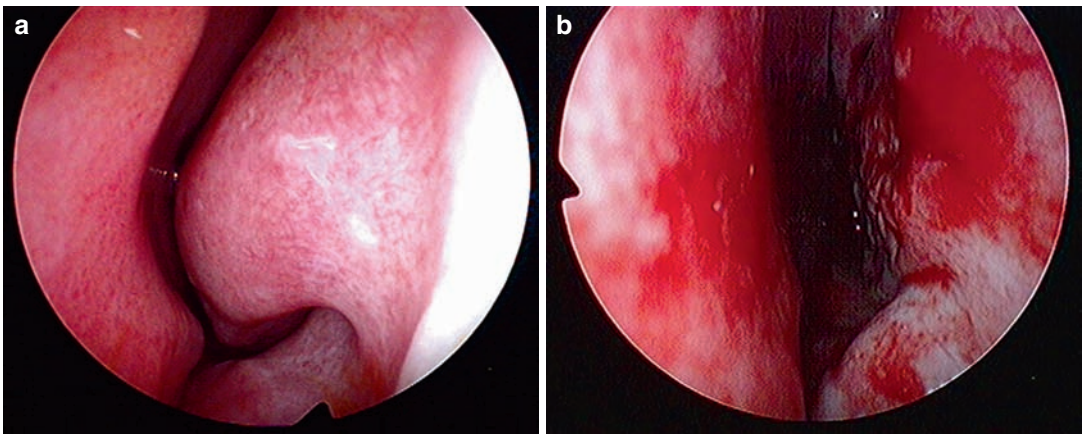

Figure 9.4. (**a**) Preoperative view of left inferior turbinate. (**b**) Postoperative view of endoscopically reduced left inferior turbinate.

crusting, acoustic rhinometry, cross-sectional area, and nasal cavity volume. A similar study by Gupta et al.[13] performed an outcome analysis on endoscopic inferior turbinate reduction via a patient questionnaire confirming the long-term effectiveness of endoscopic turbinate reduction for the relief of nasal obstruction. Retrospective data collected at our own institution using this procedure for the past 2 years indicated that 90% of patients have complete or near-complete relief of nasal obstruction. Greater than 90% of the patients were satisfied with the outcome and would recommend this procedure to others. Further objective analyses that include measurement of nasal airway and rhinometric studies are underway to determine objective outcomes with this technique.

Similar endoscopic techniques can be used to address isolated septal spurs as well. Limited local incisions can be made in the mucosa close to the site of interest and only the anatomic area of the problem can be addressed, without having to raise widely undermined flaps or perform extensive septal surgery.

The learning curve for some of these procedures is steep. However, with time, many of these procedures can be done very efficiently.

Endoscopic Breast Surgery

Endoscopic breast augmentation has now been practiced for more than 10 years. The commonly used submammary and periareolar incisions have the disadvantage of placing a scar on the aesthetic unit of the breast. To limit the visible scarring associated with these procedures, an endoscopic guided creation of a subglandular, subpectoral, or subfascial pocket through camouflaged incisions has been popularized using several routes to access the breast. The transumbilical endoscopic breast augmentation (TUBA) and the transaxillary endoscopic breast augmentation have been successfully and safely used with reportedly good outcomes.

This procedure involves creating subglandular or subpectoral pockets with blunt dissection using long instruments with blunt tips passed subcutaneously through a supraumbilical tunnel.[14] Equipment required for this procedure include a high-resolution endoscopic video camera, a monitor, tube, long suction instrument, and an obturator with a rounded tip. A periumbilical incision is made and dissection is taken down to the level of the rectus sheath. A subcutaneous tunnel is created above the rectus sheath on each side using the tube and obturator as one unit to the level of the inframammary crease. The endoscope is inserted into the tube to confirm the cannula position by visualizing the mammary tissue and the pectoralis muscle. The tube is removed, and the obturator is inserted into the tunnel and using blunt dissection, a subglandular, or subpectoral pocket is created. The dissection is done blindly aided by preoperative markings. The endoscope is then used to ensure entrance into the right plane of dissection and to check for bleeding in the dissected pocket. A significant portion of the dissection is done hydraulically with the implant sizer, which is rolled like a cigar and pushed through

the tunnel into the dissected pocket. Only saline implants, not prefilled and preferably smooth, can be used for this procedure. The TUBA avoids a scar on the aesthetic unit of the breast. The main disadvantage of this technique is the limited choice of implants since prefilled saline and silicone implants are not suitable for transumbilical insertion. Contraindications for TUBA include thoracic wall deformity and the presence of an incisional hernia or scarring in the pathway of the tunnel.

Several series in the literature describe the technique and outcomes of the TUBA.[5] Dowden[9,10] recently reported good results with the technique. He reported no complications in 597 cases (479 prepectoral and 118 subpectoral) except for one patient who had to be explored for asymmetrical edema. Brennan and Haiavy[3] similarly reported good results with a low complication rate. The learning curve for these procedures is steep. Some inherent difficulties with this procedure include the added dissection of the abdominal wall, the possibility of imprecise or imperfect pocket creation and location, difficulty controlling the inframammary fold, and difficulty controlling postoperative bleeding. Despite its introduction in breast augmentation surgery, it has not gained wide popularity.

Although the blind transaxillary breast augmentation approach has been used successfully for many years, there were instances where it was difficult to address the inframammary fold adequately. Bleeding could also be difficult to control. To address these problems, the technique was modified from a blind blunt dissection to endoscopic-guided cautery dissection. The endoscopic dissection is performed through a single port using an endoscope-retractor system. Electrocautery dissection in the subglandular or subpectoral pocket is performed under direct vision. The axillary incision is made 1 cm posterior to the posterior edge of the pectoralis lateral border and extends posteriorly through a dot in the apex of the axilla. Care must be taken to avoid dissection of the axillary fat pad to avoid injury to the intercostobrachial nerve branches and branches of the lateral thoracic artery and vein. Suboptimal retractor positioning can also pose the risk of injury to the brachial plexus. After the lateral edge of the pectoralis major muscle is identified, a tunnel is created in a plane immediately adjacent to the muscle, and using the endoscope-retractor system, the pocket is dissected with needle electrocautery in the desired plane along the preoperative breast markings. Both saline and gel-filled implants can be inserted through the axillary approach. Larger size implants require larger, more conspicuous incisions. As with the TUBA, the main advantage of this technique is locating the scar off the aesthetic unit of the breast. Disadvantages include longer operative time and the need for previous training to handle endoscopic equipment. Dissecting near the axilla can also cause sensory denervation changes, seroma formation, and may limit postoperative arm motion.

Tebbetts[28] described his 28-year experience with this approach and compared 331 patients who underwent blind blunt dissection with 359 patients who underwent endoscopic electrocautery dissection. He reported less capsular contracture, less transient arm sensory changes and less axillary lymphatic banding in the latter group.

Endoscopic Techniques in Body Contouring

Endoscopic rectus plication and mini abdominoplasties can be done with limited abdominal incisions. Through ports introduced in the lower abdomen or around the umbilicus, the rectus diastasis can be tightened with endoscopic suturing of the rectus fascia. These procedures have relatively limited use for carefully selected patients. Although most patients with lax abdominal wall fascia have significant skin excess as well, there are those patients who present with significant rectus diastasis and minimal skin excess. Such patients can undergo limited incision endoscopic surgery to perform rectus plication.

Minimally Invasive Techniques in Facial Aesthetic Surgery

Many devices have been developed recently to aid the plastic surgeon in performing aesthetic and reconstructive facial surgery using endoscopic and minimally invasive techniques. This includes a variety of Coapt devices, suture/string/thread devices, and implants. The Coapt devices (Coapt systems, Palo Alto, California) are products that are made of bioabsorbable implants for use in plastic surgery for fixation of

soft tissue and bone, which are shaped and sized according to need. Once such device, the endotine forehead 3.5, was approved by the U.S. Food and Drug Administration for endoscopic browlifts. These polylactide homopolymer products have tines on the superior surface for engaging the deep soft tissues. These tines are capable of penetrating soft tissue and allow elevation and distraction of these tissues, which can then be anchored to a predetermined desired location. The posterior surface that abuts the anterior table of the skull has a post for setting into a cranial drill hole. Once the forehead and temporal soft tissues are adequately released and elevated, a Coapt endotine device can be secured to the anterior table of the skull on one surface. The other surface that has the prong is then forcefully imbedded and allowed to anchor the deep soft tissues that are held in place. Another Coapt device has been designed for midface or cheek elevation. This device is longer and wider, is placed through a temporal incision, and with endoscopic assistance or through an additional intraoral incision, is used once the subperiosteal cheeklift is completed.

More recently, some innovative surgical devices that use minimal access incision have been developed, but instead of using resorbable tined devices, barbed sutures are used to anchor and distract and elevate soft tissues. One procedure that is done in this manner is a lower facelift that is given many names such as a thread-lift or string-lift. A small incision is made in the temporal, preauricular, or other locations through which a long needle that is attached to a barbed suture is introduced. This is passed through the soft tissues of the face to a desired distance such as the nasolabial fold and is then looped around and passed back through the same incision. The barbed suture has the ability to imbed and distract and elevate the intervening soft tissues which is then anchored in the temporal region. Most reports describing this technique are anecdotal and long-term objective outcome studies are currently lacking.[12,27,32]

Limited-Incision Facial Aesthetic Surgery

Short-scar facelift surgery has gained recent popularity. Several names are used to describe such procedures such as the short-scar facelift, the MACs-lift, and the S-lift. All such procedures use a variation of the more standard facelift techniques. The incision is limited to the preauricular and temporal areas, and the amount of dissection and fixation of the deeper tissues are limited. These procedures are designed to address mild to moderate changes in the facial aging. Such procedures are best used in carefully selected younger patients with minimal or mild facial aging. These techniques are also useful in secondary facelifts when minimal or no neck dissection is needed, or when formal sub-SMAS dissection is not planned or contraindicated (Figure 9.2). With proper patient selection, aesthetic goals and gratifying results can be achieved.

Often in the elderly patients, a combination of smaller procedures can achieve significant improvement in facial aging while reducing the complexity and the potential morbidity of larger procedures. In these situations, direct excisions of skin excess can replace traditional facelift surgery. The incisions are planned where skin excess is greatest, the trade-off being a temporarily visible scar. However, since these elderly patients tend to heal with excellent scars, the relatively long-term result can be quite gratifying. Such procedures include direct excision of nasolabial folds (Figure 9.5), direct excision of neck skin and Z-plasty for the correction of the "turkey gobbler" deformity (Figure 9.6), and/or the direct excision of skin excess in the marionette line area.

The direct excision of the nasolabial fold is also a quite reasonable procedure post facelift in those patients with extremely deep nasolabial folds who have not responded ideally to facelift surgery. In these instances, the interval between facelift surgery and direct nasolabial fold excision is generally 3 months.

Finally, the direct browlift is a reasonable alternative to open browlift surgery in the elderly male who is concerned about larger incisions or greater downtime.

An alternative procedure to necklift surgery is the anterior-only approach or the anterior lipectomy and platysmaplasty. This procedure avoids a preauricular incision and the postauricular extension. It is ideal for those patients who are interested in a change in their profile only and have no concerns about the midface. The procedure is performed under local anesthesia with conscious sedation. It is begun through a 3 cm submental incision. The skin is completely released

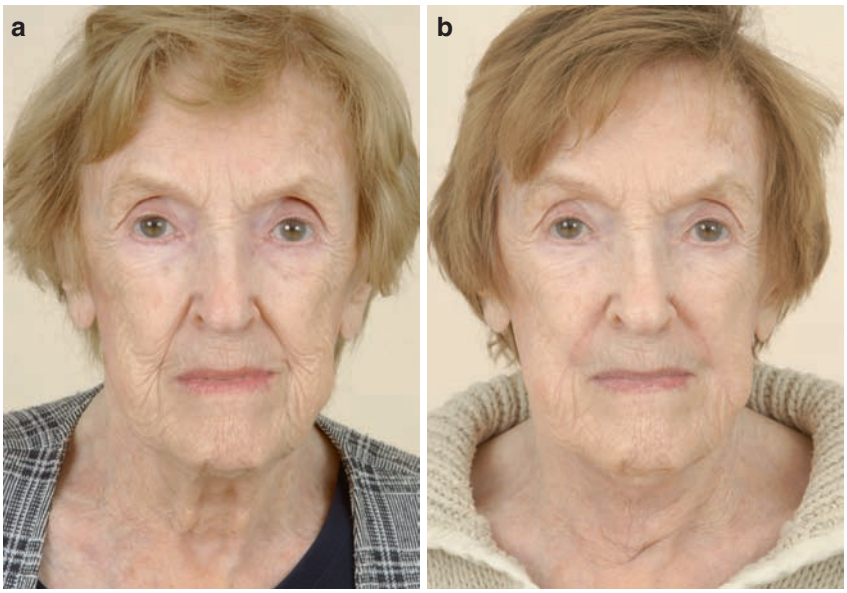

Figure 9.5. An 89-year-old woman with deep nasolabial folds, cheek laxity, and neck laxity. (**a**) Preoperative view. (**b**) 2.5 months following direct excision of the nasolabial folds.

from the underlying platysma to the extent of skin laxity. Submental and submandibular fat resection is performed superficial to the platysma and then the plastyma is opened. Subplatysmal fat is removed and a platysmaplasty of choice is then performed.

This procedure takes advantage of the inherent ability of the neck skin to contract over time. The extent of skin undermining should be similar to the amount of undermining that would be performed if the patient under consideration was to have a traditional necklift operation. Good and long-lasting results have been demonstrated in the literature using this technique.[11,18,33] The procedure can be done in conjunction with a filler to the nasolabial folds, marionette lines, and other areas of the face.

Zins and Fardo[33] classified these patients into three categories: (1) patients with obtuse cervicomental angles and good skin elasticity who may be treated with liposuction alone, (2) patients with subplatysmal fat or mild to moderate skin and muscle laxity who are best treated with anterior lipectomy and platysmaplasty, and (3) those with marked skin excess or severe skin laxity who are treated with a traditional lower face and necklift or a direct Z-plasty skin excision. Good long-term results can be achieved with such minimally invasive procedures (Figure 9.7).

Minimally Invasive Techniques in Skincare and Rejuvenation

Skincare has evolved significantly in the past few years. The advent of a variety of laser and light-based technology using nonablative techniques has changed skincare in the hands of many surgeons. Traditional, more aggressive, skin-resurfacing techniques such as the CO_2 laser, dermabrasion, and other intermediate and deep resurfacing techniques have lost popularity with the development of a variety of nonablative laser resurfacing modalities. Nonablative laser therapies can improve skin quality, reduce hyperpigmentation and skin irregularities, reduce redness and, perhaps, tighten skin without the surface injuries and prolonged erythema associated with more traditional ablative techniques. The current options include fractionated erbium and CO_2, intense-pulsed light and radiofrequency, radiofrequency alone, and photodynamic therapy. The large variety of laser and light-based modalities attests to the fact that

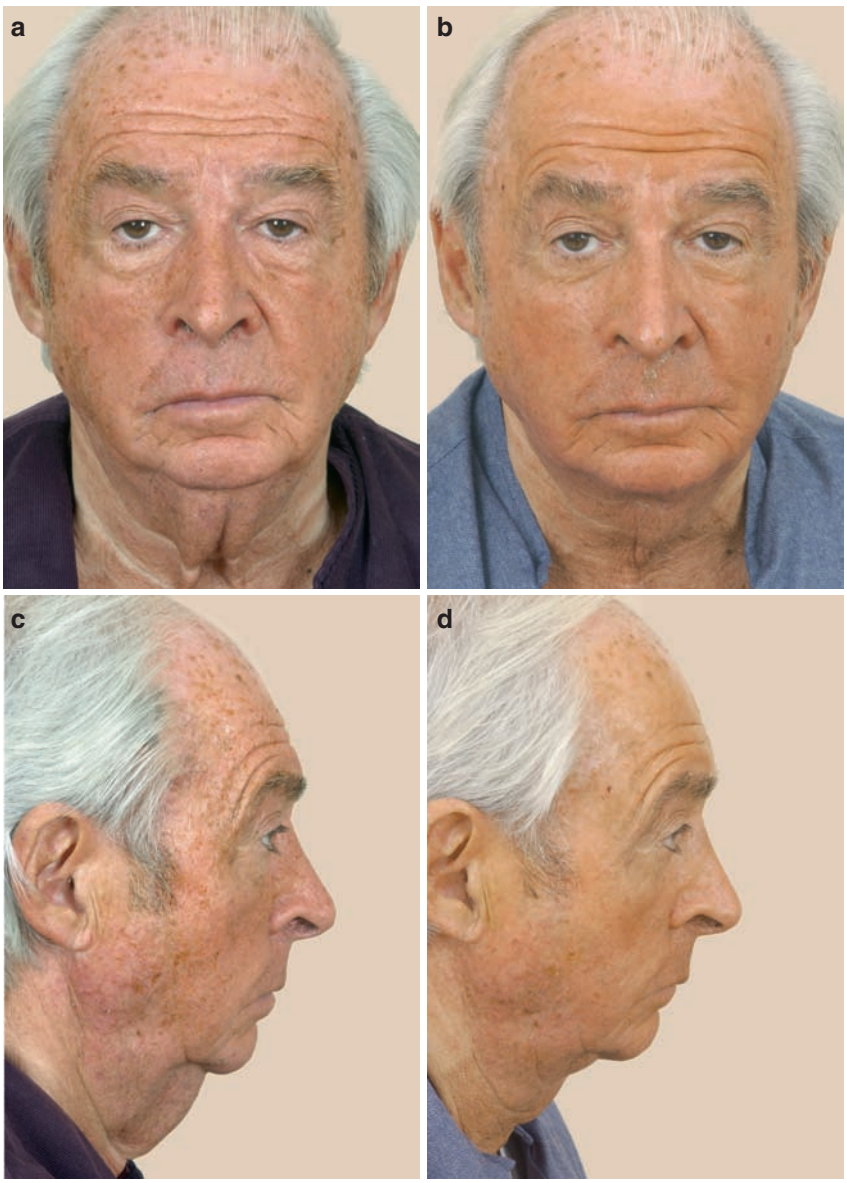

Figure 9.6. Preoperative view of a 74-year-old man with neck laxity and "turkey gobbler" deformity. (**a**) Preoperative frontal view. (**b**) One-year postoperative frontal view. (**c**) Preoperative profile view. (**d**) One-year postoperative profile view.

no one method has demonstrated clear superiority over the others. Clearly, this technology will continue to evolve and newer techniques will inevitably become available. This changing the landscape can make it difficult for the practitioner to choose the best course. This is compounded by the fact that the physician incurs significant cost with each of these light-based or laser modalities.

Stem cell research is one of the most rapidly evolving and most promising areas of basic science medical research today. Advances in adult-derived, adipose stem cells are promising and may lead to clinical improvements in current fat transfer or injection outcomes. Fat grafting and dermis-fat grafting have a long history in plastic surgery dating back to the 1950s.[2,21,22] Coleman's technique or Coleman variants are now used by

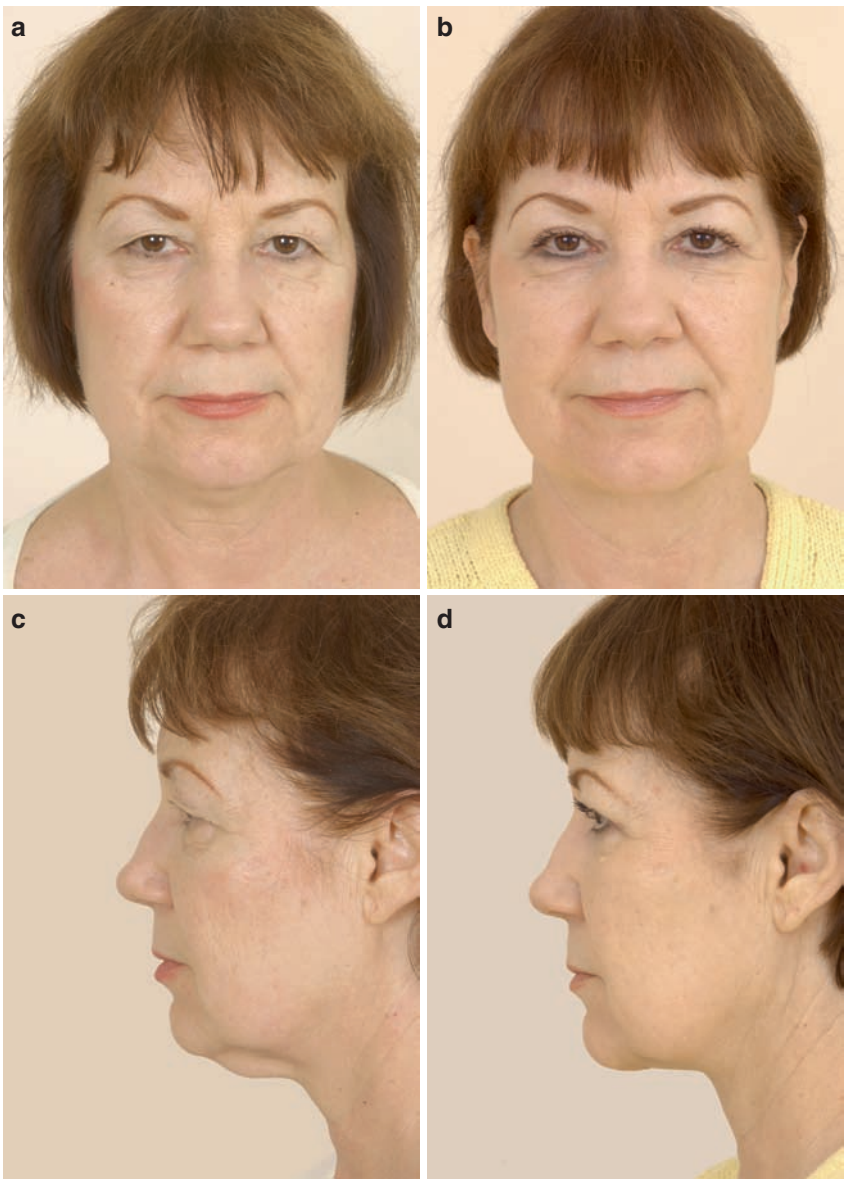

Figure 9.7. (**a**) Preoperative frontal view of a 63-year-old woman with skin excess and neck laxity. (**b**) Postoperative view 2 months following necklift without preauricular incision. (**c**) Preoperative profile view. (**d**) Two months postoperative profile view. (Reprinted from Zins and Moreira-Gonzalez. A, Advances in facial aesthetic surgery: new approaches to old problems and current approaches to new problems. In: Siemionow M, ed., Tissue Surgery, with kind permission of Springer Science and Business Media. © 2005.)

plastic surgeons nationally and internationally.[6–8] This has occurred despite the lack of vigorous outcome measures.[16]

Coleman's technique involves the atraumatic harvest of fat from the patient's abdomen, thighs or buttocks. The fat is then centrifuged to isolate the fat from oils and other debris and is then reinjected into desired areas of the face. The areas of the face most often treated include the nasolabial folds, the marionette lines, the infraorbital rim or the nasojugal groove, the upper eyelid, and the malar/cheek area. Coleman uses the multipass, multi-injection technique where the injectate is placed in various layers of

the skin and subcutaneous tissues. There may be a secondary effect from the edema that further improves the ultimate outcome.

In addition to the use of autologous fat transfer to facial aesthetic and reconstructive procedures, some innovative surgeons have introduced this technique in breast augmentation, revision breast surgery, and body contouring procedures. Large quantities of fat are harvested from the abdominal wall or the lateral thighs and processed atraumatically. Significant volumes of this autologous fat are then injected into breast tissues to correct various contour irregularities and deflation. This has been used in both primary breast augmentation and ancillary reconstructive breast procedures. Preliminary data have shown long-term improvement in breast size and shape. Recently, Spear et al.[26] have shown that autologous fat transfer is safe and reliable in the improvement of contour irregularities in reconstructed breasts.

More data will undoubtedly be gathered and reported to study this exciting new use of the autologous fat transfer further. Using similar principles, autologous fat transfer can be performed in many other parts of the body to improve contours, shapes, and sizes.

Recent research has also shown the secondary benefits to the skin from these fat injections. Recent reports claim improvement in the quality, complexion, pigmentation, and general health of the skin from fat injection. The hypothesis that the injected fat may contain various amounts of stem cells that, when they survive, can repopulate the areas with healthy cells that improve the skin as a secondary benefit has been brought forward but as yet is unproved.

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10

Liposuction Techniques

Dennis J. Hurwitz

Summary

Liposuction treats lipodystrophy and reduces the thickness of body contouring flaps. Patients are evaluated for suitability of deformity, skin quality, and understanding of the procedure. Inelastic and hanging skin is contraindicated for aesthetic lipoplasty. In general, a circumferential approach is taken to maximize skin shrinkage and harmonize the result. The traditional technique of suction-assisted lipectomy is presented with emphasis on smoothing and delivering the fat by the helping hand. Large-volume liposuction requires attention to maintain normothermia, fluid balance, and deep vein thrombosis (DVT) prophylaxis. Of the special energy sources, power assisted, laser, and ultrasonic, the author prefers and elaborates on ultrasonic and radio-frequency usage. By reducing, undermining, and gentle fat removal, liposuction can safely and effectively be combined with extensive body contouring surgery.

Abbreviations

IPC	Intermittent pneumatic compression
LVL	Large-volume liposuction
PAL	Power-assisted lipoplasty
SAL	Suction-assisted lipectomy
UAL	Ultrasonic-assisted lipoplasty
RFAL	Radio-frequency-Assisted lipoplasty

Introduction

Liposuction is aspiration of fat from the subcutaneous tissue. Liposuction can be applied to aesthetic lipoplasty or combined with body contouring surgery. Aesthetic lipoplasty is commonly called suction-assisted lipectomy (SAL). Blunt-tipped cannula, high-vacuum method of SAL was introduced in the United States from Europe and was rapidly embraced during the 1980s.[22] SAL is repeatedly surveyed as the most common aesthetic procedure performed by plastic surgeons.[4] Liposuction treats lipodystrophy, which is characterized by gender-specific deforming accumulations of fat. Men tend to seek reduction of gynecomastia, flank, and central abdominal fat. Women desire removal of fat from the central neck, lateral to the breasts, through the mid torso, along the hips, lateral thighs, inner thighs, and knees.

Liposuction is a closed technique that applies destructive energy to the subcutaneous tissue followed by aspiration of the emulsion. The usual energy is high-pressure vacuum pulling and avulsing fat through side openings in a hollow cannula. Alternative energy systems are power-assisted, laser-assisted, ultrasonic-assisted lipoplasty and radio-frequency-assisted.

Evaluation of the Patient

Ideal candidates for SAL complain of localized bulges of fat. They are young, healthy, and

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of normal weight, with good skin turgor and understand the objectives, risks, and postoperative management of their planned treatment. In most areas, hundreds of cc's of fat emulsion can be removed, and normal skin retracts to the smaller volume. Commonly successful locations are the male breasts, hips, lateral thighs, medial thighs, and knees. Nearly as predictable are the neck, flanks, back, abdomen, and upper arms. Prolonged swelling, contour irregularities, and inadequate results limit procedures in the calf and ankle.[43]

To avoid looseness or sagging, the skin needs to be elastic, and that determination is based on observation and palpation. There are some physical signs that predict diminished elastic recoil– a dense pattern of striae or stretch marks due to fractured dermal elastin subsequent to pregnancy, prolonged use of steroids, or rapid increase in size portents poorly for contractility after fat removal. Doughy skin is soft with poor tone and does not contract. The puckering of cellulite is indicative of disordered adipose architecture, which may be further distorted after liposuction. Excessively mobile and sagging skin is a contraindication for SAL.

A few patients describe a local area of contour bulge, and after proper evaluation, it may be treated in isolation. Most patients presenting for body sculpturing through liposuction have a three-dimensional deformity. The plastic surgeon must appreciate idealized gender-specific contours and be able to imagine them on his/her patient to plan the fat removal. Both the focal areas of concern and the adjacent areas need treatment. The focal areas are blended into each other, generally requiring a circumferential approach.

Candidates for localized reduction have limited excess fat with adequate tone and minimal striae. Localized suctioning is most suitable in the abdomen. Care must be taken to rule out epigastric bulges due to visceral adiposity and/or myofascial weakness. Extension of the fat removal through the enlarged flanks is often advantageous but can be of limited benefit in the apple-shaped torso.

Circumferential Liposuction and Planning

When contour deformity of the lower extremities is being treated, one considers circumferential liposuction, which means that the suctioning of one area of fullness is continuously blended into another. Circumferential liposuction enhances skin shrinkage. The author believes that this happens because the less vigorously treated blended zones undergo far less trauma from the liposuction. With only minimal fat removed, the connective tissue is better preserved, leading to maximal contractility. The fully suctioned bulging areas sustain greater damage to the connective tissue, which limits contraction. It is commonly accepted that superficial lipoplasty immediately under the skin assists in skin contraction. Unfortunately, this approach risks devascularization of the skin, which leads to skin necrosis. Therefore, most plastic surgeons are reluctant to take maximal advantage of superficial lipoplasty. Finally, injury to connective tissue may lead to scar formation, with resulting shortening of collagen bundles, leading to dermal skin retraction. Excessive scarring leaves the skin firm and wrinkled.

The patient stands for preoperative liposuction planning. Bulging areas are observed, palpated, and lightly stroked and pinched to map out removal. The focal area is outlined, and the magnitude of excess fat is indicated by plus marks ranging from + to+++. Skin quality is considered. Depression areas are indicated by minus marks. Lipoaugmenation may be performed for some fill in those areas. Markings are circumferential with one focal zone blending into another. There are recognized zones of adherence about the lower lateral and lower posterior thigh. It is exceptional that much needs to be removed in those regions.

Examine for symmetry and overall contour. Estimate the volume of removal to guide fluid infusion and need for hospitalization. A simple aid to estimating volume is to multiply place a 60 cc syringe in the area of planned liposuction and count the number of syringes that would be filled by the excess volume and multiply by 60. Generally, 1 cc of fluid is infused for every cubic centimeter of anticipated fat removal. Over 5,000 cc of removal prompts overnight in-hospital monitoring.[24] Patient positioning is considered. For most procedures, the patient lies supine and is turned from side to side. If considerable fat is to be removed from the back, the operation is begun while the patient lies in the prone position. Several access sites are chosen for each area. There are a variety of cannulas, aspirators, and energy-assist systems.

The surgeon's experience and judgment far outweigh the advantages of a particular tool. I suggest continuing trials with new equipment with purported improved features.

SAL Technique

As a blind procedure, SAL relies on tactile feedback, observation of the effluent, and contour change. As much as possible, the surgeon's dominant hand infuses the preparatory fluid, directs the energy-tipped probe, and aspirates the fat. The helping hand is flattened as it detects the progressive thickening of the infusing fluid through the layers of subcutaneous tissue. During liposuction, the hand smoothes and firmly compresses the target area to expedite the fat removal and sense the depth of delivery. Greatest compression is over the multiple plus areas or when the fat does not evacuate easily. Grasping the tissue, forming a cylinder and suctioning within the hand are discouraged as that method leads to ridging or depression.

SAL begins with infusion of saline containing xylocaine with epinephrine into the target subcutaneous tissues until a palpable firmness is attained. One ampule (1 mg) of epinephrine and 20 ml of 1% xylocaine plus sodium bicarbonate 10% by volume are added per liter.[28] A thin, multiple-holed, reusable, blunt-tipped needle is connected to the infusion tubing and pump that delivers fluids under desired speeds. Infusion speed, up to 450 cc/min, increases with the anticipated volume of aspirate and the thickness and firmness of the tissues. Through even diffuse infiltration of fluid, the target is enlarged, anesthetized, and vasoconstricted, making the aspiration easier, more even, and virtually bloodless. The operation starts with larger cannulas (diameters between 4 and 5 mm) with multiple holes to speed the evacuation of deeper layers of subcutaneous fat. One area is incompletely treated before advancing to a contiguous one. Then a return is made to the previous area to allow for controlled progressive reduction in volume, all the while continuously assessing the reduced thickness and evenness of the subcutaneous layer with the helping hand. Smaller cannulas (diameters between 2 and 4 mm) follow for even removal of subdermal fat and persistent bulges. When nearing the completion of liposuction, two-handed pinch and spreading evaluation is needed. Crisscross suctioning further smoothes out areas. Minor lumpiness can be corrected by firm message with a pizza type roller. For large-volume fat removal, multiple holes increase the evacuation of fat, especially when they are staggered. A 5 mm in diameter, 20 offset holed cannula is a remarkably rapid aspirator, particularly in fibrous tissue.

Fat removal in the extremities is primarily through long longitudinal strokes, supplemented with secondary shorter, roughly perpendicular strokes through staggered access incisions. The main access incisions of the upper arm are made around the elbow and the Deltopectoral groove. Incisions of the hip and lateral thighs are made along the mid lateral line. Refinement of the contour is mainly transversely oriented with smaller cannulas moved rapidly in a radial manner. Stay deep in the lateral thigh. Frequently assess the contour to avoid over-resection. Transversely oriented excessive liposuction leads to unsightly transverse depressions, which are most noticeable when standing. Multiple access sites allow for better blending of areas in a circumferential effort.

Medical Management

Circumferential liposuction takes longer than focal liposuction, with considerable exposure of the body, usually with position changes and larger volume removal. Greater quantities of aspirate simply magnify the safety considerations. Large-volume liposuction (LVL) aspirates over 5,000 cc.[24] Consideration must be given to avoiding intraoperative hypothermia, appropriate fluid resuscitation, and deep vein thromposis (DVT) prophylaxis[30].

Hypothermia

Although profound hypothermia with its attendant cardiac and coagulation instability is rare, only a few degrees centigrade drop may lead to wound infections if combined with excisional surgery.[45] Force hot air warming, warmed fluids, and warmed room are recommended. Force hot air warming of the patient in the preoperative area may be advantageous but may be difficult to organize. If the marking is performed immediately preoperatively, expect the patient to be chilled and consider a 15-min warmup period before the induction of anesthesia.

Fluids

Intravenous fluid management relates to the magnitude of subcutaneous fluid infusion for the liposuction. Fluid shifts in LVL can be quite dramatic, leading to either hypovolemia or fluid overload, necessitating in-hospital management until stable.[27,41] Overload leading to congestive heart failure or pulmonary edema is at greater risk for the larger volume aspirates since between 60% and 80% of the infiltrating wetting solution remains in the subcutaneous tissue until slowly absorbed or drained by closed suction. In addition to maintenance fluids, intraoperative replacement fluid is 0.25s cc crystalloid for each 1 cc of aspirate over 5,000 cc. A simplified and effective method of managing intraoperative fluids is to maintain a fluid ratio of 1 and a urine output of 1–1.5 ml/kg/h. The intraoperative fluid ratio is defined as tumescent fluid volume plus intraoperative replacement divided by the volume of the aspirate.[41,42] Since there is no linear correlation between the postoperative drop in hemoglobin level and the volume aspirate, measured hemoglobin levels and clinical judgment should be used.[25] Postoperative fluid maintenance should probably be less than 2 cc/kg/h with adjustments based on urine output, vital signs, and condition of the patient.

Xylocaine and Epinephrine

At about 30 ml of 1% xylocaine per liter of infusate, the analgesic effect is adequate with minimal sedation. The role of infusate xylocaine under general anesthesia is questioned because of toxicity.[26] Obviously, the risk of xylocaine toxicity is completely avoided by omitting it from the infiltration solution. The analgesic effect lasts less than 8 h, even though xylocaine and its active metabolic byproducts last up to 28 h. Patients appreciate the virtually pain-free emergence from anesthesia after major body contouring surgery; xylocaine is used in the first 3,000 cc and then reduced in further infusions.[41]

At 1 mg/l epinephrine infusion, plasma epinephrine levels may increase 3–4 times above baseline during liposuction, with peak concentrations around 300 pg/ml reached between 1 and 4 h after infiltration began.[6] Approximately 30% of the infiltrated epinephrine is absorbed, with no clinical signs of toxicity such as anxiety, restless, weakness, pallor, tremor, heart palpitations, and/or vomiting.

All patients are started on an intermittent pneumatic compression (IPC) device immediately before surgery and are continued throughout the hospitalization and until regularly ambulating. Low-dose, low molecular weight heparin is considered until ambulating in patients with multiple risk factors.[39]

Postoperative Care

After a compulsive effort is made to smooth out irregularities, the small incisions are loosely closed with 3–0 nylon sutures. Tightly fitting, encompassing, commercially available elastic garments support the operative areas. Along the extremities, circumferential compression encourages drainage and retards swelling. The expected drainage is allowed to leak out of the access incisions through the garment. Drains may be used in the thighs when large volumes are removed. There may be no practical means to immediately compress the larger thighs and that may await custom fitting some weeks later. If ace wrapping is done, choose 6-in. wide or greater, and monitor for constricting bands or pressure skin necrosis over drains. Compression is not so effective in the torso, so a 7-mm diameter suction drain is drawn across the abdomen through the two flank incisions. Thin sheets of foam are placed on the torso, especially within the flanks and where needed to smooth out skin folds. If the foam has a sticky side, multiply incise the edges for an inch or so to minimize shearing that causes blistering.

The garments and foam are removed 5–7 days later. Elastic garments are continued for 3–6 weeks, allowing removal for bathing the second week; sleeping, the third; and inactive periods, the fourth. The drain(s) is/are removed in the first postoperative visit unless there is more than 50 cc of output per day. The patient returns within a week to 10 days for check on seromas or cellulitis. Most swelling is gone by 6 weeks, but final healing may not resolve for 6 months. Unless there is severe deformity, try to postpone revision procedures until then for a more accurate appraisal.

Power-Assisted Lipoplasty

The ease and speed of fat removal can be expedited by the adaptation of a power drill, lasers, or internal therapeutic ultrasound. Power-assisted lipoplasty (PAL) is a reciprocating cannula (powered by gas or electricity) that powers through tissue and vacuums out fat with minimal effort by the surgeon. The author is familiar with the Microaire (www.microaire.com/pal) device with a 2 mm excursion at 4,000 cycles/s. The equipment is highly expensive with only a small number of surgeons using it. It is purported to be a speedier and less injurious to the patient, causing less bruising, swelling, and discomfort, and thereby leading to more rapid recovery.[44] In a 15-patient pilot study, PAL had a higher per area suction removal capacity compared with traditional SAL with comparable aesthetic results.[38] The author finds that the PAL works smoothly with minimal bleeding, but the vibration and noise are annoying.

Laser-Assisted Lipoplasty

Recently a new laser energy devise called SmartLipo by Cybnosure, Inc. (Westford, Massachusetts) has been resurrected in the form of 1064 nm ND:Yag delivered by short pulses through a 600 µm optical fiber housed in a microcannula. Ten years ago laser-assisted lipoplasty was found to have no advantage over SAL.[3] SmartLipo appears best suited for smaller contour bulges. The cannula is inserted through a small incision and with the guidance of a red helium–neon laser source a liquefied fatty emulsion is created, which may be absorbed or suctioned. A smoother result with better skin contraction is claimed.[16] Direct to consumer marketing of this minimally invasive procedure has been very effective. The author is awaiting conformational studies in the United States.

Ultrasonic-Assisted Lipoplasty

In 20% of the surveyed cases, the most common high-tech energy source for liposuction is ultrasound vibration.[4] Introduced from Europe in the mid-1990s, UAL is the internal use of probes for cavitation and percussion to emulsify undesirable fat. The goal is aesthetic recontouring of all accessible regions of the body with maximal skin contraction and rapid recovery. Unquestionably UAL is physically easier than SAL on the surgeon.[5,35] It virtually glides through the tissue, with particular advantage with more fibrous tissues such as the male breasts, the back, and flanks or when stroking through the scarred subcutaneous tissue of secondary surgery. The vibrating probe appears to bounce off the scar tissue and seeks fat. Less force or thrust by the surgeon may mean a smoother, more consistent result. Vessel disruption with bleeding is rare. Larger volumes may be removed with less effort and no fatigue.

Introduced in 1994, the LySonix,® with inline suction has a new 3000 model with a pulsed mode to lower heat generation at the tip. MySonix (Framington, New York), www.misonix.com, produces this reliable machine for Mentor Corporation (Santa Barbara, California). About 9 years ago Sound Surgical Technologies, www.soundsurgical.com, produced the VASER®, a smaller diameter, multiringed probe that is more efficient at less energy than the LySonix. The sonic energy is increasingly splayed out from one to three rings. The greater the number of rings, the more diffuse is the sonic energy and the less is the thermal injury to the tissues. High resistance to the passage of the probe will prompt the use of a single ring. The VASER® mode rapidly cycles the probe on and off, keeping the rod from generating too much heat. The sonic energy is predominantly percussive. Having extra hand pieces are essential because shutdowns occasionally occur with either machine.

The probe is rhythmically passed through the subcutaneous tissue layer by layer. I prefer to start in the subdermal plane and proceed deeper in laminated planes. The tissues are coolest at the start with less chance of damage by the probe tip. The probe should be moved continuously as a motionless probe is not cooled and, hence will generate undesirable focal thermal injury. The entry points are placed in inconspicuous locations and asymmetrically staggered. The helping hand messages the target tissue to the probe, taking care to flatten curvatures of the dermis to avoid end hits. End hits are the thermal damage done to the underside of the skin due to force by the vibrating probe held in place with blanching pressure against the skin.

Evacuation of the emulsion is performed by more rapid-stroke liposuction. Sound Surgical Technologies provide a vented suction system, called Ventx®, which theoretically causes less damage to the connective tissue. When the threshold vacuum pressure is reached, the cannula releases the tissues, avoiding avulsion of connective tissue. Accordingly, the Ventx® cannulae rarely clog. For both UAL and traditional SAL, drains are used for very large-volume removals.

For those experienced in its use, UAL causes less morbidity and more rapid recovery.[13] The result is smoother than I can routinely achieve with SAL (Figure 10.1). UAL often results in good skin contraction, but that is not predictable. UAL is effective in correction of gynecomastia,

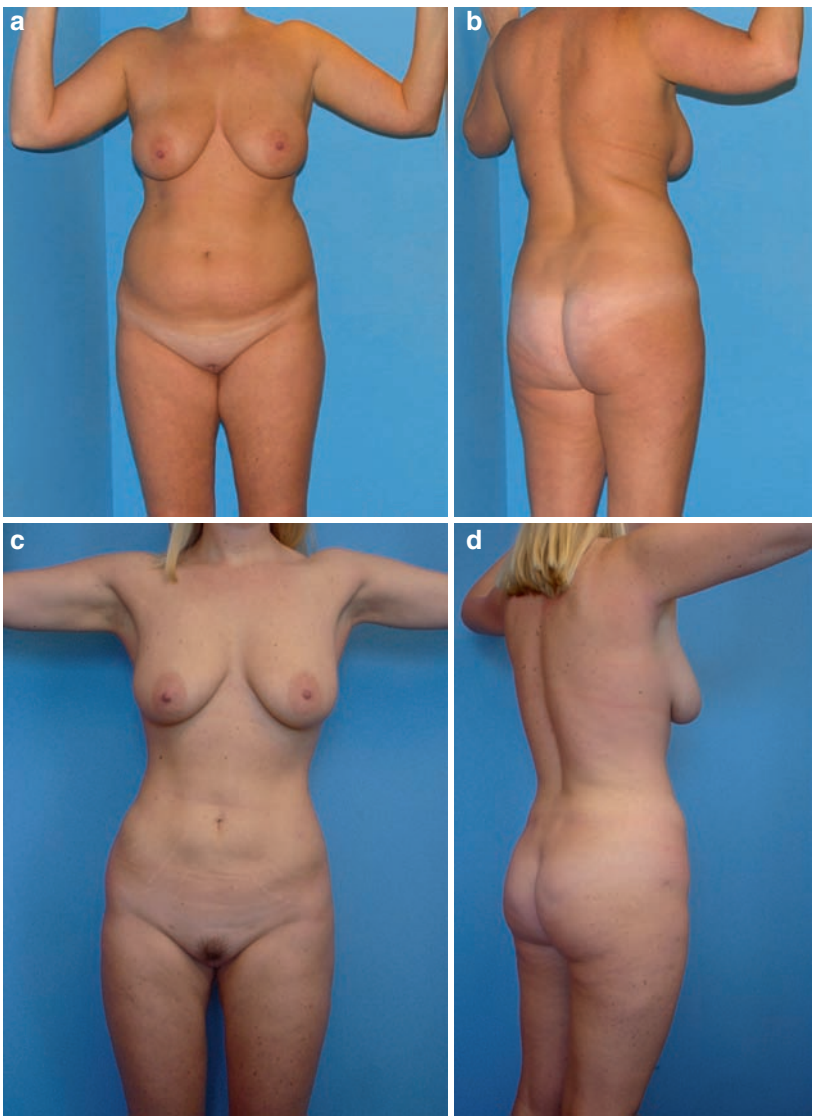

Figure 10.1. Cosmetic UAL. The before (**a** and **b**) and 5 months after (**c** and **d**) UAL (VASER) of the abdomen, flanks, hips, thighs, arms, and submental region in a 35-year old, weighing 160 lb (BMI 26). She was infused with 3,300 cc of saline with xylocaine and epinephrine. A two-ring probe with VASER was on pulse mode and she was treated for 30 min across the torso and 8 min for each inner and outer thigh. A total of 3,700 cc of fatty emulsion was aspirated via Ventx, of which 2,400 cc was removed from the torso and 800 cc from each thigh.

including mild ptosis. Broad UAL application extending over the anterior to the lateral chest and onto the abdomen with disruption of the inframammary fold leaves a smooth even contour. This bloodless operation is usually followed by a partial glandular excision pull-through removal.[20]

Clearly, over treatment leaves a leathery appearance due to subcutaneous scar and altered pigmentation. Furthermore, the incisions may be traumatized by torque and hot probe, end hits burn skin, and with resistance being the major end point, prolonged subcutaneous induration can occur.

The initial reports of blistering skin burns, skin loss, prolonged postoperative pain, and high seroma rates hampered the adoption of UAL.[15,35] The troubling pain may be due to blunt trauma, demyelination injury, or soft tissue scar contracture with entrapment. When neuralgia occurs, resolution of the pain takes months to even years. It is most likely to occur in the anterior thighs and flanks. These problems are avoided by using less power and emulsifying over shorter periods of time. The initial teaching with the inline suction was the end point of energy usage was the desired contour. This led to prolonged use of the probe, and higher power settings were then necessary. The current teaching is to use only as much power as needed to easily move the probe through the tissues. The power should be off when the probe is not moving. The end point of probe passage is when tissue resistance is low. The LySonix inline suction should be seen as a guide to the quality of aspirate only.

Although adipose is most sensitive, the destructive forces of ultrasound energy are not specific for fat. I believe there is a UAL system that comes closer to optimal fat emulsion and that is the Surround Aspirating System developed by El Hassane Tazi of Casablanca, Morocco. The vibrating probe is encased in a firm Teflon-coated cylinder that has a short, enclosed chamber at the end. High suction delivers the fat to this small chamber for rapid fragmentation and removal. Over the past 10 years, Tazi has repeatedly shown excellent fat removal with good skin retraction in large and very large-volume liposuction (Figure 10.2).[21] We are attempting to introduce this system into the United States.

Radio-frequency-Assisted Lipoplasty

The author is a co-investigator on a recently completed IRB approved trial on the safety and effectiveness of using bipolar radiofrequency internal probe energy for lipolysis tissue healing and skin tightening during lipoplasty of the arms, abdomen and thighs, with attention to technique and skin temperature the rapid removal of fat was followed by up to 30% contracture.

Large-Volume Liposuction

For the many patients with primarily contour excesses and inability to lose weight, an alternative is large-volume liposuction (LVL).[1,9,11,18,41] Regardless of the technique, LVL for obesity is controversial. Large amounts of fat removal is hours of trauma to the body. As mentioned, intraoperative and immediate postoperative care consider major fluid and electrolyte shifts and concerns of lidocaine and epinephrine toxicity.[41,42] Retained damaged tissue and bleeding may be a source for prolonged drainage and rare serious infection. Damage to connective tissue and neurovasculature reduction may prolong postoperative swelling and reduce the capacity for the skin to shrink down to the reduced volume. Inadequate contour improvement, sagging skin, and regain of weight are further problems.

Studies conflict on the medical improvement such as reduction of cardiac risk factors, blood pressure, and insulin levels. There are studies showing benefits of liposuction on cardiovascular risk factors, diabetes, and insulin requirements.[12,14,17,19] Italian researches prospectively studied the medical effects of LVL (average of 3,540 cc) in 30 obese (BMI 30 to 45) and 30 non-obese (under 26) subjects.[19] Their data showed than 2–3 l of fat removal is safe and associated with improvement of some metabolic (insulin resistance) and inflammatory (cytokines and CRP) markers, which are indicators of cardiovascular risk. In fact, a predominant part of the literature shows that LVL improved cardiac risk and vascular inflammatory markers, along with beneficial effects on reduced insulin resistance and vascular inflammation.[12] This health

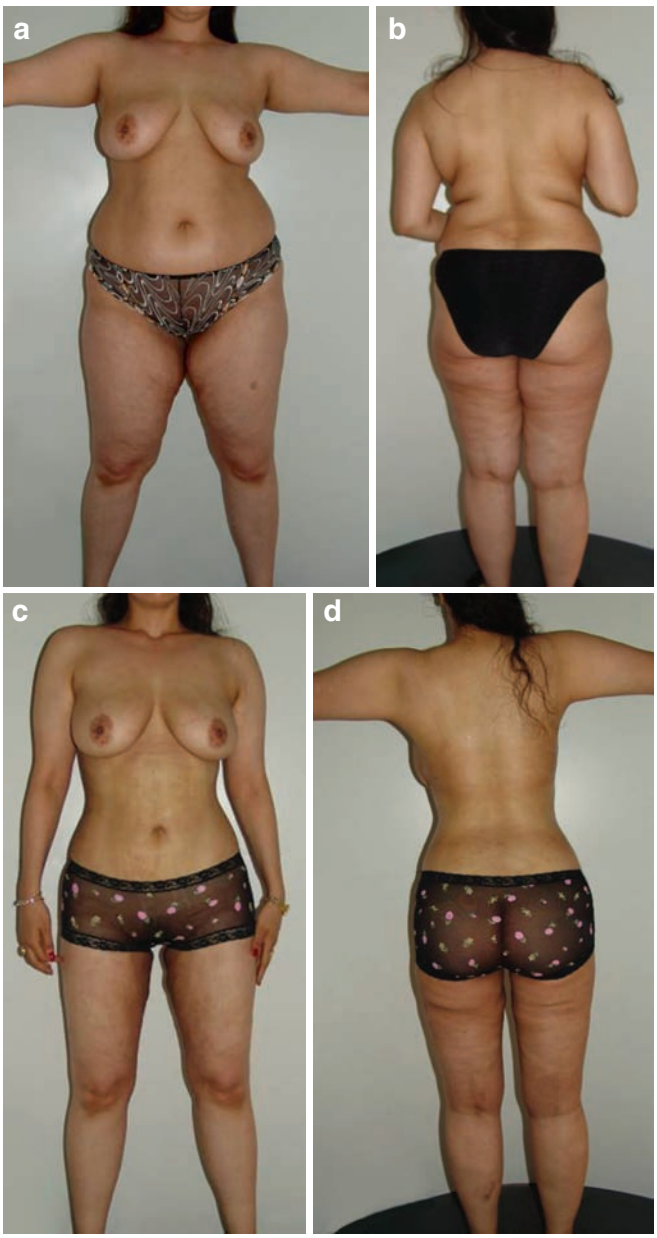

Figure 10.2. Large-volume liposuction as performed by Dr. Hassane Tazi with SAS/UAL. Preoperative (**a** and **b**) and postoperative (**c** and **d**) views after 17.5 l removal of fat.

improvement was due to reduced adipocyte reduction of adipokines such as Interleukin-6 and tumor necrosis factor alfa and increased production of anti-inflammatory adiponectin and interluekin-10. They suggest that plastic surgery should be incorporated into a multifaceted program of lifestyle changes for the obese for both contouring and health.[12]

A group from St. Louis reported in the *New England Journal of Medicine* no change in insulin action or risk factors for coronary heart disease in 15 women 3 months after lipoplasty of approximately 9.5 kg of fat from the subcutaneous tissues.[29] Patients who went on to lose further weight were eliminated from the study. Clearly, when suction of bulging fat encourages

an improved lifestyle with weight loss, there is an overall health benefit.

Body Contouring and Liposuction

Patients seeking generalized liposuction should be considered for excisional techniques if there is excess skin. Loose skin after weight loss and/or aging will sag further after liposuction. Excisional body contouring surgery improves body contours first by removing excess skin and closing tightly. Second, adipose is selectively left behind, transferred as a flap or removed by liposuction. With increasing presentation of overweight patients for body contouring, combining liposuction with body contouring has become more prevalent. Liposuction maybe performed in a neighboring region or within the advanced body contouring flap. Preservation of blood supply is critical to the safe healing of liposuctioned flaps, which are usually closed under considerable tension. Blood supply is preserved by minimal undermining of flaps and least traumatic fat suctioning.

Lockwood advanced the mid torso ideal of a contoured lateral trunk and inguinal region with a deep waist concavity, convexity of the hypogastrium, concavity of the epigastrium, and a valley between the rectus muscle bulges, with a vertically oriented umbilicus.[32] He advocated limited undermining during his high lateral tension abdominoplasty. After the abdominoplasty flap is closed, then liposuction is performed in the epigastrium. A remarkable flattening of the bulging tissue occurred. Other leading plastic surgeons have also advocated liposuction in conjunction with full abdominoplasty[10,23,33,36,40] (Figure 10.3).

Cardenas–Camarena declared his large-volume torso liposuction (defined as > 1,500 ml, mean of 4.3 kg) and extensive abdominoplasty (mean pannus resections of 1.3 kg) as safe and effective[8] (http://online5.hsls.pitt.edu:5551/gw2/ovidweb.cgi - 79#79). Complications included seromas in three patients, a dehiscence, and one distal flap necrosis.

In 2001, Brazilian plastic surgeons emphasized abdominoplasty without surgical undermining.[37] Instead, flap mobility is enhanced by discontinuous undermining provided by liposuction. French plastic surgeons have advanced excision site profound liposuction as a means to best preserve deep subcutaneous tissue lymphatic channels, thereby reducing the seromas and lymphocele formation during abdominoplasty, brachioplasty, and vertical medial thighplasty.[31,34]

Over the past 4 years, I have adopted these principles of lipoabdominoplasty with minimal undermining of flaps, maximal preservation of underlying connective tissue, and vascularity throughout all body contouring operations. The results are most dramatic in the overweight patient. On the other hand comes a point at which the adiposity is too excessive to allow for combined therapy. At that extreme, the region is tensely swollen with fat and has no discernable laxity. In those patients, UAL may be all that is necessary or just the first stage of contour correction.

Rohrich is a proponent of concomitant, moderate-volume liposuction with abdominoplasty.[36] His group averages 4 l of UAL (up to 8,450 cc) of the upper lateral flank, lower back, and upper buttocks, hips and medial thighs, and entire abdomen during his central body lift, circumferential body contouring in 151 patients. He reports high patient satisfaction, excellent results, and a low rate of complications: three major (2%) (two deep vein thrombosis and 1 PE) and 32 minor (21.1%). Surgical revision was required in eight patients (5.3%).[36]

In fact, Lockwood believed there is a safety factor in discontinuous undermining of the abdominal flap by means of liposuction. He felt it was almost as efficacious as direct undermining with the advantage of improved blood supply[32]. The ultimate test of this approach is the Brazilian lipoabdominoplasty[37]. All undermining and fat removal are by liposuction, and only skin is excised. Others have reported that the combination of multiple trunk excisional procedures with liposuction do not have a greater number of complications than isolated abdominoplasty[36]; however, obese patients have significantly increased morbidity regardless of the number of procedures.[10]

Cardenas–Camarena combined abdominoplasty and circumferential liposuction in 310 women patients during a 7-year period, yielding excellent body contouring in a single surgical procedure. There were less than 25% complications even in mostly overweight women.[7] Gentle manipulation of the tissues prevents cutaneous compromise.

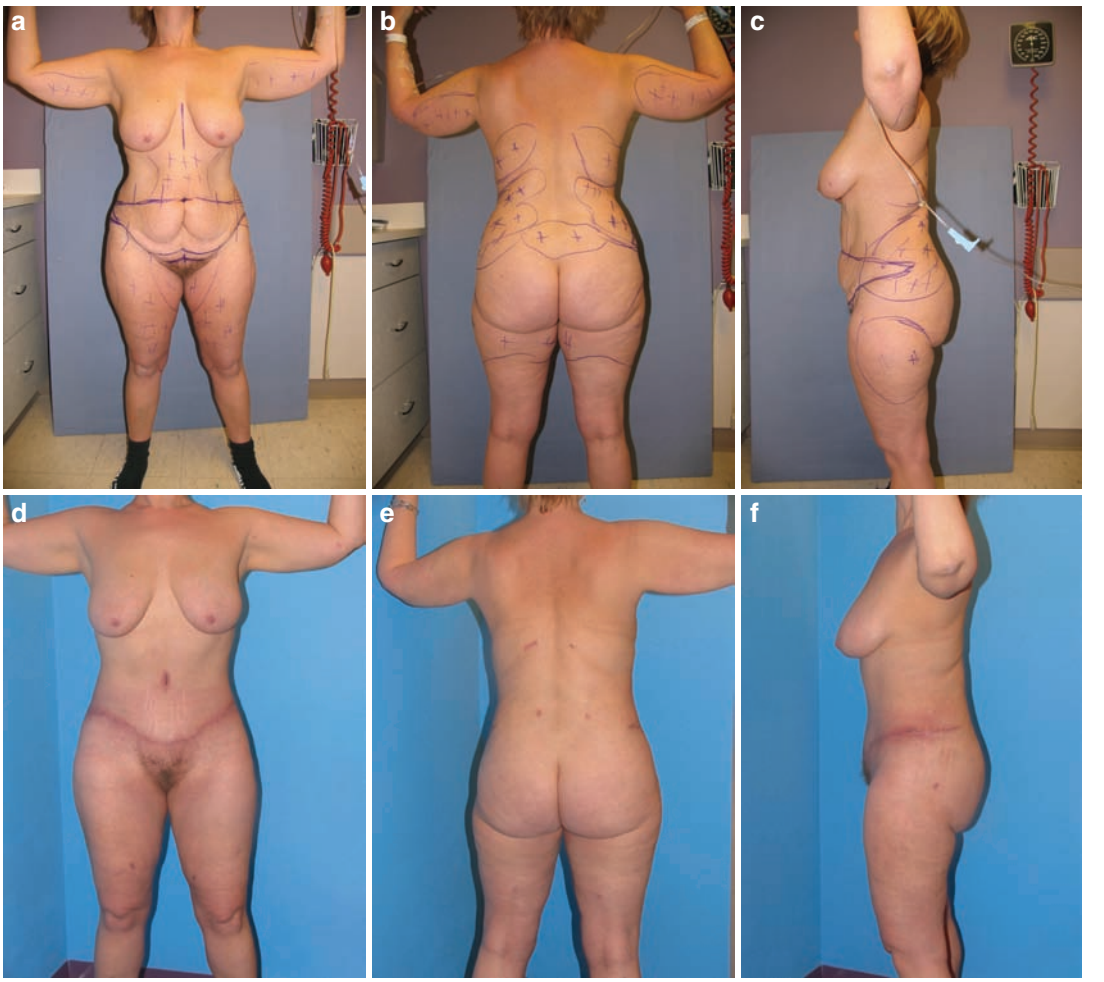

Figure 10.3. Combining body contouring with UAL. The before (**a**, **b**, and **c**) and 1 month (**d**, **e**, and **f**) after UAL (VASER) of the flanks, abdomen, and flanks with an extended abdominoplasty in a 44-year old, 148-lb woman.

I agree with this author's conclusion that the combination should be used not as a means to lose weight but as a surgical procedure that improves the body contour in patients with differing degrees of obesity.

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11

Biomaterials in Craniofacial Surgery

Earl Gage, Claude-Jean Langevin, and Frank Papay

Summary

Bone substitutes are increasingly used in craniofacial surgery. This chapter discusses the characteristics of an ideal bone substitute and briefly reviews the evolving history of the biomaterials with a particular emphasis on craniofacial reconstruction. Some of the most important bone substitutes, including calcium phosphate and hydroxyapatite (HA) ceramics and cements, bioactive glass and polymer products, are discussed. Areas of active research and future directions include tissue-engineered products and an increasing emphasis on bioactivity of the implant material.

Introduction

Bone substitutes are increasingly used in craniofacial surgery. This trend stems, in part from their ease of use and handling, improved safety profiles, intraoperative cost and time advantages, and their adaptability to a variety of clinical challenges. A wide variety of bone substitute materials have been developed during the last 50 years. Some of the most commonly used bone substitute materials are summarized in Table 11.1. Biomaterials used in the osseous reconstruction of the craniofacial skeleton can be broadly categorized into calcium phosphate-based ceramics and cements, synthetic polymers, and, more recently, tissue engineered bone substitutes. This chapter reviews some of the most important biomaterials in each of these categories.

Properties of an Ideal Bone Substitute

The ideal bone substitute should have a number of important properties. First, it should be biocompatible. A substance that is biocompatible is well tolerated by the host and does not evoke an adverse inflammatory response. Second, the ideal bone substitute should be easily molded to the bony defect it is intended to fill and have a fast setting time. Third, it should be durable, holding its shape and volume over time. Fourth, it should be radiolucent to allow radiographic assessment. The ideal bone substitute should also be thermally nonconductive, bioactive, sterilizable and readily available. Finally, in our era of skyrocketing healthcare cost, the ideal bone substitute should be inexpensive to purchase and use.

Some additional basic terminology is useful in discussing the biologic characteristics of bone substitutes. A substance that is osteoconductive is one that provides a conductive surface for bone growth. A bone substitute that is osteoinductive has the capacity to induce osteogenesis by stimulating immature cells to become prosteoblasts. Finally, osseointegration refers to stable anchoring of an implant material to the surrounding bone.

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Table 11.1. Commonly used calcium-based and polymer bone substitutes.

Compound	Sample of commercial products
Hydroxyapatite	Pro-Osteon, Bio-Oss, Endobon, Calcitite
Tricalcium phosphate	Vitoss
Hydroxyapatite cements	Norian CRS, Bone Source, Mimix Bone Void Filler
Bioactive glass	NovaBone
Methylmethacrylate polymer	Hard Tissue Replacement
Porous polyethylene polymer	Medpor

History of Bone Substitution

Earliest Origins of Bone Substitution

Although the last 50 years have seen the most significant and accelerated advances in biomaterial technology, the roots of bone substitution reach back to ancient civilizations. The earliest evidence of implanted material used to fill bone defects can be traced to ancient Peruvian civilizations. Trephination, the oldest known surgical procedure, was practiced as early as 3000 BC by pre-Incan surgeons, and there is evidence to suggest that a variety of alloplastic materials, including gourds and metallic plates, were used to fill trephination defects as long ago as 2000 BC. The first recorded description of cranioplasty using a gold plate to cover a cranial defect has been attributed to Fallopius in the 16th century.[56]

The 17th Century

In the 17th century, Job Janszoon van Meekeren, a surgeon from Amsterdam, was the first to report a bone xenograft in cranial reconstruction.[35,56] Although not the surgeon who performed the procedure, van Meekeren reported the case of a Russian nobleman who sustained a sword blow to the head, which resulted in a soft tissue and cranial bone defect. This was reconstructed using canine calvarium, and the patient made a full recovery. Ironically, the patient was later threatened with excommunication from the Christian church for having been defiled by the canine xenograft.[56]

The 18th and 19th Centuries

The 18th and early 19th centuries saw an increased understanding of the physiology and dynamic nature of bone healing as documented by the work of Ollier, Barth, Axhausen and others. Attempts to cover craniotomy defects with autogenous tissue began in earnest at the beginning of the 20th century with a variety of tissues being used to fill bony defects, including split cranial grafts, autogenous bone chips, split rib grafts and temporoparietal fascia, to mention only a few.[56]

The Early 20th Century

The first half of the 20th century is also notable for expanded attempts to find alloplastic materials for use in craniofacial reconstruction. This effort was driven, at least in part, by wartime injuries during World Wars I and II. A variety of metallic alloplastic implants were trialed during this time, including gold, silver and aluminum, continuing work begun at the end of the 19th century and beginning of the 20th century by researchers such as Booth, Curtis, Gerster and Sebieau.[56] Additional early metallic implants included lead, platinum and various other alloys. Perhaps the most important alloy for its strength, low cost, inertness and relative radiolucency is titanium, which was developed for use as a craniofacial alloplast in the 1960s, as first reported by Simpson.[56,57]

The Mid 20th Century to the Present

Methylmethacrylate, an acrylic resin, was first introduced as a bone substitute during the 1940s and remains a popular choice for cranial reconstructions for its strength, moldability, low cost and relative radiolucency.[56] Since its initial introduction into the surgeon's armamentarium, methylmethacrylate has been combined with various metallic meshes to facilitate fixation and provide additional strength. The latter half of the 20th century has also seen the evolution of hydroxyapatites and calcium phosphate-based cements and ceramics and more recently efforts to develop tissue-engineered products that incorporate bone growth factors and mesenchymal stem cells. The most important of these bone substitutes are discussed in more detail below.

Calcium Phosphate-Based Compounds

Hydroxyapatite (HA) Ceramics

Hydroxyapatite [$Ca_{10}(PO_4)_6(OH)_2$] is a calcium phosphate compound that is the primary mineral component of teeth and bone. For the last 30 years, it has found increasing use in craniofacial and orthognathic surgery for filling bony defects and smoothing bony contour abnormalities. Initially developed as a potential bone substitute in the 1970s, early forms of HA consisted of preformed ceramic products. Created through a heating process called "sintering," these early ceramic forms of HA consisted of thermally fused crystals and were notable for their strength, biocompatibility and stability against resorption.[14] However, although these ceramics were well tolerated after implantation, they were awkward to handle and shape and, as a result, had applications limited primarily to a few orthognathic and dental surgery procedures.[14,39,40]

In 1991, Costantino et al.[14,27] were the first to characterize and use a new hydroxyapatite cement paste as a bone substitute in a cat model. This form of hydroxyapatite differed from the earlier ceramics in its preparation and did not require the sintering process. HA cement paste is prepared by mixing tetracalcium phosphate and dicalcium phosphate with aqueous solution, resulting in an isothermic reaction that yields hydroxyapatite.[10] Subsequent investigational studies have shown that setting time can be accelerated by addition of a sodium phosphate buffer solution.[10,14] Although not as strong as HA ceramic and demonstrating more bioresorbability, HA cement paste is osteoconductive,[14] easy to prepare and apply in the operating room and highly sculptable, allowing its use in filling a variety of defect shapes. Disadvantages include its brittleness and insufficient strength for load-bearing applications. Commercially available HA cement products are discussed below.

HA ceramics comes in both naturally occurring and synthetic forms. Clinically available naturally occurring forms of HA include the coral-based products Interpore and Pro-osteon (Interpore International, Inc – Irvine, CA) as well as bovine-derived products such as Bio-Oss (Geistlich Biomaterials – Geistlich, Switzerland), Osteograf-N (CeraMed Co – Denver, CO) and Endobon (Merck Co – Darmstadt, Germany). Synthetic HA products include Calcitite (Sulzer Calcitek – Carlsbad, CA). HA ceramics comes in a variety of forms including granules and porous blocks. In addition, HA is frequently used as a coating on orthopedic and dental implants to promote bony in-growth.[44]

Carbonate- and Silicon-Substituted Hydroxyapatite Ceramics

Although HA accounts for nearly 70% of the mineral content of teeth and bone, the naturally occurring HA in the human body exists in a substituted form wherein carbonate and silicates, among other ions, may replace hydroxyl or phosphate groups of the apatite structure. Basic science investigators have attempted to produce carbonate- and silicon-substituted synthetic HA in an effort to produce HA that more closely resembles the mineral content of native bone and simultaneously enhance bioactivity and new bone in-growth.[3]

Carbonate substitution can occur at the hydroxyl position, the phosphate position or both within the apatite structure resulting in types A, B or AB carbonate hydroxyapatite (CHA), respectively.[3,43] Carbonate substitution produces a ceramic product that is more dense and able to be sintered at lower temperatures, approximately 200°C[31,54] compared to stoichiometric HA, which is sintered at temperatures between 600 and 1,300°C.[14,44] Additionally, the resultant crystal size of CHA is smaller, with a crystalline structure that is superior to noncarbonate-substituted HA and is more bioresorbable.[31,54] The benefit of CHA appears to lie in its biologic properties, which may promote more osseointegration compared to nonsubstituted HA. Despite its increased hardness, attributable to its greater density and smaller crystalline granule size, it still should not be considered for high load-bearing applications.[31]

Silicon dioxide (SiO_2) is thought to be important in bone formation and calcification.[6,7,30] This has led researchers to postulate that the bioactivity of HA might be enhanced by substitution of silicates into the apatite structure in ways similar to the enhanced bioactivity observed in CHA. Multiple attempts have been made to produce a silicon-substituted HA (Si-HA) with only

modest success.[4,45,55,59] One of the more successful approaches produced incorporation of a small amount of silicon (0.4 wt%) into HA via an aqueous precipitation reaction done at room temperature as described by Gibson and colleagues.[30] Si-HA is postulated not only to enhance osteoclastic absorption at the bone-scaffold interface but also to potentially promote osteoblast activity.[36] Although early studies regarding enhanced bioactivity in Si-HA ceramics have been encouraging, more studies are needed.

Although there are few CHA or Si-HA products in clinical use at this time, hydroxyapatite substitution is likely to remain an active area of research.

Tricalcium Phosphate [$Ca_3(PO_4)_2$]

Tricalcium phosphate (TCP) is synthetically created by sintering precipitated calcium-deficient apatite with calcium phosphate in a ratio of 1.5. That ratio is less than the Ca:P molar ratio of 1.67 found in HA.[44] TCP is more soluble than HA as a result of its small granule size and porosity. A pure TCP product is commercially available as Vitoss (Orthovita, Inc – Philadelphia, PA). This product is engineered to resemble cancellous bone and to fill traumatic cancellous bony defects. This particular product has not found broad use in craniofacial reconstruction due to its rapid dissolution, but many other calcium phosphate and HA cement products are widely used.

Hydroxyapatite Cements

As mentioned previously, HA cements were first offered for clinical use in the early 1990s. Since then, calcium phosphate cements have become one of the most versatile bone substitutes for filling calvarial defects and smoothing contour defects of the facial skeleton (Figure 11.1a–e). These calcium phosphate-based cements do not have sufficient tensile and compressive strength to be useful in load-bearing applications, but their sculptability has made them extremely useful in addressing bony contour irregularities. A comparison of the three most commonly used HA cements is summarized in Table 11.2.

Norian CRS bone cement (Synthes – Paoli, PA) is a moldable calcium phosphate cement that is mixed in vivo and forms dahllite, a carbonated apatite, once set. Originally approved by the FDA for use in distal radius fractures,[26] it has found broad application in craniofacial surgery as well. Norian CRS bone cement is prepared by mixing sodium phosphate solution with calcium powder to form a putty. This putty begins to harden in 2 min in a mildly exothermic reaction that may reach as high as 42°C and is set in 10 min. Maximum compression strength is reached in 24 h.[26] Norian is absorbed over time and is not intended for use in load-bearing applications or in the presence of active infection.

Bone Source (Stryker-Leibinger – Kalamazoo, MI) is a self-setting calcium phosphate cement originally approved for use in filling burr holes and for facial skeleton augmentation.[22] It is prepared by mixing calcium phosphate salts in a sodium phosphate buffer to form a putty that remains moldable for approximately 20 min. Bone Source hardens into hydroxyapatite, and, like other hydroxyapatite cements, is very slowly absorbable over time. It is not intended to fill defects over 25 cm^2 and lacks sufficient strength for load-bearing applications.

Mimix Bone Void Filler (W. Lorenz Surgical – Jacksonville, FL), like Bone Source, won FDA approval for use in filling burr hole and craniotomy defects and in smoothing facial skeletal contour abnormalities over a surface area no larger than 25 cm^2.[23] This cement product is prepared by mixing dry components of calcium phosphate powder and sodium citrate dehydrate with an anhydrous citric acid solution. As it cures, Mimix hardens into hydroxyapatite and is mildly exothermic. Mimix Quickset is rapidly prepared, remains malleable for 3–4 min and is completely set in 4–6 min, offering a potential advantage over other commercially available HA cement products that take longer to set and cure.[32]

Since the introduction of HA cement products in the mid 1990s, they have found broad use and application in reconstructing cranial, facial and orbital defects. Overall results have been excellent. The largest review of experience using HA cement in craniofacial reconstruction was published by Burstein and colleagues[5] in 2006. They reviewed 150 patients who underwent orbitocranial reconstruction using Bone Source and Mimix HA cements over a 7-year period of time. The majority of patients were reconstructed using an onlay technique with or without adjunctive absorbable or titanium mesh. The average amount of cement used was 26 g with a range of

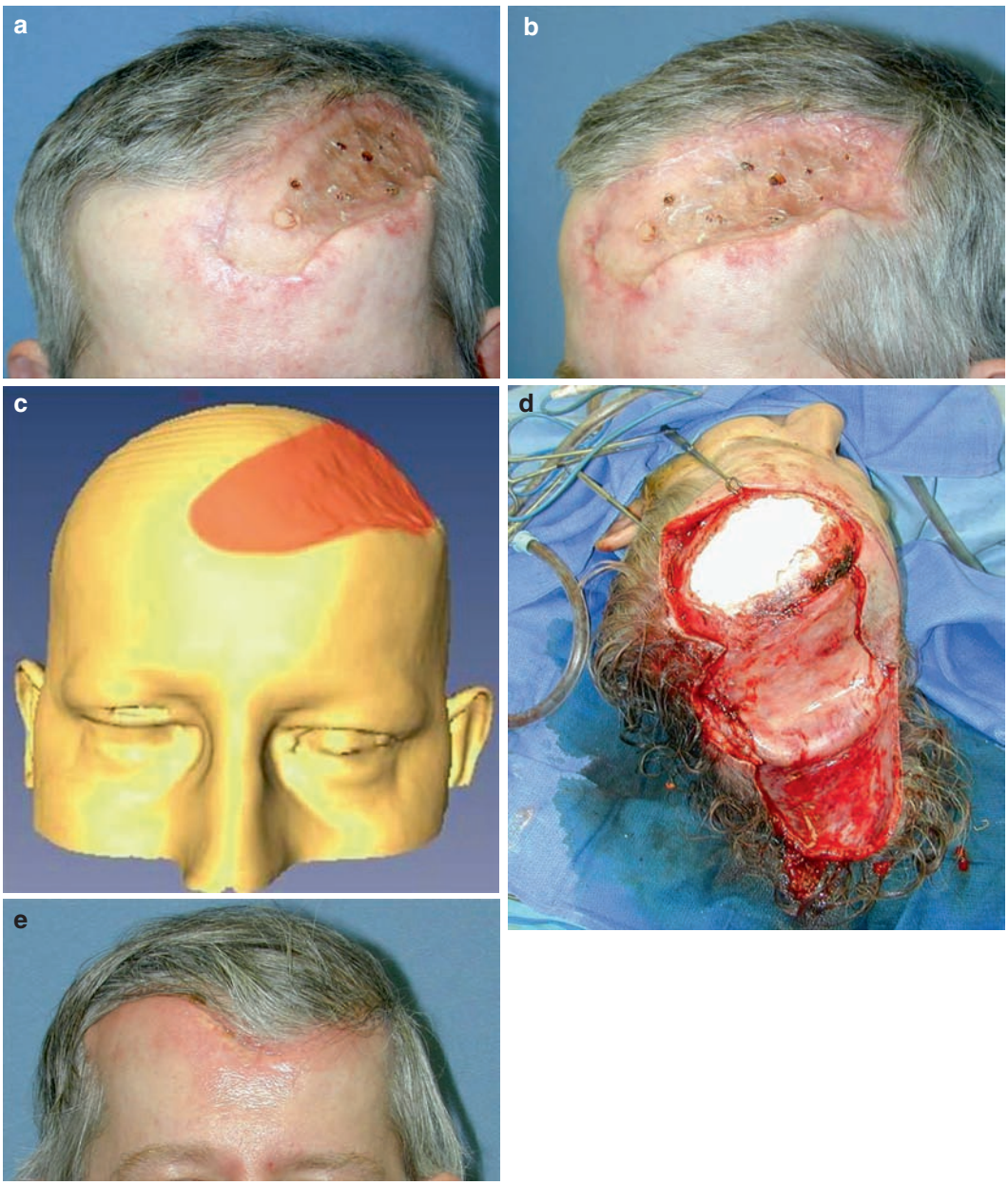

Figure 11.1. (**a**) Anterior view of post-traumatic calvarial defect in a 48-year-old male. (**b**)The same patient is seen from an oblique angle. (**c**) Preoperative assessment of the calvarial defect included high-resolution, three-dimensional computed tomography to quantify the size of the defect. (**d**) Intraoperative view of the application of the BoneSource bone cement to the calvarial defect after first stage tissue expansion of the adjacent scalp. (**e**) Postoperative result after recontouring of the calvarium with bone cement and advancement of the expanded adjacent scalp soft tissue. (Photographs courtesy of Dan Medalie, MD.)

8–125 g. Excellent results were reported, with 92% of patients in the study having a satisfactory contour result over a minimum follow-up of 1 year (mean follow-up, 26 months). Seven patients had a seroma that required aspiration in the first week following surgery. Four patients developed chronic seromas, and three of them required reoperation for removal of microfragmented HA

Table 11.2. Comparison of the properties of calcium phosphate cements.

	Norian	Bone Source	Mimix
Base component	Monocalcium phosphate, tricalcium phosphate, calcium carbonate	Tetracalcium phosphate	Tetra-tri-calcium phosphate
Compression strength (psi)	4,350	7,396	3,300
Resorbability	Complete	Minimal	Minimal
Pore diameter (μm)	0.03	0.002–0.005	211
Initial set time (min)	10	10–15	3–4
Final set time	1 h	4 h	4–6 min
Sets in a moist environment	Yes	No	Yes
Osteoconductive	Yes	Yes	Yes

cement. One patient required reoperation for overcorrection of an orbital contour abnormality, and another patient had to return to the operating room for removal of a postoperative drain that had become adherent to the HA cement. The overall complication rate was 9%. No infections were reported.

Other series have reported much higher rates of infection/exposure.[2,33,51,62,65] Moreira-Gonzalez et al.[51] found that infection or extrusion occurred in 22.4%, with an increased likelihood of infection when reconstruction is undertaken in the vicinity of the frontal sinus. Increased rates of infection when the frontal sinus is involved are corroborated by Verret et al.[62] In their review of 102 patients undergoing craniofacial reconstruction for traumatic and malignancy-related defects, they found a 12% rate of infection/foreign body reaction requiring implant removal with tissue irradiation and frontal sinus involvement, both increasing the risk of these complications. When irradiated patients were not considered, an infection rate of approximately 5% was noted.

Microfragmentation has also been reported as a complication of HA cement use.[2,5,47,65] Losee et al.[47] attribute microfragmentation to brain pulsations in cranial reconstruction and suggest that this risk can be mitigated in large defects by including a mesh as an adjunct to cement use. Combined use of both titanium and absorbable mesh products with HA cements has been shown to be safe and effective by several authors[15,29,47] (Figure 11.2a–d). The choice of HA cement may affect the rate of microfragmentation, with Norian having recently been shown to have the highest mean fracture force for fracturing a standardized test piece.[50] Zins et al.[65] reviewed 121 patients undergoing craniofacial reconstructions using Norian and Bone Source with and without mesh adjuncts and found an overall major complication rate of 15%. However, in the subset of patients undergoing reconstruction for large (>25 cm^2) defects, they report major complications in 10 of 16 patients (62.5%). Of these, 2 represented minor contour problems that required re-operation, 3 had fragmentation with infection and 5 had fragmentation without infection. As a result of these findings, Zins recommends autogenous reconstruction instead of HA cement for large cranial defects, even if a mesh is used.

Bioactive Glass

Bioactive glass is a synthetic, osteoconductive silica-containing particulate bone filler, which forms an osteoconductive apatite layer at the bone–implant interface, enhancing bone attachment and promoting new bone growth.[10] Collagen, mucopolysaccharides and glycoproteins are recruited from the adjacent bone and facilitate early bonding of the bioactive glass with surrounding bone. Once mature, this bond has been shown to be stronger than the native bone itself, with fracturing more likely to occur within the native bone or within the bioglass substance rather than at the interface between the two.[10,42] In addition to its osteoconductive properties, bioactive glass has also been noted to be osteoinductive as the bioactive surface becomes coated with osteogenic stem cells in response to the controlled release of soluble silicon from the glass surface.[34,63]

NovaBone (Porex Surgical – College Park, GA) is a commercially available bioactive glass intended for filling of surgical or traumatic bone voids.[25] It is composed of 45% silica dioxide, 45% sodium oxide, 5% calcium and 5% phosphate.[10] NovaBoneAR, a second-generation NovaBone

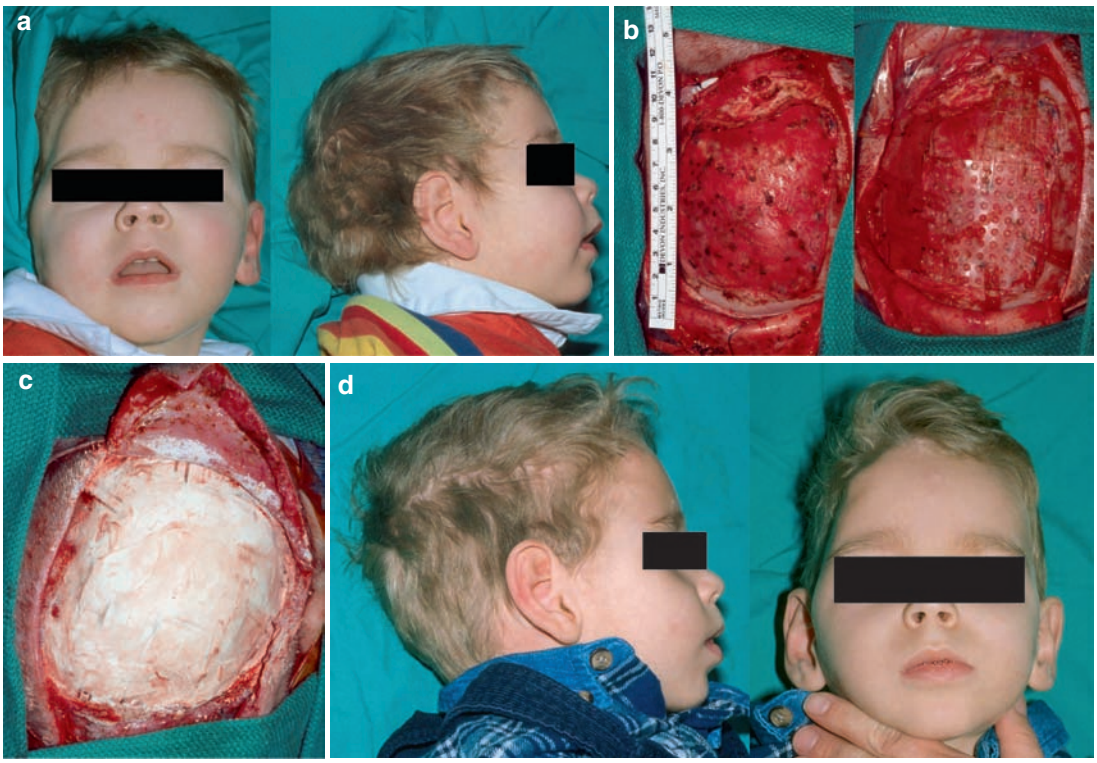

Figure 11.2. (**a**) A toddler with a tight temporoparietal bone defect as seen from anterior and lateral views. (**b**) Intraoperative views demonstrating the calvarial defect (*left*) and the defect as it appears after partial reconstruction with an absorbable plating system (*right*). (**c**) Hydroxyapatite cement is next applied over the absorbable plate and allowed to set. (**d**) Postoperative anterior and lateral views demonstrating improved cranial contour.

product, is composed of two components – a slowly absorbing, melt-derived, calcium phospho-silicate bioglass component and a more rapidly absorbed, solution-gelation, calcium phospho-silicate component. The latter component is more rapidly absorbed, leaving more space for bone infiltration in the interstices between the more slowly absorbed melt-derived component. NovaBone thus acts as a scaffold for new bone in-growth and is substantially resorbed within 6 months. NovaBone Putty[24] is similar to NovaBoneAR with the exception that the bioglass particulate material is mixed with a gelatin binding agent to form a malleable putty that can be gently packed into osseous defects.[14] The gelatin component will reabsorb over time leaving the osteoconductive bioglass matrix to promote bone in-growth. NovaBone is not intended for heavy load-bearing applications before hard-tissue in-growth has occurred.

Gosain et al. have reviewed the role of bioactive glass in craniofacial surgery, detailing its use in periodontal, alveolar, orbital floor, maxillofacial and cranial applications.[10,34] Bioactive glass has been mixed with autogenous bone particles as well as demineralized bone matrix, resulting in accelerated bone healing time compared to bone grafting alone in some craniofacial applications.[13,34,58,63] Complication rates from use of bioactive glass in craniofacial reconstruction are not well delineated in the literature but may be as high as 20%.[16]

Polymers

Polymers are extensively used in both bone and soft tissue reconstruction. There are a variety of polymer products in use clinically, including polytetrafluoroethylene (PTFE), polyesters,

polypropylene, nylon, silicone, polymethylmethacrylate (PMMA) and polyethylene. Not all of these polymers have applications and uses in craniofacial reconstruction. This chapter focuses on the most important ones with craniofacial applications.

Polymethylmethacrylate

Polymethylmethacrylate is an acrylic-based resin, which has broad medical and nonmedical uses. It may be prepared as a cement by mixing powdered methylmethacrylate polymer and liquid methylmethacrylate monomer, which polymerize in an exothermic reaction. PMMA also comes in block form. PMMA cement has been used for many years to secure orthopedic prosthetic devices and to fill craniofacial defects. This polymer is hard, biologically inert and minimally reabsorbed. It is also relatively inexpensive and easy to obtain.[10,48] Disadvantages of its use include its lack of bioactivity, excessive heat associated with the polymerization reaction, lack of remodeling or replacement by bone in-growth and lifelong susceptibility to infection or extrusion.

Hard Tissue Replacement (HTR) (Walter Lorenz Surgical, Inc., Jacksonville, FL) is a PMMA product that is fabricated by sintering a polyhydroxyethyl and calcium coating over a PMMA core. The polyhydroxyethyl and calcium coatings interface with surrounding tissue, whereas the PMMA does not. The outer coating imparts hydrophilicity, an extensive porosity (150–350-μm interbead pore size and a 200-μm intrabead pore size, which results in a 20–30% material porosity), a negative surface charge (−8 to −15 mV) and substantial compressive strength (50,000 lb/in.^{2} in particulate form and 5,000 lb/in.^{2} in molded form) despite its porous nature.[18,19] HTR alloplastic implants are preconstructed based on high-resolution computed tomography models of the bone defects. The custom-made implants come packaged sterile for immediate use in the operating room and provide an out-of-the-box implant that can fit any number of complex-shaped defects.

Advantages of the HTR include good strength, durability, surface osteoconductivity, biocompatibility and some degree of tissue in-growth and revascularization. Additionally, there is no need for intraoperative mixing of reagents or waiting for cements to set and cure, leading to decreased OR time. Disadvantages include lifelong risk of infectious complications, since it is not entirely reabsorbed, and the need to plan procedures well in advance to allow time for prefabrication.

Eppley has written extensively regarding the clinical use of PMMA and HTR in craniofacial reconstruction.[18–21] In 1990, he evaluated HTR polymer in reconstructing cranial defects in a rabbit model using both inlay and onlay grafts.[19] Histologic evaluation was performed at 60, 120 and 240 days. He reported the HTR polymer material to be biocompatible, with no evidence of infection, inflammatory reaction or bone resorption observed around any of the implants. He also noted that the best bony in-growth and osteoconductive effects were observed when implants were exposed to bleeding cortical marrow as inlay grafts.

Eppley also reviewed his experience in seven patients who had cranial reconstructions using preformed PMMA implants constructed based on 3D computed tomography data.[21] The anticipated defect was calculated preoperatively and the implant fabricated according to those calculations. Intraoperatively, Eppley reports the need for minor modifications based on discrepancies between the predicted and actual bony defects after tumor excision. These discrepancies most often were the result of the actual defect exceeding the size of the implant. The discrepancies were managed by using HA bone cements to fill the defect or by using the sterile back-up of the implant as an addition to the original implant. In cases where the frontal sinus was in proximity to the implant, it was either cranialized and obliterated with a pericranial flap or obliterated with hydroxyapatite cement. Eppley reported excellent cosmetic results and no complications with a minimum 1 year follow-up.

More recently, Eppley looked at hardness of the various forms of PMMA, including intraoperatively cured and preformed implants fabricated to thicknesses approximating that of native bone. Mean failure weights were reported of 3.9 lb (Cranioplast), 4.2 lb (Cranioplexx), and 4.0 lb (HTR polymer). He concluded that all forms of PMMA compare favorably with native bone in terms of measured impact resistance.

Infection rates for methylmethacrylates used in cranial reconstruction have been estimated at 5%, with the risk of infection rising when the frontal sinus and nose are reconstructed.[10,48]

Porous Polyethylene

Medpor (Porex Surgical – College Park, GA) is a biocompatible, porous, high-density polyethylene that has been used extensively in orbital reconstruction and facial recontouring for the last 20 years.[64] Coming in sheets, blocks or preformed shapes, Medpor's high degree of porosity, with an average pore size of 100 µm and pore volume of around 50%, promote tissue ingrowth. The material is flexible enough to bend yet rigid enough to cut sharply and has good handling characteristics. Medpor alloplastic implants may be placed subperiosteally and may be adequately secured in place by reapproximation of periosteum and soft tissue over the implant. Alternatively, titanium or absorbable screw fixation may be used. Newer generation Medpor products are also available, with titanium plates extending from the periphery of the implant to allow easy screw fixation as well as products that have a titanium mesh incorporated within the polymer for additional structural support.

Yaremchuk[64] has reviewed his Medpor experience with 370 implants in 162 consecutive patients over 11 years. Implants were placed for a variety of acquired, aesthetic and congenital craniofacial deficits. All implants were placed in a subperiosteal plane and most of them were secured using titanium screw fixation. The author reported infection complications in 3% (n = 4) and an overall reoperation rate of 10% (n = 16). Among those that underwent reoperation, nine underwent recontouring procedures, three operations were performed to remove the implant at the patient's request, and four had operations for infection. No implant extrusions were reported.

Cenzi[8] also reported a series of 285 Medpor grafts in 187 patients placed between 1992 and 1999. Grafts were used almost exclusively for craniofacial reconstruction and were placed as both onlays and inlays. Over a mean follow-up of 60 months, Cenzi reports a global complication rate of 6.3%, with implant exposure and infection being the most common. Risk factors for implant extrusion and infection included placement in the maxilla or ear and placement in areas where soft tissue coverage was thin and/or scarred from irradiation or previous surgeries.

Menderes et al.[49] reviewed their experience reconstructing craniofacial defects using 83 high-density porous polyethylene implants in 71 patients between 1996 and 2003. Grafts were placed for malar/infraorbital (n = 30), mandibular (n = 14), temporofrontal (n = 13), paranasal (n = 4) and maxillary alveolus augmentation (n = 2) as well as ear reconstruction (n = 3). Subperiosteal placement was performed in the vast majority of patients, and fixation was accomplished using titanium screws, absorbable screws and miniplates or stainless steel wire circulage. At a minimum of 1 year follow-up, the authors report that 7 patients (9.8%) required a second intervention. Three of the seven secondary interventions were for contour alignment, and four interventions (5.6%) were for extraction of the implants because of extrusion or infection. Menderes concludes that use of porous polyethylene is safe, easy and effective and associated with low morbidity.

Tissue Engineering

The rapidly expanding field of tissue engineering in the context of bone substitutes seeks to combine the stimulatory effects of bone growth factors, such as bone morphogenetic protein-2 (BMP-2) and osteogenic protein-1 (OP-1), with bone substitute carriers to provide structural support during healing, deliver critical growth factors to the fracture site and promote more rapid bone growth and healing. Bone mesenchymal cells have also been explored as a potential component in engineered bone substitutes for similar reasons. Potential delivery systems have included demineralized bone matrix, collagen composites, fibrin, calcium phosphate, polylactide, polylactide-co-glycolide, polylactide-polyethylene glycol, HA, dental plaster, titanium and bioglass.[9,11,12,17,28,37,38,41,46,52,53,60,61] Much of the work in these areas remains preliminary but underscores the increasing emphasis not only on the physical properties of the implant material but also on the biologic effects on new bone growth.

The Future of Biomaterials

The early history of bone substitution in craniofacial surgery emphasized the physical properties of the material itself, such as inertness, malleability and strength, among many others. Over the last 30 years, the science of biomaterials

and bone substitution has focused increasingly on the biologic interactions of the implant materials with the surrounding tissue.[1] It seems certain that a mechanistic approach to biomaterials, which seeks to understand and develop new products with an eye toward biologic interactions between alloplastic implant and host, will guide future endeavors.

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12

Tissue Engineering

Michael R. Pharaon, Thomas Scholz, and Gregory R.D. Evans

Summary

Tissue Engineering is an interdisciplinary field that applies the principles of engineering and life sciences to develop biological substitutes with the purpose of restoring and regenerating damaged or injured tissues. This chapter provides an overview of the field of tissue engineering and outlines its potential to provide solutions to the field of regenerative medicine. It gives an overview of important aspects used in tissue engineering and discusses the use of stem cells, cytokines and growth factors, gene therapy, and materials used to create bioartificial scaffolds and tissue-engineered constructs. Present and future challenges in the clinical application of tissue-engineered products are discussed in the context of products used for skin, cartilage, bone, peripheral nerve, breast, tendon, and skeletal muscle.

Abbreviations

ADSC	Adipose-Derived Stem Cell
ACT	Autologous Chondrocyte Transplantation
BMSC	Bone Marrow Stem Cell
BMP-2 and BMP-7	Bone Morphogenetic Protien-2 and 7
cDNA	Complementary deoxyribonucleic acid
DNA	Deoxyribonucleic acid
FGF	Fibroblast Growth Factor
FGF-10 and FGF-2	Fibroblast growth factor-10 and 2
FDA	Food and Drug Administration
GAGs	Glycosaminoglycans
GM-CSF	Granulocyte-Macrophage Colony-Stimulating Factor
IGF-1	Insulin-like Growth Factor-1
IL-1	Interleukin-1
IL-6	Interleukin-6
KGF	Keratinocyte Growth Factor
MHC-I	Major Histocompatibility Complex I
MSC	Mesenchymal stem cell
μm	Micrometer
rPTH	Parathyroid Hormone-related peptide
PDGF	Platelet-Derived Growth Factor
PEGDA	Poly (ethylene glycol) diacrylate
PGA	Poly (glycolic acid)
PLA	Poly (lactic acid)
PLGA	Poly (lactic-*co*-glycolide)
PCL	Poly (ε-caprolactone)
SNT	Somatic-Cell Nuclear Transfer
TGF-β	Transforming Growth Factor-β
VEGF	Vascular Endothelial Growth Factor
3D	3-Dimensional

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Introduction

Over the last several decades, the field of tissue engineering and regenerative medicine has begun to move from the research laboratory to the clinical setting. In 1987, the National Science Foundation first defined tissue engineering as "an interdisciplinary field that applies the principles of engineering and the life sciences towards the development of biologic substitutes that restore, maintain, or improve tissue function."[84] In 2001, the National Institutes of Health Bioengineering Consortium defined regenerative medicine as "the regeneration and remodeling of tissue in vivo for the purpose of repairing, replacing, maintaining, or enhancing organ function, and the engineering and growing of functional tissue substitutes in vitro for implantation in vivo as a biological substitute for damaged or diseased tissues and organs."[2] Many authors have used the terms tissue engineering and regenerative medicine synonymously. The term "regenerative medicine" is a broader field that includes the therapeutic techniques of cell therapy, tissue engineering, and bioartificial organ construction.[107] Regenerative medicine's subdivisions of cell therapy, tissue engineering and bioartificial organ construction are based on different basic approaches that restore tissues and organs. Because each major subdivision of regenerative medicine employs different techniques, they are associated with different governmental and institutional regulations.[107]

Different Techniques of Regenerative Medicine

Cell therapy and tissue engineering techniques have many potential applications in the field of surgery. These techniques may be able to provide tissues de novo that can be used for reconstruction, eliminating the problem of donor-site morbidity.[58]

Cell Therapy

Cell therapy uses living cells, including stem cells, to restore, enhance, or maintain specific tissues. Cell therapy does not use cell culture expansion, cell differentiation, growth factors, or other bioactive molecules nor does it use extracellular matrixes or scaffolds.[63,111] For example, the isolation of adipose-derived stem cells (ADSCs) from liposuctioned tissue followed by the reimplanting of ADSCs as soft tissue fillers would be considered cell therapy. The FDA considers this process "minimal manipulation."[148] Most general hospitals are able to meet the FDA regulatory requirements and are able to perform autologous cell therapy. Generally, governmental restrictions regarding autologous cell therapy involves compliance with regulations intended to prevent the transmission of communicable diseases.[1,145–148]

Tissue Engineering

Tissue engineering is an extension of the cell therapy approach and includes the use of stem cells and the addition of "more than minimal manipulation." Tissue engineering scaffolds are used as extracellular matrixes to provide a 3-dimensional (3D) supporting structure to the cells, resulting in a *tissue construct*. Cell culture expansion and amplification, directed cell differentiation, coculture with multiple cell lines, genetic modification of cells, cell exposure to cytokines from an ex vivo or in vivo environment, and the attachment of cells to a scaffold, which results in mechanical interactions, are all examples of cell manipulation used by tissue engineers.

Advancement in the tissue engineering approaches to regenerative medicine are becoming more complex, and, therefore, more stringent regulatory policies apply.[1,3,145–148] Currently, fabrication of tissue engineering products is beyond the capacity of most hospitals, and manufacturing usually occurs in the facilities of a biotechnology or pharmaceutical company.[107]

Various biomaterial scaffolds being researched include naturally occurring biodegradable polymers, synthetic organic biodegradable polymers, hydrogels, and nonorganic bioactive glasses and ceramics. These various biomaterial scaffolds have been demonstrated to have an effect on the cellular activity of cells within and adjacent to a tissue construct.[121] Recently, ceramic and synthetic polymer scaffolds have been designed to provide a sustained local release of cytokines.

Bioartificial Organs

An extension of tissue engineering is the construction of bioartificial organs. Organs and individual tissues differ. Physiologically, organs function independently as a unit, whereas tissues do not. This independent function allows the constructed bioartificial organ to be placed either intra- or extracorporeally. In contrast, tissue-engineered constructs require a tissue construct to be implanted intracorporeally to function appropriately. Intracorporeal and extracorporeal placement of tissues may be associated with different degrees of regulatory oversight, especially if xenotransplanted tissues are used. Although the FDA considers both an extracorporeal and intracorporeal xenotransplant similar, the European Union has a higher degree of regulatory oversight with the use of intracorporeally placed xenotransplanted organs compared to extracorporeally placed organs.[107,144]

Basic Approaches to Tissue Engineering

Tissue engineering uses three major means to repair, restore, maintain, or enhance tissues. These include the use of cells, cytokines, growth factors, and scaffolds to create a tissue construct. A comprehensive understanding of cell and developmental biology, cytokine activity, molecular biology (gene regulation and gene therapy), and biomaterials is necessary to successfully create a tissue construct that replaces damaged or missing tissues. This usually requires close cooperation between biologists, material scientists, and clinicians.[84]

Tissue engineering uses two different approaches, the *substitutive* approach and the *histoconductive* or *histoinductive* approach. The substitutive approach consists of a living tissue construct that is composed of living cells on a scaffold or extracellular matrix. This tissue construct with living cells is manufactured ex vivo and implanted into the patient. The second approach, the histoconductive or histoinductive approach implants an acellular, nonliving scaffold, or matrix material. This implant is designed to optimize, enhance, or increase the native autogenous cell's regenerative ability to repair and regenerate tissues in vivo.[154]

Substitutive Approach

The substitutive approach to Tissue engineering involves the ex vivo proliferation of cells and their application on a scaffold or extracellular matrix. The result is the formation of a 3D structure known as a tissue construct. The 3D architecture of ex vivo formed tissue constructs is due to cell–cell signaling, cell–matrix interactions, and the interactions of cells within the local environment, (including mechanical forces applied to a cell's surface proteins by local biomolecules).[171] Ex vivo proliferation uses progenitor cell cultures that have been expanded, differentiated, and modified before implantation. This modification can include the addition of genes that code for growth factors using established molecular biology and gene therapy techniques.[11,109] Tissue constructs may be fully functional at the time of implantation, but more commonly, they require maturation to be incorporated into the surrounding native tissues.[9]

The development of such living tissue constructs before implantation generally takes place in a *bioreactor*. Used by chemical engineers and biologists, a bioreactor is a vessel that supports and allows living cells to grow into a 3D structure. Bioreactors used in mammalian cell cultures and tissue engineering are complex devices that use multiple sensors and feedback loops to keep the environment constant. This involves regulation and maintenance of conditions such as temperature, pH, levels of cell culture medium, and gas levels, including air, oxygen, nitrogen, and carbon dioxide.[4,52]

Histoconductive/Histoinductive Approaches

In the histoconductive/histoinductive approach, cell proliferation and differentiation occur in vivo from native progenitor cells that migrate into an implanted acellular scaffold. Growth factors and cytokines used within the scaffold are intended to increase recruitment of native progenitor cells and enhance cell expansion and differentiation in vivo. This approach uses the patient as a "self-bioreactor," allowing for cellular differentiation and expansion and eventually tissue formation.

Clinical Applications

In many surgical specialties today, the use of tissue constructs and bioartificial organs has obvious clinical utility. The urologist could use tissue constructs and bioartificial organs to reconstruct and replace injured or surgically resected parts of the urinary tract, such as the bladder, ureters, and kidneys.[134] The cardiovascular surgeon could use tissue constructs as vascular grafts for bypass procedures, replace cardiac valves, and repair injured myocardium after a myocardial infarction.[36] The orthopedic surgeon could use tissue constructs to replace or repair tendons, ligaments, cartilage, and bone.[30]

For the plastic surgeon, the clinical application of tissue engineering has the potential to limit or eliminate donor-site morbidity and to generate tissue banks that can be used for reconstruction, improving overall outcomes. Tissues of special interest to the plastic surgeon are skin, fat/soft tissue fillers, muscle, tendon, cartilage, bone, peripheral nerves, and nerve conduits, all of which could be used for reconstructive surgery. Autologous tissue transfer/transplantation using Tissue Engineered Constructs is an option for reconstruction after mastectomy, resection of head and neck cancers, repair of soft tissue defects after trauma or surgical resection, and for the coverage of burn wounds.[42] Currently, there are FDA-approved tissue-engineered products on the market, which may be used to repair or regenerate skin, cartilage, and bone. Simple collagen scaffolds are available for use as nerve conduits as well.[12,13,165]

Cell Types Used in Tissue Engineering

One of the key components of tissue engineering is the use and modification of cells to regenerate and replace injured or lost tissues. Both the substitutive and histoconductive/histoinductive approaches are dependent on living stem and progenitor cells. These cells expand and differentiate forming the basis of regenerated tissues.

Stem Cell Classifications

Stem cells are incompletely differentiated cells capable of self-renewal by cellular division and replication. They have the capacity to differentiate into multiple specific cell lines. Stem cells are broadly divided into two groups. (1) The embryonic stem cell present in the early stages of embryogenesis and derived from the inner cell mass of the blastocyst. (2) The adult stem cell, stem cell obtained from a postnatal organism.[56,164]

Stem cells can also be classified based on their differentiation potential, also known as *potency*. The four major classes of stem cells based on potency are as follows: (1) the totipotent stem cell, (2) the pluripotent stem cell, (3) the multipotent stem cell, and (4) the unipotent stem cell. The totipotent stem cell has the greatest differentiation potential, while the unipotent stem cell only retains the ability to differentiate along a single lineage[138,170] (see Figure 12.1).

Totipotent stem cells are derived from early divisions of the zygote. Totipotent stem cells can be derived from the developing embryo only until the 8-celled stage morula.[164] Each totipotent stem cell is able to generate an entire organism. It also produces extraembryonic tissues, such as the placenta and yoke sac.[56]

Pluripotent stem cells are not able to form extraembryonic tissue. They are derived from the inner cell mass of the blastocyst and are able to form tissues from all three embryonic germ cell layers, (ectoderm, mesoderm, and endoderm). Both totipotent and pluripotent stem cells are considered to be *embryonic* stem cells.[49]

Multipotent and *Unipotent* stem cells are considered types of adult stem cells. These stem cells are obtained from postnatal organisms and classified based on their origin from one of the three germ cell lineages (ectoderm, mesoderm, or endoderm).[17]

Classically, cell differentiation has been thought of as a unidirectional progressive process with cells proceeding from a completely undifferentiated totipotent stem cell to a completely differentiated cell of a particular tissue type. This unidirectional process has proven not to be the case. The term *plasticity* describes an adult stem cell's ability to cross the embryonic germ cell line boundaries between endoderm, mesoderm, and ectoderm tissues.[16,152] Recent experiments demonstrating the plasticity of adult stem cells have shown that homogenous cloned bone marrow-derived mesenchymal stem cells are able to produce ectodermal tissues such as nerves and airway epithelium.[76,157,163]

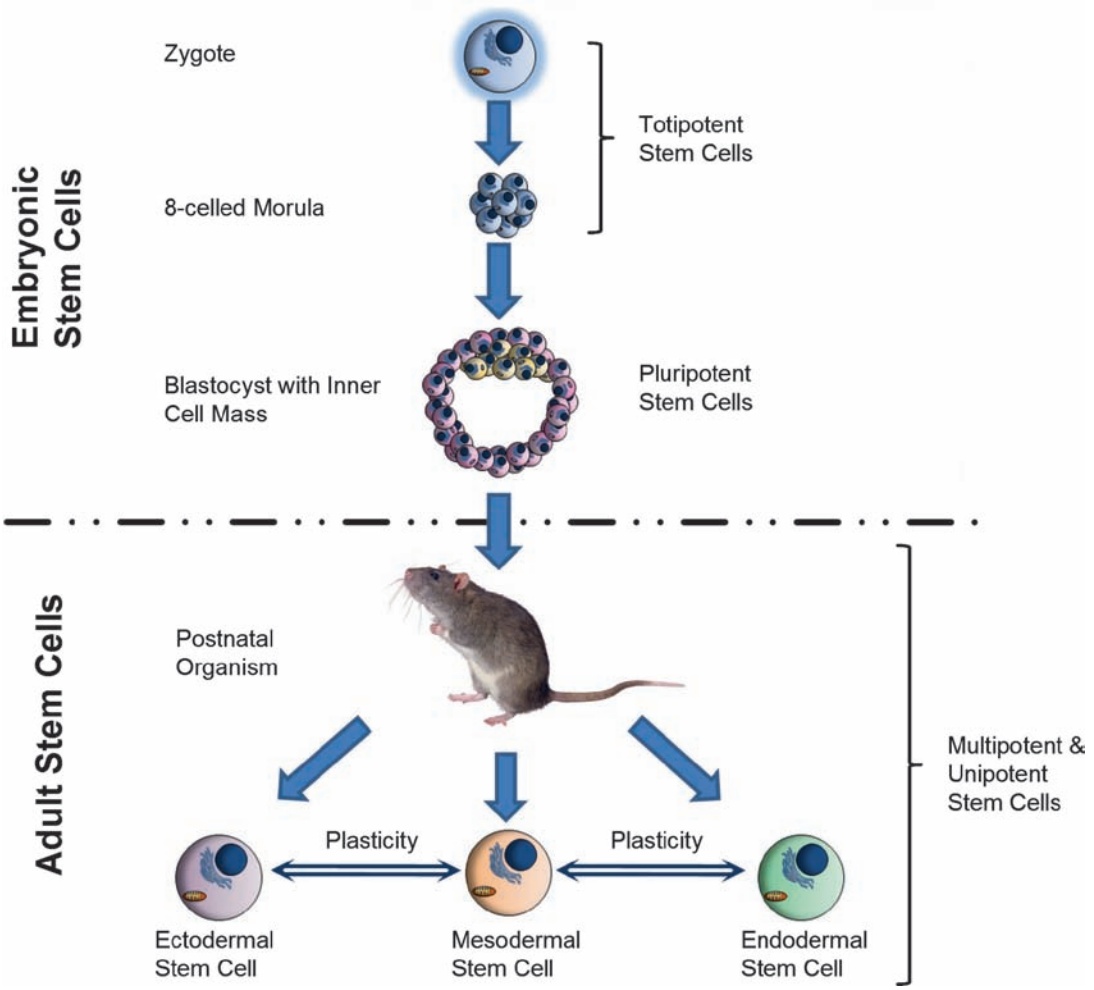

Figure 12.1. Stem cell types, stem cell differentiation, and stem cell plasticity.

Autogeneic vs. Allogeneic Stem Cells

A stem cell has the potential to be either autogeneic or allogeneic.

Ideally, tissue engineering cells have the following traits: (1) a cell capable of self-renewal; (2) a cell that is immunocompatible with the organism in which it is implanted, (including the mature daughter cells that make up the tissue construct once it has been incorporated fully); and (3) a cell that has regenerative potential.

Autogeneic Stem Cells

Autogeneic stem cells are derived from the same organism in which they are used and contain the same genetic material. Autogeneic stem cells are by definition immunocompatible cells. The only exception is autoimmune disease, in which the immune system attacks the cells derived from the autogeneic stem cell. Autogeneic stem cells can be isolated from all three germ cell lineages and from multiple different tissue types within each germ cell linage.

Ectodermal adult stem cell sources include tissues such as the retina, hair follicle bulge, cerebral cortex, olfactory bulb, and the inner ear.[8,34,39,88,90]

The identified mesenchymal, adult stem cell sources are bone marrow, adipose, skeletal muscle, peripheral and umbilical cord blood, vascular pericytes, stromal fibroblastic

cells, synovium, trabecular bone, and periosteum.[26,30,81,102,130,135,165,174]

Endodermal adult stem cells have been isolated from tissues such as gastric and intestinal epithelium as well as from the pancreas.[18,19,131]

The number of adult stem cells in a given tissue is dependent on many factors. These factors include the tissue source of the stem cells and the age of the organism.[128] For example, the number of bone marrow stem cells (BMSCs) present in an individual is variable between different aspirations and may only be 1 out of 10^6 to 10^7 nucleated cells.[96,124] In addition, the number of stem cells decreases as the organism ages. Mesenchymal stem cells in a newborn have been reported as high as 1 per 10,000 cells, whereas in an 80-year-old person, the number is closer to 1 per 2 million cells.[128]

Presently, the autologous stem cell approach is attractive because it avoids immune rejection. It is however, limited by extensive costs and time. The difficultly lies in the fact that the cells need to be harvested from the individual, expanded, and differentiated in ex vivo cultures, then successfully reimplanted. Ex vivo serial passage of cells affects the differentiation potential of autogeneic adult stem cells and may limit their use.[155] The prevention of bacterial and viral contamination while the cells expand ex vivo must also be ensured. This approach may be limited by the availability of adult stem cells for autotransplantation. Problems with the use of autogeneic stem cells such as harvest-site morbidity and time delays between harvest and reimplantation exist. The risks associated with the harvest of autogeneic stem cells are dependent on the tissue type and the anatomic location from which they are obtained.

Allogeneic Stem Cells

Allogeneic adult stem cells can be expanded ex vivo and banked as a source of stem cells or prefabricated into tissue constructs before an individual patient's need. This allows for tissue-engineered products to be available acutely, unlike the vast majority of autogeneic tissue constructs available today, which require time for ex vivo expansion and differentiation after autogenous harvest. A disadvantage of allogeneic adult stem cells is the potential for transmission of infectious disease and immunorejection.

Processed Lipoaspirate Cells and Adipose-Derived Stem Cells

The recent description of a multilineage mesenchymal adipose-derived stem cell (ADSC) by Zuk et al. in 2001 is responsible for increasing the number of adult mesenchymal stem cells available for research. Processed lipoaspirate cells have been reported to contain an ADSC density as high as 1 per 4,000 cells.[174]

The multilineage potential of ADSCs has great utility as an adult autogeneic stem cell source and has been shown to produce adipocytes, myocytes, chondrocytes, osteoblasts, endothelial cells, and neuron like cells, neuroendocrine cells that secrete insulin, somatostatin, and glucagons.[120,127,142,174]

The higher density of mesenchymal stem cells from processed lipoaspirate cells compared to bone marrow aspirates has even greater potential. It is a plentiful, easily accessible source of adult stem cells, with a great potential utility and clinical importance to the plastic surgeon.[42] The method by which the tissue is harvested from the patient is familiar to most plastic surgeons and simply entails the same process by which routine liposuction is performed. The cell slurry from the liposuction is then processed to obtain processed lipoaspirate cells. Further processing then allows for the isolation of ADSCs. With approximately 400,000 liposuction procedures annually in the United States, (each producing 100 ml to 3 l liposuctioned fat), the amount of tissue that could be used for stem cell research is vast, potentially 40,000 to 1.2 million liters annually.[79]

Immunocompatibility

Genetically identical, autogeneic stem cells are generally immunocompatible. Allogeneic stem cells are not identical genetically but appear to have some degree of immune-suppressive effects. Recent studies have demonstrated that adult autogeneic and allogeneic stem cells secrete a diffusible factor that has immunosuppressive properties on both cytotoxic and helper T-lymphocytes.[43,123] Despite the secretion of this factor, immunorejection remains a problem with the use of both adult and embryonic allogeneic stem cells.[49]

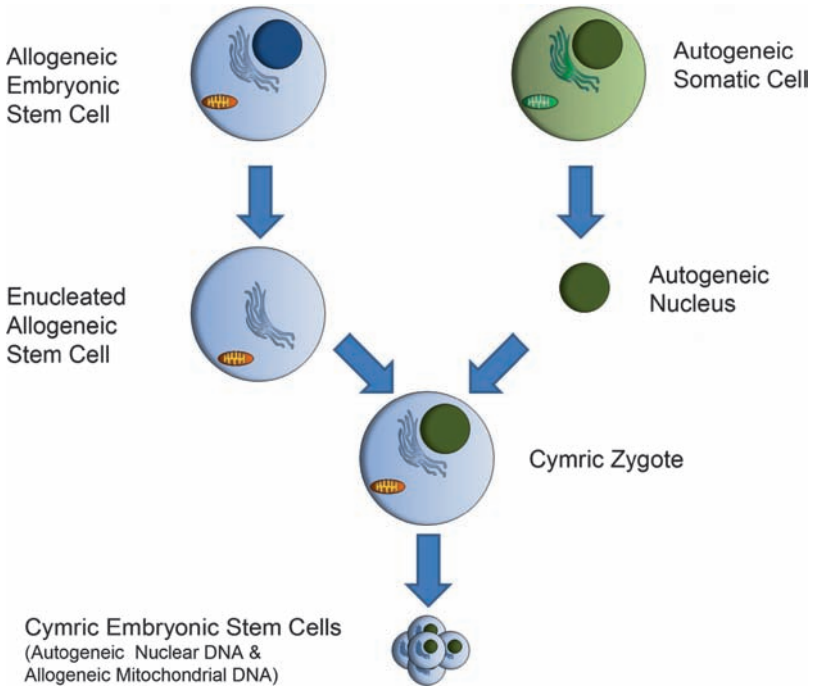

Figure 12.2. Somatic-cell nuclear transfer (SNT) using an allogeneic embryonic stem cell to create immunocompatible cymric stem cells.

Allogeneic human embryonic stem cells have been demonstrated to increase the number of major histocompatibility complex I (MHC-I) proteins on their surface after the differentiation process. This increase in MHC-I allows for increased foreign antigen presentation to cytotoxic T-cells and can trigger an immunorejection response.[44] Further techniques are under investigation, which may solve the immunorejection problem associated with the use of allogeneic stem cells.

Somatic-Cell Nuclear Transfer and Allogeneic Embryonic Stem Cells

The technique of somatic-cell nuclear transfer (SNT) is one method under investigation that could potentially circumvent the problem of immunorejection associated with the use of allogeneic embryonic stem cells.[48] This technique was used to create "Dolly" the cloned sheep.[28] Researchers are currently investigating the use of SNT in human cell lines, in an attempt to provide a solution to immunorejection of allogeneic embryonic stem cells.[64] The SNT technique entails taking a nucleus from a somatic cell from the patient and transferring it into a donor oocyte forming a *cymric cell*. This cymric cell can then be used as a source of embryonic stem cells. The cymric cell formed has autogeneic nuclear DNA and allogeneic mitochondrial DNA (see Figure 12.2). Due to the unlimited proliferation capacity of embryonic stem cells, teratoma and teratocarcinoma formation is a potential problem.[153] Despite the many uses, ethical and political considerations affect funding and the amount of embryonic stem cells available for research.[29,41] The use of allogeneic embryonic stem cells, like that of adult allogeneic stem cells, carries the risk of infectious disease transmission either from the donor cells or from an acquired infection during the ex vivo cell expansion and differentiation process.[49]

Stem Cell Type Considerations

Each cell type has benefits and drawbacks. A more practical approach will likely involve the use of

different stem cell types based on the clinical applications and desired outcomes.

In general, clinical situations that need emergent use of a tissue-engineered product (such as a vascular graft for an emergent coronary artery bypass procedure) will require the use of a product based on allogeneic stem cells. Conversely, a delayed breast reconstruction after mastectomy allows for time to harvest, expand, and differentiate autogeneic stem cells ex vivo before reimplantation.

Cytokines and Growth Factors

The use of cytokines and growth factors plays an important role in tissue engineering. A further understanding of the complex interactions between cell signaling molecules and their interactions between cells will assist in finding strategies and solutions for the field of regenerative medicine. Advances in genetic engineering, cell biology, pharmacology, and material sciences will allow for a more controlled modification and manipulation of cellular signaling pathways, enhancing the regenerative and restorative processes.

The cellular signaling process is complex and involves cell–cell interactions and cell–matrix interactions. Cell signaling is used to regulate the maintenance of cells, cellular differentiation and proliferation, and apoptosis. Soluble factors such as cytokines, growth factors, and hormones result in cell–cell interactions. These soluble factors can be classified based on the distance between their release and the location of their action (autocrine, paracrine, and systemically active cytokines and growth factors).[35]

Autocrine Cytokines and Growth Factors

Autocrine acting cytokines and growth factors have the potential to play an important role in both in vivo and ex vivo tissue engineering products. Cytokines, which have autocrine activity, are released from the cell that they act on. Fibroblasts are known to release autocrine acting cytokines such as transforming growth factor-beta (TGF)-β. Increase in fibroblast proliferation, secretion of other tissue growth factors, and an increase in collagen synthesis have been demonstrated to occur in response to TGF-β.[71] Bioartificial skin tissue constructs with fibroblasts present capitalize on this autocrine activity[165] (see Figure 12.3).

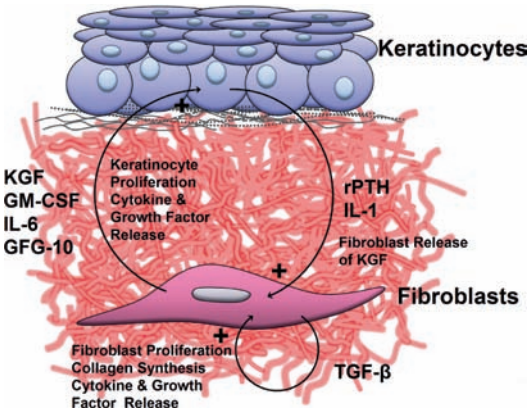

Figure 12.3. Positive feedback loops using autocrine and paracrine cytokines and growth factors between fibroblasts and keratinocytes.

Paracrine Cytokines and Growth Factors

Paracrine acting cytokines are released locally and effect nearby surrounding cells. Recent advances, in material sciences and gene therapy, have allowed for the manipulation of paracrine acting cytokines and hold great promise to tissue engineers. Paracrine acting cytokines act locally without the downside of systemic activity.

The paracrine activity of fibroblasts has also been used to improve keratinocytes growth and differentiation in skin tissue constructs. Fibroblasts secrete multiple paracrine acting cyto-kines and growth factors, such as keratinocyte growth factor (KGF), granulocyte-macrophage colony-stimulating factor (GM-CSF), interleukin-6 (IL-6), and fibroblast growth factor-10 (FGF-10), all of which have a stimulating effect on keratinocytes.[23,98,159,160] This stimulation of keratinocytes by paracrine cytokines and growth factors from fibroblasts results in an increased production of IL-1 and parathyroid hormone-related peptide (rPTH). An increased level of IL-1 and rPTH in turn stimulates the fibroblasts to produce more KGF.[20,94] This paracrine intercellular signaling between

epidermal keratinocytes and dermal fibroblasts has been capitalized on by corporations that manufacture FDA-approved cocultured skin tissue constructs such as OrCel™ and Apligraf® (see Figure 12.3). Both products are allogeneic cocultured tissue constructs approved by the FDA for the treatment of burn wounds. They contain living keratinocytes in the epidermal layer supported by living fibroblasts in the dermal layer. After implantation of such tissue constructs, maturation and long-term regeneration of the injured skin is dependent on the stimulation and ingrowth of autogeneic progenitor cells.[165]

Systemic Cytokines and Growth Factors

Systemically administered cytokines with pleiotropic effects on various tissues, such as TGF-β, FGF types a and b, vascular endothelial growth factor (VEGF), and platelet-derived growth factor (PDGF), have been demonstrated to have adverse effects on tissues distant to the area of interest. Owing to the short half-life of most cytokines involved in the stimulation of angiogenesis, systemic delivery has been problematic and requires high doses to achieve measurable effects. Systemic delivery may result in the growth of occult preexisting tumors due to increased neovascularization.[108,167] FGF delivered systemically in an attempt to induce angiogenesis can result in intimal thickening systemically and has been shown to have a negative effect on renal function, hematocrit levels, and platelet counts.[86] Tissue engineering has found a potential solution to this problem. Scaffolds designed for controlled local release of cytokines and growth factors have been manufactured. These scaffolds have circumvented the need for repetitive, high-dose cytokine administration.[55] A further understanding of the interactions between cytokines, growth factors, and cells will allow future tissue engineers to make advances in tissue regeneration.

Gene Therapy

Gene therapy is the science of the genetic transfer of material into a cell for the purpose of altering the molecular and cellular function of that cell.[59] The science of gene therapy is being employed by tissue engineers in an attempt to provide local delivery of desired growth factors without the complications associated with systemic delivery or repetitive local delivery methods.[46] Gene therapy has been shown to be a promising technique for the local delivery of growth factors.[106] The ideal growth factor in tissue engineering is released locally at the site of interest and has predictable pharmacokinetics, such as local concentration, half-life, and duration of release. It should be upregulated during the regenerative process and downregulated to basal levels once this process is complete. Given that the release of cytokines and growth factors occurs at the cellular level, tissue engineers have attempted two different approaches to use gene therapy. The first involves the substitutive approach with the *transfection* of cells ex vivo followed by implantation within the tissue construct. The second approach is more novel and is related to the histoconductive/histoinductive approach. Scaffolds with incorporated plasmid DNA are implanted, resulting in in vivo transfection using liposomal or naked cDNA uptake techniques.[10,21]

Gene therapy techniques have been shown to have a positive effect on in vitro and in vivo experiments investigating bone regeneration in craniofacial reconstruction. These experiments used transfected genes such as TGF-β, insulin-like growth factor (IGF)-1, and bone morphogenetic protein (BMP)-2.[5] The major drawback of the gene therapy technique is the concern for patient safety. This problem was highly publicized after the deaths of several patients enrolled in various gene therapy trials. Recently, the FDA has allowed the resumption of clinical trials using gene therapy, which may lead to further advancement in tissue engineering.[140] However, the potential still exists for the abnormal regulation of cell growth, cytokine secretion, and malignancies due to unexpected gene expression from transfected genes.

Currently, gene therapy methods are unable to deliver accurate doses of desired cytokines and growth factors for predetermined periods. Some tissue engineers believe that the risks of gene therapy, at this time, are outweighted by the potential benefits. This is largely due to the potential risks of abnormal regulation of transfected genes and the inability to predict the dosage.[13]

Biomaterials and Scaffold

Biomaterials and scaffolds are components used by tissue engineers. Manufactured scaffolds should have the following properties: (1) biocompatibility, including degradation products, both of which must not elicit an inflammatory response; (2) noncytotoxic, including both the material itself and its degradation products; (3) noncarcinogenic; (4) sterilizable; (5) predictable physical and mechanical properties, including elasticity, load bearing, and shear stress capacity that are appropriate to the tissue which they intend to replace; (6) surgical manipulation, including suturing as required for soft tissue implantation, and being able to be drilled and hold screws and hardware as required for bone and cartilage scaffolds; (7) porosity with at least an open pore size of 100–150 μm to allow for cellular migration and vascularization and allow for permeation of nutrients, cytokines, and waste; (8) histoconductivity, which guides and stimulates proliferation of autogenous progenitor cells that migrate from surrounding tissues into the scaffold; (9) histoinductivity that induces proliferation and differentiation of autogenous progenitor cells that have migrated from surrounding tissues into the scaffold; and (10) have sites that allow for cellular binding as well as in vivo drug delivery, including growth factors and genes.[125,154]

Tissue-engineered biomaterials used in scaffold construction can be classified into many different subtypes and include (1) biodegradable, naturally occurring polymers[97]; (2) biodegradable, synthetic organic polymers; (3) bioactive ceramic, most commonly calcium phosphate hydroxylapatite based; and (4) bioactive glasses.[125]

Naturally Occurring Biodegradable Polymers

Living organisms synthesize a variety of different polymer macromolecules. These naturally occurring polymers can be classified into three major groups: (1) proteins, such as collagen, fibrin, fibronectin, and glycosaminoglycans (GAGs)[27,117,143]; (2) polysaccharides, from plant, animal, and microbial sources[125]; and (3) natural polyesters derived from microbes such as poly (hydroxybutyrate) and poly (hydroxybutyrate-*co*-valerate).[31,173] Due to advances in biotechnology techniques, these naturally occurring polymers can be produced in bulk from microorganisms in a bioreactor or by in vitro enzymatic synthesis.[82,162] They may also be produced by tissue extraction from plants[51] and animals.[73]

As naturally occurring polymers are similar to the extracellular matrix, it is believed that these materials may avoid the complication of chronic inflammation that is associated with synthetic biodegradable polymers.[97] However, nonmammalian-based proteins such as silk, soybean, and casein proteins have been shown to have the propensity to incite an inflammatory response.[97,151,157]

Recently designed hybrid hydrogels that contain polypeptide segments in conjuncture with synthetic organic compounds, such as poly (ethylene gylcol), have been created. These hybrid hydrogels are designed to have a controlled release of growth factors only after cells have migrated into the hydrogel.[92] Migrating cells release metalloproteinases, which hydrolyze the scaffold, thereby releasing the growth factors trapped within.[6,93]

Synthetic Organic Biodegradable Polymers

This class of biomaterials is synthetic in nature and can be manufactured under more controlled conditions, resulting in products that have fewer impurities. Synthetic organic biomaterials have more reproducible mechanical properties, such as elasticity, tensile strength, sheer stress capacity, and degradation rates, compared to naturally occurring biomaterials. This allows for more precise manufacturing of tissue engineering scaffolds. These products, however, have a higher potential for toxicity and can trigger an immunogenic response. This immunogenic response can be from the material itself or from the acidic degradation products they form. The result is chronic inflammation at the implantation site.[125] Commonly used organic biodegradable polymers include saturated polyesters, [poly (lactic acid) (PLA), poly (glycolic acid) (PGA), poly (lactic-*co*-glycolide) (PLGA), and poly (ε-caprolactone) (PCL)], and polypropylene fumarate. These different compounds possess different degradation times ranging from 1 month to as long as 5 years.[45,65,126]

Bioactive Glasses and Ceramics

Bioactive glasses and ceramics in tissue engineering are generally used to create bone forming scaffolds. Both materials have similar processes by which hydroxylapatite forms on their surfaces. This hydroxylapatite has similar chemistry and structure as the mineral phase of bone.[141] Bioactive ceramics include calcium phosphates as well as less commonly used metallic hydroxides, such as titanium, zirconium, niobium, and tantalum.[57,89,104,149] Calcium phosphate-based ceramics have been shown to bind directly to bone and allow for the attachment of osteoblasts and mesenchymal cells.[25,74]

Vascularization and Fabrication Techniques

The vascularization problem associated with larger tissue constructs is a complex problem and has limited the clinical use of prefabricated tissue constructs to date. Scaffolds larger than 200 μm require a preformed vascular network for the supply of adequate nutrients, gas exchange, and for the removal of waste from the cells within the scaffold.[85] Several different fabrication techniques have been in use to create such scaffolds, including particle leaching, freeze drying, phase separation, fiber mesh formation using melt- or solution spun techniques, electrospinning fiber formation (Figures 12.4 and 12.5), and solid free-form fabrication.[22,60,68,69,75,168] The purpose of these different fabrication techniques is to create a scaffold with adequate porosity,

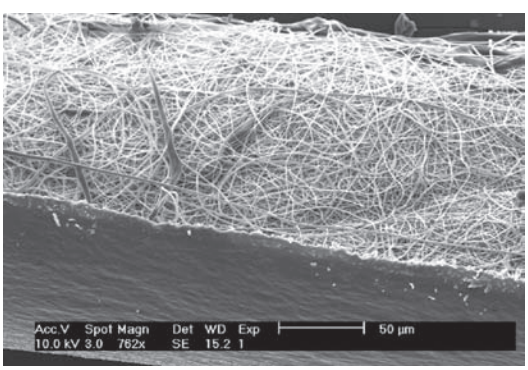

Figure 12.4. Electron microscopy image of electrospun poly(lactic acid) scaffold. (Courtesy of Dr. Anup Kundu and Dr. Darren Tyson, Dept. of Urology, University of California, Irvine.)

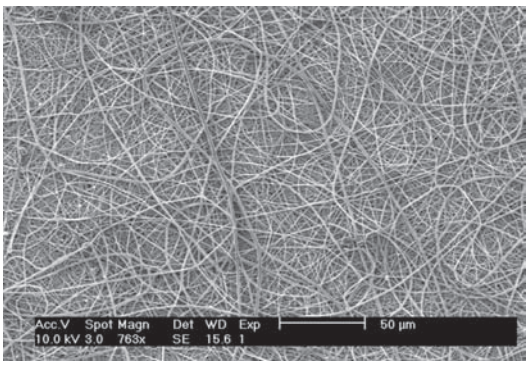

Figure 12.5. Electron microscopy image of electrospun poly(lactic acid) scaffold. (Courtesy of Dr. Anup Kundu and Dr. Darren Tyson, Dept. of Urology, University of California, Irvine.)

allowing for cellular migration and for adequate vascularization of the scaffold, thereby allowing for cell survival within the core of a larger tissue construct.

Three major techniques attempt to provide a vascular supply to larger tissue constructs. The first technique involves implantation of endothelial cells into the scaffold. After implantation these endothelial cells may result in angiogenesis and more rapid neovascularization of the tissue construct.[66] A second technique is dependent on the histoconductive/histoinductive approach to tissue engineering. This approach entails the implantation of an acellular scaffold followed by cellular migration of endothelial progenitor cells, resulting in material vascularization. Fibronectin arranged in an oriented layered fashion has recently been shown to increase endothelial cell adhesion and vascularization of scaffolds.[27] This process may also employ the use of growth factors released from the scaffold to induce vascular ingrowth into the acellular scaffold.[111] A more recently developed third technique involves the use of the free-form microfabrication.

The process of free-form microfabrication was initially developed for the construction of microprocessors and has recently been employed by tissue engineers. Its application to tissue engineering has allowed for the construction of artificial microvascular channels and capillary networks on polymer films. Theses films are then stacked, resulting in a prefabricated 3D vascular network within the tissue scaffold. Currently, tissue constructs with prefabricated

vascular networks used in combination with endothelial progenitor cells are under investigation. If successful, this technique would allow for a preformed vascular tree, ex vivo, which could then be implanted.[22]

Applications of Tissue Engineering Products

The current approach of using autogenous tissue transfers of skin, soft tissues, muscle, tendons, cartilage, bone, and nerve grafts to achieve reconstruction may be replaced by implantation of tissue constructs. These tissue constructs may be composed of an acellular scaffold alone or of cells on a scaffold. Implanted tissue constructs then result in a process that leads to regeneration and restoration of damaged or missing tissues.

Skin Tissue Engineering

Skin tissue-engineered constructs have the greatest number of FDA-approved products on the market and are the most widely used clinically. Multiple FDA-approved products using tissue-engineered skin constructs are available for the treatment of burns and chronic wounds. The development of skin substitutes is based on their uses as temporary dressings or as replacements to traditional skin grafts.[165]

Integra™ and Hyalomatrix® are acellular constructs that are based on a silicone membrane as an epidermis, with an underlying biologic-based acellular dermal scaffold. They are histoconductive/histoinductive and allow for fibroblast proliferation and migration into the dermal scaffold.

Laserskin® is an epidermal tissue construct composed of a hyaluronic acid membrane with implanted keratinocytes. The hyaluronic scaffold has laser-drilled pores, which allow for cellular ingrowth and vascularization from the dermal layer to the implanted keratinocytes.[122] Epicel®, an autologous epithelial tissue construct, is available for the treatment of burns, chronic wounds, and scar revisions.[165]

Products with allogeneic cells in the dermal layer (TransCyte®, Dermagraft®) are used to increase the concentration of FGF and other growth factors in the wound. They increase native fibroblast and keratinocyte growth and proliferation. (see section on Systemic Cytokines and Growth Factors) Dermagraft® uses viable allogeneic neonatal foreskin fibroblasts, while TransCyte® has nonviable fibroblasts as a source of FGF.[7,100]

Products that use autologous dermal layer fibroblasts are also approved for use. Hyalograft-3D® is a product similar to Hyalomatrix® but uses autogeneic cells expanded ex vivo.[137] Isolagen® is an autograft fibroblast product used for the treatment of facial rhytids, scars, and deformities.[67]

More complex tissue constructs with living allogeneic cells in both the epidermis and dermis are additionally available, (Apligraf®, OrCel™). They are used for the treatment of venous ulcers, diabetic ulcers, and burns. A recent Cochrane review on the treatment of venous ulcers found that the use of bioartificial skins in conjunction with compression bandaging results in improved healing of chronic venous ulcers compared with compression bandaging alone.[77]

Cartilage Tissue Engineering

Due to cartilage's limited ability to self-repair and its general avascular nature, it is an ideal candidate for tissue engineering.[32] Autologous chondrocyte transplantation (ACT) with ex vivo cellular expansion has been in use since 1987 and is the basis of Carticel®, a tissue construct used to treat full-thickness cartilage defects.[24] ACT often results in fibrocartilage (type I collagen), which is not suitable for joint use, as articular (hyaline) cartilage is generally composed of type II collagen.[99] In order to avoid donor-site morbidity associated with the harvest of articular cartilage for ACT, nonarticular sources of chondrocytes have been investigated. These nonarticular sources of chondrocytes included auricular, nasoseptal, and costal cartilage. Fibroblasts and stem cell sources have also been developed.[72]

One of the major challenges tissue engineers face in constructing articular cartilage is the need to duplicate the three distinct zones of articular cartilage.[38] Several different fabrication techniques and biomaterials have been used in an attempt to replicate these distinct zones. These biomaterials included natural polymers, synthetic polymers, and hybrid products, which

have been used to produce hydrogels, sponges, and meshes used as scaffolds for chondrocytes in cartilage tissue constructs.[32,113]

Multilayered poly(ethylene glycol) diacrylate (PEGDA) hydrogels have been shown to support subpopulations of chondrocytes similar to that of native articular cartilage.[132] Scaffolds with heterogeneous pore sizes throughout have resulted in tissue constructs with a similar distribution of GAGs and type II collagen as native cartilage.[112]

Using rapidly biodegradable scaffolds allows for new cartilaginous formation but may compromise structural support of the matrix, resulting in a thinner cartilage layer in an osteochondral defect. Conversely, nonbiodegradable and slowly degradable scaffolds result in thicker cartilage but are associated with surface fissures and cracks.[136]

Bone Tissue Engineering

Bone tissue engineering has been in clinical practice for a relatively short time. The engineering of bone tissue constructs is a more complex problem compared to cartilage. Bone is vascularized and has more extreme mechanical forces applied to it. Bone must withstand compression, flexion, and torque forces. Biomaterials employed in the manufacturing of bone tissue constructs must have adequate porosity allowing for cellular and vascular ingrowth. Yet excessive porosity results in the inability of biomaterials to maintain mechanical strength during the incorporation and maintenance phase of bone regeneration.[125]

Bioactive glasses and ceramics are employed in the construction of bone tissue constructs. Their surfaces form hydroxylapatite layers, which are similar in chemistry and structure to mineralized bone.[141] Bioactive glasses are important to tissue engineers and have been shown to induce differentiation of mesenchymal stem cells to osteoblasts, stimulate vascularization, and induce osteoblast adhesion and growth.[54,80,91] Bioglass® is an amorphous mixture of calcium, sodium, phosphate, silicon, and oxygen and is degradable by chemical and cellular mechanisms. This degradation allows Bioglass® to be replaced with bone, resulting in less than 6% remaining after 2 years.[83] Bioglass® has been shown to increase vascularization of tissues based on its ability to stimulate the release of VEGF from cells.[40] Bioactive glasses with silver oxides have been shown to have antimicrobial properties after transplant in vivo as well.[87] However, the use of bioactive glasses is limited due to low fracture thresholds and mechanical strengths.

Bioactive ceramics, particularly calcium phosphate-based ceramics, are able to completely fuse with mineral bone. The mechanical properties of bioactive ceramics can be manipulated by the tissue engineer by varying the ceramic's porosity, creating scaffolds with different fracture toughness, load capacity, and flex tolerance.[25] Bioactive ceramics, however, have lower fracture toughness, load capacities, and flex tolerance compared to natural bone.

The drawback of calcium phosphate-based ceramics is that they posses long in vivo degradation times, preventing bone remodeling and regeneration. Research into the use of various biodegradable polymers with bioactive ceramics is needed to create a bone tissue scaffold with mechanical properties similar to those of natural bone.

The use of growth factors in combination with biodegradable polymers is in clinical use at this time. OP-1 putty (Stryker Biotech) is a tissue construct that combines lyophilized human recombinant bone morphogenetic protein-7 (BMP-7) to increase bone growth and regeneration.[150] Another FDA-approved bone tissue-engineered product is INFUSE® (Medtronic Sofamore Danek). INFUSE® is composed of collagen and sustained-release human recombinant BMP-2.[55] The use of INFUSE® has been shown to be comparable to autogenous bone grafts.[101]

Polypropylene fumarate is a biomaterial of special interest in bone tissue engineering, because of its double-bond structure that forms cross-links once placed in situ. This formation of cross-links in situ occurs within 10–15 min and results in a hardening of the material, changing it from a malleable, injectable material to a hardened solid structure.[118]

Nerve Tissue Engineering

Peripheral nerve injuries can result from mechanical, thermal, chemical, congenital, or pathological etiologies. Peripheral nerves possess the capacity of self-regeneration, which represents an important difference to the central nervous system. In case of loss of important

nerve tissues, the severed nerves do not spontaneously restore their function and continuity. To date, interposition of an autograft is the "gold standard" for these critical-sized nerve injuries. It is associated with numerous disadvantages such as limited availability, donor-site morbidity, incomplete and nonspecific regneration, and variable clinical outcomes.[50,95,129] Nerve regeneration requires a complex interplay between cells, extracellular matrix, growth factors, and guidance of nerve fibers. The combination of natural or synthetic nerve conduits (filled or open lumen) used as a guidance channel with local growth factor delivery has been demonstrated to show promising results during the last two decades.[119] Currently, FDA-approved collagen nerve conduits NeuraGen® and the NeuraWrap™ (Integra) are used as guidance channels in the treatment of injured peripheral nerves.[12]

Due to the important physiological role of Schwann cells, cell transplantation represents yet another strategy to create the optimal microenvironment for nerve regeneration. Studies using autogenous Schwann cells for their tissue-engineering nerve constructs were able to obtain improved axonal growth.[47,161] Directed neuronal differentiation and single or multipe protein delivery using embryonic or adult stem cells are alternatives to Schwann cell therapy.[62,103]

Breast and Adipose Tissue Engineering

Breast tissue engineering is one of the most complex problems facing the tissue engineer. The fact that human breasts naturally change shape and size as the patient ages complicates the reconstruction of a natural breast mound.[114] Tissue engineering in breast reconstruction is further complicated by the fact that adipose is a highly vascularized tissue, and adipogenesis and angiogenesis are intimately intertwined.[37] Two different tissue-engineering approaches to breast reconstruction exist, the histoconductive/histoinductive approach and the substitutive approach.

The histoconductive/histoinductive approach involves the use of an acellular scaffold to incite migration of preadipocytes from surrounding tissues. This cellular migration can be enhanced by the use of growth factors such as fibroblast growth factor-2 (FGF-2) and insulin-like growth factor-1 (IGF-1) delivered via microspheres or Matrigel (reconstituted basement membrane matrix).[139,172] The process of adipogenesis has been augmented by vascularization of the acellular scaffold using a pedicled blood supply.[156]

The substitutive approach to breast tissue engineering is even more complex. Prior attempts to use mature adipocytes in fat transplantations have resulted in poor long-term results due to their propensity to be injured by mechanical forces.[33] The use of terminally differentiated mature adipocytes in ex vivo cultures is precluded due to the fact that they do not readily expand in culture. For this reason, current approaches to breast tissue engineering have focused on the use of pre-adipocytes, processed lipoaspirate cells, and adipose-derived stem cells (ADSC) as a cellular source. One or more than one of these cell types are then placed onto a breast tissue scaffold before implantation in the substitutive approach.[115]

The problem of vascularization of an ex vivo manufactured breast tissue construct is an even greater obstacle in the substitutive approach compared to the histoconductive/histoinductive. The lack of adequate vascularization to an adipose tissue construct has resulted in few studies able to demonstrate long-term stability of adipose volume.[116,158] As aforementioned, angiogenesis and fat are intimately intertwined and thus the problem is complex. Factors from mature adipocytes have been demonstrated to induce angiogenesis.[110] Conversely, factors released from endothelial cells are known to promote preadipocyte proliferation and differentiation.[70]

Recently, the technique of fat transplantation has been revisited for breast reconstruction. "Freshly" isolated processed lipoaspirate cells have been used as soft tissue filler in breast augmentation with good long-term (12 months) maintenance of volume. This study demonstrated that the use of fresh processed lipoaspirate cells in combination with traditional mature adipocytes resulted in a greater maintenance of breast volume, compared to fat alone or fat combined with cultured ADSC.[105]

Tendon and Skeletal Muscle Tissue Engineering

The design endpoint of tendon tissue engineering is to create a substitute that is able to withstand forces that are greater than the peak forces seen in vivo.[133] Tendon tissue constructs have been made using mesenchymal stem cells (MSCs) in combination with hydrogel and

sponge scaffolds. These scaffolds are composed of polymers and proteins such as collagen.[30] Advances in ex vivo culture conditions, such as cell culture on a scaffold under tension, have been shown to increase the strength of the tendon tissue constructs both ex vivo and after implantation at 12 weeks.[14,78] Tissue engineers have also found that alteration in the cell to matrix ratio has an effect on the load-bearing capacity of tendon tissue constructs.[15]

Unlike smooth muscle, in which muscle contraction is in multiple directions, skeletal muscle requires a tissue construct that produces uniaxial contractions.[169] The cells for such a muscle tissue construct can potentially be derived from satellite progenitor cells found in adult striated muscle or from mesenchymal adult stem cells.[30,166] The use of an aligned scaffold such as collagen has been explored with different cell seeding techniques in an attempt to form a tissue construct that has the desired unidirectional orientation.[166] This has proved to be a difficult problem, and further research involving cell implantation techniques and scaffold construction will be required.

The Future of Tissue Engineering: Where Are We Going?

The "irrational exuberance"[61] of the scientific community associated with the emergence of stem cell therapy and tissue engineering has been tempered by the realization that the process of regenerative medicine is more complex and difficult to achieve than initially expected. Further basic science research in the fields of material sciences, gene therapy, and cell and developmental biology will provide better insight into the overall potential of tissue engineering and the problems it currently faces. Eventually, scientific advances in tissue engineering will allow for better tissue constructs that will improve the overall success rates in reconstructive surgery using regenerative medicine techniques.

The best source of stem cells for the tissue engineer has yet to be determined; either autogenesis vs. allogeneic cells or embryonic vs. adult stem cells. Evolving ethical and political considerations play a role in the future research of stem cells and their applications in regenerative medicine. The use of adult stem cells is attractive, because it avoids the potential ethical and political milieu that surrounds the use of embryonic stem cells.[13] Both the autogeneic adult stem cell and the allogeneic embryonic stem cell using SNT will likely require time for the manufacturing of tissue constructs and are unlikely to be used in acute fashion.

Research with tissue-engineered skin substitutes will likely involve the refinement of products that manipulate growth factors, allowing for a more rapid healing of wounds. Advancements will also likely involve the combination of skin tissue constructs with deep soft tissue constructs, such as subcutaneous tissue and fascia.

Ongoing research is attempting to identify sources of cells capable of producing articular cartilage or creating a tissue that simulates articular cartilages' hydrostatic pressures and ability to withstand dynamic compression forces. The development of a zonal cartilage tissue construct will play an important role in the development of future tissue-engineered cartilage substitutes.

Advancements in the tissue engineering of bone will likely result in tissue constructs that are composed of biodegradable polymers combined with either bioactive glasses or ceramics. This would create a product with load capacities and fracture toughness similar to those of natural bone. Bone tissue constructs will likely expand to include the use of growth factor and cytokines to increase the rate at which fracture repair and regeneration occur.

In the future, more complex devices (nerve conduits, delivery systems, bioengineered nerve grafts, etc.) will be needed. A better understanding of the complexity of growth factor therapy and genetic engineering may help find better solutions to restore functional peripheral nerve tissue.

The need for an extensive vascular support system in breast tissue-engineered constructs is one of the most limiting factors in creating a tissue-engineered breast that maintains its volume and structure. As a result investigators are researching the use of artery-venous loops with tissue constructs to improve neovascularization.[156] The future of breast and adipose tissue engineering will require additional research to solve this vascularization problem. Given the recent result using "fresh" processed lipoaspirate cells, future approaches to breast reconstruction will likely involve "fresh" processed lipoaspirate cells used in concert with a scaffold that may

or may not contain cells cultured ex vivo.[53] If a solution to the vascularization issue is found, it will have broad applications to fields of tissue engineering and bioartificial organ construction.

Tissue-engineered products used to repair and reconstruct tendons and skeletal muscle have obvious clinical significance to the plastic and orthopedic surgeons. The need for clinically applicable tissue-engineered products that can replace tendon and skeletal muscle will drive the advancement of such products. Such tissue constructs could be used to repair extremity injuries that otherwise would result in a nonfunctional hand or extremity. There would also be a utility of such products with facial reconstruction and facial re-animation.[166] Research into tendon tissue engineering will involve the evaluation and manipulation of cell matrix interactions along with the use of growth factors to maximize the mechanical similarities between bioartificial tendons and native tendons. Advances will also be directed at decreasing functional recovery time after implantation. Skeletal muscle tissue engineering, however, will require further research to be able to create a tissue construct with uniaxial contractions.

As a whole, tissue engineering has great potential for significant advancements in plastic surgery. The advancements made in engineering and life sciences have changed how we approach the problems of reconstruction after traumatic injury or surgical resection due to neoplasm. Future advancements will result in tissue-engineered products that improve patient care and continue to change how plastic surgeons practice reconstructive surgery.

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Part III

Skin and Adnexa

13

Skin Anatomy and Physiology

Shashidhar Kusuma, Ravi K. Vuthoori, Melissa Piliang, and James E. Zins

Summary

If plastic surgeons are to provide state-of-the-art care in the techniques of skin rejuvenation and minimally invasive treatments for aging, a thorough understanding of skin, anatomy, and physiology is required. With the changing population demographics, the plastic surgeon must also be knowledgeable about ethnic variations in skin anatomy and physiology. The changes that occur with aging skin, inherent developmental disorders, and development of skin cancers are complex. This chapter provides a practical guide to the understanding of skin and its application to clinical conditions.

Skin

The average thickness of skin is between 2 and 3 mm. The thickness of the epidermis and dermis at the sacrum is significantly greater than that of the abdominal wall, groin, lateral gluteal area, and gluteal fold areas.[10] In the face, an area of particular interest to plastic surgeons, the epidermis is relatively constant in thickness measuring approximately 150 μm. However, dermal thickness varies considerably. For example, the dermal thickness in the periorbital area is approximately 200–250 μm, whereas the dermal thickness of the lip and forehead regions is 900–1,000 μm[8] (Figure 13.1). This variability has significant consequences when skin resurfacing is performed.[8] An area of skin that measures about 6 cm^2 can contain upwards of 20 blood vessels, 650 sweat glands, 60,000 melanocytes, and thousands of nerve endings.

Other functions include excretion of sweat and piloerection for temperature regulation. Skin provides sensation, helping us determine hot from cold, pressure, injury, vibration, and light touch. The use of local anesthetics is targeted to inhibit the sodium pumps in these sensory fibers to effect afferent signals to the brain to painful stimuli. The skin can also act as an interface for the diffusion of substances into the body including gases (CO_2, O_2, and N_2 in minute amounts). Delivery of topical medications is also based on the absorption and diffusion from the skin.

Skin Embryology

Skin is derived from both ectoderm and mesoderm. The epithelial layers are formed from the ectoderm. The various skin appendages including the pilosebaceous glands, sweat glands, and hair follicles are ectodermal elements. Specialized cells including melanocytes and neural elements are derived from the neuroectoderm. The cells of the dermal layers that include the fibroblasts, mast cells, blood vessels, lymphatic channels, and the adipocytes are derivatives of the mesoderm. Macrophages, Langerhan's cells (LCs), and Merkel cells are also derived from the mesoderm

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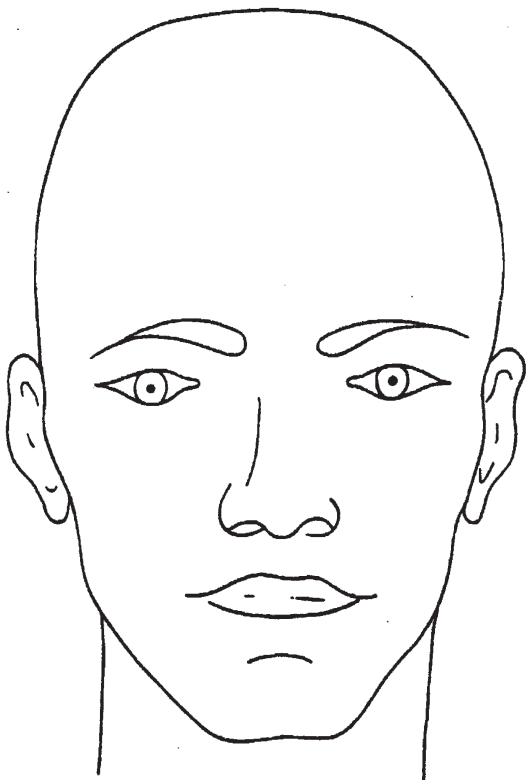

Figure 13.1. Variation of the epidermis and dermis about the face. While the epidermis is relatively uniform from location to location, the thickness of the dermis varies significantly. (Adapted from Gonzalez-Ulloa et al.[8]).

as are the components of the dermal layer. There are many inherent processes that are coordinated during embryogenesis for successful formation of the skin covering. Any alterations in these processes can lead to abnormal skin formation. Such congenital skin diseases include cutis laxa and Ehlers–Danlos syndrome.

Components of Skin

There are three distinct layers of the skin: the epidermis, the dermis, and the hypodermis (Figures 13.2 and 13.3). The epidermis is the primary defense layer against organic elements

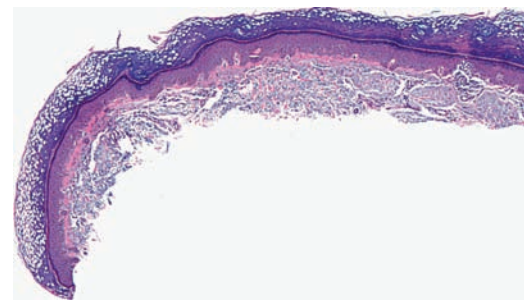

Figure 13.2. Histology of aging skin of the face. Note that solar elastosis and the loss of rete pegs are clearly visible.

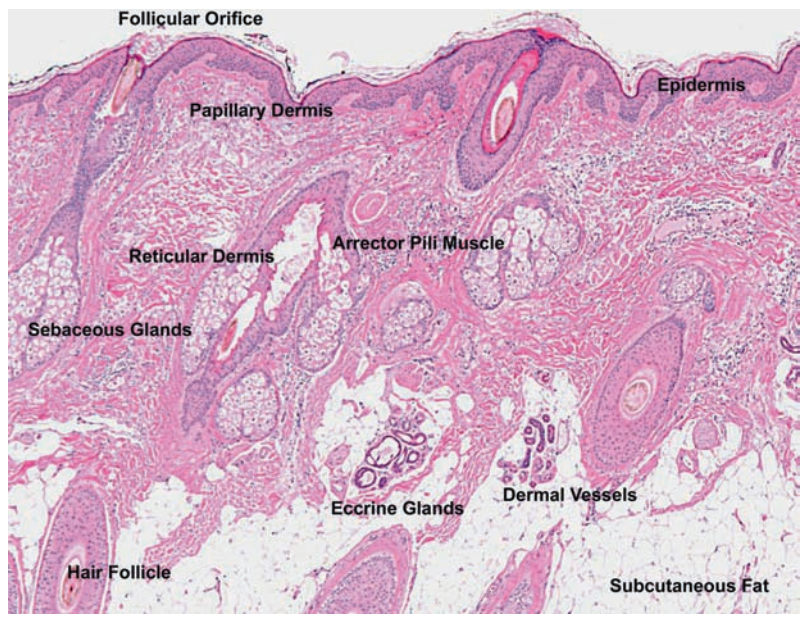

Figure 13.3. Skin histology of the scalp.

such as bacteria, viruses, parasites, and other organisms. In addition, the presence of a thick and tough stratum corneum (the keratin layer) in conjunction with the melanocytes provides protection from photodamage. The epidermal layer of skin also protects us from harsh elements in the external environment. The epithelial cells of the epidermis, also known as keratinocytes, sit on an underlying basal lamina. This layer of skin contains no blood vessels. The nourishment for the keratinocytes comes from a process of diffusion from the underlying dermis. In addition to keratinocytes, melanocytes, Langerhans', and Merkel cells are also present in the epidermal layer of the skin.

The thickness of the epidermis can vary in different regions of the body. It is approximately 150 μm in the eyelids and almost 1.5 mm in the soles of the feet. Regional variations in epidermal thickness result from the different number of keratinocytes and the length of the rete pegs. Such anatomical changes should be kept in mind when handling tissues. Areas where skin is quite thin are particularly prone to surgical injury when roughly handled.

Layers of the Epidermis

There are five distinct cellular layers of the epidermis. These layers include (from superficial to deep) the stratum corneum, stratum lucidum, stratum granulosum, stratum spinosum, and stratum germinativum. During mitosis, cells from the stratum germinativum migrate superficially to populate the more superficial layers of the epidermis. The deeper layers of cells are columnar in structure, but as they migrate toward the surface, they become flatter in appearance as cellular differentiation takes place. As keratinocytes migrate upwards, they mature and fill with keratin and lipids. Keratinocytes undergo apoptosis in the stratum granulosum and subsequently are shed to make way for newer cells once they reach the stratum corneum. This process, known as keratinization, happens continuously throughout life.

The stratum corneum is sometimes described as the "horny" layer. Composed mainly of dead keratinocytes, the stratum corneum is the final stop for skin cells before they are shed. The cells in this layer contain mainly keratin and lipids. The protein keratin is of vital importance, because it keeps the human body hydrated by minimizing evaporative water loss from the skin. While preventing dehydration, keratin can also absorb outside moisture. The thickness of the stratum corneum varies in different parts of the body. Skin that covers body parts exposed to constant wear and tear, such as the hands and feet, have thicker stratum corneum than other parts of the body. Also skin that is located on pressure points and joint surfaces such as the sacrum, elbows, and knees is much thicker. Beneath the stratum corneum is the stratum lucidum, so named for its translucent appearance. The stratum lucidum is a thin layer of dead cells that contain Eledin, a clear intermediate form of keratin that contributes to its "lucid" appearance. Usually, thicker parts of the body, that is, palms and soles, contain the stratum lucidum.

The stratum granulosum is found just below the stratum lucidum and is present in all regions. Characterized by small basophilic granules in the cytoplasm of squamous cells, the stratum granulosum is the outermost layer where living cells are found. The granules in these cells contain phosphorylated histidine-rich and other cystine-rich proteins through which keratin bundles traverse. The stratum granulosum also helps bundle keratin through a protein called filagrin. "Membrane-coated" lamellar granules are also present and contain lipids. The lamellar granules are exocytosed in this layer to generate a waterproof barrier. This physical barrier to evaporative water loss also prevents life-sustaining nutrient diffusion for these cells and thus leads to the characteristic cell death of the outer layers of keratinized epithelium.[7,11]

The next layer of the epidermis is the stratum spinosum. Sitting underneath the stratum granulosum, the stratum spinosum consists of cuboidal cells in a multilayered fashion. The keratinocytes flatten out as they progress through the stratum spinosum. When these cells shrink during staining, they sometimes look spiny from the desmosomes that connect them together, hence, the name "spinosum." Cells of the stratum spinosum actively synthesize intermediate filaments called cytokeratins. These intermediate filaments are anchored to the desmosomes joining adjacent cells to provide structural support, helping the skin resist abrasion.[13]

The basal layer of the epidermis is the stratum germinativum. This layer is composed of columnar cells that undergo mitosis to populate the epidermis and gradually migrate superficially.

This layer of cells lies directly on the basal lamina of the skin and forms the dermal/epidermal junction.

A desmosome or macula adherens functions in cell-to-cell adhesion. Found in the cell membrane between keratinocytes, desmosomes are important in controlling shearing forces in the epithelium by anchoring cells to each other. As complexes of proteins, desmosomes help link cell surface proteins to intracellular keratin cytoskeletal filaments. An autoimmune disease known as pemphigus vulgaris is caused by auto antibodies to desmosomes. Such an aberrant process leads to acantholysis or separation of the adherent cell layers and results in the characteristic sloughing of skin and formation of painful blisters.

Melanocytes are cells found in the stratum germinativum of the epidermis and in the middle layer of the eye. Melanogenesis, which is the production of melanin by melanocytes, can be altered by various stimuli. The production process is not completely understood at this time, but isobutylmethylxanthine, retinoids, melanocyte-stimulating hormone (Melanotan), metabolites of vitamin D, cholera toxin, forskolin, UV light, ACTH, and diacylglycerol all stimulate the process of melanogenesis.[14] In addition, DNA damaged by UV radiation can lead to the formation of thymidine dinucleotide (pTpT) fragments that can stimulate melanogenesis. Once made, melanin is moved along dendrites in a special container called a melanosome. Melanosomes are organized into a cap and protect the DNA in the nucleus of the keratinocyte from ultraviolet light. One keratinocyte provides melanin for 4 to 10 keratinocytes.[6] Melanin provides pigment to the skin. The skin helps protect against harmful UV irradiation and contains enzymes to help repair DNA damage from UV light.

Differences in skin pigmentation between races can be directly associated with the latitude of various continents. More melanin provides greater protection against increased UV radiation near the equator and gives individuals a darker pigmentation. Data show that skin tone is independent of geographic origins of a human race, being derived from ancestral pigmentation.[12]

The Langerhans' cells, also located in the epidermis, play a vital role in the immune response. Langerhans' cells are classified as dendritic cells or antigen trapping dendrites. After capturing the antigens, these cells travel from the epidermis to local and regional lymph nodes. While in transit, the Langerhans' cells (LCs) become activated and expose the captured antigens to circulating T cells. Although the main function of LCs is to aid in host protection, dysfunction of these cells can lead to neoplastic changes. Langerhans' cell tumors are a result of cellular atypia.

Dermis

The second major layer of the skin is the dermis composed of collagen, elastin, salts, water, and a gel of glycosamin proteoglycans. All these proteins and molecules give significant density to the dermal layer of the skin. The dermis varies considerably in thickness from location to location. It can be as thin as 200 μm in the eyelid and as thick as 3 mm on the back skin. The dermis provides protection from stress and strain by providing a cushion. Hair follicles, sweat glands, sebaceous glands, apocrine glands, and blood vessels all partially exist within the dermis and exit through the dermis. Nourishment and waste removal of the dermis are dependent on the blood vessels that exist in the vicinity.

Dermal fibroblasts help control the production and maintenance of the dominant structural components of the dermis. Fibroblasts make up the majority of cells in the dermis along with interspersed mast cells and tissue macrophages.

The tensile strength of the dermis comes from collagen, which accounts for a significant amount of the fat-free dry weight of skin. The majority of collagen in the dermis is Type I collagen and constitutes up to 80% of the collagen in skin. Type III collagen constitutes about 15%, while Type V and VI account for the remainder. The typical ratio of Type I collagen to Type III collagen is 4:1. This ratio is maintained even in scars after wound healing.

Collagen is the most abundant protein in mammals and is found mainly in connective tissue. As a long fibrous structural protein, bundles of collagen or collagen fibers are the major constituent of the extracellular matrix. The collagen fibers provide support for tissue and cell structures. Collagen has significant tensile strength and is found in the fascia, ligaments, tendons, bone, teeth, and cartilage. Collagen maintains skin elasticity and strength in a synergistic manner with elastin. Tissue development is also aided by collagen because

of the support it gives to blood vessels. With decreased production and turnover of collagen, the signs of aging including rhytids, loss of skin elasticity, and thinning of skin become apparent. In conjunction with collagen, elastin fibers help maintain structure and allow for flexibility of the skin.

Papillary Dermis

The dermis comprises two layers: pars papillaris and pars reticularis. The papillary dermis is the thinner and more superficial layer of the dermis zepidermis and dermis are contoured by ridges and folds of papillae that arise from the papillary layer of the dermis, which give integrity and increase the surface area of the dermal/epidermal junction. The folds and ridges are referred to as rete pegs. With aging, these rete pegs diminish and subsequently lead to a decrease in the surface area of the dermal–epidermal junction. This phenomenon can lead to epidermal gliding and shearing. Papillae are most prominent in the hands (palms and fingers) and the feet (soles and toes) as these areas must withstand the greatest frictional forces. Rete pegs are also known as friction ridges, because the exaggerated rete pattern gives the hands and feet the ability to grasp objects through friction.

Elastin and collagen fibers are dispersed more widely and are arranged in a more haphazard fashion in the papillary dermis than in the reticular dermis. However, the papillary dermis has a higher amount of ground substance and connective tissue cells. Blood vessels and lymphatic plexuses are found in the papillary region directly below the dermal papillary ridges.

Reticular Dermis

Directly below the papillary dermis is the reticular dermis. Thicker than its more superficial counterpart, the reticular dermis is avascular and acellular, but contains a high amount of collagenous and elastic tissue. Type III and V collagen fibers are seen mostly in the epidermal–dermal junction. However, they are also found in both the papillary and reticular dermis as sheaths for epithelial appendage structures, vessels, and nerves.

These collagen fibers are arranged in an interwoven, crisscross pattern in a plane parallel with the skin. The arrangement of the collagen fibers was first observed by Langer (1861) who described the arrangement as a "lattice-like network with much extended rhomboidal meshes." The pattern performs important functions especially with regard to skin extensibility. An anatomic feature in this layer of the dermis is the even distribution in thickness of the collagen and elastin fibers.

Dermal Ground Substance

An important component of the dermal connective tissue is what is known as ground substance. This substance is composed of a broad class of anionic polysaccharides or glycosaminoglycans, which comprise the milieu for cells of the dermis.[5] Hyaluronates, dermatan sulfate, chondroitin-4-sulfate, and heparin sulfate constitute the ground substance in the skin and are regulated by fibroblasts and mast cells. Existing as a viscoelastic solgel of hydrophilic polymers, the ground substance in the dermis has complex interactions involving water binding, flow resistance, collagen, and other glycosaminoglycans.

Clinical Correlation: Skin Expansion

When there is a shortage of skin because of injury or skin needs replacement because of tumor resection, plastic surgeons may take advantage of the viscoelastic nature of the dermis. This can be accomplished by expansion of the residual skin. Skin expanders are placed subcutaneously and are gradually inflated to stretch the skin taking advantage of the process called "creep." The collagen and elastin fibers located in the dermis are stretched and the ground substance located in the dermis is displaced out of the area of expansion allowing for skin expansion. As a result, the dermis gradually thins and the epidermis thickens. The underlying subcutaneous tissues and muscle also undergo some degree of atrophy and thinning. Any underlying bone will also undergo some resorption or remodeling. Such expansion is ideally performed over months and is done in areas adjacent to the area in need of reconstruction. Once the skin is expanded and used in reconstruction, it gradually undergoes a reversal of the changes noted prior to expansion and can return close it its normal anatomic and physiologic composition (Figure 13.4).

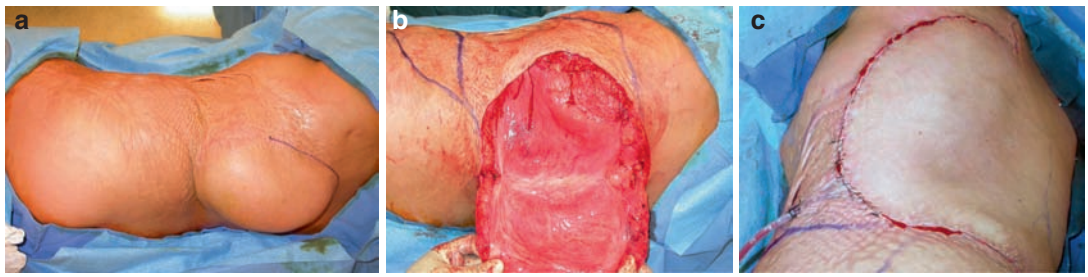

Figure 13.4. (**a**) Skin expander in the lateral back. Healed burn with skin contracture noted. (**b**) Expander removed with the expanded skin shown. (**c**) The reconstructed skin contracture with the expanded skin.

Skin Circulation

Cutaneous branches of the musculocutaneous arteries are the main suppliers of blood for the skin. Branches from these cutaneous arteries form a small vessel plexus within the dermis. Some of these branches protrude outward as perforating arteries morphing into arterioles as they reach the papillary dermis layer of the skin. The small arterioles and venules constitute the microvasculature/microcirculation of the skin. The circulation in the skin plays a vital role in maintaining the body temperature in concert with the temperature-regulating control centers in the hypothalamus. Chronic changes that occur from diabetes, smoking, and other vascular disorders can lead to changes in skin circulation and can cause significant soft tissue injury. Some of the autoimmune disorders such as Raynaud's lead to decreased circulation in the skin of the digits and result in pain and discoloration and cold intolerance.

Nerve

Within the deep dermal plexus, branches of myelinated sensory nerves lie parallel to the skin surface. These branches project upward into the dermis to form a web in the superficial dermal plexus. These nerves convey sensations from the skin to the brain through specialized receptors for touch, pressure, temperature, and pain. These nerves also carry autonomic fibers that innervate the smooth muscles of the cutaneous blood vessels, pilomotor units in the hair follicles, and the sweat and sebaceous glands.[4] Cholinergic fibers use acetylcholine, adrenergic fibers use norepinephrine, and the purinergic terminals use ATP as a neurotransmitter.

Clinical Correlation

The use of botulinum toxin for hyperhydrosis of skin is specifically targeted to inhibit the cholinergic fibers that innervate the sweat glands. Excellent results can be obtained with appropriate use of this medication in patients with hyperhydrosis. The appearance of rhytids or wrinkles as a result of the action of the underlying facial mimetic musculature can also be improved with selective weakening of the mimetic muscles with botulinum toxin. This minimally invasive procedure involves injection of small quantities of the toxin directly into the muscles of interest that results in improvement in rhytids caused by the facial mimetic muscles (Figure13.6).

Hair Follicles

Hair follicles in the skin contain lanceolate terminals, Merkel cell–neurite complexes, and Ruffini corpuscles. The lanceolate terminal is composed of axon endings and Schwann cell membranes located over the hair bulb on the sheath. For fine body hair, the terminals encircle the whole shaft, whereas in terminal hairs, they are less evenly distributed. Hair pigmentation is dependent on the amount of melanin in the hair follicle. The melanin present produces eumelanin and pheomelanin giving hair a specific color. It has

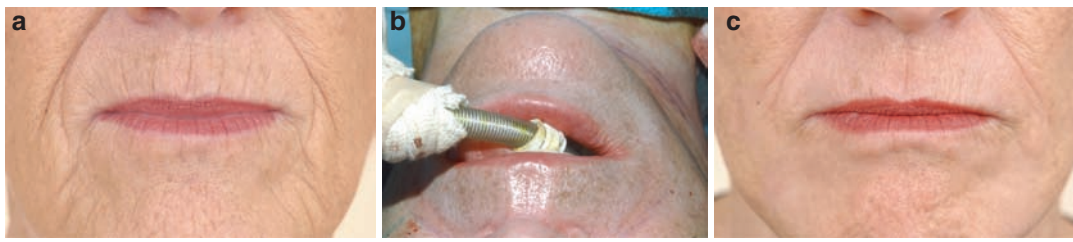

Figure 13.5. Skin resurfacing using phenol-croton oil peeling. (**a**) Preoperative. (**b**) Intraoperative view after application of peel. (**c**) Eight-month postoperative result.

been shown that genetics plays an important role in hair color.[17] Production of melanin decreases with age. The new hairs that grow in the elderly grow without melanin, which results in grey hairs. This process is not limited to the elderly but can present as early as adolescence in some individuals. Stem cells responsible for the maintenance of the melanin are reduced with increasing age, resulting in a decrease in melanin production.[15]

Clinical Correlation

Another important function of the hair follicle is its involvement in the regenerative capacity of the epidermis. Progenitor epithelial cells located along the hair bulb and shaft migrate outward to areas of de-epithelialization gradually repopulating it with new keratinocytes. This regenerative capacity of skin is of vital importance in burns and other trauma. This principle applies in the use of split-thickness skin grafts as well. Preservation of these skin appendages is crucial in the proper execution of skin resurfacing procedures as well. Loss of these appendages from trauma, burn, or iatrogenic causes will lead to wound healing with delayed re-epithelialization. If re-epithelialization is significantly delayed, hypertrophic scarring becomes more likely. In situations of full-thickness skin loss, it is important to recognize the extent of the injury early and perform reconstructive surgery to prevent hypertrophic scarring and possible scar contractures. This is especially true in preservation of the functional anatomic areas, such as the joints, hands, feet, neck, and face.

Any skin resurfacing procedures should be done with precise knowledge of the desired depth of skin injury. Laser resurfacing or deep chemical peeling procedures must extend deep enough to eradicate wrinkles but not so deep as to destroy epidermal progenitor cells in the reticular dermis if healing is to proceed without hypertrophic scarring. This depth is controlled by limiting the fluence (energy) and/or the number of passes with an ablative laser. Similarly, the number of coats of any chemical peeling agent should be limited and the appearance of skin and the resultant desired changes with application should be understood to prevent unwanted injury to skin (Figure 13.5).

Eccrine Glands

Eccrine sweat glands are distributed over the entire body surface but are particularly abundant on the palms of hands, soles of feet, and the forehead. Eccrine glands play an integral role in temperature regulation of the body. Eccrine sweat glands are coiled tubular glands derived from squamous epithelium that extends into the dermis. The sweat glands are controlled by Sympathetic cholinergic nerves, which are controlled by a center in the hypothalamus. The hypothalamus senses core temperature directly via thermoreceptors in close proximity to circulating blood and also has input from temperature receptors in the skin. The hypothalamus maintains homeostasis by modifying sweat output, along with other thermoregulatory processes.

Apocrine Glands

Apocrine glands develop during early to mid puberty. They help regulate sweat production. Apocrine glands are mainly found in hair-bearing areas such as the axillae and genitalia. The sweat produced by these glands can have an odor due to the bacteria that break down the organic compounds in the sweat. Emotional stress increases the production of sweat from the apocrine glands.

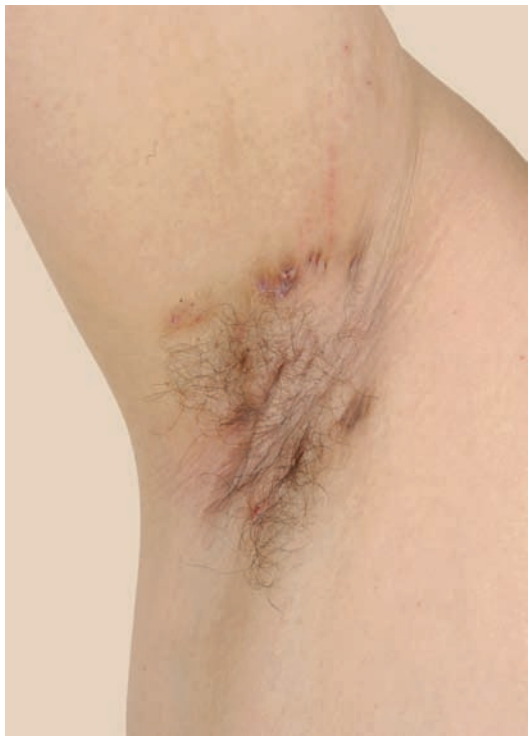

Figure 13.6. Hidradenitis suppurativa of the axilla. Note areas of folliculitis.

Clinical Correlation

Hidradenitis suppuritiva, which results in multiple abscesses, is a disease of the apocrine sweat glands and occurring predominantly in hair-bearing areas. An alternative mechanism of this condition is thought to occur from acne-like follicular obstruction and hence is referred to as acne inverse by some dermatologists and dermatopathologists. It is associated with repeated bouts of inflammation, infection, and abscess formation. Treatment usually consists of multiple courses of antibiotics. Severe cases not responsive to conservative treatment require radical debridement and secondary wound healing or reconstructive procedures (Figure 13.6).

Acne and Inflammatory Conditions of the Skin

Early in the teenage years acne can cause physical and emotional stress. There have been many products and procedures recommended for its treatment. However, none has proved to be uniformly successful. This condition is caused by the production of excess sebum associated with follicular hyperkeratinization. This leads to the formation of a keratin plug that obstructs the follicular opening and is clinically seen as a comedo. Subsequently, local inflammation and secondary contamination by skin flora lead to the formation of papules, pustules, and skin nodules. This is thought to be heavily influenced by the hormonal changes that occur during the teenage years. The general treatment is to target the various processes that are affected. Benzyl peroxide is used as an antibacterial agent and as a comedolytic agent. Topical antibiotics such as clindamycin are used to target the secondary bacterial overgrowth. Retinoids are the mainstay of acne treatment. They target the initial events in acne formation – follicular hyperkeratinization, excessive sebum production, and inflammation. They also improve the vascularity and turnover of the dermal components. Such retinoids include tretinoin in a topical format and isotretinoin given in a pill format. Isotretinoin should be avoided in patients with liver disease or lipid abnormalities. It is a potent teratogen. Women of child-bearing potential must use two forms of birth control and be closely monitored with monthly pregnancy tests while taking the medication. Systemic retinoids should also be avoided before any skin resurfacing procedures as they interfere with the re-epithelialization of the skin. Some agents such as vitamin C and plant extracts may be used as antioxidants. Various peels that use alpha hydroxyls, such as glycolic acid, kojic acid, and salicylic acid, are used to exfoliate the superficial dead layers of skin, thus clearing obstructed pores. More invasive peels using potent chemicals that penetrate deeper can be combined with the more superficial alpha hydroxyl agents to resurface the skin.

Skin Aging

Skin aging is a product of a variety of factors.[2] With age, the dermis changes anatomically and physiologically. Vital functions such as vitamin D production, sensory perception, wound healing, and other functions have been shown to decline.[3,9] The most evident component of skin change is dermal atrophy resulting from decreased production and turnover of collagen. Enzymes associated with post-translational processing of collagen decrease with age.

Hydroxylation and glycosylation of the collagen proteins also decline and cross-linking between collagen molecules tends to decrease with age. Fibroblast numbers that produce collagen and blood vessels decrease with age.[3] In women, collagen decline can be reduced with exogenous estrogen as there is a direct correlation between skin collagen production and estrogen.[16] It has been shown that estrogen has a positive effect on wound healing as estrogen increases the production of TGF-β secretion.

Skin thickness also changes with age. These changes can be evident as early as age 20 and continue into late adulthood. In women, decline in skin thickness can reach up to 1.13% per year. The thickness of skin is directly related to and dependent on the components of the dermis, including collagen, elastin, and the ground substance.

Wound healing can also be impaired with the aging process. Proper wound healing is dependent on neovascularization, granulation, collagen deposition, and re-epithelialization, which all decrease in efficiency with age.

Clinical Correlation

Treatments exist to help slow down the signs of skin aging and in some instances reverse the aging process. These treatments target various processes in the skin and include sunscreen to reduce the harmful ultraviolet radiation of the sun preventing photodamage to skin, antioxidants (vitamin C, vitamin E, coenzyme q10, lipoate) to combat the oxygen-free radicals, DNA repair agents (niacin, folate, reemergence) to help repair damaged cells, cell turnover stimulants such as retinoic acid and niacin, and epidermal barrier enhancing agents (niacin). The newest of these agents are topical prodrugs aimed at delivery of molecules that penetrate the skin and function as precursors at various stages to improve skin health. Other agents target the loss of the ground substance and the decrease in the collagen content of the skin. Such products include the various fillers used to augment the soft tissues (hyaluronic acid fillers, collagen-based fillers, hydroxyapatite fillers, PMMA fillers, and other agents). The plastic surgery and dermatology literature is replete with articles that describe results with these various agents.

The use of hyaluronic acid for soft tissue augmentation is a popular treatment gaining widespread acceptance in plastic surgery and dermatology. This product is associated with minimal side affects and requires no skin testing. Maintenance of volume enhancement can be seen with time as the product is hydrophilic, recruiting water molecules and maintaining the augmentation of the soft tissues. A new calcium hydroxyapatite treatment mixes calcium hydroxyapatite with a polysaccharide gel for soft tissue augmentation use. This treatment serves as a base for collagen growth and has been shown to be safe in humans.

The use of dermal fillers is rapidly increasing. Recent estimates by the American Society of Plastic Surgeons estimate that more than 5,00,000 patients were injected with hyaluronic acid in 2006.[1] It should be noted that these fillers are FDA approved for nasolabial fold correction only. Any other use is *off-label*.

Classification of Skin Types

Often in plastic surgery and dermatology, skin is classified in terms of its response to sun exposure and the resultant changes that occur with photodamage. This is termed Fitzpatrick classification (Figure 13.7). The resultant pigmentation/tanning changes that occur with sun exposure are due to alterations in the production of melanin. There is a paucity of melanin in the lighter skin types and is usually only located in the basal layer. In the darker skin types, there is an abundance of melanin and it is also present in more superficial layers of the skin. The presence of melanin plays a protective role in minimizing photodamage to skin.

Clinical Correlation

A thorough knowledge of skin type and the ability to correctly assign a skin classification to a patient are important when skin resurfacing or nonablative laser or light-based treatment is being considered. Such classification can help a plastic surgeon decide on the appropriate skin care or rejuvenation procedure. Inappropriate selection of skin rejuvenation techniques performed in skin types IV through VI, including chemical peels, laser resurfacing, or dermabrasion, can result in hypopigmentation or hyperpigmentation (Figure 13.7). In general, melanin suppression for a minimum of 6 weeks using bleaching agents such as hydroquinone 4% should be used before superficial, intermediate, or deep peeling in darker skinned individuals (Fitzpatrick IV–VI).

Figure 13.7. The Fitzpatrick skin classifications. I (never tan, always burns) to VI (dark, African-American skin type).

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14

Congenital Malformations

Jennifer Lucas, Christopher Gasbarre, and Allison T. Vidimos

Summary

Vascular birthmarks are a common cutaneous finding in newborns and infants. Characterization of these vascular anomalies is challenging and the terminology has historically been confusing. A classification system was proposed by Mulliken and Glowacki and modified by the International Society for the Study of Vascular Anomalies in 1996 to help clarify this confusion. It separates vascular anomalies into two distinct categories, tumors and vascular malformations, based on clinical appearance, natural history, and pathologic characteristics. The term hemangioma is reserved for congenital vascular tumors, while vascular malformations include a variety of lesions with varying clinical presentations. Proper identification and classification of these lesions have important implications in their management and treatment.

Abbreviations

AVM	Arteriovenous malformations
BRBN	Blue rubber bleb nevus
CM	Capillary malformations
CMTC	Cutis marmorata telangiectatica congenital
GVM	Glomuvenous malformations
ISSVA	International Society for the Study of Vascular Anomalies
KTS	Klippel–Trenaunay syndrome
LM	Lymphatic malformation
NICH	Noninvoluting congenital hemangiomas
PWS	Port wine stains
PHACES	Posterior fossa, Hemangioma, Arterial, Cardiac defect, Coarctation of the aorta, Eye anomaly, Sternal clefting, or Supraumbilical raphe
PDL	Pulsed dye laser
RICH	Rapidly Involuting Congenital Hemangiomas
SWS	Sturge–Weber syndrome
VM	Venous malformations

Infantile Hemangiomas

Introduction

Vascular birthmarks are a common cutaneous finding in infants and children. Historically, characterization of these vascular anomalies was challenging, and the term hemangioma has been used indiscriminately. A classification system purposed by Mulliken and Glowacki and modified by the International Society for the Study of Vascular Anomalies in 1996 has helped to clarify this confusion. It separates vascular anomalies into two distinct categories, tumors and vascular malformations, based on their clinical appearance, natural history, and pathologic characteristics (Table 14.1).[9]

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Table 14.1. Vascular anomaly classification.

Tumor	Malformation
Endothelial cell hyperplasia	Vascular hamartoma
Present in early infancy	Present at birth
Rapid proliferation	Growth with the child
Spontaneous involution	No involution

Epidemiology

Infantile hemangiomas are the most common tumor of childhood affecting approximately 1–2% of neonates and up to 10–12% of 1-year-old infants.[17] Present in all races, Caucasians are most commonly affected. For unclear reasons, there is a female predominance, with a female-to-male ratio ranging from 2:1 to 5:1.[17,23] Other correlations include prematurity, low birth weight, and chorionic villus sampling at gestational weeks 9–12.[10,23,42] Inheritance is sporadic; however, in six families an autosomal dominant inheritance has been demonstrated.[6]

Clinical Features/Natural History

The clinical appearance of an infantile hemangioma varies depending on the depth of the tumor and its stage of development. Clinical subtypes include superficial (50–60%), deep (15%), and mixed (25–35%) hemangiomas.[17] Superficial hemangiomas, commonly referred to as "strawberry hemangiomas," are bright red, raised, incompletely blanching plaques (see Figure 14.1). Deep hemangiomas, often referred to as "cavernous hemangiomas," are soft, bluish, ill-defined nodules or masses (see Figure 14.2). Mixed hemangiomas have a deep and superficial component

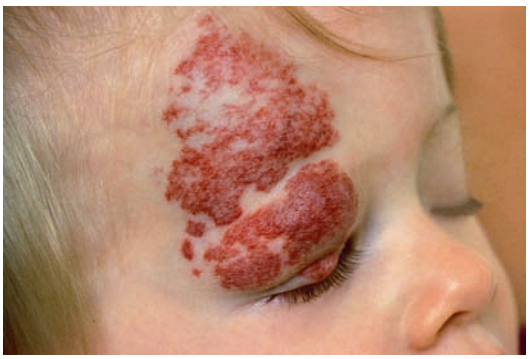

Figure 14.1. Superficial hemangioma of the forehead and upper eyelid causing obstruction of vision.

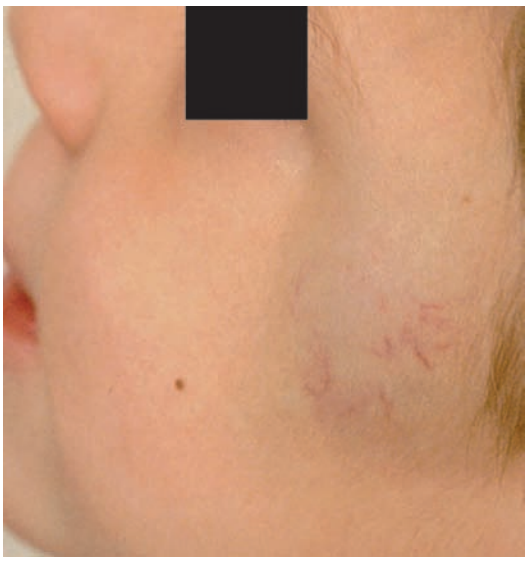

Figure 14.2. Deep hemangioma of left cheek with overlying telangiectasias.

(see Figure 14.3a and b). The typical presentation is of a solitary tumor but 15–30% of the time multiple lesions are present.[17] The most common location is the head and neck (60%) followed by the trunk (25%) and extremities (15%).[9]

Hemangiomas have a predictable natural history. Typically absent at birth, a precursor lesion, ranging from a telangiectatic or pale macule to a scratch or bruise, is observed in half the infants. Within weeks to months of birth, the proliferative phases ensue and the hemangioma becomes evident. Growth is most rapid in the first 3–6 months, and maximal size is obtained by 9–12 months of age. Involution classically ensues by 12–18 months, correlating clinically to centrifugally spreading pallor and compressibility. Complete involution follows at a rate of 10% per year (Rule of 10), such that by 9 years of age, 90% have involuted.[9] Residual changes persist in half the patients, especially if involution occurs after 6 years of age.[16] The changes range from telangiectasias and atrophic wrinkling to redundant skin or scarring.

Clinically Important Presentations

Segmental Hemangiomas

Segmental hemangiomas are large, plaque-like, linear, or geometric tumors associated with an increased risk of complications (Table 14.2).

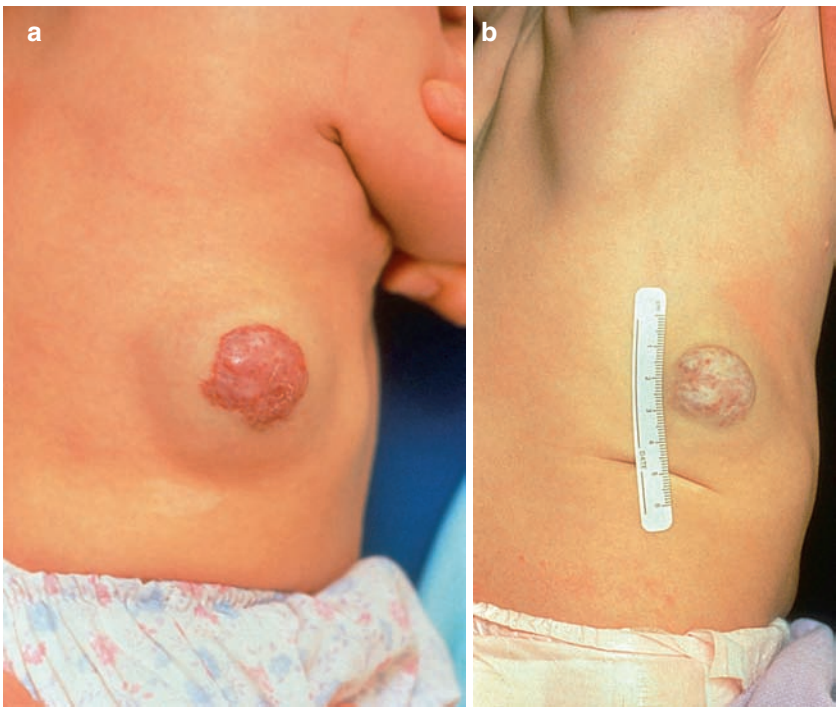

Figure 14.3. (a) Superficial and deep hemangioma (mixed) of the back with partial involution of superficial component. **(b)** Mixed hemangioma showing further involution of superficial and deep component with no intervention.

Table 14.2. Complications of cutaneous hemangiomas.

Complication	Hemangioma characteristics
Ulceration	Perioral, perineal, skin fold location
Airway obstruction (laryngeal and/or subglottic involvement)	Beard distribution
Permanent visual deficits	Periorbital
PHACES syndrome	Large segmental cervicofacial
Spinal dysraphism, tethered cord	Lumbosacral and segmental
Genitourinary anomalies	Lumbosacral and segmental
Hypothyroidism	Large hemangiomas productive of 3-iodothyronine deiodinase

They carry a higher risk of ulceration and are often a marker for visceral involvement, syndromic associations, and poorer outcome.[35]

Ulcerated Hemangiomas

Ulceration is the most common complication affecting approximately 10% of hemangiomas. Asso-ciated with chronic irritation, they are located in areas of high friction, such as the perioral skin, skin folds of the neck, and anogenital regions (see Figure 14.4). Segmental morpho-logy, large size, and mixed clinical type are other predisposing features. The ulcerations usually present around 4 months of age during the tumor's proliferative phase. They are more likely to bleed (41%) and have an increased risk of infection (16%).[12]

Airway Hemangiomas

Beard-like, segmental hemangiomas of the mandibular area involving the preauricular skin, lower lip, chin, and anterior neck often correlate with an underlying visceral hemangioma of the upper airway (larynx or subglottic) (see Figure 14.5a and b). Rapid airway obstruction and respiratory distress can occur as the visceral hemangioma proliferates. The classic presentation is a 6- to 12-week-old infant with worsening inspiratory and/or expiratory stridor, cough, respiratory distress, hoarseness, and/or cyanosis. Imaging with direct laryngoscopic visualization is often required, and up to 40% of these children

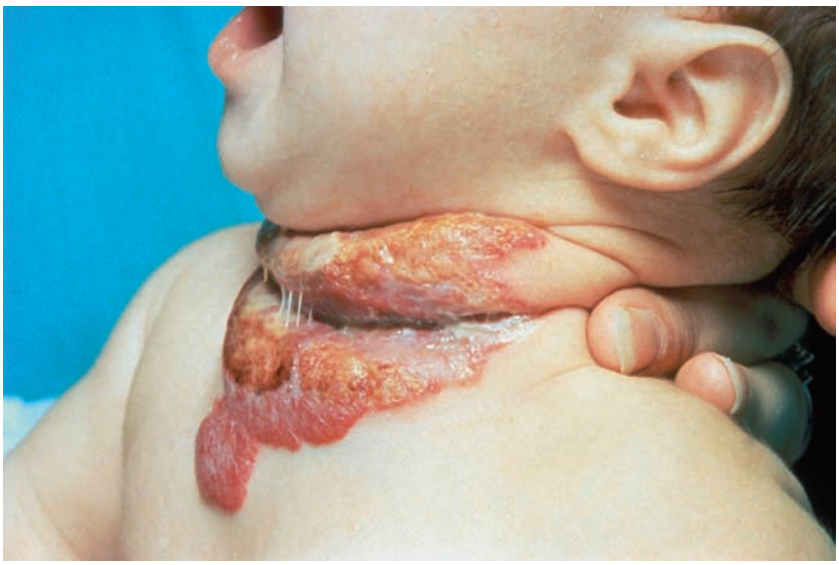

Figure 14.4. Ulcerated and infected extensive superficial hemangioma of skin folds of the neck in an infant.

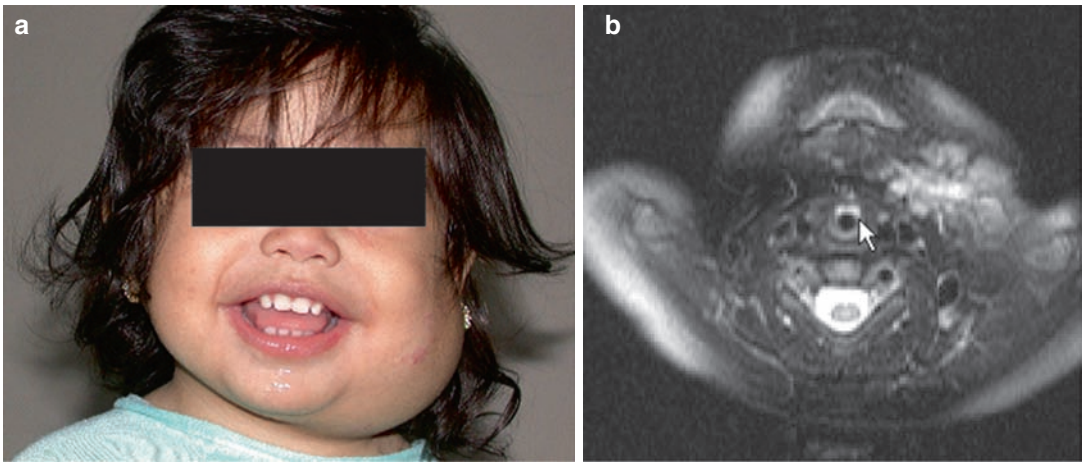

Figure 14.5. (**a**) Eighteen-month-old girl with large hemangioma of left side of neck and chin. Patient developed stridor. Laryngoscopy revealed a subglottic hemangioma, and patient required intubation and oral corticosteroids. (**b**) MRI of patient in (**a**) illustrating enhancing hemangioma along the anterior wall of subglottic trachea. The signal void of the airway lumen represents an endotracheal tube that flattens the free margin of the hemangioma.

require a tracheotomy. Close observation of these infants is essential, especially for the first 3–4 months of life.[35]

Periorbital Hemangiomas

Periorbital hemangiomas are an ophthalmologic emergency as their rapid growth can lead to visual compromise and permanent visual deficits (see Figure 14.6a and b). Potential sequelae include amblyopia, refractive errors, strabismus, astigmatism, and ptosis. Urgent ophthalmologic evaluation should be obtained and treatment started to avoid permanent visual disturbances.[11]

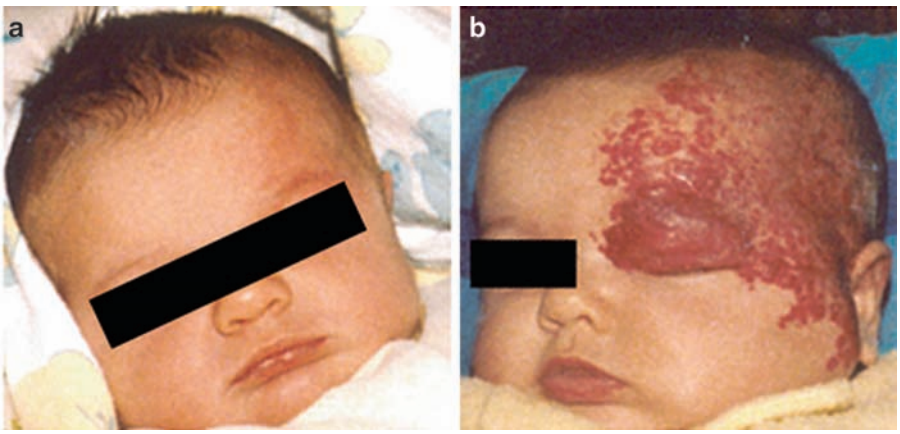

Figure 14.6. (**a**) Newborn baby girl with faint blanchable patches of left side of forehead and upper eyelid (*left*). (**b**) Baby girl at 4 weeks of age with rapidly growing superficial hemangioma causing closure of left eye. Cushingoid appearance is due to oral corticosteroids (*right*).

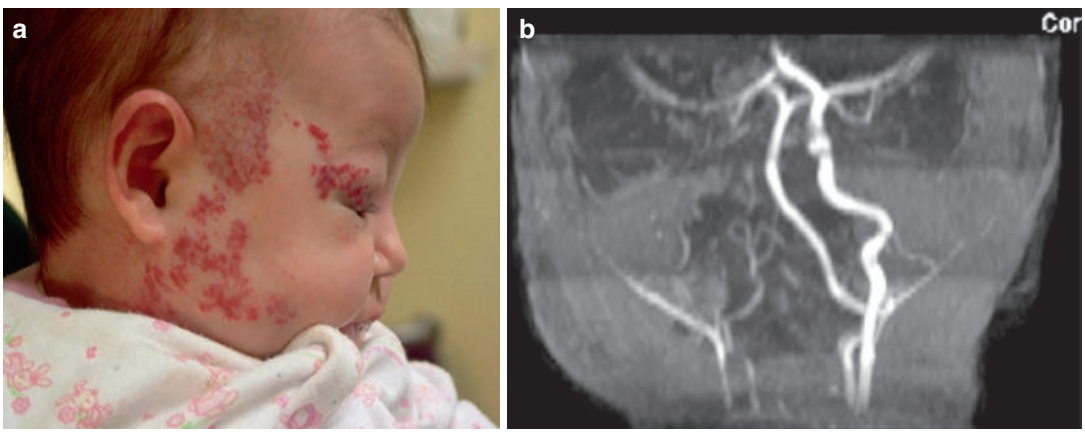

Figure 14.7 (**a**) Two-month old baby girl with large plaque-like hemangioma of right side of face, upper eyelid, and neck. Radiologic evaluation was performed to rule out PHACES. Patient was evaluated by a pediatric ophthalmologist who instituted oral prednisolone. (**b**) MRA of patient shown in (**a**) with occlusion of the right internal carotid artery. Branches visible on the right are external carotid branches. Carotid bifurcation is below the field of view. Right distal vertebral artery also shows poor flow-related enhancement in comparison with the left. Cardiac evaluation was normal.

PHACES Syndrome

PHACES syndrome is a multisystemic association of anomalies consisting of a large segmental cervicofacial hemangioma (see Figure 14.7a) and at least one of the following: Posterior fossa malformation (most commonly a Dandy–Walker malformation), hemangioma, arterial anomaly (carotid [see Figure 14.7b] and cerebral artery anomaly, persistent embryonic arteries), cardiac defect, coarctation of the aorta, eye anomaly (micro-ophthalmia, cataracts, glaucoma), sternal clefting, or supraumbilical raphe. The majority of patients (70%) present with only one extracutaneous finding. A structural cerebral or cerebrovascular anomaly accounts for 72% of cases. Patients with cerebrovascular disease are at increased risk for ischemic strokes. Approximately 90% of affected infants are female.[36,37]

Segmental Lumbosacral Hemangiomas

Midline lumbosacral segmental hemangiomas are associated with an increased risk of spinal dysraphism, a tethered cord, and/or genitourinary anomalies. Spinal dysraphism has been demonstrated in 17.5% of these hemangiomas. A tethered cord can result in permanent neurologic deficits. As such, aggressive investigation with a thorough neurologic examination and spinal imaging (MRI, ultrasound) should be pursued. A deviated supragluteal cleft is a particularly concerning sign. Syndromic findings have also been reported (anorectal anomalies, renal anomalies, abnormal genitalia, bony sacral abnormalities, and spinal cord abnormalities).[21,48]

Benign/Diffuse Neonatal Hemangiomatosis

Benign neonatal hemangiomatosis refers to multiple (≥5) discrete cutaneous hemangiomas present at birth or within the first few weeks of life. This syndrome runs a benign course. Diffuse neonatal hemangiomatosis, in contrast, consists of multiple cutaneous hemangiomas in association with visceral involvement. Left untreated, the mortality rate is as high as 77%.[30] The most common extracutaneous site of involvement is the liver, but any site including the gastrointestinal tract and brain can be affected. When associated with an arteriovenous malformation (AVM), high-output cardiac failure, hepatomegaly, and/or anemia can occur. Infants with ≥5 hemangiomas should have a complete history and physical examination and may require adjunctive studies to rule out extracutaneous involvement, including an abdominal ultrasound with Doppler to assess hepatic involvement, complete blood count, liver function tests, coagulation studies, guaiac of the stools, chest x-ray, CNS imaging, echocardiogram, electrocardiogram, and consultation with a pediatric cardiologist. Close follow-up is essential.[9]

Hypothyroidism

Large hemangiomas, especially hepatic hemangiomas, produce high amounts of 3-iodothyronine deiodinase, which inactivates thyroxine. Infants cannot compensate for this deactivation, resulting in hypothyroidism. This places them at risk for irreversible mental sequelae.[26]

Congenital Hemangiomas

Congenital hemangiomas account for 3% of all hemangiomas and are present in their mature form at birth. In utero development, including a proliferative phase, is believed to account for their different clinical appearance and course. As a group, they are solitary tumors composed of high-flow vasculature. Histologically, they can be differentiated from infantile hemangiomas by staining negative for GLUT-1. A subset of these hemangiomas, rapidly involuting congenital hemangiomas (RICH), undergo early involution that is usually completed by 6–14 months of age. Noninvoluting congenital hemangiomas (NICH) continue to proliferate after birth and may worsen with maturity (see Figure 14.8). NICH lesions may require surgical excision, while RICH lesions can be observed for involution.[29]

Kasabach–Merritt Phenomenon

Kasabach–Merritt phenomenon is a consumptive coagulopathy associated with kaposiform hemangioendotheliomas and tufted angiomas. Hemangiomas are not associated with this phenomenon. The clinical presentation is that of a rapidly enlarging vascular tumor in association with thrombocytopenia, anemia, and disseminated intravascular coagulation. Given the high mortality rate, early aggressive treatment and close observation are essential.[32]

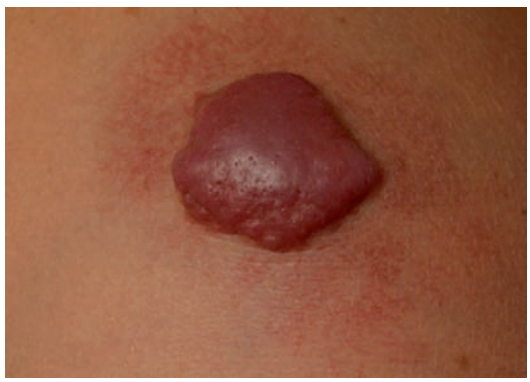

Figure 14.8. Noninvoluting congenital hemangioma (NICH) of the left shoulder of a 14-year-old girl. The central vascular nodule grew rapidly at puberty. MRI/MRA confirmed the findings of a hemangioma.

Pathology and Pathogenesis

Infantile hemangiomas are composed of lobules of endothelial cells with few lumens. During the proliferative phase these cells enlarge and rapidly divide. During involution the vascular lumens dilate and the endothelial cells are replaced with fibrofatty tissue. Mast cells are numerous during proliferation and involution. Hemangiomas stain positive for placenta-associated vascular antigens GLUT-1, FcyRII, Lewis Y antigen, and merosin (North et al. 2001). They lack Weibel–Palade bodies.

The pathogenesis remains unclear and is likely multifactorial. Current hypotheses include sequestered angioblasts, a placental or trophoblastic origin (given their positive staining), an embryonal developmental field defect, or mutations of the cytokine regulatory pathway leading to deranged angiogenesis.[3]

Treatment

Most infantile hemangiomas (80–90%) are uncomplicated and amenable to treatment with active nonintervention.[17] Family counseling and frequent, regular follow-up, especially during the proliferative phase, are essential. The family must be counseled on a hemangioma's natural history, potential for complications, and realistic expectations of the final cosmetic outcome.

High-risk hemangiomas require treatment, that is, those threatening the function of a vital organ, structure, or life of the patient. Other hemangiomas requiring treatment are those at risk for bleeding, scarring, disfigurement, or pain. Many of these complications are seen with ulceration. Large, plaque-like hemangiomas and those of the nose, lip, and ear have a high propensity for disfigurement. The psychosocial impact on the patient and family should also be minimized.[24]

Systemic corticosteroids are a mainstay of treatment for proliferating hemangiomas with a response rate of approximately 84%.[4] Prednisone 2–3 mg/kg daily (or an equivalent dosage of prednisolone) is given as a single morning dose for at least 1 month. Responsiveness can be determined within the first few weeks of treatment, but a taper must continue often for several months. Early discontinuation will result in rebound proliferation. Severe or life-threatening hemangiomas often require higher dosages. Rare side effects may include cushingoid appearance, transient growth retardation, personality changes, gastric irritation, hypertension, and adrenal suppression.[24]

Treatment with intralesional or ultrapotent topical corticosteroids is most effective in small, superficial hemangiomas. These are most extensively studied in tumors of the periocular location. Intralesional corticosteroids (triamcinolone acetonide10–40 mg/mL up to a total dose of ≤3 mg/kg and repeated every 6–8 weeks as needed) although effective can induce adrenal suppression, retinal artery occlusion, subcutaneous fat atrophy, dystrophic calcifications, dyspigmentation of the periocular skin, and eyelid necrosis.[11,24] Ultrapotent topical steroids have shown a 74% good or partial response with daily or twice-daily application to superficial hemangiomas.[20]

Other systemic treatment options include interferon alpha and vincristine. Interferon-alpha 2a and 2b at a dosage of 1–3 million U/m^2 body surface area daily can inhibit angiogenesis and induce shrinkage. The high rate of side effects (spastic diplegia, fever, malaise, neutropenia, anemia, and liver transaminitis) has, however, limited its use.[24] Vincristine (0.05 mg/kg in children < 10 kg or 1.5 mg/m^2 in children > 10 kg IV weekly) has replaced interferon alpha as a second-line systemic treatment.[9] The most significant side effect is an acute, mild, and transient peripheral neuropathy. Given the caustic nature of the medication, placement of a central line is often required.

Lasers targeting intravascular oxyhemoglobin can be used to treat superficial and symptomatic hemangiomas via photothermolysis. Specific indications include treatment of the proliferative phase, ulcerations, and bleeding. They can also be used to treat the residual telangiectasias of an involuted hemangioma. There is often transient erythema and bruising after treatment. Much less commonly hyperpigmentation, hypopigmentation, or scarring may occur following laser treatment. The most commonly used lasers are the 585 or 595 nm flashlamp pulsed dye laser (PDL). For thicker hemangiomas, the 755 nm alexandrite, 800–940 nm diode, or 1064 nm Nd:YAG may be used for deeper penetration of the laser light. Residual telangiectasias following involution can be

treated with the 532 nm KTP or the 585 or 595 nm PDL. Several treatments are often required on a 6–8 week basis. (author's experience[43])

Ulceration is the most common complication of a hemangioma and is associated with significant morbidity. Local wound care is the mainstay of treatment and often consists of topical antibiotics (mupirocin, bacitracin, metronidazole), barrier pastes (zinc oxide), and occlusive bandages or petroleum gauze. Vascular laser and systemic corticosteroids are sometimes required. Unique to ulcerated hemangiomas, becaplermin 0.01% gel, a recombinant human platelet-derived growth factor, has proven an effective treatment bringing about healing in 3–21 days.[38]

Other treatment options may include cryotherapy, surgical excision, or embolization. Topical imiquimod 5% cream applied 3 times weekly for a mean of 17 weeks may also induce improvement in superficial hemangiomas. In a retrospective study, 4 of 22 patients had complete remission.[25]

Vascular Malformations

Introduction

Vascular malformations are a broad category of disorders characterized by errors in vascular development that occur in 0.3–0.5% of the population.[18] As such, they are less common than infantile hemangiomas, lesions with which they have historically been confused. Historically, congenital vascular tumors were classified according to their clinical and/or histological appearance regardless of biologic behavior, leading to confusing terminology. Mulliken and Glowacki proposed the first classification scheme to incorporate biologic behavior of these lesions, separating hemangiomas from vascular malformations.[39] Unlike hemangiomas, vascular malformations have an equal gender distribution and, while also present at birth, do not undergo rapid growth followed by involution. Instead, vascular malformations tend to grow proportionally with the child and exhibit greater prominence at puberty. Histologically, they demonstrate mature and nonproliferative vascular or lymphatic channels, lacking expression of the proliferative markers and antigens associated with hemangiomas. The updated International Society for the Study of Vascular Anomalies (ISSVA)/Mulliken classification of 1996 therefore distinguishes vascular tumors (which include hemangiomas) and vascular malformations.[15] Despite this separation, the term "hemangioma" continues to be inappropriately used in the literature to describe various vascular malformations. Vascular malformations are further subdivided based on their flow characteristics and likely primary vascular component: slow-flow lesions typically resulting from venous, capillary, or lymphatic malformations (LMs), fast-flow lesions representing arterial malformations, or a combination of the two.

Capillary Malformations

Clinical Features

Capillary malformations (CM), also referred to as port wine stains (PWS), are very common, affecting approximately 3 of 1,000 infants.[18] Usually present at birth, they have an equal gender distribution with a wide variety of clinical presentations. Initially, they present as pale pink macules or patches and may be seen on virtually any cutaneous surface. The head and neck are particularly common sites, and mucosal surfaces may also be involved in these cases (Figure 14.9). Clinical behavior seems to vary

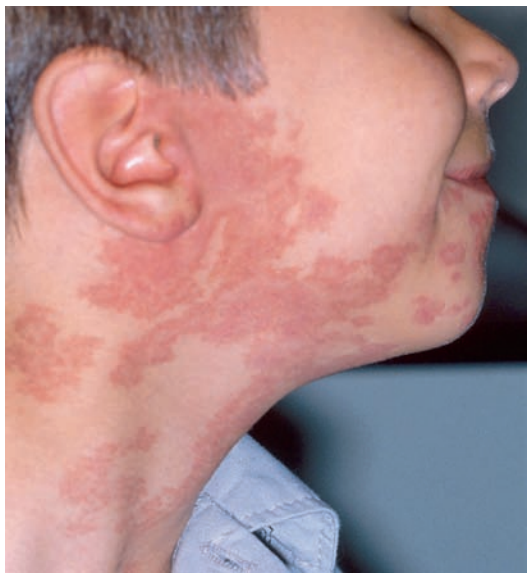

Figure 14.9. Capillary malformation (port wine stain). No tissue hypertrophy or vascular blebbing is noted.

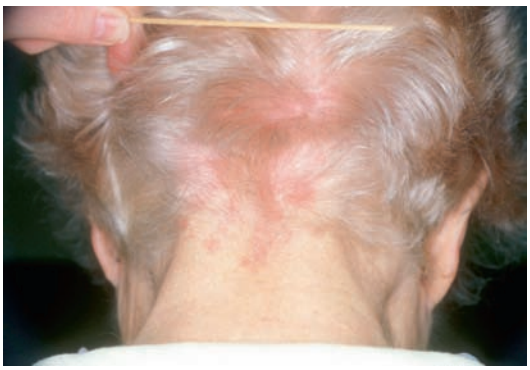

Figure 14.10. Capillary malformation. Located on the posterior neck, these lesions are often referred to as a "stork bite nevus" or nevus flammeus.

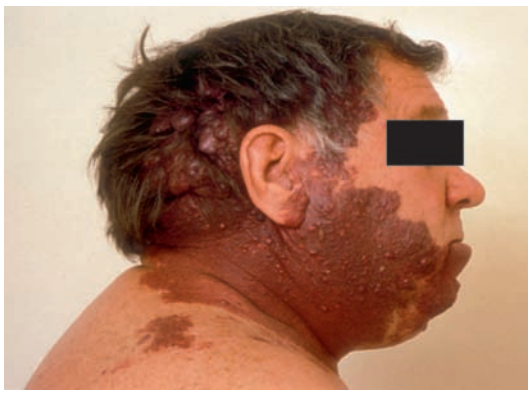

Figure 14.11. Longstanding capillary malformation with cutaneous hypertrophy and vascular blebs.

based on anatomic location. Lesions located on the nape of the neck and central forehead or face, referred to as "stork bite nevi" and "angel's kiss," respectively, tend to lighten and/or disappear within the first few years of life and do not demonstrate tissue hypertrophy or vascular blebs when persistent (Figure 14.10). Other terms used in the past to refer to these lesions include nevus simplex, nevus flammeus, vascular stain, and fading macular stains. These lesions likely represent a distinct clinical entity separate from true capillary malformations. "True" capillary malformations of the head and neck have a tendency to darken and thicken over time, often with hyperkeratosis and vascular blebs (Figure 14.11). Lesions on the trunk and extremities may be red at birth and fade over time. Hypertrophy and vascular blebs, when seen on the extremity, usually are associated with a lymphatic or lymphatic-venous malformation (VM). Extremity lesions may also be associated with bone and soft tissue hypertrophy. Clinically, extremity lesions are more likely to have well-defined, rather than patchy, borders. Midline lesions may be associated with occult spinal dysraphism, especially when associated with a cutaneous pit or underlying mass, while bilateral facial lesions have a higher association of associated eye and brain abnormalities.[18,33,47]

Pathogenesis

The appearance of capillary malformations is due to increased number of ectatic vessels of the papillary and reticular dermis, with a mean vessel depth of 0.46 mm, though this varies with anatomic location. (Eubanks, 2001) Lesions of the extremities and V3 dermatome tend to be deeper lesions. With age, mean vessel area increases, correlating with the change in clinical appearance. Lesions have also been noted to exhibit a decreased number of associated neurons (Rydh, 1991). It is unclear if abnormal neuronal control contributes to the abnormal flow characteristics of these lesions or if this finding is simply secondary to the malformation. While several gene loci have been associated with some capillary malformations, a definitive pathogenesis has yet to be elucidated. They are slow-flow malformations.

Diagnosis

The diagnosis of capillary malformations is based largely on clinical grounds. Doppler ultrasonography may be used to detect the presence of an arteriovenous fistula, as erythema overlying arteriovenous malformations (AVM) may mimic CMs. The physical examination should be directed toward detection of possible deeper vascular malformations and/or associated congenital defects. If suspected, magnetic resonance imaging (MRI), magnetic resonance angiography (MRA), duplex Doppler ultrasonography, lymphoscintigraphy, and bone radiographs may be of help. A discussion of the more common congenital syndromes is included at the end of this chapter.

Management

The treatment of choice for CMs is the flashlamp pulsed dye laser (PDL), effectively lightening up to 80% of all lesions (Reyes, 1990). These lasers

(577, 585, 595 nm) selectively target oxyhemoglobin, resulting in intravascular coagulation and thermal damage to the vessel wall. Though lesions are unlikely to clear completely, the majority of patients experience satisfactory cosmetic results. Generally, lesions associated with a more favorable response include those of younger patients, lighter in color, and located on the trunk.[45] Longstanding or hypertrophic lesions as well as those on the central face or extremities are more likely to respond poorly or require a greater number of treatments. The timing of intervention is somewhat controversial, but most agree that treatment during early childhood is preferable in order to reduce the psychological impact of these lesions and possibly produce a greater response.[1] Treatment of young patients with extensive lesions often requires general anesthesia. Recent evidence suggests that recurrence may occur as soon as 3–4 years after treatment, however.

Some studies suggest that as many as 35% of CMs will respond poorly to treatment.[27] Treatment options for these lesions include newer PDL (595 nm) with longer pulse widths and dynamic cooling devices. Stubborn lesions that were initially responsive to treatment often have a deeper component for which treatment with longer wavelengths such as the long-pulsed (millisecond) alexandrite (755 nm) and Nd:YAG (1,064 nm) lasers are helpful. Multiple-pass treatment has also been reported to be beneficial.

Venous Malformations

Clinical Features

Venous malformations are much rarer than capillary malformations and typically present as soft, compressible blue nodules or masses that enlarge with activity, crying, or in a dependent position. While typically present at birth, they may not be evident until the patient gets older, most notably between infancy and puberty. There is no palpable thrill or temperature change with these lesions, and they may be located on any site. Head and neck VMs may be more extensive than apparent on examination and may involve musculature, mucosa, and parotid gland (Figure 14.12). As the lesions enlarge, bony abnormalities may result. There may be associated recurrent bleeding, airway obstruction, cosmetic deficit, dental abnormality, and speech impediment.

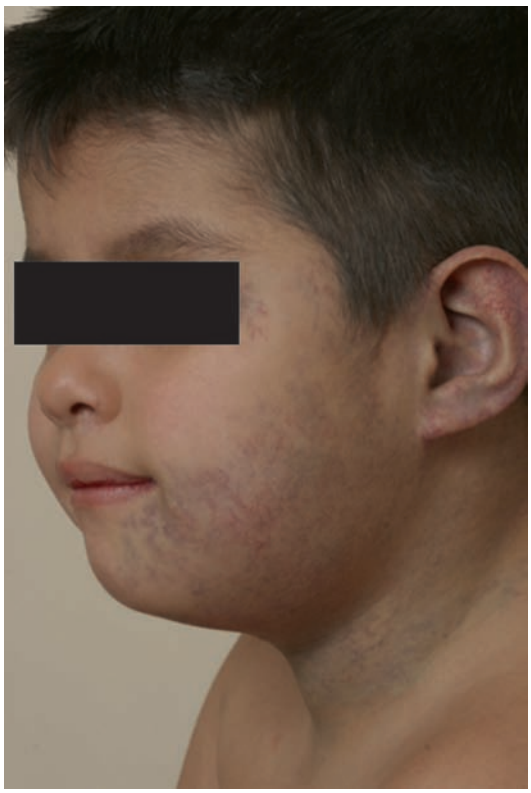

Figure 14.12. Venous malformation. This patient presented with enlargement of his left cheek and neck.

Lesions of the extremity are usually local, but they may extend into underlying joints or musculature, resulting in decreased limb circumference and/or slight hypertrophy. This should be distinguished from Klippel–Trenaunay syndrome (KTS), which is discussed later. Lesions without a significant superficial component may go unrecognized for some time, until the patient presents with functional impairment or pain. Also associated with VMs are phlebolith formation and coagulopathy due to localized intravascular coagulation within the lesion. This is distinct from the Kasabach–Merritt syndrome associated with infantile vascular tumors.

Pathogenesis

Venous malformations (VM) are slow-flow vascular malformations composed of numerous ectatic and irregular venous channels in the dermis. There is no known molecular basis for sporadic VMs, though some familial cases have

been linked to mutations in the endothelial receptor Tie2, which plays a role in the branching and sprouting of the capillary plexus. Previous terminology has included venous angioma, cavernous angioma, cavernous hemangioma, and phlebangioma. These terms may imply similarity with true hemangiomas (a proliferative lesion) and should be avoided.

Diagnosis

Ultrasonography may help to diagnose a VM, but it will not be able to evaluate the size of the lesion. MRI may be useful to evaluate the extent of the VM. MRA may help to identify a large feeding vessel amenable to sclerotherapy. A coagulation profile will rule out underlying inwwtravascular coagulopathy when suspected.

Management

Management of VMs requires a multidisciplinary approach. Treatment modalities include sclerotherapy, surgical excision, or a combination of both. VMs of the head and neck are often amenable to sclerotherapy by an interventional radiologist.[44] Multiple treatments may be required. The lesions are unlikely to fully resolve but will shrink significantly. Surgical excision with reconstruction may be a reasonable option following sclerotherapy. Surgical resection alone is not generally recommended due to the risk of bleeding and recurrence. VMs of the extremities are often more extensive and less amenable to interventional treatment. Eighty-eight percent of patients with large lesions exhibit chronic localized intravascular coagulation and mildly decreased platelet count (Enjolras, 1997). Partial treatment with sclerotherapy may decrease pain and swelling but is not curative. Compression garments help decrease pain, swelling, and intravascular coagulation.

Glomuvenous Malformations (Glomangioma)

Clinical Features

Glomuvenous malformations (GVM) are relatively rare vascular malformations characterized by purple to blue nodules with a "cobblestone" or "pebbled" surface. They are often firm and painful with palpation and typically involve only the skin and subcutis. Approximately 60% of cases are familial and inherited in an autosomal dominant pattern.[18] A subset called congenital, plaque-type glomuvenous malformations may be mistaken for other vascular malformations at birth but may progress with significant cutaneous thickening.

Pathogenesis

GVMs are composed of numerous ectatic vascular channels surrounded by glomus cells and are caused by mutation in the *glomulin* gene on chromosome 1p21–22. The glomus cells are believed to be aberrantly differentiated vascular smooth muscle cells.[8]

Diagnosis

Diagnosis is made on clinical grounds.

Management

Small or well-localized lesions may be amenable to surgical excision. Sclerotherapy is less effective for GVMs than for VMs. Compression garments may increase the pain associated with these lesions.

Arteriovenous Malformations

Clinical Features

Most commonly located on the head and neck, AVMs exhibit no gender predilection (Figure 14.13). About one-half of all AVMs are evident at birth, while approximately one-third

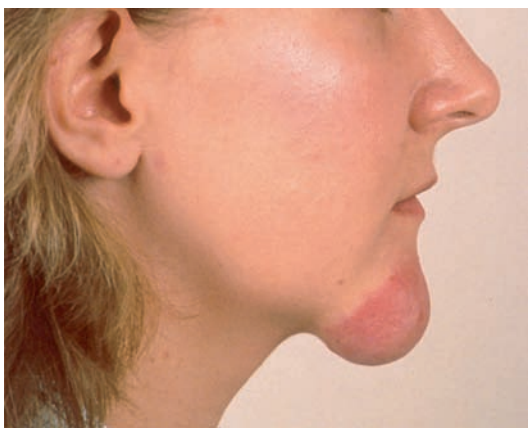

Figure 14.13. Arteriovenous malformation.

become apparent during childhood. They are staged using the ISSVA-approved Schobinger scoring system.[28] Stage I is characterized by asymptomatic and quiescent lesions from birth through adolescence that are either not clinically apparent or resemble involuting hemangiomas or CMs. Thrills, bruits, and increased warmth may be present. If progressive, stage II lesions begin during adolescence and are characterized by darkening, enlargement, and invasion of deeper structures. Tortuous vessels may appear. Stage III involves deeper tissue disruption with necrosis, ulceration, bleeding, pain, and lytic bone lesions. Stage IV occurs in the setting of cardiac decompensation due to high-output heart failure.

Pathogenesis

AVMs are considered arterial and venous vessels connected without an intervening capillary network formed due to failed regression of arteriovenous channels of the embryologic retiform plexus. They are considered high-flow malformations. Familial cases of AVMs in association with CMs have been linked to mutation in *RASA1*, a gene encoding p120-rasGAP (Eerola, 2003). Several other signaling molecules have been implicated in the formation of sporadic AVMs, though these have yet to be fully evaluated in humans.

Diagnosis

Diagnosis is made on clinical and radiographic grounds. Ultrasonography can best characterize the flow characteristics of the lesion and is especially recommended for pediatric patients. MRI may be helpful in delineating the extent of the lesion, while MRA can identify vessels amenable to therapeutic intervention.[14]

Management

Treatment of AVMs can be challenging. Due to recurrences following partial treatment, it is recommended that asymptomatic or quiescent lesions without impeding functional impairment be managed conservatively and followed closely. Conversely, stage I lesions are often more amenable to therapy, suggesting that early intervention may be a viable approach to prevent the complications associated with stage II–IV lesions. Combination treatment with initial embolization followed by surgical resection is the treatment of choice.[46] Embolization alone may be used for symptomatic improvement in many lesions.

Lymphatic Malformations

The terminology regarding lymphatic malformations is unclear. In this discussion, the term lymphatic malformation (LM) refers to congenital localized malformations. These have historically been separated as either deep (macrocystic) or superficial (microcystic) lesions. Lymphangioma circumscriptum is a term used to describe superficial skin lesions, while cystic hygroma is a form of deep LM on the neck or axilla.

Clinical Features

LMs are typically apparent by 2 years of age, though most of them are present at birth. Microcystic LMs may occur on any part of the body, though they favor the proximal limbs. They appear as clusters of shiny clear to red-black papules resembling "frog spawn." While they appear localized and discrete, they may be part of much larger and deeper lesions.[41] Lesions on the genitalia have been confused with genital warts.[13] Lesions commonly bleed or ooze clear lymphatic fluid with mild trauma. Macrocystic lesions usually present at birth as poorly defined subcutaneous masses that enlarge over time. Complications from deep LMs are dependent on the location of the lymphatic malformation. LMs on the head and neck may impair vaginal delivery, cause airway obstruction, mandibular hypertrophy, or lead to speech or feeding abnormality. Other reported location-dependent complications include ocular swelling, congenital cataract, strabismus, diplopia, abdominal distension, and volvulus.[18]

Pathogenesis

While debate still exists as to the etiology of LMs, many theorize that LMs are malformations of the lymphatic system and not true neoplasms. Some authors have proposed that LMs represent somatic or mosaic mutations in genes regulating normal lymphangiogenesis.

Diagnosis

The diagnosis of LMS is typically made clinically, especially when a superficial component exists.

A skin biopsy may confirm the diagnosis. Many macrocystic LMs are diagnosed prenatally via ultrasonography, though many of these children do not survive to delivery. Imaging of superficial lesions is important to rule out deeper involvement.[34] While MRI is the gold standard, ultrasonography may also be helpful in younger infants. Additionally, Doppler studies can distinguish between slow-flow venous malformations and no-flow LMs.

Management

LMs that threaten vital functions must be managed quickly.[7] Patients noted to have large cervical LMs on ultrasound are delivered via cesarean section with immediate airway management as needed. The most common interventional treatment for well-defined lesions is surgical excision, though multiple procedures may be necessary. When removed in their entirety, recurrence rates are estimated to be near 25% and much higher with incomplete excision. Unresectable cases may be treated with percutaneous sclerotherapy, which is more effective in treating macrocystic LMs. Ablative lasers can be used for superficial LMs, though recurrence is common.

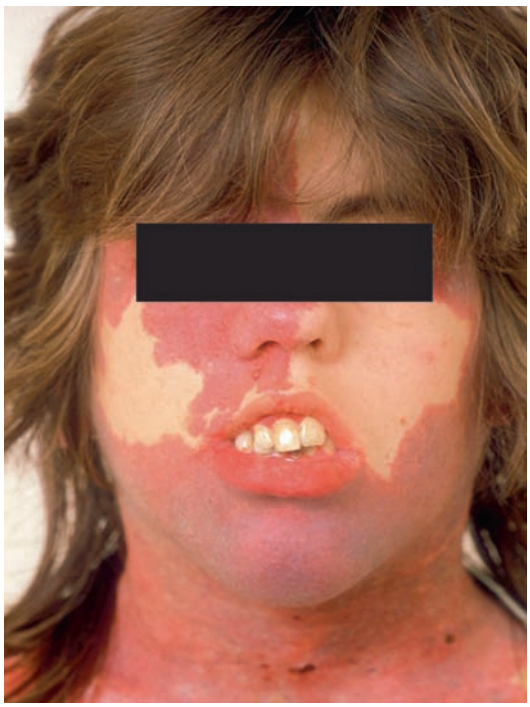

Figure 14.14. Sturge–Weber syndrome.

Syndromes Associated with Vascular Malformations

Sturge–Weber Syndrome

Sturge–Weber syndrome (SWS) is a congenital syndrome that includes the triad of facial CM, ipsilateral leptomeningeal angiomatosis, and vascular malformation of the choroid with glaucoma.[19,40] While classic SWS includes all elements of the triad, cases of partial expression have been reported. The facial CM of SWS is distributed in at least the V1 dermatome, but it may also include V2 or V3 distribution (Figure 14.14). Involvement of multiple dermatomes or bilateral involvement is associated with a higher incidence of ocular or CNS involvement. Leptomeningeal involvement is most commonly located over the occipital lobes. Associated findings include seizure, mental retardation, glaucoma, growth delay, and hemiplegia. Plain radiographs of the skull may detect "tram-track" intracranial calcifications after 2 years of age, though CT is more sensitive and may detect lesions at 1 year of age. MRI is the imaging modality of choice to detect intracranial vascular anomalies, though newer imaging modalities have been successful as well. SWS should be suspected in any patient with a CM in the V1 or V2 dermatome.

Klippel–Trenaunay and Parkes–Weber Syndromes

Klippel–Trenaunay syndrome (KTS) refers to the presence of CMs of the extremity in association with varicosities, soft tissue and bony hypertrophy, and possible deep venous malformations of the affected limb(s) (Figure 14.15). The above, in conjunction with an associated AVM, is referred to as Parkes–Weber syndrome. The affected limb will often be longer and have a greater circumference than the unaffected limb. Ultrasonography is useful to characterize the underlying vascular malformation, while MRI may detect the extent of the lesion and the presence of lymphatic anomaly. (Baskerville, 1985) KTS must be distinguished from an extensive venous malformation of the extremity. KTS exhibits minimal muscular involvement.

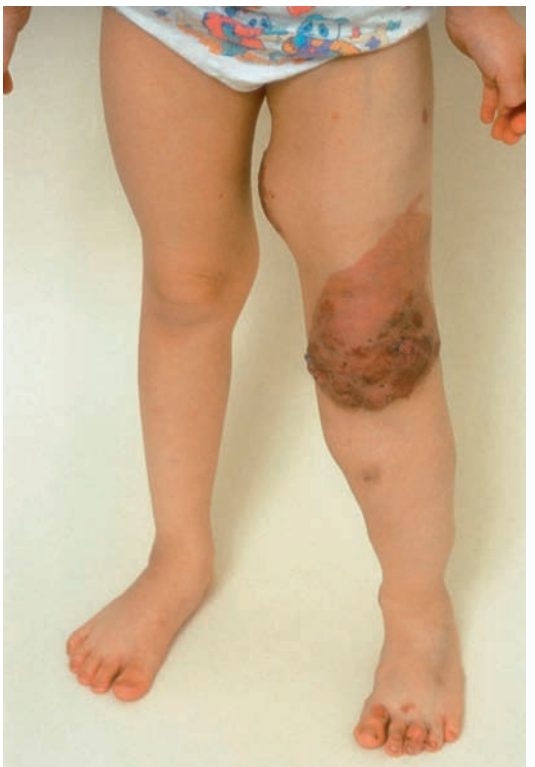

Figure 14.15. Klippel–Trenaunay syndrome.

Cutis Marmorata Telangiectatica Congenita

Cutis marmorata telangiectatica congenita (CMTC) is a congenital but mostly sporadic syndrome characterized by fixed reticulated violaceous patches that may be localized or widespread (Figure 14.16). It is differentiated by physiologic cutis marmorata by persistence after warming. Capillary malformations and varicosities may also be seen. While cutaneous lesions are most often the only manifestations of CMTC, associated findings may include overlying skin and soft tissue atrophy, limb hypoplasia or hyperplasia, congenital melanocytic nevi, dermal melanosis, scoliosis, syndactyly, anogenital abnormality, skull asymmetry, hypothyroidism, and developmental delay.[2,19] These features are most commonly present in cases where the cutaneous lesions are widespread. Other syndromes have been described of which CMTC is a part. Unlike capillary malformations, CMTC tends to lighten (but not resolve) over time, usually within the first 2 years of life. Treatment with a PDL has met with variable results.

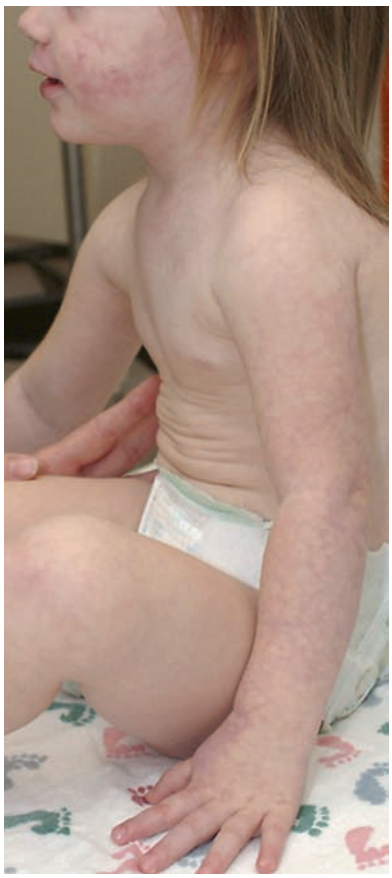

Figure 14.16. Cutis marmorata telangiectatica congenita.

Cobb Syndrome

Cobb syndrome is the association of spinal AVMs with an overlying congenital cutaneous AVM in the same dermatome. Identification of an AVM over the spinal column should raise the possibility of Cobb syndrome and possible neurologic complications.

Blue Rubber Bleb Nevus Syndrome

Blue rubber bleb nevus (BRBN) or Bean syndrome is a disorder of venous malformations occurring in the skin and gastrointestinal tract. It is characterized by the appearance of compressible or "rubbery" blue nodules that appear soon after birth and increase in size and number with time. While the trunk and extremities are the most common locations, lesions may occur anywhere on the skin or mucosa and are often tender. Orthopedic complications and pathologic

fracture may result from extension and compression of overlying cutaneous malformations.[19] The greatest degree of morbidity results from gastrointestinal lesions, typically in the colon or small intestine. Manifestations include hemorrhage, anemia, melena, abdominal pain, intussusception, and consumptive coagulopathy. Patients with multiple VMs or a clinical picture suggestive of BRBN should undergo evaluation for gastrointestinal involvement and anemia. Reportedly successful treatment methods for gastrointestinal lesions include surgical resection, laser ablation, and systemic steroids.

Proteus Syndrome

Proteus syndrome is characterized by a constellation of findings and diagnosed clinically on these grounds. Diagnostic criteria were proposed in 1998 and include one or more vascular malformations, connective tissue nevi (often of the palms and soles), epidermal nevi, disproportionate limb overgrowth, ovarian cystadenomas, parotid monomorphic adenomas, dysregulated adipose tissue deposition (lipomas or regional absence of fat), lung cysts, and distinct facies.[5] The clinical findings between patients may be quite varied, leading to diagnostic confusion.[49] Lung abnormalities (cysts, infections), renal abnormalities (cysts, vascular anomalies, diabetes insipidus), and neurologic impairment may also be seen. It is important to note that the presence of slow-flow vascular malformations may predispose these patients to deep venous thromboses and pulmonary emboli. The etiology of Proteus syndrome is still unclear, but it may be linked to mutation in the *PTEN* tumor suppressor gene. Treatment is supportive.

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15

Burn Trauma

J. Brian Boyd

Summary

Burn trauma is still a significant cause of morbidity and mortality in the United States. It causes a spectrum of disability and deformity primarily by damaging the integumentary system of its victims. However, it is the systemic effects caused by sepsis, fluid and electrolyte imbalance, shock, inhalation injury and myonecrosis that are the usual agents of death. Patients must be assessed and treated expectantly to ensure adequate rehydration, and prevent acute life-threatening complications resulting from infection, respiratory burns, poisoning and compartment syndromes. Close monitoring is required during the initial resuscitation when large volumes of fluid will need to be administered. The modern trend is for early excision of the burn wound to speed rehabilitation and lessen the risk of sepsis. This is facilitated by the increasing use of cultured skin and skin substitutes. Finally, the importance of rehabilitation and secondary surgery cannot be overstated in terms of re-integrating the burned patient into society as well as returning him or her to the workforce.

Introduction

In the United States, approximately 5,00,000 people are treated for burn injuries every year, resulting in 40,000 admissions to hospital. Burns may be thermal, electrical, or chemical; but most are thermal, resulting from house fires. Of 4,000 deaths due to burns, 3,500 follow domestic conflagrations. Today, burns constitute the third largest cause of accidental death in the United States.[1,8–10]

Pathology

Skin is the primary organ of injury in burns (Figure 15.1), and the harmful effects of burns are determined largely by the depth of injury and the surface area involved.

In the normal epidermis, the deeper layers divide to produce the stratum corneum and also contain pigment to protect against UV radiation; while the outer cells are dead, act as a mechanical buffer and form a watertight seal.

The dermis contains tough, elastic connective tissue containing sebaceous glands. Their secretions keep the skin waterproof and usually discharge around hair shafts. The dermis also contains hair follicles (that produce hair from each hair root or papilla); sweat glands; nerve endings, and blood vessels. The hair follicles, sweat glands, and sebaceous glands extend into the dermis and are lined with epithelium. Thus, there is an extensive network of epithelial cells within and occasionally – in the case of hair follicles – somewhat below the dermis as well. It is from these epithelial cells that regeneration occurs after partial-thickness burns and also after the harvesting of split-thickness skin grafts.

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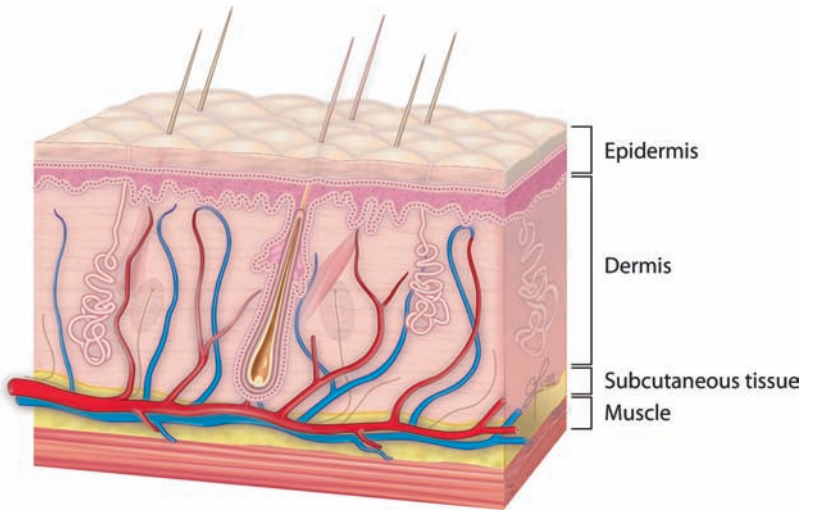


Figure 15.1. Cross section of normal skin showing the epidermis (pink) and dermis (pale pink) containing epithelial adnexial structures, such as hair follicles and sebaceous and sweat glands. Nerve endings are also present in the dermis as well as arteries and veins. Below the dermis lie fat and muscle. (With kind permission of The Cleveland Clinic Center for Medical Art and Photography © 2008.)

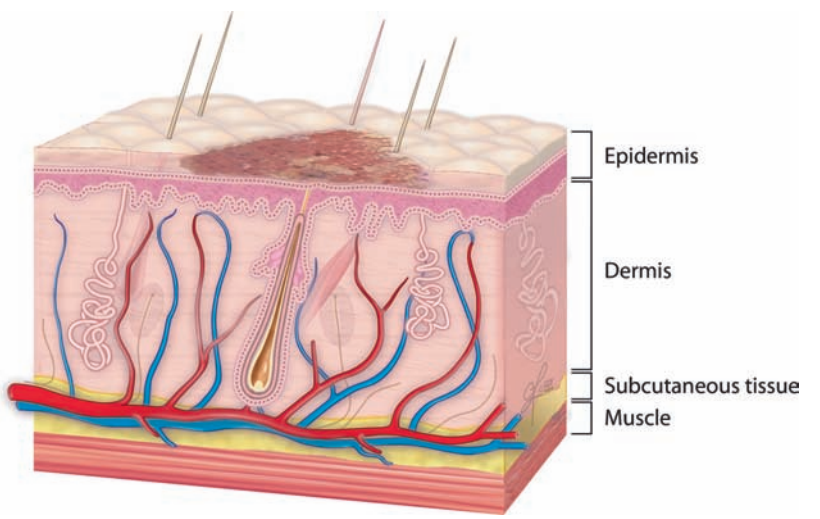


Figure 15.2. A superficial burn involves the epidermis only. There are superficial ulcers within the epithelium (peeling) but little or no bleeding. A complete recovery is anticipated. (With kind permission of The Cleveland Clinic Center for Medical Art and Photography © 2008.)

Burns are broadly classified into superficial, partial-thickness, or full-thickness. *Superficial* burns (Figure 15.2) involve the epidermis and are familiar to all as sunburn. The skin is reddened and warm, there is tenderness and pain together with edema, but blistering is absent. Part of the epidermis may peel, however. The affected skin blanches under pressure, and healing is complete in less than a week.

Partial-Thickness Burns

Partial thickness implies that there are enough epithelial remnants left deep to the burn for

spontaneous regeneration to occur. The damage extends through the epidermis and involves the dermis. However, the skin can regenerate from the epithelial lining of adnexial structures, such as hair follicles, sweat, and sebaceous glands. Partial-thickness burns are therefore characterized by blistering and loss of the epithelial layer (Figure 15.3). With severe partial-thickness burns (Figures 15.4 and 15.5), the blisters are often burned off, leaving a moist, shiny, weeping dermal surface. The burn is painful and exquisitely sensitive. It may be salmon pink to red and

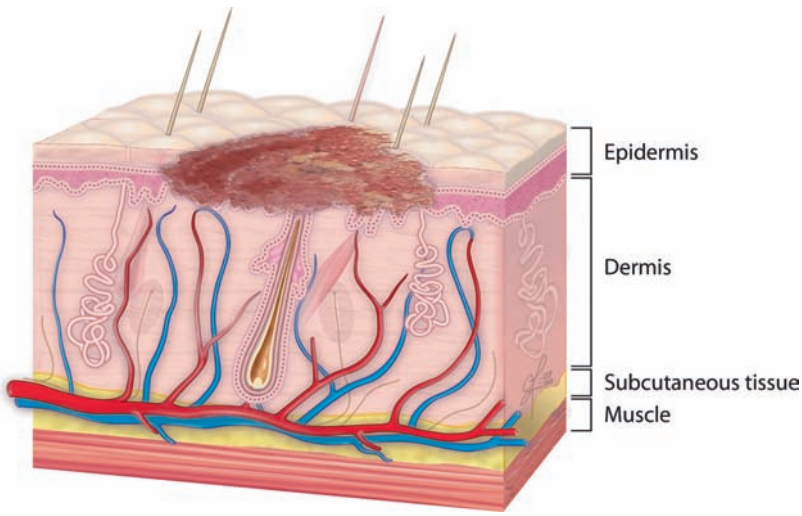


Figure 15.3. A superficial partial-thickness burn penetrates a short distance into the dermis. Blisters are characteristic, and there are plenty of epithelial remnants to permit spontaneous healing in 1–2 weeks. The burn rarely scars but can produce pigmentary changes. (With kind permission of The Cleveland Clinic Center for Medical Art and Photography © 2008.)

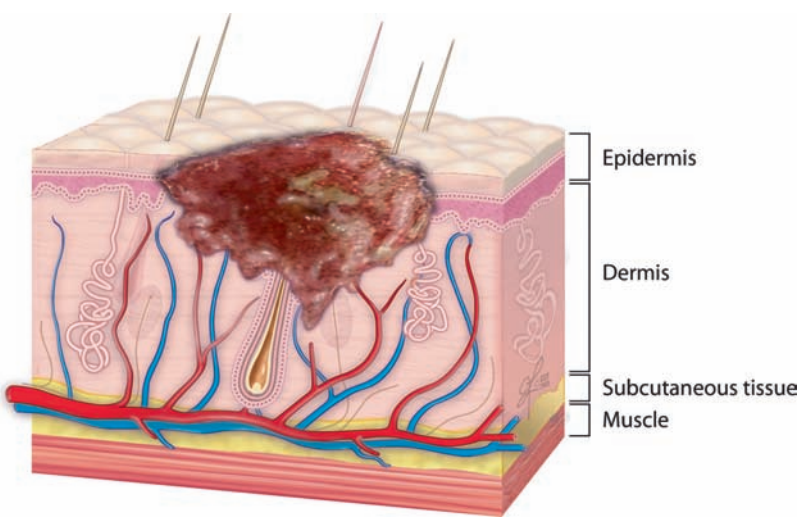


Figure 15.4. A deep partial-thickness burn. The blisters are burned off leaving a pinkish-white, moist surface. There is often a thin eschar. Healing is often prolonged 2–6 weeks and hypertrophic scarring is common. (With kind permission of The Cleveland Clinic Center for Medical Art and Photography © 2008.)

blanch to the touch; or, if deeper, it may be covered with a superficial soft yellowish-white eschar. The time taken for re-epithelialization is proportional to the burn's depth. It can vary from 7 to 21 days. The deeper ones, in the presence of infection or ischemia, can "convert" to full-thickness burns.

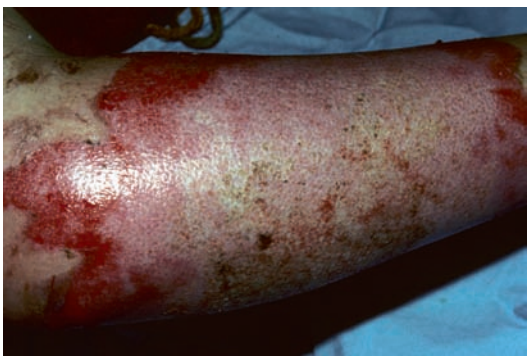


Figure 15.5. A mixed burn showing a central area of full-thickness injury (zone of coagulation) surrounded by a zone of stasis and then a zone of hyperemia. The last two zones are partial thickness. The zone of hyperemia will survive, while the zone of stasis may convert to full thickness unless adequate peripheral perfusion is reestablished and appropriate topical antimicrobial treatment applied.

Full-Thickness Burns

Full-thickness burns extend through the dermis and into fat. They leave no deep epithelial remnants and may heal spontaneously only by contraction and by migration of epithelial cells from healthy tissue at their periphery. These burns generally require skin grafting for optimal healing. Unlike with partial-thickness burns, the eschar formation in full-thickness burns is universal. It has a hard, dry leathery quality (Figures 15.5 through 15.7). It is frequently insensate to the touch and painless unless mixed with some partial-thickness elements. The color is usually pearly gray to yellowish or a charred black. The skin is completely denatured and contracted. In extensive circumferential burns, this contracture can lead to respiratory compromise – when the thorax is involved – and peripheral ischemia when the burn involves the limbs.

Some sequelae of burn trauma to the epidermis result from the destruction of its waterproofing and barrier function. Not only does the body become susceptible to the invasion of bacteria but it also leaks fluid, losing its ability to maintain normal water balance. Dead eschar can provide a breeding ground for bacteria and a focus for infection. Furthermore, the burned tissues cannot perform their normal vasoregulatory function, and

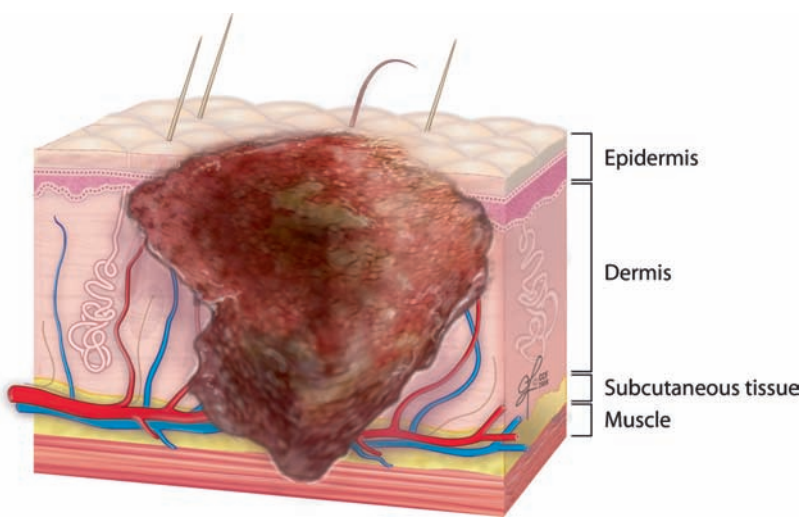


Figure 15.6. A full-thickness burn penetrating through the epidermis and dermis into the fat and destroying all epithelial remnants on the way. Healing can take place only from the periphery of the wound. This is a slow process and results in scar contracture. (With kind permission of The Cleveland Clinic Center for Medical Art and Photography © 2008.)

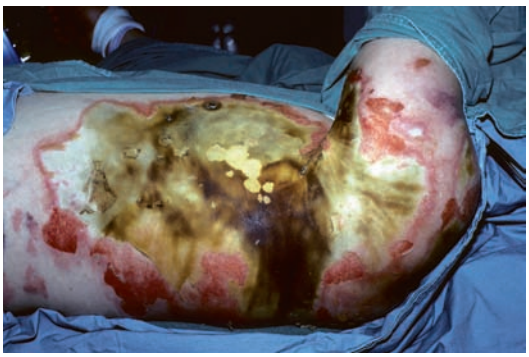


Figure 15.7. A full-thickness burn involving the axilla and left chest. The burn is quite discrete and may be excised and grafted in one stage. However, the patient will likely develop a severe adduction contracture of the axilla unless appropriate splinting is employed postoperatively.

the individual becomes unable to maintain body temperature. Secondary healing, by contraction and scarring, results in disfigurement, distortion, and functional loss.

The evolution of the thermal burn injury is divided into four phases:

The *emergent phase* (0–12 h) is characterized by catecholamine release in response to pain and the cardiovascular sequelae associated with fight and flight. The pulse rate and the blood pressure increase accompanied by peripheral vasoconstriction. The patient is anxious and apprehensive. This overlaps with the second phase: the *fluid shift phase* (6–48 h), which is associated with increased capillary permeability leading to an outpouring of fluid from the intravascular into the extravascular space. This "third space" loss is in addition to a considerable loss of fluid from the burn wound itself. The third phase – or *hypermetabolic phase* (day 1–day 30+) – marks the stabilization of the burn wound and an increased consumption of energy[6,12,15] with an elevated demand for nutrients (not predictably influenced by closure of the burn wound itself). This is required for the processes of regeneration and repair. In severe burns this period can extend several months.[14] Finally, *the resolution phase* (1–6 months+) heralds the formation of scar and the remodeling of tissue.

Systemic complications in the acute phase can include fluid and electrolyte loss leading to hypovolemia and shock. This progresses to hypothermia, infection, and acidosis. The increased catecholamine release results in vasoconstriction, which in severe cases can produce renal or hepatic failure. The need for adequate and accurate fluid replacement is evident.

Jackson's thermal wound theory (Figure 15.5) allows conceptualization of the dynamic nature of the burned wound.[2] A burn can be imagined as consisting of three concentric rings where the inner circle represents an area of full-thickness loss. This is the *zone of coagulation*. The outer circle encloses the *zone of hyperemia*. This is the peripheral area of the burn, characterized by limited inflammation and increased blood flow. The damage is reversible. Between the outer zone of hyperemia and the central zone of necrosis lies the intermediate *zone of stasis*. Here inflammation is associated with reduced blood flow. The intermediate zone may survive with appropriate treatment but may be lost with inadequate resuscitation or superimposed infection.

Susceptibility of the patient to infection is related to the depth and extent of the burn; preexisting medical conditions; extremes of age; impaired blood supply (e.g., shock, thick eschar); low wound pH; and a hot moist environment.[11] Although the mortality from burns has decreased over the past 25 years, the leading cause of death continues to be sepsis. However, due to the increased use of topical microbial agents, the sepsis is now more commonly derived from bronchopneumonia than from burn wound infection. Silver sulfadiazine and other topical products have also had the beneficial effect of reducing the conversion of partial-thickness to full-thickness burns; although by decreasing the bacterial count, they have prolonged the spontaneous separation of the burn wound slough.

Burn wound sepsis is defined by the patient's having a bacterial count of 10^5 organisms or more per gram of tissue. Prophylactic systemic antibiotics are no longer recommended due to the risks of selecting antibiotic-resistant organisms, particularly MRSA, fungi, and yeasts; but sepsis must be treated with debridement of necrotic tissue and the appropriate antibiotic.

Of particular concern in a burn patient is bacterial colonization with group A beta hemolytic streptococcus. This organism secretes a large number of proteases including streptokinase and hyaluronidase, which prevent adhesion of skin grafts – a serious problem in wound closure. The organism is generally susceptible to penicillin and should be treated "on sight," but the infrequency of

this infection combined with the problems mentioned above precludes its prophylactic use.

Assessment of the Patient with Burn Trauma

The American Burn Association has promulgated guidelines for the admission and transfer of burned patients to specialized burn units:

1. Partial-thickness burns of greater than 10% of the total body surface area.
2. Burns that involve the face, hands, feet, genitalia, perineum, or major joints.
3. Third-degree burns in any age group.
4. Electrical burns, including lightning injury.
5. Chemical burns.
6. Inhalation injury.
7. Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality.
8. Any patients with burns and concomitant trauma (such as fractures) in which the burn injury poses the greatest risk of morbidity or mortality. In such cases, if the trauma poses the greater immediate risk, the patient's condition may be stabilized initially in a trauma center before transfer to a burn center. Physician judgment will be necessary in such situations and should be in concert with the regional medical control plan and triage protocols.
9. Burned children in hospitals without qualified personnel or equipment for the care of children.
10. Burn injury in patients who will require special social, emotional, or rehabilitative intervention.

Excerpted from Guidelines for the Operation of Burn Centers (pp. 79–86), Resources for Optimal Care of the Injured Patient 2006, Committee on Trauma, American College of Surgeons.

Smaller burns may be handled in a regular hospital setting. When assessing the burned patient, a detailed history and examination are of vital importance. Patients younger than 2 years or older than 55 years develop more complications and have a poorer outlook. Preexisting medical conditions can adversely affect the individual's tolerance of the stress of injury as well as the hemodynamic trauma of resuscitation. It must not be forgotten that burn victims frequently suffer associated injuries resulting from falls, motor vehicle accidents, explosions, inhalation of toxic gasses, and tetanic contractions – conditions frequently associated with burns but often missed at initial evaluation. A high index of suspicion is indicated so that such injuries may be identified, appropriate treatment begun, and iatrogenic injury avoided.

The burn agent can significantly affect the extent of the burn and its management. Most of this discussion concerns flame burns, but electrical and chemical burns create specific problems, requiring special precautions and individual solutions. It is therefore important to determine the burn agent at the outset.

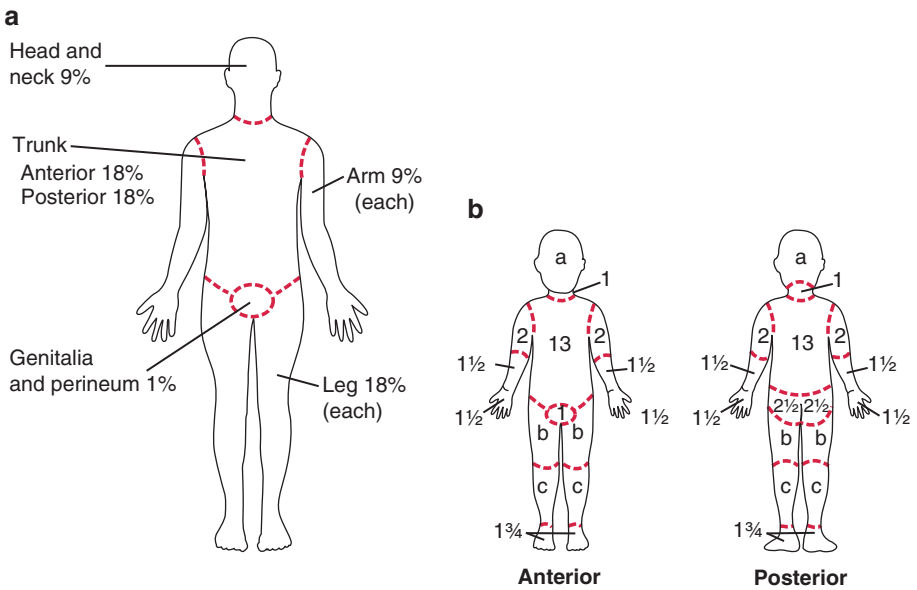
Burns in confined spaces or loss of consciousness in a burned area can lead to a life-threatening inhalation injury, the sequelae of which (asphyxiation, pulmonary edema, and death) may become evident only after the patient has been admitted and is receiving fluid resuscitation. Therefore, patients with respiratory burns require early endotracheal intubation and active airway management. This possibility of a respiratory burn must be actively ruled out by taking a careful history of the circumstances surrounding the burn as well as by examining the patient's head, neck, and chest for burns; the nares for singed or burned hairs; the patient's nasal and oral mucosa for redness and dryness; the throat for carbonaceous sputum; the voice for hoarseness, the mouth for drooling; and, if indicated, the pharynx, larynx, and trachea (via a flexible bronchoscope) for evidence of supraglottic or subglottic injury. What are the indications for performing a flexible bronchoscopy? In addition to the positive findings mentioned above, they include a respiratory wheeze, tachypnea, or pulmonary crepitations. The respiratory rate, on the other hand, is often unreliable due to the depressant effects of toxic combustion products. Positive bronchoscopic findings include redness and dryness of the mucosa and the presence of carbon particles on the respiratory mucosa. When a diagnosis of respiratory burn is made, the patient should be intubated. (Tracheotomy is avoided due to the risk of infection.) Respiratory burns are discussed in more detail later.

Circumferential burns can restrict ventilation when the chest is involved. Full-thickness

burns across a joint area can limit motion, and circumferential burns of limbs, hands, and fingers can lead to compartment syndrome and/or circulatory compromise. These conditions, like the sequelae of respiratory burns, are greatly exacerbated during the fluid resuscitation phase when tissue edema can magnify the constrictive forces produced by the burn. It is therefore important to recognize circumferential full-thickness burns at the time of the initial examination and take immediate measures to relieve their effect. An escharotomy may be performed in the emergency room or in the burn admission room immediately after the initial assessment. It consists of making a series of axial cuts completely through the burn and allowing the fatty tissue to bulge through. Each incision must be taken from uninvolved tissue on one side of the burn to uninvolved tissue on the other. Since a full-thickness burn is insensate, this is not as barbaric as it sounds. The physician should be armed with a scalpel, numerous artery forceps, and suture-ties, since large subcutaneous veins are frequently encountered and must be ligated.

The magnitude of the burn wound is assessed by calculating the surface area of involvement according to depth, on a percentage basis. For the purposes of calculating fluid requirements, partial-thickness and full-thickness burns are counted together and superficial burns are generally ignored.

The percentage surface area is calculated according to the "rule of nines" (Figure 15.8). The head and both arms each individually constitute 9%, while the anterior trunk, the posterior trunk, and the lower limbs count for 18% each. The perineum is 1%. For the purposes of burn area estimation, the "palm rule" is also



Relative percentage of body surface area (% BSA) affected by growth

	Age				
Body Part	**0 yr**	**1 yr**	**5 yr**	**10 yr**	**15 yr**
a = 1/2 of head	9 1/2	8 1/2	6 1/2	5 1/2	4 1/2
b = 1/2 of 1 thigh	2 3/4	3 1/4	4	4 1/4	4 1/2
c = 1/2 of 1 lower leg	2 1/2	2 1/2	2 3/4	3	3 1/4

Figure 15.8. Charts for the estimation of the burned wound as a percentage of body surface area (bsa) in adults (**a**) and children (**b**) In children, the head is relatively larger, and so the rule of nines is modified accordingly.

valid. The patient's palm represents one percent of his or her body surface area. By calculating the fraction of each area involved, an approximation can be made of the percent of body surface area (bsa) burned.

In the estimation of the surface area of pediatric burns, the rule of nines is inaccurate due to the larger relative head size in infants. The infant's head constitutes 18% of his or her total body surface area, and each leg only 13.5%. For each year over the age of 1 year, the treating physician must subtract 1% from the head and add 0.5% equally to each leg.

Initial Management of the Burned Patient

After the patient has been examined for associated trauma and had his burn wounds assessed, consent is obtained for possible blood transfusion and surgery. Two large peripheral intravenous (IV) lines are inserted through which the patient may then be given analgesia – such as 2 mg morphine – and, if necessary, fluid replacement. Central lines are avoided in burns due to the risk of sepsis. Patients requiring fluid replacement should have an indwelling urinary catheter to monitor urine output. In the initial 24–48 h, the hourly urine output is the best guide to the adequacy of resuscitation. Escharotomies are performed as described here and the burns dressed with 1% silver sulfadiazine cream together with multiple layers of absorbent gauze. The profuse exudate will require a dressing change at least daily. If a respiratory burn is present, the patient is intubated. Positive end expiratory pressure (PEEP) is then employed to maintain the airway. Blood is tested for H & H, and a CHEM 7 assay is performed. With burns in a confined space, and in respiratory burns in general, the blood carboxyhemaglobin (COHb) level is assessed and appropriate actions taken according to the level (see below). Prior to fluid replacement, the circulatory status is assessed.

Fluid Therapy

Although fluid replacement is an integral part of burn management, rapid-onset hypovolemic shock is quite rare: when this is present, there is often another cause. Fractures and injuries to internal structures need to be positively ruled out before proceeding with routine fluid replacement. Burns of limited surface area may not need IV resuscitation at all, since the fluid loss is limited and the patient can easily make up the deficit by mouth. As a general guideline, burns needing IV resuscitation include the following:

1. Partial-thickness burns exceeding 15% body surface area
2. Full-thickness burns exceeding 10% body surface area

It has become accepted that the appropriate fluid for burn resuscitation in the first 24 h is crystalloid. The amount of fluid given in the first 24 h is calculated by the Parkland[3] burn formula. Crystalloid, consisting of Ringer lactate, is preferred over colloid because of generalized increased capillary permeability in response to trauma. It is not desirable for large amounts of colloid to leak out of the capillaries, particularly into the lung, which is highly susceptible to pulmonary edema in the early phases of burn resuscitation. The Parkland formula[3] specifies 4 ml of Ringer lactate per kilogram body weight (bw) per percentage body surface area (bsa) burn or:

$$4\,\text{ml/kg bw/\%bsa burn}$$

Fifty percent of the total is given in the first 8 h and the rest in the next 16 h. To this must be added the normal daily requirement of about 3 l/day in the average adult. When the burn victim presents to the burn unit some hours after the injury, it is necessary to give the patient the fluid he should have had during the delay. For example, if a 70 kg man received a 40% burn 4 h ago, his total 24 h allotment according to the Parkland formula is:

$$4 \times 40(\%) \times 70(\text{kg}) = 11.21$$

Fifty percent of this, or 5.6 l, must be given in the first 8 h. However, in this example, the patient had no fluid in the first 4 h following his burn. Therefore, he must receive the whole 5.6 l in the next 4 h, and the other 5.6 l in the next 16 h. It should be emphasized that the Parkland formula is just a guide, and the amount of fluid given must be titrated according to clinical factors such as urine output and hematocrit. A Foley catheter should be in place

and vital signs monitored closely. The lungs should be ascultated for signs of pulmonary edema. The objectives are to maintain a heart rate of less than 110; a normal sensorium (awake, alert, and oriented), and a urine output of 30 ml to 50 cc in adults and 1–2 ml/kg/% burn in children. Prophylactic anti-biotics are not generally given due to the risks of inducing the formation of resistant organisms. (However, proven infections are treated according to in vitro sensitivities.)

After 24 h, fluid loss stabilizes somewhat and the capillary leak diminishes. At this time the patient may be given albumen (5% albumen at 0.5 ml/kg/bw/%bsa). The patient may also require blood, particularly if the full-thickness component exceeds 10% body surface area. The whole blood requirement is roughly 1% of the patient's normal blood volume for each 1% of deep burn.

Once the patient is stable, hyperalimentation should be commenced to make up for the massive loss of energy and protein that characterizes the catabolic phase of a major burn. The diet should be high in calories as well as protein and include necessary vitamins and minerals. If the patient is not able to take adequate supplementation by mouth, it may be given via a nasogastric tube (seeBurn Nutrition).

Inhalation Injury

As mentioned here, respiratory burns occur when an individual is burned or exposed to products of combustion in closed space. A cough is usually present, often with carbonaceous sputum. The diagnosis is made from a detailed history and examination, which may include flexible bronchoscopy.

Inhalation injuries may be supraglottic or subglottic. Supraglottic injury is more common. The mucosa is very susceptible to high temperatures, and injury may result in immediate edema of the pharynx and larynx. However, the symptoms may also present late with hoarseness, stridor, and a brassy cough. Respiratory obstruction and asphyxiation may result.

Subglottic injury is less common. It involves injury to the lung parenchyma and is usually due to superheated steam, aspiration of scalding liquid, or inhalation of toxic smoke or chemicals. Presentation here is frequently delayed. On auscultation, wheezing or crackles are heard. The patient has bronchospasm, a productive cough, and eventually pulmonary edema.

Management of Inhalation Injury

High-flow oxygen should be administered immediately via a face mask. The airway should be assessed early using fiberoptic endoscopy in patients who are at risk, and early consideration given to intubation. When airway burns are seen, the patient should be given 100% oxygen and ventilated using PEEP. A short course of high-dose steroids may be considered. In severe cases, it is sometimes difficult to separate the symptoms of "burned lung" from fluid overload. A Swan–Ganz catheter may help distinguish between the two (although, in burns, central lines are generally avoided because of the risk of sepsis).

Carbon Monoxide Poisoning

Apart from the effect of thermal damage to the airways and lung parenchyma, inhalation poisoning is a major cause of morbidity and death. Any patient in a fire has the potential of hypoxia and carbon monoxide (CO) poisoning. CO is a product of combustion of organic matter under conditions of restricted oxygen supply, which prevents complete oxidation to carbon dioxide (CO_2). CO poisoning is not always associated with respiratory burns, however, and so a high index of suspicion should be maintained in any burned patient. Initial signs include headache, nausea, apathy, and confusion.

CO binds to hemoglobin (reducing oxygen transportation), myoglobin (decreasing its oxygen storage capacity), and mitochondrial cytochrome oxidase (inhibiting cellular respiration). Hemoglobin's affinity to carbon monoxide is 250 times greater than to oxygen; so small concentrations of CO can severely reduce the oxygen-carrying capacity of the blood. With CO poisoning, the skin and mucus membranes become bright pink, but only at levels likely to be fatal (40%+). The immediate treatment is to administer 100% oxygen via a close-fitting mask and take a blood sample for COHb. Hyperbaric oxygen should be considered with a COHb level of 25% or greater. However, the efficacy of this is still unproven. The half-life of COHb in room air is 320 min, but at three atmospheres it is only 23 min.

Cyanide or Thiocyanate Poisoning

Another type of inhalation toxicity involves cyanide, or thiocyanate poisoning. This is much less common than CO poisoning but shares some common features. It can occur following the combustion of wool, silk, nylon, nitriles, rubber, and paper. The symptoms are initially flu-like, and the patient's skin becomes bright pink (cherry red) reflecting diminished tissue oxygen extraction. The patient may smell of "bitter almonds." Symptoms soon progress to weakness, headache, nausea, dyspnea, trembling, convulsions, coma, severe hypoxemia, and cardiovascular collapse. Blood gasses show a normal arterial pO_2 and an abnormally high venous pO_2 (decreased A-V O_2). There is a high anion gap, a metabolic acidosis, and a raised lactate level. An RBC cyanide level is diagnostic but takes too much time for the result to influence vital treatment. An antidote "kit" is available consisting of amyl nitrate, sodium nitrite, and sodium thiosulfate.

Amyl nitrite perles should be broken onto a gauze pad and held under the nose, over the Ambu-valve intake, or placed under the lip of the face mask. The patient should inhale for 30 s every minute and a new perle used every 3 min if sodium nitrite infusions will be delayed.

If the patient has not responded to oxygen and amyl nitrite treatment, sodium nitrite should be infused intravenously as soon as possible. The usual adult dose is 10 ml of a 3% solution (300 mg) given slowly over no less than 5 min; the average pediatric dose is 0.12–0.33 ml/kg body weight up to 10 ml infused as above. The blood pressure is monitored during sodium nitrite administration, and the rate of infusion slowed if hypotension develops.

Next, sodium thiosulfate is administered intravenously. The usual adult dose is 50 ml of a 25% solution (12.5 g) infused over 10–20 min; the average pediatric dose is 1.65 ml/kg of a 25% solution. One-half of the initial dose should be repeated 30 min later if there is an inadequate clinical response.

Amyl nitrite and sodium nitrite oxidize the ferrous iron of hemoglobin to methemoglobin. (Methemoglobin levels should not exceed 20%.) This creates an additional site for cyanide binding and promotes dissociation from cytochrome oxidase. Resultant cyanomethemoglobin may then be converted to less toxic thiocyanate through enzymes such as rhodanese or other sulfurtransferases in the presence of sodium thiosulfate. Treatment with nitrite and thiosulfate should be repeated as required.

The efficacy of hyperbaric oxygen in cyanide poisoning is unproven. It has been reported to be useful in severe cases of smoke inhalation combined with exposure to hydrogen cyanide and carbon monoxide.

Burn Wound Management

Superficial partial-thickness burns should have their blisters debrided since the contained fluid contains prostaglandins, thromboxane, and prostacycline that can stimulate the release of free oxygen radicals and cause further tissue damage.[5] In these burns, the underlying tissue is reddish pink and moist, characterized by the absence of slough. Such burns may be dressed with large sheets of (nonadherent) Vaseline-impregnated gauze such as Xeroform™ and covered with several bulky layers of burn gauze (sterile dry gauze in large sheets) to absorb the exudate. Dressings should be changed daily or more often if the exudate soaks through.

Deeper burns, which include partial-thickness burns with a yellowish slough as well as full-thickness burns, should be treated with copious amounts of 1% silver sulfadiazine cream and then covered with burn gauze as before. As well as being a powerful antiseptic, the silver sulfadiazine cream is something of a desloughing agent: it softens and loosens eschar, facilitating its ultimate removal. An alternative to silver sulfadiazine is sulfamylon, an older preparation with the distinct disadvantage of stinging on application. However, it is somewhat more effective against pseudomonas and is occasionally used when this pathogen proves problematic.

Extensive flame burns frequently have a mixed pattern of deep partial and full thickness. Often it is difficult to distinguish one from the other. The traditional treatment of such burns is to remove the dressings daily and place the patient in a "burn tub," preferably with a whirlpool feature. With the patient in the bath, the burn nurses remove all the silver sulfadiazine and gently debride any loose eschar. Three or 4 weeks of daily

dressings, bathing, and debridement usually result in healed partial-thickness burns, complete de-sloughing of full-thickness burns, and clean granulations ready for skin grafting. However, when this method is used, there is a significant morbidity and mortality related to sepsis; hospitalization is prolonged, and some partial-thickness burns may convert to full thickness. (The effect of modern antimicrobials is to actually increase the time taken for de-sloughing to occur.)

The modern trend is for early burn wound excision with immediate[7] or delayed skin grafting. Of course, it is vital that the patient is completely resuscitated and stabilized before any surgery is carried out. It is also important that the surgeon realize that by excising the burn and harvesting skin grafts to cover it, there is a danger of creating an exudating wound far larger than the original injury – producing fluid shifts and the need for further IV resuscitation. In extensive burns it is, therefore, necessary to stage excision and grafting sessions according, not only to the surface area of the burn, but also the patient's ability to withstand the surgery and the availability of suitable donor sites. Generally, such surgery is commenced 2–3 days after admission and typically a decision is taken to either excise the burn and apply skin substitutes or simply excise and graft one anatomical area at a time. The main role of skin substitutes is to temporarily stabilize wounds and "buy time" until definitive skin grafting can safely be performed.

Where the burn is obviously full thickness, it may be excised with a scalpel, and hemostasis achieved with the cautery. Where the burn is mixed or deep partial thickness, *tangential excision*[7] is indicated. Tangential excision is performed using a 12-in dermatome (such as a Humby knife) with the guard wide open (Figure 15.9). Successive thin layers of the burn slough are "shaved off" until healthy bleeding dermis is visible. This tissue may then be dressed or grafted. The concept of tangential excision is to preserve as much viable tissue as possible. Of course, in full-thickness areas, tangential excision will extend completely through the dermis until viable tissue is found.

If the patient has enough unburned skin to provide adequate donor sites, then split-thickness skin autografts are used – since they constitute definitive wound closure – provided the patient's condition is stable enough to permit their harvest. When skin graft donor sites are limited, a decision may be taken to use whatever is available to close part of the wound and skin substitutes, xenografts, or allografts to provide temporary cover for the rest. Later, as the patient's own donor sites regenerate (8–12 days), they can be "recropped" and the skin used to replace these products.

Split-thickness grafts are harvested at about 12/1000 of an inch. This produces a useful graft and a donor site that heals in about 10 days to 2 weeks. To extend the area that can be covered with such a graft, it may be meshed in a ratio of 1.5 or 3–1. The graft is spread out on a specially grooved plastic plate, which is then passed through the rollers of the meshing machine. The rollers are also grooved and a series of slits are cut in the skin allowing it to be opened up like a string undershirt. The openings allow for the drainage of blood and serum but ultimately epithelialize from the adjacent meshed skin.

Full-thickness skin grafts have limited application in burns. Although they are more durable than partial-thickness grafts and do not contract as much, "take" is a little more uncertain, donor sites are limited, and they must be closed by local tissue or by split-thickness skin grafts. Full-thickness skin grafts do have a place, however, in small localized full-thickness burns, particularly of the hands and face. In these circumstances, the burn may be excised early and replaced by such a graft. Common donor sites include the opposite upper eyelid (for eyelid reconstruction),

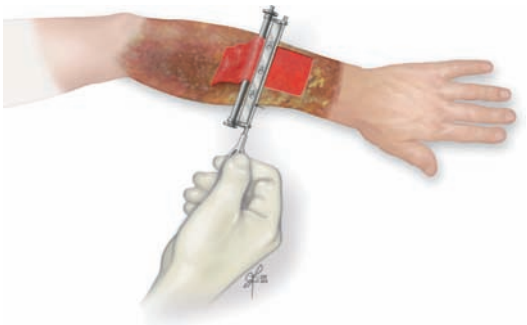


Figure 15.9. Tangential excision: a Humby knife with its roller guard wide open is used to repeatedly shave down burned tissue until the healthy bleeding bed is obtained. (With kind permission of The Cleveland Clinic Center for Medical Art and Photography © 2008.)

the groin crease (hand), the post auricular groove, and the supraclavicular fossa (face).

Skin Graft Substitutes

Biologic skin substitutes may be intended for permanent replacement or as a temporary biologic dressing until a permanent solution is available or normal skin regeneration and healing occur. They serve multiple functions: they decrease the bacterial count and promote a sterile wound; they slow the loss of water, protein, and electrolytes; they reduce pain and fever, help restore function, facilitate early motion, and provide coverage of vessels, tendons, and nerves to prevent desiccation. The ideal skin substitute is nontoxic, has little or no antigenicity, is immunologically compatible, and does not transmit disease.

Cultured Skin

Certain laboratories provide a service whereby a biopsy is taken of the patient's own epithelial tissue and is then subjected to tissue culture. Sheets of epithelial cells are produced that may be used to resurface burn wounds. Unfortunately, the grafts – lacking a dermal layer – are somewhat unstable, making them susceptible to even minor trauma. Attempts are currently being made to provide a collagen layer to be used with cultured cells in the hope of increasing the durability of the graft. One such method uses cultured skin on a pig skin mesh. It may also be placed on Alloderm once the latter has become incorporated into the wound (see below) or Integra once the silicone layer has been removed 2–3 weeks after initial application.

Allografts and Xenografts

Split-thickness cadaver skin grafts are probably the best alternative to the patient's own skin when insufficient of the latter is available. The use of cadaver allograft for temporary closure was fairly common in the past but has become less frequent due to the risks of possible HIV transmission. The allografts behave like normal skin autografts but undergo rejection several days later. Pigskin is a commercial alternative, but this too undergoes rejection. For this reason, both kinds of grafts need to be changed every 3–4 days. Alloderm is an acellular human dermis: a processed allograft. It comes in sheets of predetermined size, is freeze-dried, and has to be reconstituted in saline before use. Some of its collagen becomes incorporated in the wound during healing and may result in more durable cover once it is skin grafted.

Skin Substitutes

INTEGRA Bilayer Matrix Wound Dressing™ is an advanced wound care device comprising a porous matrix of cross-linked bovine tendon collagen and glycosaminoglycan and a semipermeable polysiloxane (silicone) layer. The semipermeable silicone membrane controls water vapor loss, provides a flexible adherent covering for the wound surface, and adds increased tear strength to the device. The collagen–glycosaminoglycan biodegradable matrix provides a scaffold for cellular invasion and capillary growth. At approximately 3 weeks, the silicone layer may be peeled off and replaced with cultured epithelial cells or thin split-thickness skin grafts.

BIOBRANE is a biosynthetic wound dressing constructed of a silicone film with a nylon fabric partially embedded into the film. The fabric presents to the wound bed a complex 3-D structure of trifilament thread to which collagen has been chemically bound. Blood/sera clot in the nylon matrix, thereby firmly adhering the dressing to the wound until epithelialization occurs.

Burn Nutrition

Large burns impose a massive metabolic requirement on the patient. There is severe catabolism in the initial stages, which is accompanied by a profound disuse atrophy of the muscles due to inactivity.

The burned patient should be nursed in a warm room with occlusive dressings to minimize the energy loss due to evaporation. Immediate rehabilitation with physiotherapy must be accompanied by a diet high in calories, protein, and essential vitamins and minerals. Both should begin after resuscitation – preferably within 48 h of the burn incident. Generally, the calorie intake should be 35–40 cal/kg/day (or

about 1.5 times the basal energy expenditure). If the patient cannot (or will not) ingest the necessary calories by mouth, then nasogastric intubation and postpyloric feeding are indicated. The protein requirement in a severe burn is 2–3 times the recommended daily requirement (RDA). The RDA is 0.8 g/kg/day, so the patient will require 1.5–2 g/kg/day. More specifically, glutamine, the most significant amino acid lost from muscle, should be given as a separate oral supplement at the rate of 10–30 g/day. Carbohydrates should provide no more than 60% of total calories. More than this is undesirable due to the risks of hyperglycemia and fat formation. Fat should provide 20–25% of the total calories. Since endogenous fat will also be used, more than this should be avoided since fat can become a substrate for immunosuppressive mediators. Vitamins and minerals are usually given in doses 5–10 times the RDA to keep up with the increased metabolism and quickly restore deficiencies.

Progress is monitored by daily weighing, recording of calorie and protein intakes, indirect calorimetry weekly, as well as nitrogen balance measures, especially if there is evidence of excessive weight loss or symptoms of excess lean mass loss.

Blood chemistry is vital in monitoring the effects of catabolism as well as the treatment. Major electrolyte shifts occur in burns. Typically intracellular electrolytes, magnesium, and phosphate levels recede to extremely low levels with the onset of nutrition as the cell mass increases. Key electrolytes must be given to prevent complications. Hypernatremia can indicate inadequate hydration. Blood urea nitrogen typically increases with protein breakdown, but the level must be assessed relative to creatinine (renal function). An elevated BUN is also a marker of inadequate hydration. Blood glucose is frequently elevated in burns, so glucose intake and insulin must be carefully adjusted. A rapidly rising level of alkaline phosphatase may suggest increasing fat deposition possibly due to overfeeding. Serum triglycerides measure adequacy of fat clearance and should not exceed 250 mg/dl. If elevated, fat intake should be decreased. Prealbumin and transferrin are markers of protein synthesis and degradation. A continually decreasing value usually reflects inadequate protein intake.

Serum electrolytes, blood urea nitrogen, creatinine, glucose, magnesium, and phosphate as well as a lipid profile, prealbumin, and transferrin should be monitored at least weekly in burned patients.

Chemical Burns

Chemical burns are generally the result of industrial accidents. Burns may be caused by acids, alkalis, or sticky substances such as tar.

Acid burns generally produce a coagulation-type necrosis creating an eschar: this is usually a self-limiting injury once the active ions are used up in the coagulation process. Bases, on the other hand, produce liquefactive necrosis, which releases more ions to penetrate deeper into the wound and produce a more extensive injury.

Burns from relatively inert chemicals such as tar occur because the substance acts as a heat reservoir and adheres to the skin. A contact burn is produced before the substance can be removed. A tar burn should be considered as a prolonged "scalding" injury.

Dry Chemicals often produce burns when they come in contact with the skin and are activated by sweat or water used to wash them off. The addition of water produces an exothermic reaction that produces the burn.

The main principle of emergency chemical burn management is to remove the chemical from the skin as soon as possible. The patient's clothing and footwear should be immediately taken off and all dry chemicals brushed away. Notwithstanding the risks of an exothermic reaction, the burned areas should be flushed for 20–30 min with copious amounts of water. This may involve placing the patient in a shower for this period of time. Neutralization by adding acid to alkali or visa versa should not be attempted because of the risk of severe heat production and further burns.

When burns involve the eye, the conjunctival sac should be copiously irrigated medial to lateral with normal saline via a plastic tube derived from an IV administration set or nasal cannulae. Contact lenses should be removed. The irrigation should last for 15–20 min at least.

Specific chemical agents are treated according to these principles:

Dry lime should be brushed off, since it is water activated, and then the skin flushed with copious amounts of saline.

Phenol is not water soluble. If available, alcohol should be used to remove the chemical before copious flushing with water. However, alcohol should not be used in the eyes.

Sodium and potassium metals react violently on contact with water. After brushing off any residue, flushing is required with copious amounts of water.

Tar burns require the use of ice packs and ice water to reduce the heat sink effect of the adherent tar. Once this has been achieved, the tar can be removed using mineral oil – a nontoxic solvent.

Sulfuric acid generates heat on exposure to water. The affected area should be washed with soap to neutralize it and then irrigated with large quantities of water.

Hydrofluoric acid burns are extremely serious and often fatal. Fluoride ions penetrate and form insoluble salts with Ca^{2+} and Mg^{2+}. However, soluble salts formed with other cations dissociate rapidly releasing F^{+} ions. These F^{+} ions then continue to penetrate, causing further and deeper destruction. Hydrofluoric acid burns are highly destructive and toxic, burning right through flesh and into bone without stopping. The pain is severe and systemic effects are hypocalcemia, hyperkalemia, hypomagnesemia, and sudden death – even with burns as small as three percent body surface area. Treatment involves the application of 2.5% calcium gluconate gel to the burned area and the IV infusion of 0.5 ml calcium gluconate solution per square centimeter of surface burn.

Electrical Burns

Electrical burns arise from contact with an electrical conductor. This may consist of domestic wiring, electrically powered devices, power transmission lines, transformers, or lightening. Damage depends on the intensity of the current (I). However, although current produces tissue damage, voltage (V) determines if the current enters the body. Ohm's law states that I = V/R where R represents the resistance of the conducting medium. The current generally follows the shortest path to the ground, and it is this path – the course of the current through the body – that governs the extent and location of the injury.

Low voltage usually cannot enter the body unless the skin is broken or moist. (Once in, however, it can follow blood vessels and nerves due to their lower resistance.) A common low-voltage injury occurs when a child bites or chews on a domestic electrical wire. Classically, a full-thickness burn is produced at the corner of the mouth (Figure 15.10). This takes a long time to heal and frequently results in microstomia due to scarring and contracture. The best treatment for this is a specially made splint that attaches to the teeth and holds the corner of the mouth in a lateral position. Healing is still prolonged, but microstomia does not develop and the aesthetic result is excellent.[13]

High-voltage injuries are often devastating. The magnitude of the voltage easily overcomes skin resistance and the current enters the body. The current then passes through the tissues, which act as a "volume conductor." In other words, the current passes through all tissues. However, since I = V/R, the current is maximal where there is least resistance. Typically the nerves and blood vessels sustain the most damage; however, with the passage of massive currents, no tissues are spared. Myonecrosis is a prominent feature of high-voltage electrical burns due to the large volume of muscle in the limbs. Factors affecting the severity of the electrical burn include the width or extent of the current pathway, the tissues through which it passes, the duration of the contact, and whether the current is alternating or direct.

Alternating current (AC), in addition to direct tissue damage, can produce tetanic contraction of muscles, resulting in muscle injury, tendon rupture, joint dislocation, and fractures. It can also produce cardiac arrhythmias, apnea, seizures, and spasms, which may keep the patient from getting free of the current source.

Tissue damage, as mentioned earlier, is due to heat as current flows through tissues. The exit and entry burns can be trivial looking, but everything in between can be "cooked." It should be noted, however, that higher voltage results in less trivial external burns.

There are various types of electrical burns:

Contact burns occur when the individual touches a conductor and an electrical current passes through his or her body, producing heat and tissue damage. There is often an entry

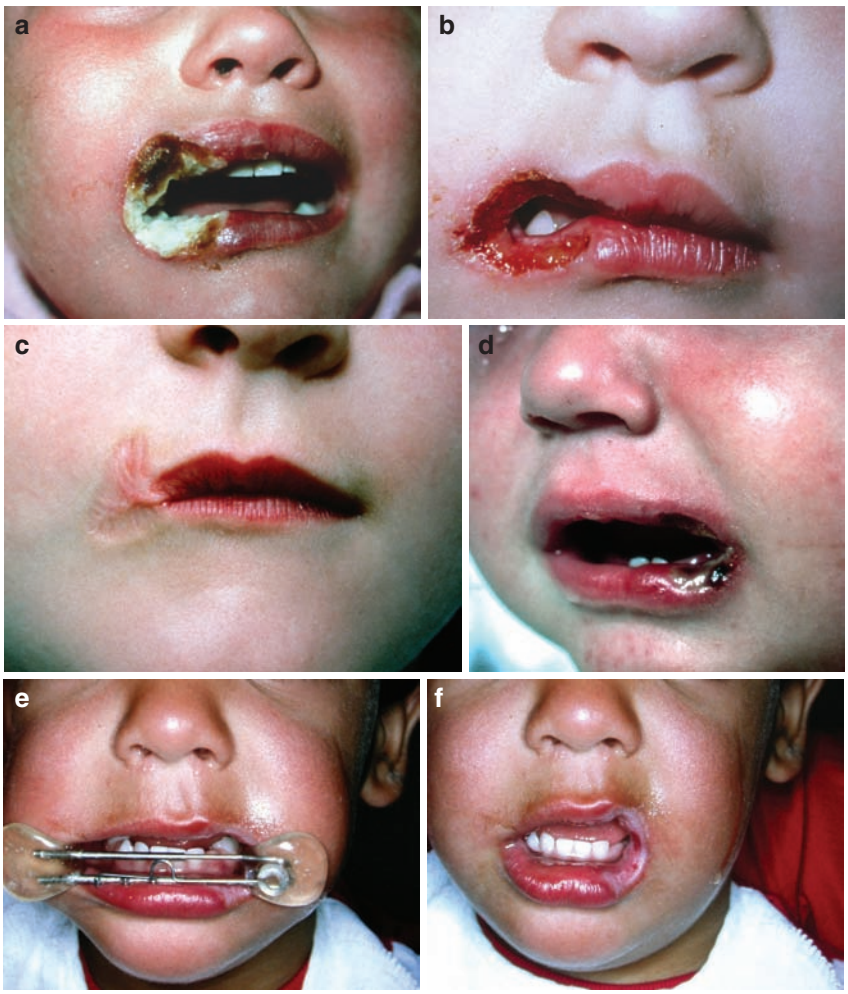


Figure 15.10. Electrical burns caused by children chewing electrical cords. These commonly cause tissue destruction at the corners of the mouth. (**a**) Acute burn. (**b**) Result of excision. (**c**) Repair. (**d**) A second patient with an acute burn. (**e**) Fitted with a splint and (**f**) Final result 1 year later. (Reproduced by permission of Dr. R. M. Zuker, Hospital for Sick Children, Toronto.)

wound and a much larger exit wound. The injury is greater than that expected by calculating the body surface area of the burn, since much of the damage is internal and involves other tissues in addition to the skin.

Flash burns occur when the current arcs and a large amount of radiant heat is generated. The current may or may not enter the patient, but the patient is burned nevertheless. Such burns are often superficial or partial thickness due to the short duration of the flash.

Flame burns result when clothing ignites. Frequently, high-voltage electrical injuries from, say, a transformer have a mixed appearance, combining contact with flash and flame burns.

Lightening usually produces a high-voltage pattern of burning. However, here too, injury may result from a direct strike, a side flash, or a flashover. Severe injuries, often of a mixed pattern, are common.

The management of electrical burns differs from that for regular burns in a number of key respects:

Extensive internal damage is common. Electrical burns are usually worse than they appear. As a rule affected individuals should all be admitted

to hospital. In the initial assessment, the fluid requirement calculations should allow for the fact that there is an internal burn in addition to the external one, so the "rule of nines" will underestimate the fluid requirement. The electrical burn may involve many tissues or organs. A full general examination with EKG and chest x-ray should be used to rule in or rule out cardiac or respiratory injury as well as determining the extent of any damage to the peripheral or central nervous systems. In addition, the following laboratory testing should be performed to help assess the extent of the injury: CBC, serum electrolytes, creatinine, urinalysis, creatinine kinase (CK) level, urine myoglobin, and serum myoglobin.

Associated injuries are more frequent. A detailed history must be obtained from the patient and witnesses. A detailed examination must be performed to rule out musculoskeletal injury. Fractures must be appropriately immobilized to prevent further damage. Cardiac, respiratory, and neurological problems must be addressed. The patient should be carefully assessed for musculoskeletal injury, particularly fracture or dislocation of the cervical spine, since electrical burns are frequently associated with falls from ladders or electrical structures. Moreover, the tetanic effect of AC may produce musculoskeletal injury in its own right. It must also be remembered that individuals suffering electrical burns in confined spaces may also be the victims of respiratory burns or inhalation injury (see above).

Rhabdomyolysis considerations are vital. Fluid resuscitation must be monitored carefully and dehydration avoided at all costs. The patient's fluid requirement will be far greater than anticipated. Ringer lactate is given according to surface calculations and titrated upward to maintain urine output. Muscle damage is extremely common in high-voltage electrical injuries. This can produce rhabdomyolysis and renal injury. The urine should be routinely tested for the presence of myoglobin. If urinary pigment is present, the patient should be challenged with two ampoules of sodium bicarbonate and 50 g of mannitol. Sufficient IV fluids should be given to maintain the urine output at 100–125 ml/h until the urine clears.

Compartment syndrome is common, and it is often necessary to take the patient to surgery during resuscitation for emergency fasciotomies. In addition to assessing the external burn, the physician should try to imagine the path of the current and make some assessment of the internal burn. He or she should look for evidence of compartment syndrome (a tense painful limb, severe pain on passive extension of the digits, an interstitial tissue pressure of greater than 30 mm Hg) and arrange urgent fasciotomies if present.

Early and repeated surgical debridements are carried out due to the difficulty in identifying living tissue from dead. Such procedures are performed early and often. Regrettably, amputations are frequently part of this picture. Often vital structures such as nerves and blood vessels become exposed as necrotic tissue is removed, necessitating early flap coverage. However, this should not take place until the viability of the underlying tissue is known.

Rehabilitation and Reconstruction

Rehabilitation of burns begins immediately. Burned hands should be splinted in the ideal position of function with the wrist extended; the metacarpophalangeal joints of the index, long, ring, and little fingers flexed at 90°; and the interphalangeal joints splinted straight and the thumb held abducted and in opposition. In the arms, burns that cross flexion creases require splintage of the resting joint in extension to prevent the formation of contractures. In the burned lower limb, the knee should be splinted straight, and the foot should be splinted in a neutral position between dorsi- and plantar-flexion. Hypertrophic scarring may be treated by compression garments until the scars mature. Physiotherapy is commenced immediately to mobilize the joints, preserve a normal range of motion, maintain muscle strength, and facilitate normal ambulation.

Surgical reconstruction is usually commenced after 6 months or more, by which time the wounds should have healed and the scars matured. A number of operative procedures may be contemplated. However, the principle of the reconstructive ladder should be considered. Here, the simplest method of reconstruction is performed if at all possible – provided it meets the goals of the operation. Only if it does not, does one consider the next step on the ladder. For example, a scar may be excised and a wound produced. The simplest concept is to close it directly using sutures. If the defect is too wide for this, then a skin graft may be

contemplated. If bone or vital structures are exposed, then a local flap may be necessary. If there is not enough local tissue for this, then a regional flap would be preferable. On this stairway, a free flap, or, perhaps, a composite tissue allograft may represent the final step.

Contractures may be released by incisions carried through them into the underlying unburned tissue. The spaces opened up when the joint is extended can then be filled with skin grafts or flaps. Scar revisions may be performed to make burn scars more acceptable both from an aesthetic as well as from a functional standpoint. Revisions may include the narrowing of a scar, its complete excision, or attempts to realign it into a less conspicuous position. Narrow scars that form a web across a joint may be lengthened using a Z-plasty; but sheets of scar causing a contracture usually require grafts. Scars may sometimes be removed and replaced by a local tissue rearrangement. Scars that have proven unstable with recurrent skin breakdowns may be excised and resurfaced with a skin graft or a flap. To facilitate the resurfacing of a scar using limited adjacent tissue, the adjacent skin may be subjected to tissue expansion prior to the scar's excision.

Specific structures require special attention in the reconstruction of the burned patient. Nowhere is this more evident as in the reconstruction of the burned hand. Burns are more likely to be full thickness on the dorsum of the hand, since the skin is thinner and has less keratin than the palm. Furthermore, the hands are frequently used to protect other areas from injury. In such situations, the dorsum of the hand inevitably faces the flame. However, full-thickness burns of the palm certainly do occur especially when the burn is a contact burn. While the burned hand is being dressed and receiving topical treatment, it is important to splint it in a position of function as described above. The splint is removed for physiotherapy. Due to their asymmetrical attachments, the collateral ligaments of the finger joints are under maximum stretch when the hand is in the functional position. Any shortening is thus resisted and the full range of movement maintained, setting the stage for definitive reconstruction. This may involve resurfacing, scar releases, the correction of burn syndactyly, local and distant flaps, fusions, tendon reconstructions, and amputations.

Burns involving the axilla can result in a severe adduction contracture of the shoulder. Assuming the contracture is refractory to physiotherapy, it will require a release with the interposition of healthy tissue such as a graft or flap. Postoperative splinting and physiotherapy are designed to maintain and consolidate the gains produced by surgery.

Another area of concern is the female breast. Frequently, young children receive a mixed partial and full-thickness flame burn involving the anterior chest. If the nipple–areola complex is damaged, then the progenitor cells of the breast bud may be destroyed leading to underdevelopment. If it remains intact, the breast will develop normally but may be constricted by overlying scarring. Even with early wound excision and skin grafting, the area remains tight and inelastic. As the patient matures, this can adversely affect the growth of the breast. Frequently, it becomes necessary to place a subpectoral tissue expander in the affected side in order to stretch the skin and permit the placement of an implant. Successively larger implants may need to be placed until growth is complete. A decision is then made whether to carry out a symmetry operation on the other side and whether to consider autogenous reconstruction.

A particularly disfiguring recipient site for burns is the face. Frequently, facial burns look much worse than they are due to severe swelling and blistering. Often partial-thickness burns may initially appear to be full thickness. Since complete healing can occur when least expected, a conservative approach has been adopted in the treatment of burns of the face. Closed dressings are seldom used but silver sulfadiazine cream is applied topically and then washed off before each new application (twice daily). Tangential excision has been recommended to remove eschar in certain cases.[4] Reconstructive surgery is performed long after healing is complete, and it takes the form of the options mentioned above. Of particular importance in dealing with the face is to reconstruct in "aesthetic units" (Figure 5.11) to avoid the appearance of a patchwork quilt. In other words, when resurfacing is contemplated with the use of either grafts or flaps, then an entire unit is replaced, not just its burned component.

Finally, long-term reconstruction of underlying structures is frequently necessary following electrical burns. Extensive injury to muscles and nerves may necessitate arthrodeses, tendon transfers, nerve grafts, flaps, and occasionally functioning muscle transfers.

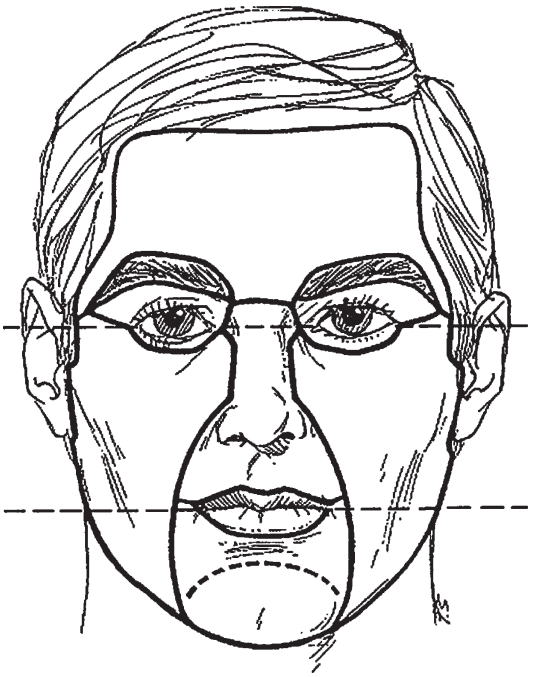


Figure 15.11. Aesthetic units of the face. (Reprinted from Gonzalez-Ulloa M, Restoration of the face covering by means of selected skin in regional aesthetic units. *Br J Plast Surg*. 1956;9:212. With permission from Elsevier © 1956.)

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16

Benign and Malignant Skin Tumors

Risal S. Djohan, Rebecca Tung, Esteban Fernandez-Faith, and Laszlo Karai

Summary

Skin neoplasms are common concerns for which patients seek medical attention. Familiarity with these benign and malignant tumors is essential for appropriate evaluation and management. In the current era, skin cancer is the most common neoplasm in humans – more than 1 million new cases will be diagnosed in the United States this year. Pigmented lesions pose a particular challenge because melanoma, the potentially lethal form of skin cancer, is always part of the differential diagnosis. Fortunately, early detection and prompt treatment of skin cancer, especially in the case of melanoma, improve overall prognosis and survival.

This chapter reviews the epidemiology, pathogenesis, clinical presentation, histopathology, and management of common benign and malignant skin tumors including seborrheic keratoses, melanocytic nevi, actinic keratoses, squamous cell carcinoma (SCC), basal cell carcinoma (BCC), and melanoma.

Abbreviations

AJCC	American Joint Committee on Cancer
BCC	Basal cell carcinoma
DFSP	Dermatofibrosarcoma protuberans
EMPD	Extramammary Paget's disease
FAMMM	Familial atypical multiple mole melanoma syndrome
MAC	Microcystic adnexal carcinoma
NMSC	Nonmelanoma skin cancer
PUVA	Psoralen plus ultraviolet A radiation
SLNB	Sentinel lymph node biopsy
SCC	Squamous cell carcinoma

Introduction

Skin neoplasms are a common source of concern for patients, who seek advice from primary care physicians, family physicians, plastic surgeons, and dermatologists. Commonly, patient's concerns are focused on cosmesis and the potential of malignancy of the specific skin conditions. Knowledge about common benign and malignant skin neoplasms is crucial for an appropriate evaluation and management. Of particular importance is the ability to recognize clinical features that raise the suspicion of malignant changes.

Seborrheic Keratosis

Definition and Epidemiology

Seborrheic keratoses are common, benign neoplasms of the skin with characteristic clinical and histopathologic features. Whether appearing as solitary or multiple lesions, the incidence increases with age. Seborrheic keratoses are rarely present before the third to fourth decades

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and have a prevalence of 80–100% in people older than 50 years.[26,48]

Pathogenesis

The pathogenesis of these common growths is not entirely known but is likely to be multifactorial. The proposed risk factors include aging, ultraviolet (UV) light exposure, and mutations in fibroblast growth factors.[21,26]

Clinical Presentation

The face, neck, and trunk are commonly affected sites, while the palms and soles are spared. Early lesions appear as hyperpigmented macules, later evolving into round-oval, light brown to black papules or plaques with sharp demarcation. The surface is waxy or verrucous with a "stuck-on" appearance (Figures 16.1 and 16.2).

Histopathology

Seborrheic keratoses are benign squamous proliferations with variable degrees of acanthosis, hyperkeratosis, and papillomatosis. They are composed of cells with basaloid morphology, which in reaction to irritation form structures known as "squamous eddies." The presence of horn pseudocysts and melanin pigment is a common finding.

Treatment

Seborrheic keratoses have traditionally been considered as benign neoplasms; however, different types of skin cancers have been reported in association with seborrheic keratoses. Moreover, recent data suggest the potential for malignant transformation of seborrheic keratoses.[30] Biopsy should be considered in lesions that appear irritated or have undergone clinical changes.

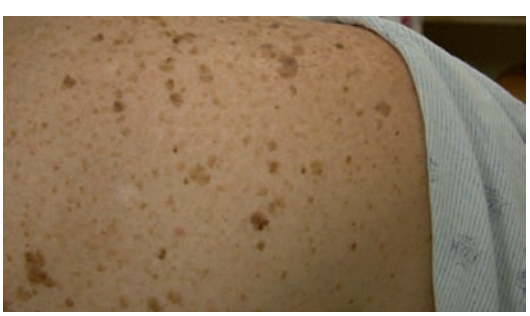

Figure 16.1. Multiple early seborrheic keratoses as brown macules and verrucous papules on the back.

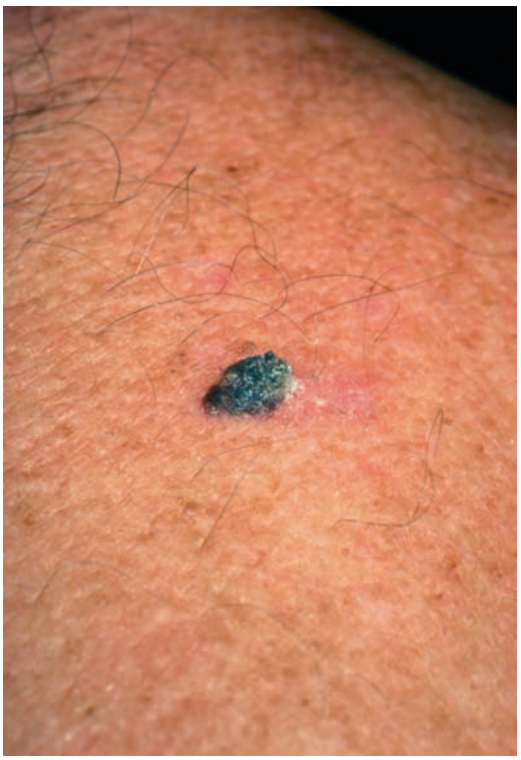

Figure 16.2. Seborrheic keratosis as dark keratotic papule with "stuck-on" appearance.

If malignancy is not a concern after clinical evaluation, treatment of seborrheic keratoses is done for cosmetic reasons or to alleviate potentially associated symptoms of pruritus, inflammation, or bleeding. Widely used treatments include removal with cryosurgery (liquid nitrogen), curettage, CO_2 laser ablation, focal chemical peeling (trichloroacetic acid), electrodessication, or surgical excision.

Melanocytic Nevi: Congenital, Acquired, and Atypical

Definition and Epidemiology

Melanocytic nevi or moles are very common benign skin neoplasms that result from the proliferation of nevus cells, which are slightly altered melanocytes.

Depending on the time of appearance, these neoplasms are subdivided into congenital or acquired.

The prevalence of acquired nevi depends on several factors including skin type, age, genetic predisposition, and sun exposure. These common neoplasms typically appear after 6–12 months of age; increase in number during childhood and adolescence; peak in the third decade; and tend to disappear with increasing age.

Congenital melanocytic nevi are, by definition, present at birth; although sometimes they are not noticed until later during the first year of life. Their incidence has been calculated between 0.2% and 2.1% of newborns.[25] Traditionally, congenital nevi have been classified according to their size as small (<1.5 cm), medium (1.5–19.9 cm), and large or giant (>20 cm). This classification is based on the greatest diameter of the nevus in adulthood.

The atypical or dysplastic nevus is a somewhat controversial term, which refers to melanocytic nevi with abnormal or unusual clinical and/or histopathologic features. As opposed to acquired melanocytic nevi, atypical moles begin to appear around puberty and may continue to develop past the fourth decade. The prevalence of dysplastic nevi is variable, ranging from 7% to 18%.[35]

Pathogenesis

Multiple factors are involved in the pathogenesis of acquired melanocytic nevi and dysplastic nevi. These factors include skin type, genetic predisposition, and sun exposure. Congenital melanocytic nevi develop between weeks 5 and 25 of gestation. They are thought to result from a dysregulated growth and arrest of melanocytes during migration from the neural crest to the skin.[6] Genetic and familial predisposition is particularly important in a subset of patients with a condition known as familial atypical multiple mole melanoma (FAMMM) syndrome. Patients with FAMMM syndrome have large amounts of acquired melanocytic nevi, some of which are atypical, and have increased risk of melanoma.

Clinical Presentation

Melanocytic nevi present as well-defined, round or oval, symmetric lesions measuring from 2 to 6 mm in diameter. The clinical appearance depends on the level where the nevus cells are located. Nevi with a predominant epidermal component (junctional nevi) appear flat with a uniform brown to almost black color (Figure 16.3). When the nevus cells involve both the epidermis and the dermis (compound nevi), the nevus will rise above the skin surface and show lighter shades of brown when compared to the junctional counterpart (Figure 16.4). An intradermal nevus (nevus cells predominantly in the dermis) is typically a raised, dome-shaped papule, with pigmentation ranging from light brown to flesh color (Figure 16.5).

The clinical features of dysplastic nevi include a diameter larger than 5 mm, irregular pigmentation, ill-defined or irregular borders, asymmetry,

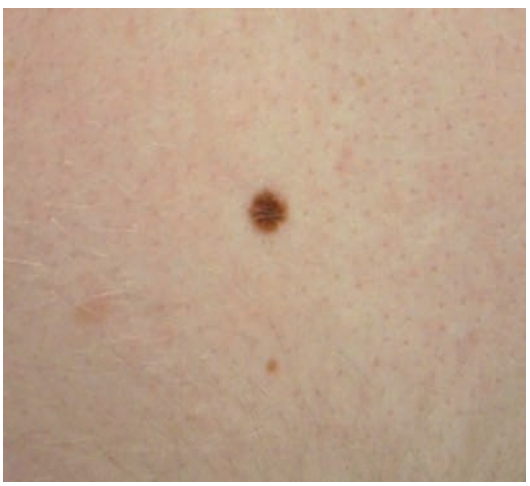

Figure 16.3. Junctional nevus: symmetric, brown macule with regular borders.

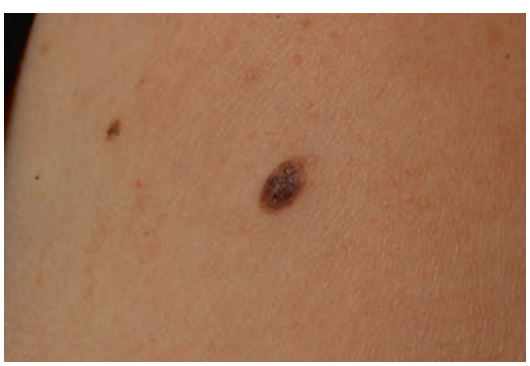

Figure 16.4. Compound nevus: oval, brown, symmetric papule with regular borders.

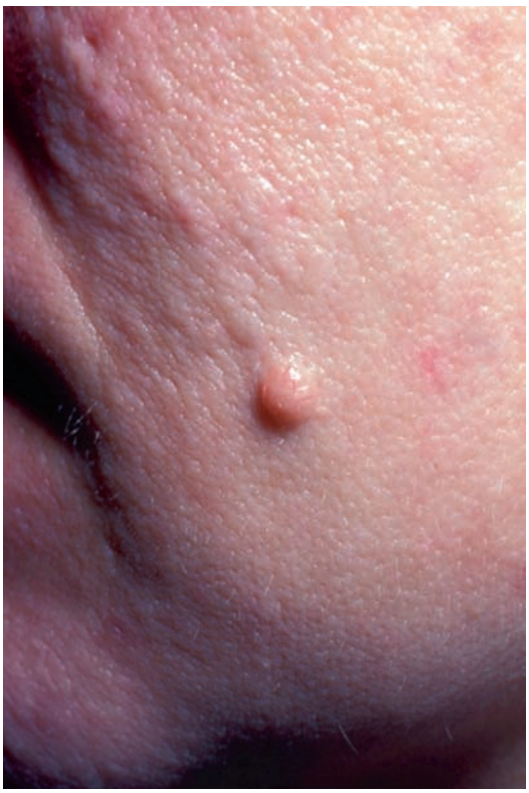

Figure 16.5. Intradermal nevus: flesh-colored, dome-shaped papule.

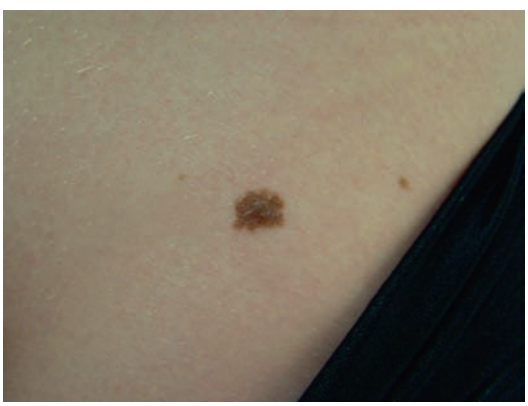

Figure 16.6. Dysplastic nevus: brown asymmetric macule with irregular borders and irregular surface.

and irregular surface (Figure 16.6). They are most commonly located on the trunk, although they may present anywhere in the skin.

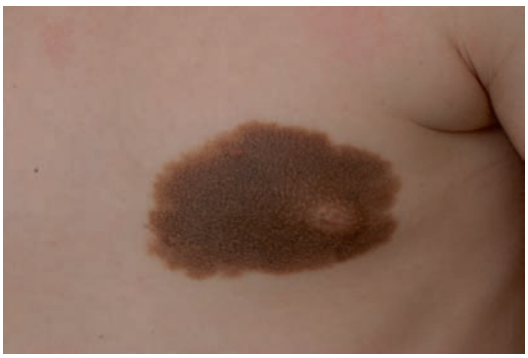

Figure 16.7. Congenital nevus on trunk with mild surface changes.

Congenital nevi occur most often on the trunk, followed by the extremities, head, and neck. Congenital nevi are usually light brown in the first few weeks of life and frequently undergo multiple clinical changes, including darkening, development of hair, nodules, verrucous texture, erosions, or ulcerations (Figure 16.7). These changes are seen particularly in large congenital melanocytic nevi, not so often in small or medium sized nevi. Patients with large congenital nevi may also present satellite nevi, which refers to small or medium-sized congenital nevi distant from the larger lesion.

Recommended follow-up for patients with atypical nevi or a family history of skin cancer is at least an annual full skin examination. Patients should also be encouraged to perform a self-skin examination on a monthly basis. Any changing, growing, or bleeding lesion should be evaluated by the physician. Since sun exposure is the most preventable risk factor for skin cancer, sun safety tips such as daily application and reapplication of broad spectrum sunscreen, wearing sun protective clothing (with sunglasses and hats), seeking shade during peak sun hours (10 a.m. to 4 p.m.), and avoiding tanning beds should be suggested to all patients.[3]

Histopathology

Congenital and acquired nevi may share several histologic features; therefore, the diagnosis of congenital nevi is heavily dependent on the presence at birth as part of the clinical history information. Certain features suggestive of this type of

nevi include involvement of arrector pili muscles, sebaceous and eccrine glands and splaying of melanocytes in between collagen fibers.

Dysplastic nevi show cytologic atypia and architectural abnormalities such as elongated rete ridges with exuberant proliferation of melanocytes, bridging between nests of melanocytes and lamellar fibroplasia. The presence of a variably dense lymphocytic infiltrate is also a common feature.

Management

The rationale for removal of acquired melanocytic nevi, typical and atypical, relies on their associated potential risk for melanoma, and, in some cases, aesthetics. Although melanomas can arise from melanocytic nevi, most melanomas will develop as de novo lesions; and most nevi (typical and atypical) will not progress to melanoma. For this reason, "prophylactic excision" is generally not recommended, unless there are concerning clinical changes that suggest melanoma. Although lasers, cryosurgery, and chemical peels have been used to remove benign-appearing melanocytic nevi, these modalities are generally not considered appropriate therapy for a nevus with atypical features, since no tissue will be available for histologic interpretation and an undetected melanoma could potentially be present in the residual lesion. If an atypical nevus is suspected, an excisional biopsy is preferable.

Treatment of congenital nevi is based on the size and the location of the lesion. Large congenital nevi have an increased risk of melanoma, particularly during childhood and adolescence.[25] Management remains controversial as some authors believe that the risk is not significant enough to warrant large, complicated surgical procedures.[6] However, if a congenital nevus, regardless of size, is impairing a child's self-confidence or social development, possible excision should be investigated. Treatment options include surgical excision alone or in conjunction with tissue expansion and/or skin grafting, curettage, dermabrasion, chemical peeling, and lasers.

The risk of melanoma in small and medium congenital nevi is not well determined and is thought to be similar to the risk in acquired melanocytic nevi. For this reason, surgical excision should be considered on an individual basis.

Nevus Sebaceous and Epidermal Nevus

Although not as common as melanocytic nevi, nevus sebaceous and epidermal nevus are two nonmelanocytic congenital conditions with potential important implications.

Nevus sebaceous is a congenital oval or linear, hairless verrucous plaque most commonly found on the scalp and face (Figure 16.8). It typically undergoes distinct phases of growth during childhood, puberty, and adulthood. Different benign and malignant neoplasms can potentially arise in a nevus sebaceous, particularly during puberty and adulthood. Basal cell carcinoma (BCC) is the most commonly reported malignant neoplasm in this type of nevi. Treatment of nevus sebaceous is surgical excision.

Past recommendations for removal of all lesions are now questioned since the incidence of malignant transformation is low. Current practice advocates observation and removal of lesions clinically suspicious for malignancy.[41]

Epidermal nevus present within the first year of life as well circumscribed, linear, or whorled plaques commonly found on the trunk and extremities (Figure 16.9). With time, the surface may become more verrucous, and pigmentation can vary from skin color to pink to hyperpigmented.

In a subset of patients, the epidermal nevus as well as the nevus sebaceous and other less common skin lesions can have associated systemic involvement, termed epidermal nevus syndrome.

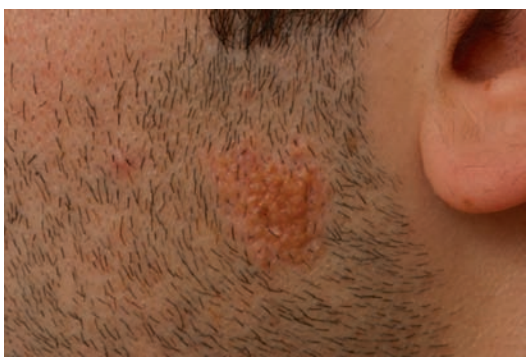

Figure 16.8. Nevus sebaceous: orange-yellow waxy plaque on the face.

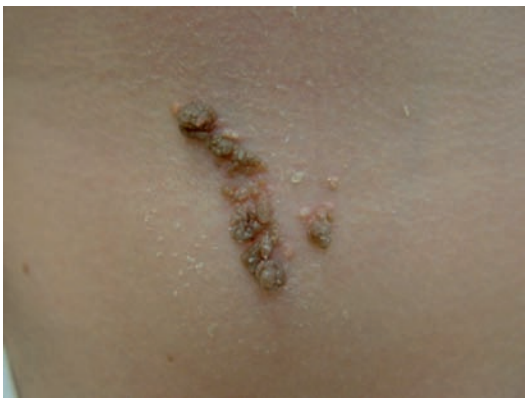

Figure 16.9. Epidermal nevus: linear, light brown, verrucous/papillomatous plaque.

The most common extracutaneous manifestations involve those in the central nervous system, skeletal system, and eyes.[44]

Actinic Keratosis

Definition and Epidemiology

Actinic keratoses are ultraviolet (UV) light-induced, in situ epidermal dysplasias, also known as solar keratoses. Historically considered a premalignant neoplasm with the potential to develop into a squamous cell carcinoma (SCC), recent debate has centered on the controversy of whether they represent a precancerous condition versus an in situ SCC.

Actinic keratoses occur primarily in fair-skinned individuals with a history of chronic sun exposure. With skin phototypes I–III, the prevalence in patients older than 40 years has been calculated at 40%. In patients older than 60 years, the prevalence increases to 80%.[15]

Pathogenesis

Natural UV radiation, mainly UV-B (290–320 nm), is the main associated risk factor in the development of actinic keratoses in fair-skinned individuals. Other known causes include prior exposure to x-irradiation, repeated UV light exposure from artificial sources, and exposure to chemicals, including polycyclic aromatic hydrocarbons and arsenic.[39]

Clinical Presentation

Most commonly, actinic keratoses present as red, scaling papules or plaques on sun-exposed areas, mainly on the face, scalp, dorsum of hands, and shoulders (Figures 16.10 and 16.11). Although usually presenting as multiple lesions, single actinic keratosis can occur. On average, they measure 1–3 mm in diameter, but larger or confluent lesions can also be present. The surface is rough on palpation, and early actinic keratoses can be more easily felt than seen. Not infrequently, the patient may report pruritus, tenderness, and burning sensation.

Given the causal effect of UV light exposure, the surrounding skin typically reveals signs of

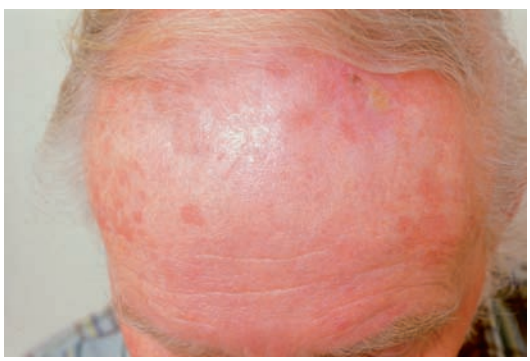

Figure 16.10. Actinic keratoses: red, rough plaques on forehead and scalp.

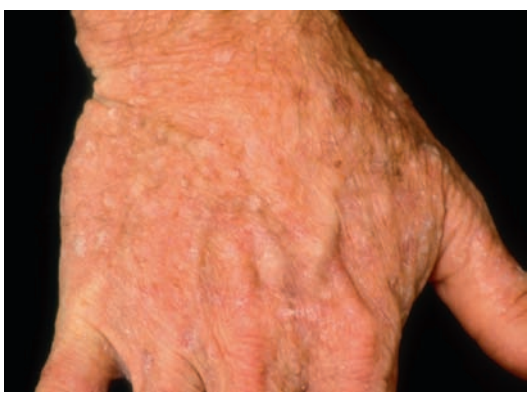

Figure 16.11. Actinic keratoses: multiple rough, scaly papules on the dorsum of the hand.

sun damage, including telangiectasias and blotchy pigmentation.

Other clinical presentations include the pigmented actinic keratosis, the cutaneous horn, actinic cheilitis, and lichen planus-like keratosis. The cutaneous horn is a hypertrophic variant of special consideration, since up to 8.9% of these lesions are actually SCCs.[49] Since the pigmented actinic keratosis can easily be confused with a solar lentigo or a lentigo maligna, histopathologic analysis is required for differentiation.

Histopathology

The hallmark of actinic keratosis is the presence of dysplastic keratinocytes in the epidermis associated with prominent parakeratosis, which can alternate with orthokeratosis. In early lesions, dysplastic cells are scattered and involve the basal layers only. As the dysplasia spreads to the full thickness of the epidermis, the diagnosis of in situ SCC is warranted.

Treatment

The main rationale for treating actinic keratoses is to prevent malignant transformation into SCC, but cosmesis and symptomatic relief may also play a role.

Multiple treatment modalities, including surgical and medical options, are available.[34,39] The treatment of choice must be tailored to the individual. Factors such as the number and location of the lesions, clinical subtype, and patient preference, must be taken into account. In many cases, a combination of surgical and medical treatments is optimal. Whenever the clinical diagnosis is not clear, a biopsy should be considered.

Procedural options include cryosurgery, curettage and electrodessication, dermabrasion, laser ablation with CO_2 or erbium-YAG lasers, photodynamic therapy, medium-depth chemical peeling, and surgical excision.

Medical treatment options are used mainly for patients with multiple or widespread lesions. These options include 5-fluorouracil cream/solution, imiquimod cream, diclofenac gel, oral and topical retinoids, and interferon-α-2b.

Regardless of the treatment modality chosen, photoprotection must always be advised in an attempt to prevent or reduce the number of actinic keratoses in the future.

Nonmelanoma Skin Cancer: Squamous Cell Carcinoma and Basal Cell Carcinoma

Definition and Epidemiology

Nonmelanoma skin cancer (NMSC) is a broad term that includes skin neoplasms arising from cells other than melanocytes. Although multiple different types of such malignancies have been described, most of them are represented by BCCs and SCCs. More than 1 million cases of NMSCs are diagnosed annually,[4] with BCCs leading the count in a ratio of approximately 4:1 when compared with SCCs.[38]

The risk for developing NMSC increases with age, particularly in white populations with a history of chronic sun exposure.

Pathogenesis

Several factors have been implicated in the pathogenesis of NMSC, the main one being UV radiation. A history of chronic sun exposure (recreational and occupational) and a history of sun burns along with other factors including geographic location, ethnicity, and skin color have a role in pathogenicity.[5] Mutations in the p53 tumor-suppressor gene from UV radiation have been implicated in the molecular basis of NMSC.[8,20] Artificial UV radiation also increases the risk of SCC and BCC, particularly when the first exposure occurs in the first two decades of life.[24]

Other less well-studied lifestyle behaviors have been linked with the development of NMSC (especially SCC), including smoking and diets high in fat content.[10,14]

Special consideration should be given to certain populations with higher risk of NMSC. These groups include transplant patients, chronic immunosuppression, patients treated with ionizing radiation or PUVA (psoralen plus UV A radiation), exposure to carcinogenic chemicals such as arsenic, and certain hereditary disorders including xeroderma pigmentosum and oculocutaneous albinism.[2]

Clinical Presentation

Squamous Cell Carcinoma

Squamous cell carcinomas may develop in the skin of any body site or in mucous membranes;

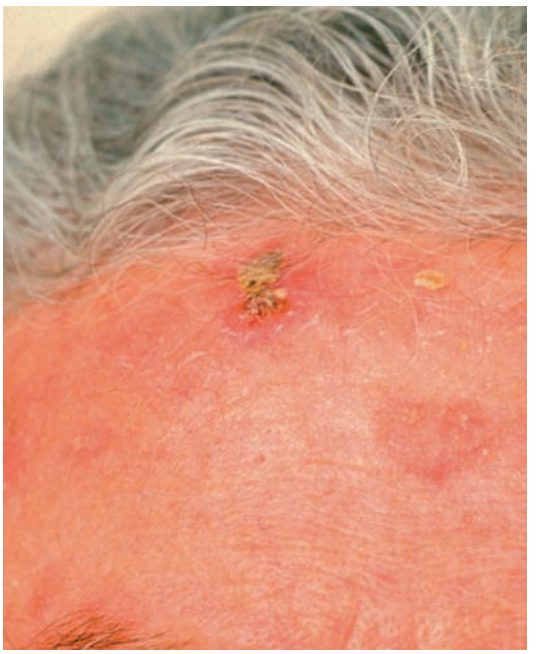

Figure 16.12. Squamous cell carcinoma: keratotic plaque on an erythematous base on the forehead.

nevertheless, the most common locations are the scalp, ears, face, lower lip, neck, and dorsum of the hands (Figure 16.12). As mentioned above, a number of SCCs develop from actinic keratoses, which could be clinically indistinguishable. SCC in situ presents as sharply demarcated, erythematous, scaly papules or plaques. This early form of SCC is known as Bowen's disease. Erythroplasia of Queyrat is the name given to SCC in situ when occurring on the glans penis of uncircumcised men.

More advanced lesions of SCC present as enlarging, indurated, erythematous, scaly papules, plaques, or nodules. Itching, pain, or bleeding may be concomitant symptoms. Ulceration and crusting may be associated features, which, in certain cases, signal invasion of underlying structures with development of regional lymphadenopathy.

Basal Cell Carcinoma

In contrast to SCC, BCC usually arises de novo on sun-exposed areas, particularly the head and neck. Different clinical variants have been described, the most common being nodular and superficial. Nodular BCC presents as a pearly or waxy papule

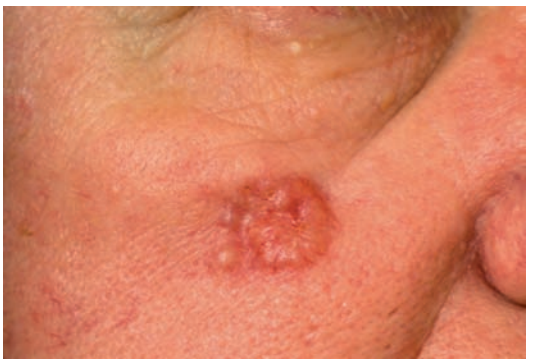

Figure 16.13. Basal cell carcinoma: pearly nodule with rolled borders and telangiectasias.

or nodule with a rolled border and overlying telangiectases (Figure 16.13). Superficial BCC presents an erythematous, scaly patch or plaque. The morpheaform or sclerosing clinical variant is an indurated yellow to white scar-like plaque with indistinct borders and atrophic surface. Even though this is an uncommon variety, its aggressive and invasive growth pattern has important treatment and prognostic implications.

Histopathology

Basal cell carcinoma is composed of deep blue cells due to a high nucleus to cytoplasm ratio. There is prominent peripheral palisading commonly associated with artifactual cleft formation between the tumor and the stroma, the latter being rich in mucin. As previously mentioned, there are several subtypes of BCC of which the sclerosing or morpheaform is significant for a more aggressive behavior.

The precursor lesion of an SCC is the in situ carcinoma, which is sometimes difficult to differentiate from superficially invasive lesions. Well, moderately, and poorly differentiated forms are identified together with specific subtypes. Histological features such as desmoplastic reaction around keratinocytic islands, perineural invasion, or intravascular spread are diagnostic of malignancy.

Treatment and Prognosis

In general, prognosis of primary NMSC is excellent, with low recurrence rates and risk of metastasis when the appropriate treatment modality has been

chosen. SCC has a less favorable prognosis than BCC. The 5-year recurrence rate of primary cutaneous lesions has been estimated as 8% for SCC versus 4.8% for BCC.[2,43] The risk of metastasis is higher in SCC ranging from 0.1% to 9.9% versus 0.0028% to 0.55% in BCC.[40,47] Several risk factors for the development of recurrence and metastases of NMSC have been identified. Particularly important features include size (larger than 2 cm in diameter) and depth of invasion, aggressive histologic pattern, perineural and perivascular involvement, lesions arising in previous radiation sites, location on the mid-face, ears, lips, and genitals, and immunosuppression.[12,40,47]

Several treatment modalities, surgical and nonsurgical, can be employed for the treatment of NMSC. The treatment of choice will depend on the specific characteristics of the patient and neoplasm, such as age, location, risk of recurrence and metastasis, histologic subtype, and history of previous NMSC. The gold standard of treatment is Mohs micrographic surgery, because it maximally preserves healthy tissue and offers the lowest 5-year recurrence rates: 1.4% for primary BCC, 4% for recurrent BCC; 2.6% for primary SCC and 5.9% for previously recurrent SCC.[27–29]

Mohs micrographic surgery is a precise margin-controlled surgical technique that allows complete examination of all margins of tissue removed. Other surgical options include conventional excision, electrodessication and curettage, and cryosurgery. Nonsurgical methods are topical chemotherapy with 5-fluorouracil, intralesional interferon, imiquimod, retinoids, photodynamic therapy, and, in specific circumstances, radiation therapy.[37]

During Mohs surgery, serial horizontal sections of tumor are removed, mapped, processed by frozen section, and analyzed microscopically. In contrast to standard, vertically oriented histopathology sections, which assess less than 1% of the tumor margin, this technique provides up to 100% of the epidermal and deep margins for examination, allowing more accurate tumor mapping and cancer clearance.[16,37] This technique is usually done with local anesthesia in an outpatient setting. Mohs surgery can be used for multiple types of tumors including basal cell carcinoma, SCC, melanoma, sarcomas, and other nonmelanoma skin cancers, including dermatofibrosarcoma protuberans (DFSP), microcystic adnexal carcinoma (MAC), extramammary Paget's disease (EMPD).[45] The indications for Mohs micrographic surgery include[16,37,42] the following:

- Recurrent tumors
- Tumors greater than 2 cm in size
- Aggressive histological growth patterns
- Tumors with ill-defined clinical margins
- Incompletely excised tumors
- Perineural involvement
- Tumors in areas with high risk of recurrence (central face, periorbital, periauricular areas)
- Tumors arising in irradiated skin
- Tumors in areas in which tissue preservation is mandatory

In most of the cases, the Mohs micrographic surgeon performs the reconstruction of the defect once the tumor is cleared. However, a multidisciplinary approach including plastic surgeons, oculoplastic surgeons, and/or head and neck surgeons can be advantageous for the excision of deeply invasive tumors or in the repair of complex defects.[37,42]

Melanoma

Definition and Epidemiology

Cutaneous melanoma is a neoplasm that arises from melanocytes as a de novo lesion, but it may also develop from congenital or acquired nevi. Other potential sites in which melanomas can form include mucous membranes, retina, leptomeninges, lymph nodes, and gastrointestinal and genitourinary tracts.

Around the world, the incidence of melanoma has been increasing steadily, with non-Hispanic men older than 65 years showing the highest increase in rate.[17] The estimated number of cases of melanoma in 2007 was 59,940 (33,910 in males, and 26,030 in females) according to the American Cancer Society,[4] giving men an approximately 1.5 times higher risk of developing melanoma when compared to women. The peak incidence of melanoma is among people aged 20–45 years, in contrast to nonmelanoma skin cancer, which occurs mainly in older patients.

Mortality rates show variable patterns depending on the geographic location. Even though the mortality rate in the United States has remained stable in men and even decreased among women, the worldwide trend is for uniformly increasing mortality rates. This increase in mortality is particularly noticeable in older men and women.[17] Deaths from melanoma in 2007 were estimated to be 8,110.[4]

On the other hand, early detection and education programs have led to 5-year survival rates exceeding 90% in certain countries including the United States. This highlights the importance of adequate clinical diagnostic skills to detect early disease.

Although anyone can develop melanoma, the particular risk factors include advanced age, male gender, family history of melanoma, personal history of melanoma or nonmelanoma skin cancer, organ transplant recipient, low socioeconomic status, atypical nevi, and fair-skinned individuals.[17]

Pathogenesis

Melanoma develops from a combination of constitutional predisposing and environmental factors, particularly UV radiation. The role of sun exposure and melanoma formation is complex, and both natural and artificial UV light have been linked to the development of melanoma, particularly when exposure occurs before the age of 35 years.[23] The host factors associated with increased risk of melanoma include number of melanocytic nevi (both dysplastic and nondysplastic), family history, immunosuppression, and certain phenotypic characteristics such as blue or green eyes, blond or red hair, and skin sensitivity to the sun.[32] Genetic and molecular abnormalities associated with some of these host factors, and therefore linked with melanoma, include mutations in *CDKN2A* gene and melanocortin-1 receptor.

The model of progression from normal melanocytes to melanoma was proposed by Clark. This model refers to stepwise histologic changes, starting with the acquired melanocytic nevus undergoing aberrant differentiation and nuclear atypia resulting in the formation of primary melanoma, which initially has a radial growth phase followed by a vertical growth phase, ending with the development of metastatic melanoma.[13]

Clinical Presentation

Clinical evaluation of pigmented lesions can be complicated, because melanoma is part of the differential diagnosis. A conventional guide, particularly for evaluation of nevi, is the ABCDE acronym, which lists clinical characteristics that can be associated with melanoma. The acronym stands for *A*symmetry, *B*order irregularity, *C*olor variation, *D*iameter larger than 6 mm, and *E*volving referring to changes in size, shape, surface, shades of color, or presence of symptoms such as pruritus and pain[1] (Figure 16.14). Although not perfect, it represents an appropriate general guide both for health care providers and for patients. The "ugly duckling sign" is another useful clinical finding that refers to the atypical appearance of a pigmented lesion when compared with surrounding nevi.[19] Dermoscopy or epiluminescence microscopy has gained popularity as the one aiding in the early clinical diagnosis. This is a noninvasive technique using a high-resolution, optical, handheld device or dermoscope to enhance visualization of microscopic structures of pigmented lesions.

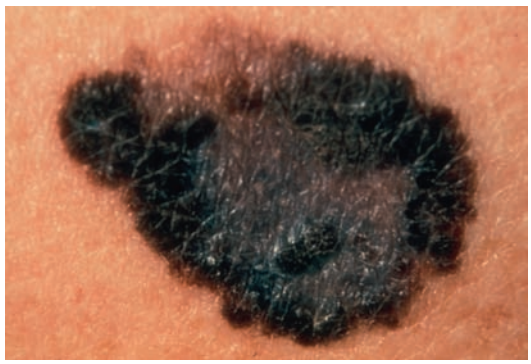

Figure 16.14. Melanoma: large, irregularly pigmented, asymmetric plaque.

A clinicopathologic classification divides melanomas into superficial spreading, lentigo maligna, nodular, and acral lentiginous. Approximately 70% of cases of melanoma are superficial spreading melanoma, most often occurring on the back of the legs of women and on the backs of men. Although acral lentiginous is in general an uncommon subtype, it represents the most common type of melanoma among Asian, Hispanic, and African patients.

Histopathology

Malignant melanoma can present in normal, atrophic, hyperplastic, or ulcerated epidermis, the latter being an important prognostic feature. There is asymmetrical, nonrandom, cytologic atypia throughout the lesion with nuclear hyperchromasia, irregular nuclear outlines, and the presence of prominent nucleoli. Intraepidermal

spreading of malignant single cells in a so-called pagetoid or "buckshot" pattern is a useful histological finding. Radial (intraepidermal) and vertical (invasion into the dermis) growth phases are defined. The most significant histological characteristic is the Breslow thickness of the tumor. Other important features are the type of lesion (superficial spreading, lentigo maligna, nodular, and acral), number of mitotic figures per square millimeter, perineural invasion, intravascular spread, cytologic type, presence or absence of satellite lesions, regression, lymphocytic infiltration, and involvement of the margins of the tumor.

Staging and Prognosis

In 2001 the Melanoma Staging Committee of the American Joint Committee on Cancer (AJCC) published the most recent melanoma TNM staging classification.[7] Depth of invasion is the most important histologic prognostic parameter in primary melanoma. Breslow depth and Clark level are two different classifications of depth of invasion that have been recognized for decades. Breslow depth is a quantitative measurement of the depth of invasion by measuring the tumor thickness with an ocular micrometer. Clark's staging refers to the histologic level of invasion, using the epidermis, papillary dermis, reticular dermis, and subcutaneous fat as the histologic boundaries. With the 2002 AJCC staging classification, tumor thickness measured by Breslow depth was determined to be the primary factor for T staging. The presence of ulceration was found to be a powerful predictor of survival, and hence it is incorporated in the staging system.

Lymph node involvement, determined with a sentinel lymph node biopsy (SLNB), is the most powerful predictor of recurrence and survival. Sentinel lymph node status also determines the eligibility for clinical trials and need for adjuvant therapy. This technique identifies and resects the first lymph node(s) to drain lymphatic flow from the primary tumor site by using Technetium-99 m-labeled radiocolloids and vital dye. SLNB is considered a staging and possibly therapeutic procedure. The resected lymph nodes are then evaluated by hematoxylin–eosin and immunohistochemical analysis such as S-100, HMB-45, and MART-1. Ninety-five percent of the time, the sentinel lymph node can be identified with only a less than 5% false negative rate. Indications for SLNB include tumors at least 1.0 mm thick and tumors less than 1.0 mm thick that present with ulceration or Clark's level IV involvement. Thinner melanomas (less than 0.8 mm thick) usually do not warrant SLNB, since the likelihood of finding a metastasis is only 1%.[18]

The parameters used to determine the TNM stage also establish the melanoma clinical stage, on which prognosis and therapeutic options are based. Four clinical stages are described; stages I and II represent localized melanoma, whereas stage III disease includes regional metastases and stage IV, distant metastases. Prognosis varies greatly with 10-year survival rates ranging from 100% in cases of melanoma in situ to less than 16% in stage IV disease (distant metastasis).[33]

Treatment

The current practice for invasive melanomas involves excision of cutaneous and subcutaneous tissue down to the underlying fascia, without removing it, with a suggested margin of excision as listed in Table 16.1. Appropriate surgical treatment should be based on histologic confirmation of tumor-free margins. Recent literature suggests that in some cases of melanoma in situ, the standard margin of 0.5 cm may be insufficient for complete excision.[11,22]

Patients with metastatic melanoma (stages III and IV) are candidates for adjuvant therapy. This includes interferon alpha, granulocyte-macrophage colony-stimulating factor, cancer vaccines, and systemic chemotherapeutic agents such as dacarbazine and interleukin-2. A series of novel melanoma treatment modalities are under investigation, including cancer vaccines, angiogenesis inhibitors, and cytotoxic agents.

Radiation therapy also has a role as primary treatment of certain subtypes of melanoma, such as ocular melanoma and lentigo maligna melanoma. More commonly, it has been used as adjuvant and palliative therapy.

Table 16.1. Recommended margins of excision in melanoma.[36,46]

Melanoma thickness (mm)	Radius of excision (cm)
In situ	At least 0.5
<1.0	1
1.1–2.0	1–2
2.1–4.0	2
>4	At least 2

Skin Cancer: Early Detection and Follow-Up

Nationwide campaigns have been established for prevention and awareness of the increasing incidence of skin cancer. These campaigns focus on sun protection, particularly in the first decades of life. Patients who have had a non-melanoma skin cancer are at increased risk of developing a new primary lesion, especially within the first 3 years of diagnosis and treatment of the initial cancer. Patients diagnosed with a BCC have a 44% risk at 3 years of developing a second primary BCC, whereas the risk for an SCC is 18% at 3 years after the diagnosis of the first SCC. The main risk factor for developing subsequent skin cancers is the number of previous NMSC. A doubled 3-year cumulative risk has been reported in patients with three or more prior NMSCs.[31]

Patients with a history of melanoma should also be followed closely for the risk of recurrence and development of a second primary melanoma. Recurrence rates of melanoma depend mostly on the thickness of the primary lesion and have been reported between 3% and 30%. On the other hand, up to 12% of patients diagnosed with melanoma will develop a second primary melanoma.[9]

Given this increased risk, recommendations have been made to follow up patients for skin examinations at least twice a year as well as for education and self-examination, particularly during the first 3 years after diagnosis. Patients with a history of melanoma should have a detailed skin examination initially every 3 months for 2 years, then every 6 months for 3 years, and once yearly thereafter.

Despite the increasing incidence of skin cancer, the overall mortality and survival remain stable, and in some cases, a decreasing tendency is evident. Early detection of skin cancer, particularly melanoma, is of utmost importance for an appropriate management. Key elements for this task include education of the general public about sun exposure and periodic skin examinations, particularly in at-risk populations. Current and novel treatment options hold promise for the treatment of the most common human malignancy.

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17

Esthetic Skin Treatments (Fillers)

Michel E. Pfulg and Serge Lê-Huu

Summary

Injectable soft tissue fillers play an important role in the aesthetic treatment of the ageing face. Staving off wrinkles and folds accounts for the most popular minimally invasive procedures performed.

Since the acceptance of collagen as a filler in 1977, new reabsorbable and non-reabsorbable implants have appeared with varying degrees of success. Most of the early dermal-filling materials, of historical interest, were potentially long lasting, even permanent. Today, as we know more about products and their potential complications, a more accurate treatment plan can be arranged for the patient.

The ideal desired characteristics for a soft tissue filler are that they must be safe, biocompatible, easy to inject, readily prepared, easy to store, affordable, have long lasting cosmetic effect, and not provoke any complications. In this chapter, we go through the history of dermal-filling materials, mentioning the most important biodegradable, semipermanent, and nonbiodegradable fillers. Technical guidelines are given. The conclusion is that today injectable fillers based on hyaluronic acid hold many of the sought-after properties of the ideal filler and please patients' demand for products with little associated risk. Nonbiodegradable fillers can give a definitive correction but involve the risk of severe and permanent adverse reactions.

Introduction

The demand in aesthetic plastic surgery and medicine has grown dramatically in the last 10 years. During this period, the field of cosmetic medicine changed as the demand for noninvasive methods grew substantially.

Injectable soft tissue fillers play an important role in the aesthetic treatment of the ageing face. Staving off wrinkles and folds accounts for the most popular minimally invasive procedures performed. Volume enhancement is now becoming an indispensable component of modern facial rejuvenation as it is well accepted by patients who are not yet inclined to procedures involving surgical lifting. Besides that, it is an appropriate approach for patients who have already undergone a surgical lifting.[1,17,41]

Volume enhancement does require appropriate use of a product. Today more than 35% of the procedures performed by surgeons are no longer surgical. The use of soft tissue fillers responds perfectly to the younger population's expectations, which constitutes a growing part of aesthetic consultations.

Since the acceptance of collagen as a filler in 1977, new reabsorbable and non-reabsorbable implants have appeared with varying degrees of success. The latter group especially has sometimes demonstrated dramatic late complications. The development of these late complications, which were due to a lack of information, technique, and expertise, resulted in doctors and patients

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being unwilling to use these products.[18,36,40,42] European surgeons demonstrated far more expertise than US practitioners in using reabsorbable and non-reabsorbable fillers. The initial product used by surgeons was Zyderm by injection. New products soon appeared on the market, so the accumulated experience is now immense. Today, as we know more about products and their potential complications, a more accurate treatment plan can be arranged for the patient. Ethical and medicolegal issues have to be addressed, so a precise understanding of soft tissue injectable filler substances is compulsory.

History

For many years, physicians and investigators worldwide have been looking for the ideal soft tissue filling material for aesthetic treatment for various areas of the face. One of the earliest agents used for soft tissue augmentation was autologous fat, already used more than 100 years ago. Most of the early dermal-filling materials, of historical interest, were potentially long lasting, even permanent, and were not necessarily native to the intended site. Paraffin, for instance, was used at the turn of the nineteenth century, but it fell into disrepute by the 1920s because of the formation of severe granulation tissue. Nevertheless, its use continued in Asia throughout the 1960s. Pure injectable silicone was used by a relatively small group of physicians with markedly mixed outcomes. The US Food and Drug Administration (FDA), concerned by its effect, banned injectable silicone from cosmetic procedures.[16,32,37]

Finally, practitioners have come to a better understanding of benefits and limitations as well as appropriate application when it comes to injecting filling agents to patients. In addition, factors such as technique, which contributed to untoward events, are now well controlled. This also applies to silicone, as injections show good results with minimal complications when administered by skilled surgeons.[4,49] Zyderm I was the first filling agent approved by the FDA for human treatment. In the early 1970s, a group of investigators from Stanford worked on a potentially useful injectable bovine collagen implant. This later led to the development and approval of Zyderm I (Allergan, Irvine, California) bovine collagen implant in 1977. At this point, claims were that this filler could result in "collagen replacement" with more long-lasting results. Zyderm II was approved in 1983 and Zyderm III in 1985. "Zyderms" are the three types of collagen products derived from bovine dermal collagen suspended in physiologic phosphate-buffered sodium chloride solution and 0.3% lidocaine. Considering they were the first agents introduced in the United States, they were directly used for treatment of facial lines, shallow furrows, and scars, with volume enhancement usually limited to the lip area. Results generally lasted for several months, but poor injection techniques and protocols often showed a shortening in duration. Rare occurrence of severe localized allergic reactions pointed out new issues in the field of agent injection. Skin tests were required and quickly became mandatory as physicians' understanding of collagen reactions increased in accuracy. Other animal protein collagen-like products were then introduced. For instance, porcine-derived collagen and other bovine products (Fibrel) appeared outside the United States.[10,14,19–21,30] The noxious potential of bovine products led to the concept of a nonallergenic human collagen. The first agent commercially available in the United States was Autologen (Autogenesis Technologies, Acton, Massachusetts). At this point, research and development culminated in the ability to surgically extract human dermis with intact collagen fibers for further injection. With autologous dermal tissue matrix, no more skin testing was required, and concerns about allergic inflammation and potential transmitted disease were ruled out. Further thoughts about a readily available injectable human tissue matrix spawned the idea for a cadaver-based allogeneic agent. Dermalogen (Collagenesis, Inc., Beverly, Massachusetts), identical to Autologen in structure and substance, was conceived, but the origin, rather than being autologous, was skin obtained from approved tissue banks. As observed with most injectable products, rare reactions, related to product impurities, occurred in the early stages. However, later complications did appear. In order to address allergenicity issues, CosmoDerm and CosmoPlast (Inamed division of Allergan, Santa Barbara, California) were introduced in 2003. CosmoDerm I, CosmoDerm II, and CosmoPlast were the first approved bioengineered human collagen dermal fillers. According to the manufacturer, those products presented no allergenic

risk and did not require any skin testing before injection[29,39]. The newcomer within the injectable filler spectrum is the hyaluronan family. The concept of using hyaluronic acid in tissue enhancement resulted from years of research done by Balazs and coworkers. Its use was justified by its structural and elastic properties as well as its ability to maintain skin hydration, even partially.[3] Clinically, hyaluronic acid was used as a viscoelastic injectable material for intraocular surgery, to protect delicate structures such as the cornea during instrumentation of the anterior segment. The concept of cross-linking, well known in the collagen industry, was then applied to hyaluronic acid products in an attempt to improve persistence, by fortifying the molecule against enzymatic degradation. In the late 1980s, investigators reported the potential for injectable cross-linked hyaluronan to have a prolonged residence time in tissue and yet showing the same biocompatibilities as hyaluronan.[23] In 1991, Piacquadio initiated a study of cross-linked hyaluronan acid (Hylan B) for tissue enhancement.[34]

Since their introduction, hyaluronans have become the leading filling agent worldwide and have considerably popularized soft tissue enhancement by injection as a highly acceptable procedure for facial rejuvenation. The hyaluronic acids have been long awaited as a solution in terms of longevity and allergenicity, as well as a guarantee for good aesthetic results. For many years, these agents had been used for other nonaesthetic applications and presented a proven track record of biocompatibility with both intraocular and intra-articular uses. The awareness of this substance as a primary component of skin, characterized by its hydrophilic properties, as well as its ability to produce it in a variety of ways, sparked the interest of many. As the long-lasting effect of hyaluronic acid in its nature was known to be dramatically transient in vivo, it was enhanced with a host of chemical manipulations, including cross-linking techniques and concentration optimization. Besides that, improved injection techniques and greater product persistence and versatility have facilitated the high level of treatment outcomes.[5–7,22]

Restylane was the first hyaluronic acid to receive the US FDA approval, years after having been used in Europe. Others were soon to follow. A multitude of hyaluronic acid agents are now currently available worldwide with variations due to their individual characteristics (Hydrafill Soft and Max, Juvederm 18, 24, and 30, Surgiderm, Belotero, Hyaluderm, etc.). This includes source derivation (animal versus bacterial), cross-linking (both chemical method and degree), concentration, amount of free hyaluronic acid (noncross-linked), and particle size/uniformity (structure).[38]

Biodegradable Fillers

Hyaluronan

Hyaluronan (also called hyaluronic acid or hyaluronate) is a nonsulfated glycosaminoglycan distributed widely throughout connective, epithelial, and neural tissues. It is one of the chief components of the extracellular matrix and contributes significantly to cell proliferation and migration. The average 70-kg man has roughly 15 g of hyaluronan in his body, one-third of which is turned over (degraded and synthesized) every day.

Hyaluronan is also a major component of skin, where it is involved in tissue repair. When skin is excessively exposed to UVB rays, it becomes inflamed (sunburn) and the cells in the dermis stop producing as much hyaluronan, increasing the rate of its degradation. Hyaluronan degradation products also accumulate in the skin after UV exposure.

Hyaluronan is naturally found in many tissues of the body, such as skin, cartilage, and the vitreous humor. It is therefore well suited to biomedical applications targeting these tissues. The first hyaluronan biomedical product, Healon, was developed in the 1970s and 1980s, and is approved for use in eye surgery (i.e., corneal transplantation, cataract surgery, glaucoma surgery, and surgery to repair retinal detachment). Other biomedical companies also produce brands of hyaluronan for ophthalmic surgery.

In 2003, the FDA approved hyaluronan injections for filling soft tissue defects under the trade name Restylane.

By its nature, hyaluronic acid retains water like a sponge, absorbing more than 1,000 times its weight. This helps to attract and maintain water within the extracellular space, hydrating the skin and increasing its volume and density. Hyaluronic acid is also involved in the transport of essential nutrients to the skin's viable cells.

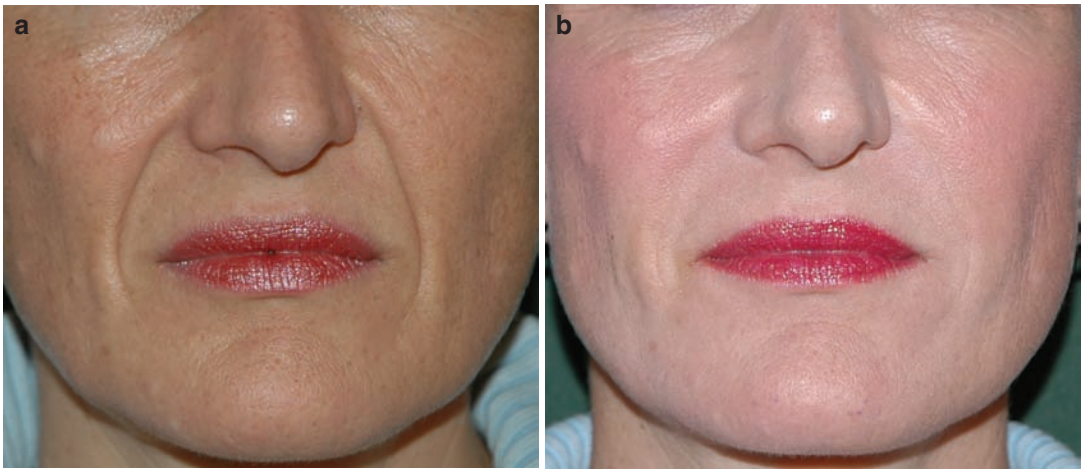

Figure 17.1. Correction of the nasolabial folds with hyaluronic acid (Hydrafill Softline). (Courtesy of Dr. H.P. Frey, Luzern, CH.)

Hyaluronic acid provides volume, helping to contribute to the skin's overall appearance.

Hyaluronic acid can be derived from bacterial or avian sources, and each product has its own specific characteristics.

Cross-linked hyaluronic acid of avian origin became the first noncollagen filler to be widely used. The Hylaform product family is based on hyaluronic acid derived from rooster combs.

Typical examples for bacterial hyaluronic acid products are the Restylane and Juvederm/Hydrafill families. These hyaluronic acid fillers are proven to deliver a longer-lasting effect than traditional bovine collagen.

Bovine collagen is derived from animals, that is cows, and requires an allergy test.

The nonanimal-based fillers can be administered without pretesting. Immediate treatment is therefore available. The viscoelastic properties, stabilizing role, and protective action on cell afforded by hyaluronic acid make it an ideal material with which to fill skin depressions. Very quickly, hyaluronic acid products surpassed collagen to become the new "gold standard" for soft tissue fillers.[7,18,31,38]

Indications

Hyaluronic acid dermal fillers can help to temporarily replace the lost hyaluronic acid and restore the skin's volume and smooth natural appearance. They are indicated for injection into the mid to deep dermis for correction of moderate to severe facial wrinkles and folds (such as nasolabial folds) (Figure 17.1). They are very useful for deeper folds, lips, and irregularities such as soft acne scars, nasal deformities, and areas that require more sculpting. One of the main indications for hyaluronic acid is treatment of the perioral area and augmentation of the lips (Figure 17.2).

Hyaluronic acid should not be injected into the eye contours (eyelids, eye wrinkles). The application in the bags (tear trough deformity) under the eyes is reserved for specialists specifically trained in this technique and having a sound knowledge of the physiology of that particular area.[22] Hyaluronic acid should not be injected into the blood vessels (intravascular) and not be used in patients who tend to develop hypertrophic scarring. It should not be injected in women who are pregnant or breastfeeding and in children. Overcorrection is to be avoided. It should not be injected into areas presenting cutaneous inflammatory and/or infections processes (acne, herpes, etc.).

In association with Botox, hyaluronic acid can produce an excellent rejuvenation effect on the face (Figure 17.3).

It should not be used in association with laser therapy, chemical peeling, or dermal abrasion. For surface peeling, it is recommended not to inject the product if the inflammatory reaction generated is significant.

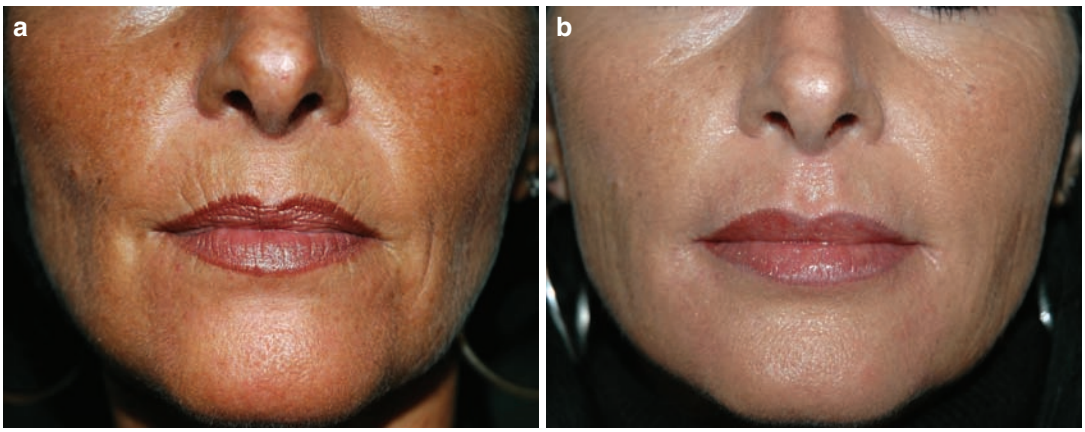

Figure 17.2. Treatment of the perioral area with hyaluronic acid (Hydrafill Softline). (Courtesy of Dr. H.P. Frey.)

Complications

Side effects were usually mild to moderate, lasting 7 days or less, and included temporary injection-site reactions such as redness, pain, firmness, swelling, and bumps.

Collagen

Collagen is the major structural component of the dermis and is responsible for providing strength and support to human skin. It is an essential protein complex found in the human body. Collagen molecules form fibrils that produce necessary fibers for our bodies. The configuration of the fibers is the foundation for tissue formation. Collagen supports the skin, bone, cartilage, and blood vessels in our bodies. The dermal matrix in adult skin is composed of type I (80–85%) and type III collagen (10–15%), in addition to glycosaminoglycans and elastin fibers.

In the 1970s, animal- and human-derived collagens were studied for soft tissue augmentation. Since then, several studies have been performed on bovine- and human-derived collagen, and injectable collagen implants are now recognized as a well-accepted treatment modality for cosmetic purposes. Collagen fillers were one of the first injectable fillers to be offered as an antiaging treatment.[1,6,11,14,18] They are classified as two types:

- Collagen fillers containing human collagen much like the one found in the skin. Manufacturers of this type of collagen fillers include CosmoDerm and CosmoPlast.
- Bovine-based or animal-based collagen fillers. Manufacturers of this type of collagen fillers are Zyderm and Zyplast.

Injected collagen is quite quickly degraded by the body's enzymes.

Bovine collagen tends to have a short life span of up to 3 months, whereas the more expensive human collagen may last up to 6 months. The problem is that the results for either type are totally unpredictable, and some women have been known to reabsorb collagen filler in the lip area in under a month. For that and many other reasons, hyaluronic acid fillers are taking over from collagen as the fillers of choice for many surgeons, as there is less risk of allergy and they are longer lasting.[39]

The greatest risk is that of allergy, but, provided the allergy test is clear, this should not be a problem. Human collagen is less likely to lead to allergy than bovine collagen, which carries a 3–7% risk of this happening.

Indications

Collagen products can be injected into the nasolabial folds, into the vermillion border, and the body of the lip. The melolabial folds and the mental folds can also be increased with collagen. More superficially, it can be used to correct glabellar lines, perioral lines, and other fine lines (crow's feet).

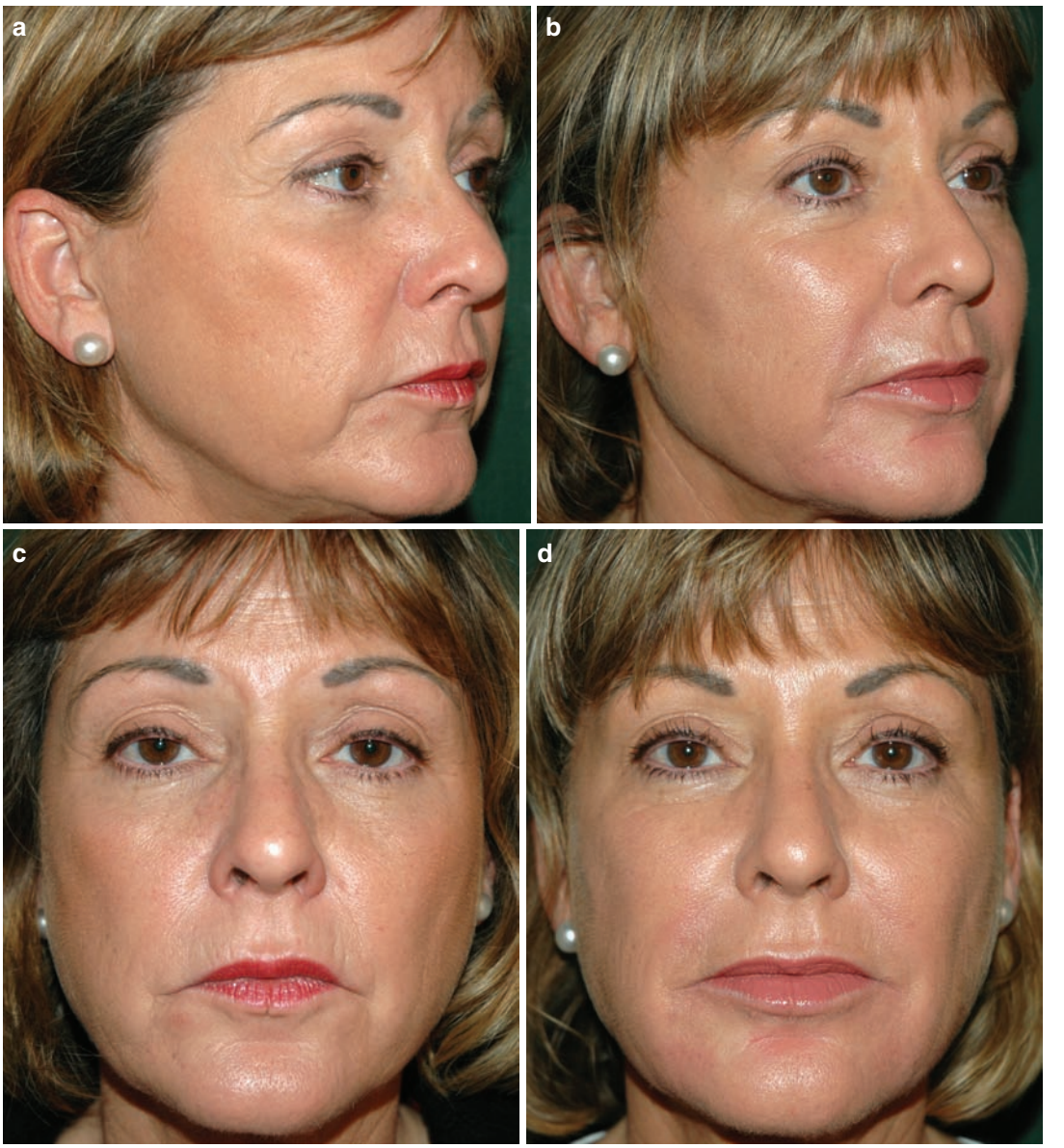

Figure 17.3. Rejuvenation of the face combining Botox and hyaluronic acid injections. (Courtesy of Dr. H.P. Frey.)

Complications

Complications from collagen fillers are typically minimal. Adverse reactions to bovine collagen implants are of two types: nonhypersensitive and hypersensitive. Ecchymosis, bacterial infections, herpes virus infection, beading, development of cysts, local necrosis, and abscess formation are examples of nonhypersensitive-type reactions. Patients with hypersensitivity reactions to bovine collagen may be reassured that it usually resolves within 4–24 months.[33,35]

Semipermanent Fillers

These materials are derived from synthetic or natural means and are used as a trigger mechanism to boost fibroblast and collagen production.

Hyaluronic Acid and Dextranes

A combination of hyaluronic acid Hydroxypropylmethylcellulose and dextranes, marked as Matridex, is thought to be more durable than other products. The combination of Dextranomer microparticles with hyaluronic acid is highly biocompatible. Products consisting of the above combination have been used for a long time for wound healing and as a bulking agent in the treatment of urinary incontinence (Urodex).

Investigations show that Dextranomere implants with a positively charged surface stimulate the formation of soft tissue in the skin. The main component of matridex is dextranomeres, which are cross-linked Dextran molecules. The Dextranomeres in Matridex are microparticles with a positively charged surface and a diameter of 80–120 µm (DEAE Sephadex 25).

There is an immediate augmentation of the wrinkles because of the combination with Hylan Gel copolymer and excellent long-term results by stimulating new collagen and regeneration of the dermis by microspheres.[8,9]

Indications

Matridex is used for the treatment of face wrinkles and folds (glabella folds, lip contour, lip augmentation, oral commissures, fine lines, perioral lines, periorbital lines, nasolabial folds) and for contour correction

Complications

There should be, according to the manufacturer, no long-term inflammations or irritations after the injection of Matridex.

Calcium Hydroxylapatite (Radiesse, Radiance)

Calcium hydroxylapatite is composed of a suspension of 30% synthetic calcium hydroxylapatite microsphere of 25–45 µm in diameter, suspended in a 70% gel consisting of 36.6% sterile water, 1.3% sodium carboxymethyl cellulose, and 6.4% glycerin. The calcium hydroxylapatite microspheres present the same chemical composition as the inorganic constituent of teeth and bone. After 1 month, fibrin and scant cellular tissue surround the microspheres that act as a scaffold for new tissue formation. At 3 months, macrophages, fibrin, and fibroblasts form a capsule around the microspheres. After 9 months, the calcium hydroxylapatite is absorbed and the microspheres lose their forms. The clinical effect of calcium hydroxylapatite may last 9–18 months. No skin testing is required before use as calcium hydroxylapatite is immunologically inert. This product is FDA approved for use in oromaxillofacial defects and laryngeal and vocal fold augmentation. The use of calcium hydroxylapatite as a soft tissue facial filler is off-label.[2]

Indications

Calcium hydroxylapatite has been used for soft tissue filling of nasolabial folds, facial lipodystrophy, wrinkles, glabellar lines, acne scars, and liposuction contour defects.

Complications

Application in lip augmentation remains controversial as a high rate of nodule formation (up to 50%) was observed. The main risk factor for nodule formation is the excessive volume injection in a mobile zone such as the lips.

Poly-L-Lactic Acid (Sculptra, New-Fill)

Poly-L-lactic acid (PLLA) is a biodegradable, synthetic polymer of L-lactic acid, which has been used in a variety of human medical applications for over 40 years (poly-L-lactic acid has been safely used for many years in different medical devices as reabsorbable plates, screws, and suture materials). It is approved for the treatment of HIV-associated facial lipoatrophy.[46] This filler consists of poly-L-lactic acid microspheres (1–63 µm in diameter), mannitol, and sodium carboxymethylcellulose, completed with sterile water for injection. Poly-L-lactic acid stimulates ingrowths of type I collagen, with a long-term tissue filling effect. The poly-L-lactic acid microspheres are progressively metabolized to carbon dioxide and water and are then replaced by ingrowths of type I collagen. Nine months after implantation, no polymer or remnant cicatricial fibrosis can be detected

histologically, demonstrating good biocompatibility of the poly-L-lactic acid microspheres, and this causes volumetric expansion with the passage of time. Unlike other dermal fillers that are intended to correct discrete facial wrinkles or folds, poly-L-lactic acid provides volumetric expansion of volume-deficient areas. The effect of poly-L-lactic acid may last 2 years and no skin testing is required before use, as it contains no animal proteins.[43,44,47]

Indications

This product, which has been used in Europe for many years, is FDA approved in the United States since 2004 as a soft tissue filler for lipoatrophy of cheeks and for HIV patients who are under highly active antiviral therapy.

Further off-label uses are for temples, upper zygoma, nasolabial and malar regions, periorbital and preauricular regions, and for the jaw line. It is injected into the deep dermis or subcutaneous layer using a 26-ga needle with a tunneling technique, and massage of the product is recommended after injection. No overcorrection is required, but most patients will require a series of three to four injections spaced 4 to 6 weeks apart.[47]

Complications

Localized ecchymosis and edema can occur at the site of injection.

Late reactions include subcutaneous nodule formation, lasting up to 2 years after injection, and granulomatous formation at 9–14 months has been described.[13,15]

Nonbiodegradable Fillers

Injectable nonbiodegradable fillers can also be used for aesthetic treatment of the ageing face. The advantage of these products is longevity. However, there are certain undesirable effects that can occur immediately or after a significant period of time, such as granulomatous formation, migration, or late allergic reaction. The most common subacute or late reaction to permanent fillers is granulomatous development (Figure 17.4). Treatment of an adverse reaction to a nonbiodegradable filler material is therefore much more difficult than that to reabsorbable products, because it will provide a permanent stimulus for the surrounding tissue.[28,48]

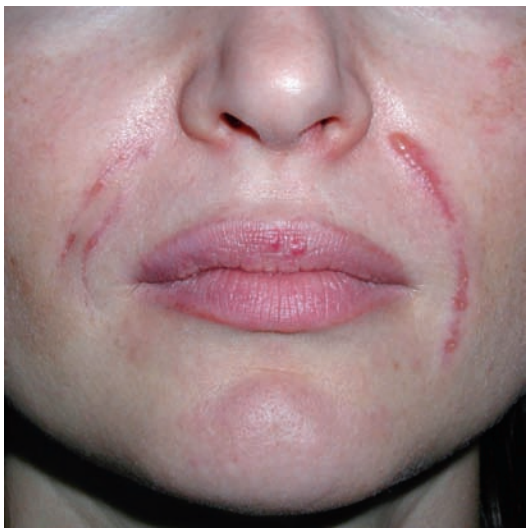

Figure 17.4. Granulomatous reactions in the nasolabial folds and cheeks following Dermalive (hyaluronic acid and acrylic hydrogel) injections.

Polymethylmethacrylate and Collagen (Artefill)

Artefill is composed of polymethylmethacrylate microspheres (20 vol %) in a suspension of denatured 3.5% bovine collagen (80 vol %). It is the successor product to Artecoll introduced at the end of the 1980s but with a smaller sphere size that measures 30–42 μm in diameter with a smooth and round surface. These properties permit encapsulation by the patient's own collagen fibers, preventing dislocation after bovine-collagen degradation within 1–3 months. The polymethylmethacrylate microspheres are nonbiodegradable and too large to be phagocytosed by macrophages. They act as a matrix for the host of fibroblasts that progressively replace the bovine collagen and stimulate tissue ingrowth to bring volume to fill the wrinkle. Artefill brings not only volume but it also stimulates the patient's own collagen production around the microspheres. This material is injected deeply into the lower third of the dermis, the reticular dermis, using a 26-ga needle with a tunneling technique, moving the needle back during the injection. The needle should never be visible.[12,27]

Indications

Artefill can be used to treat the glabellar lines, nasolabial folds, radial upper lip lines, and the corner of the mouth. It brings a long-lasting result.

Complications

Adverse events with Artefill are much less important than those with Arteplast (2.5% foreign body granulomas) or Artecoll, as the diameter of the microspheres has diminished and the purification and washing technique was introduced.[24–26]

The effects of Artefill are stable for at least 4–5 years with a low late adverse events rate. Allergy testing is required to minimize the risk of hypersensitivity reactions with the bovine collagen. In rare cases, foreign body reaction (granuloma) to Artefill can occur, 6 months to 2 years after treatment. They are first treated by intralesional injection with corticosteroids or surgical excision if there is no response. Small white granules can be visible if the filler is injected too superficially.

Polyacrylamide (Aquamid, Amazingel, Argiform, Bioformacryl, Evolution, Outline, etc.)

Polyacrylamide is composed of 97.5% water and 2.5% cross-linked polyacrylamide hydrogel. At first, a fibrocellular capsule surrounds the acrylamide gel without foreign body reaction. The capsule gets thicker with fibroblasts and macrophage accumulation. The chemical properties of polyacrylamide with a high proportion of water cause fewer foreign body reactions. The noncovalent bonds of the molecular structure and the high level of water result in a smooth surface, preventing phagocytosis by mononuclear cells and macrophages. However, after injection of small quantities of aquamid (0.1 cc), the product is absorbed within 9 months in human skin.

Indications

This product is the first choice for facial soft tissue augmentation, such as cheek, chin, or mandibular augmentation. Polyacrylamide adds volume to the subcutaneous tissue, thereby restoring or augmenting facial and body contours. It is also used for lip augmentation, nasolabial folds, perioral wrinkles, glabellar frown lines, and depressed mouth corners. This product must be injected deeply in the subcutaneous tissues, in a retrograde fashion.

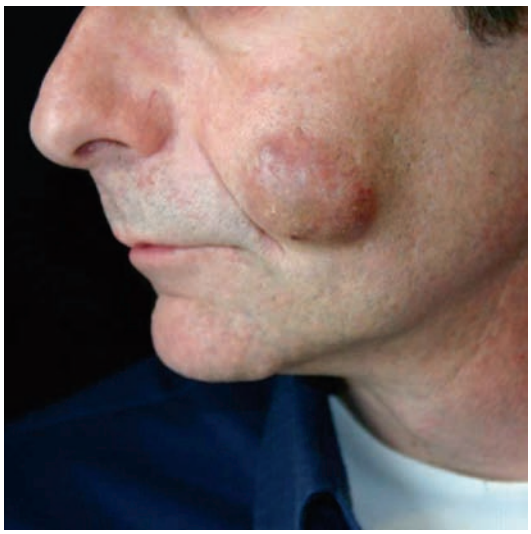

Figure 17.5. Abscess formation after injection of Aquamid (hydrogel composed of 97.5% sterile water bound to 2.5% cross-linked polymers) in the left cheek.

Complications

Local events like hematomas, edema, itching, changes in skin pigmentation, or moderate pain have been observed. Gel accumulation with nodule formation is also described. Infections (Figure 17.5), granulomata, and migration are uncommon but reported[48].

Silicone (Adato Sil 5000, Bioplastique, Biopolimero, Dermagen, Silikon 1000, Silicex)

Injectable silicone is one of the oldest injectable filler materials used. It is a synthetic polymer containing elemental silicon (dimethylpolysiloxane). It appears to fulfill most of the criteria for being the ideal implantable substance: permanent, stable, and minimally antigenic. However, this product is very "controversial" as it tended in the past to migrate, harden, and cause inflammation and skin necrosis. This is caused by a lack of standardization and wrong indications in

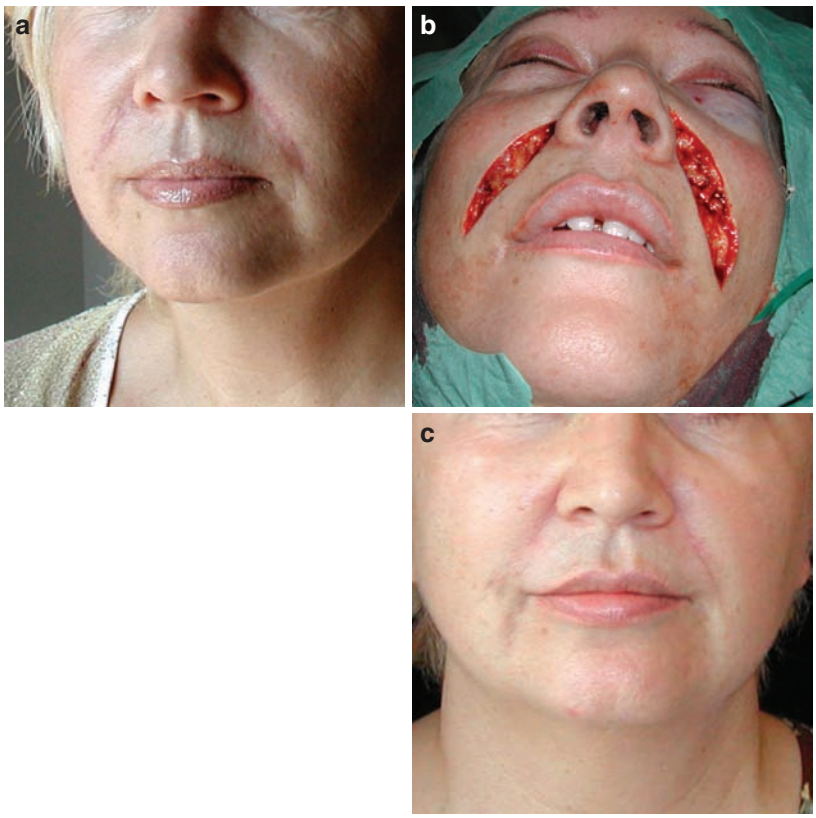

Figure 17.6. Surgical excision of the nasolabial folds following a chronic long-lasting granulomatous reaction after injection of Dermalive. (**a**) Presurgery; (**b**) during surgery; (**c**) postsurgery.

the use of silicone, with large volumes injected and poor injection technique[4,16,32].

Indications

Silicone must be used in very small quantities (0.01 ml) with a micro droplet method. Tiny droplets of silicone are deposited in the deep dermal or subdermal layer by a series of injections spaced 3–10 mm apart. Overcorrection must be avoided.

Nasolabial folds, marionette lines, glabella, tear troughs, cheek hollows, chin, lips, and cheek bones can be treated with injectable silicone.

Liquid silicone (Adato Sil 5000, Silikon 1000) is approved by the US FDA for use in the eye for retinal detachment. Use in soft tissue augmentation indication is "off-label."

Complications

Chronic cellulitis, foreign body reactions, extrusion, ulceration, nodules formation, and migration of material can occur many years after silicon injection. When these complications are observed, the injected silicone must be removed by surgical resection (Figure 17.6).

Technical Guidelines

The demand for fillers has grown dramatically in the last 10 years because of longevity of life (ageing population) and the influence of the media. Combinations of fillers may achieve maximum benefit. The degree and duration of the correction depend on the product and on the character of the defect treated, the tissue stress at the implant site, the depth of the implant in the tissue, and the injection technique. Contraindications are autoimmune diseases, hypersensitivity to a component of the product, pregnancy or breast feeding, a wrong indication (location), a flawed or faulty technique, herpes facialis, bleeding factors (salicylate, plavix,

sintrom, vitamin E, herbs, anti-inflammatory drugs), or a Koebner phenomenon (psoriasis, pyoderma gangrenosum).

Minor complications can occur after injection of any filler. Common side effects following treatment are bruising, erythema, and edema. If the product is injected too superficially or in excessive quantity, small nodules can occur. Acute infections are treated by antibiotics focused on skin infection. Granulomatous reactions may also happen, which is a generic immune response against a foreign body. Treatment of granulomas, if they appear, involves the use of topical steroids or systemic steroids. If there is no response after steroid injection, 5-fluorouracil can be added to the initial product. If there is still no sign of improvement, a surgical procedure should be considered (Figure 17.6b).

Conclusion

All fillers result in dramatic improvements if used correctly and precisely. Thanks to a better understanding of facial ageing, specifically soft tissue deflation noted between youth and middle age, the indications for injectable filler agents have significantly evolved. Injectable fillers are a common option to postpone the surgical procedure. With the improvement of products and techniques, results using fillers have become more consistent, thereby increasing patient and physician satisfaction. However, as a great variety of injectable fillers is available, especially in Europe and South America, it can be sometimes difficult to choose the right filler for the right indication. No agent meets all the criteria of the "ideal filler," and selection should be based on anatomical parameters and practitioner preference. It is important to note that the results achieved with each filler are dependent on a learning curve, and some fillers are more forgiving than others. Many of the new fillers available are longer lasting and have shifted the paradigm between permanent or nonpermanent fillers. Use of permanent fillers allows less room for error as it can produce irreversible changes in facial shape that may not retain the aesthetic modifications as the patient ages. With the introduction of hyaluronic acid derivatives in soft tissue augmentation, a safer, longer-lasting, and yet temporary alternative has been made available.

Physicians dealing with fillers have to gain expertise in choosing the best possible product for the correction of lines, folds, defects or scars, wrinkles, and for tissue augmentation. Many national health authorities and academic societies encourage the use of biodegradable instead of nonbiodegradable injectable facial implants[45]. Nonbiodegradable fillers can give a definitive correction but involve the risk of severe and permanent adverse reactions.

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Part IV

Head and Neck

18

Head and Neck Embryology and Anatomy

Arunesh Gupta, Gopal Malhotra, Oladimeji Akadiri, and Ian T. Jackson

Summary

In the first section of this chapter, a brief account of the embryogenesis of the human head and neck is presented. The discussions are focused on the formation of the branchial apparatus and their derivatives and development of the tongue, thyroid gland, palate, and face. In the second section, a concise survey of the anatomy of the head and neck is provided with an attempt to simplify the description of this complex anatomical region. The region is discussed under two subsections, the "Hard Tissue Framework" and the "Soft Tissue Envelope" to describe the skeletal anatomy and the connective tissues, nerves, and vascular anatomy of the region. Some important clinical considerations in plastic and reconstructive surgery are highlighted.

The head and neck region is perhaps the most complicated anatomical region of the human body because of its complex and delicate anatomical architecture. It also has significant impact on the mental, social, and emotional disposition and overall self-image of the individual. It is probably the region of the body for which reconstructive surgery is most required. A plastic surgeon may be confronted with significant congenital defects in the head and neck region or reconstruction of complex traumatic facial problems. For this, it is essential that he/she has adequate knowledge of the regional anatomy and embryology. No matter how artistically it is designed and sculpted, surgery based on insufficient knowledge will ultimately result in a poor aesthetic result and a patient who is anatomically as well as functionally compromised.

Embryology of the Head and Neck

Tissues required for development of the head region are contributed as follows –

(A) Mesenchymal tissue arises from the following:

1. Paraxial mesoderm.
   (a) Calvarium by forming the parietal, occipital, and petrous temporal bone
   (b) All voluntary muscles of the craniofacial region
   (c) Connective tissue and dermis in the dorsal calvarium
   (d) Meninges distal to the forebrain
2. Lateral plate mesoderm forms the arytenoids and cricoid cartilages with their connective tissue.
3. Neural crest cells from the brain migrate into the pharyngeal arches and facial region to form their skeletal structure together with other tissues.

M.Z. Siemionow and M. Eisenmann-Klein (eds.), *Plastic and Reconstructive Surgery*, Springer Specialist Surgery Series,

(B) Ectoderm is derived from ectodermal placodes. Cells from neural crest and ectodermal placodes form the neurons of the fifth, seventh, ninth, and tenth cranial sensory ganglia.

Branchial Apparatus Development and Derivatives

The derivatives of the branchial apparatus provide a major contribution to the development of the head and neck. The term *branchial* is used for the cranial region of an early embryo as it resembles a fish embryo at comparable stage. The branchial apparatus consists of branchial arches, pharyngeal pouches, branchial grooves, and branchial membrane (Figure 18.1).

Branchial arches appear early in the fourth week as ridges of mesenchymal tissue on the future head and neck regions. The branchial arches are separated from each other externally by branchial grooves and are numbered in a craniocaudal sequence. Each branchial arch is lined on the outside by ectoderm and on the inside by endoderm with a central core of mesenchymal tissue, which receives a substantial number of neural crest cells.

The neural crest element of each arch gives rise to the skeleton of the face (Figure 18.2), and the mesenchymal portion is the origin of the muscular component (Figure 18.3). This muscular component has its own cranial nerve, and wherever these muscle cells go, they carry their original nerve supply with them (Figure 18.4). Tables 18.1 through 18.3 detail the various structures derived from the branchial apparatus.

Clinical Correlates

1. Branchial Malformations:

Congenital malformations of the head and neck region mostly represent the remnants of the branchial apparatus that normally disappear as these structures develop.

These include malformations such as the following:

Congenital auricular sinuses and cysts – These are remnants of the first branchial groove commonly found in a triangular area anterior to the ear.

Branchial sinus – Mostly, these occur due to failure of the second branchial groove to obliterate. Typically, they are external and open

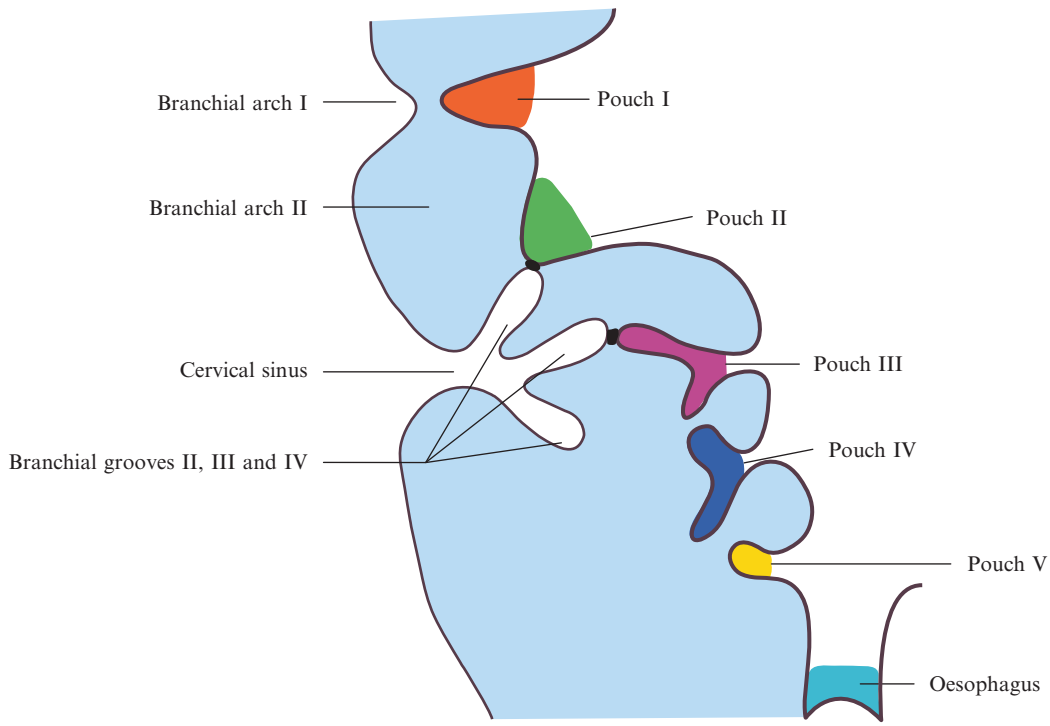

Figure 18.1. Relationship between branchial arches and pouches.

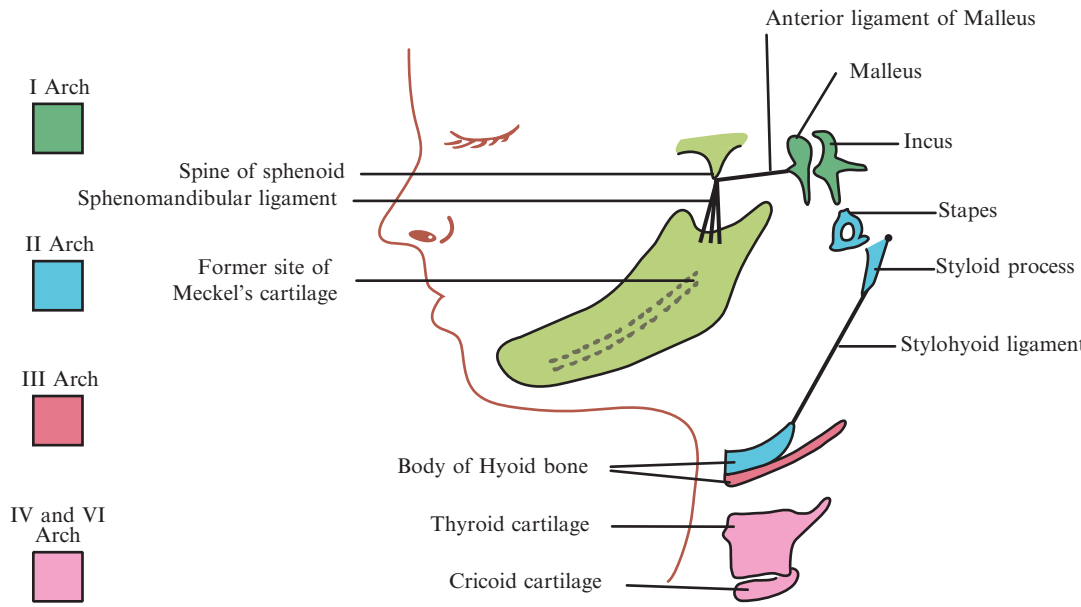

Figure 18.2. Adult derivatives of branchial arch cartilages.

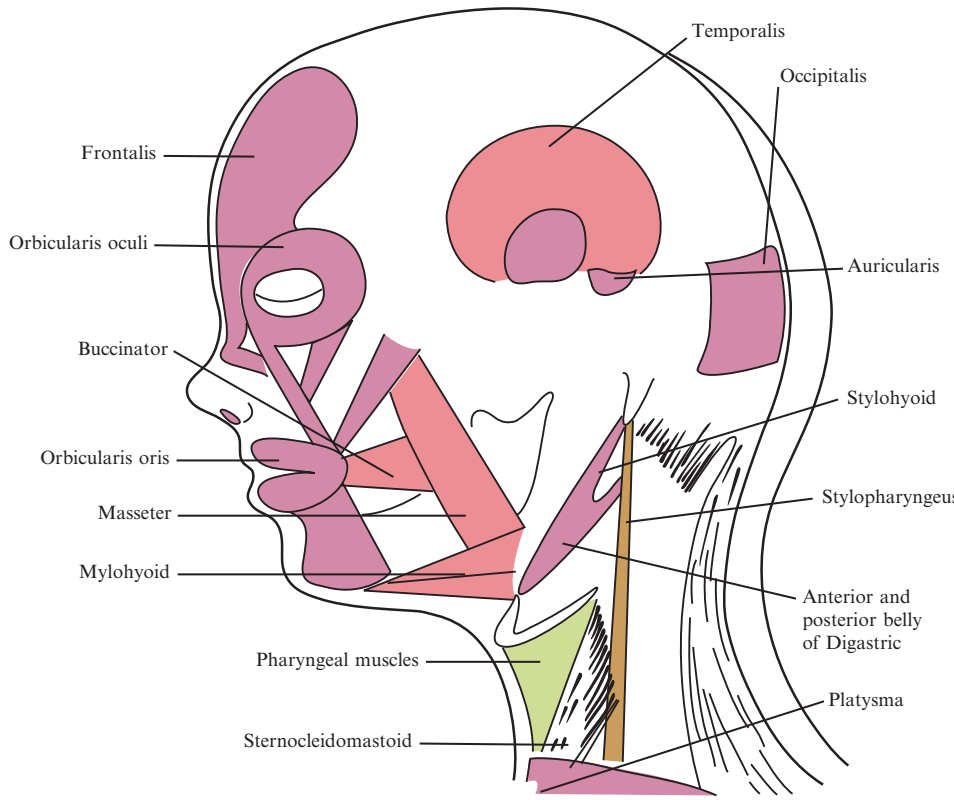

Figure 18.3. Adult derivatives of branchial arch muscles.

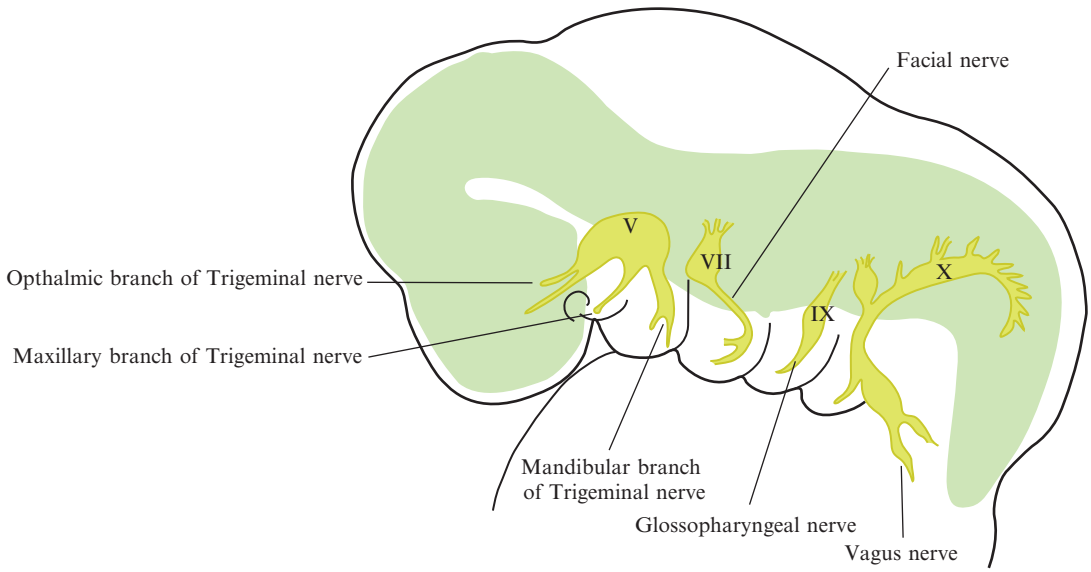

Figure 18.4. Nerve supply of the pharyngeal arches.

Table 18.1. Branchial arches.

Pharyngeal arch	Nerve	Muscles	Skeleton	Blood supply
I or Mandibular	V. Trigeminal: maxillary and mandibular divisions	Mastication (temporal; masseter; medial, lateral pterygoids); mylohyoid; anterior belly of digastrics; tensor palatine, tensor tympani	Premaxilla, maxilla, zygomatic bone, part of temporal bone, Meckel's cartilage, mandible, malleus, incus, anterior ligament of malleus, sphenomandibular ligament	Maxillary
II or Hyoid	VII. Facial	Muscles of facial expression; posterior belly of digastrics; stylohyoid; stapedius	Stapes; styloid process; stylohyoid ligament; lesser horn and upper portion of body of hyoid bone	Stapedial degenerates
III	IX. Glossopharyngeal	Stylopharyngeus; superior and middle constrictors	Greater horn and rest of hyoid bone	Common and internal carotid
IV	Superior laryngeal	Inferior constrictor	Thyroid and cuneiform cartilage	Left aorta and right subclavian
V/VI	Recurrent laryngeal	Trachea, intrinsic laryngeal muscles except cricothyroid muscle	Cricoid; arytenoids, and corniculate cart	Ductus arteriosus and pulmonary artery (L)

Table 18.2. Branchial pouches.

Pouches	Structures
I	Eustachian tube, middle ear (mastoid air cells), medial tympanic membrane
II	Supratonsillar fossa, palatine tonsils, middle ear
III	Epithelial reticulum of thymus, inferior parathyroids
IV	Thyroid parafollicular cells (C cells), superior parathyroids

Table 18.3. Branchial clefts.

Clefts	Structures
I	External auditory canal, outer tympanic membrane
II–V	Obliterates

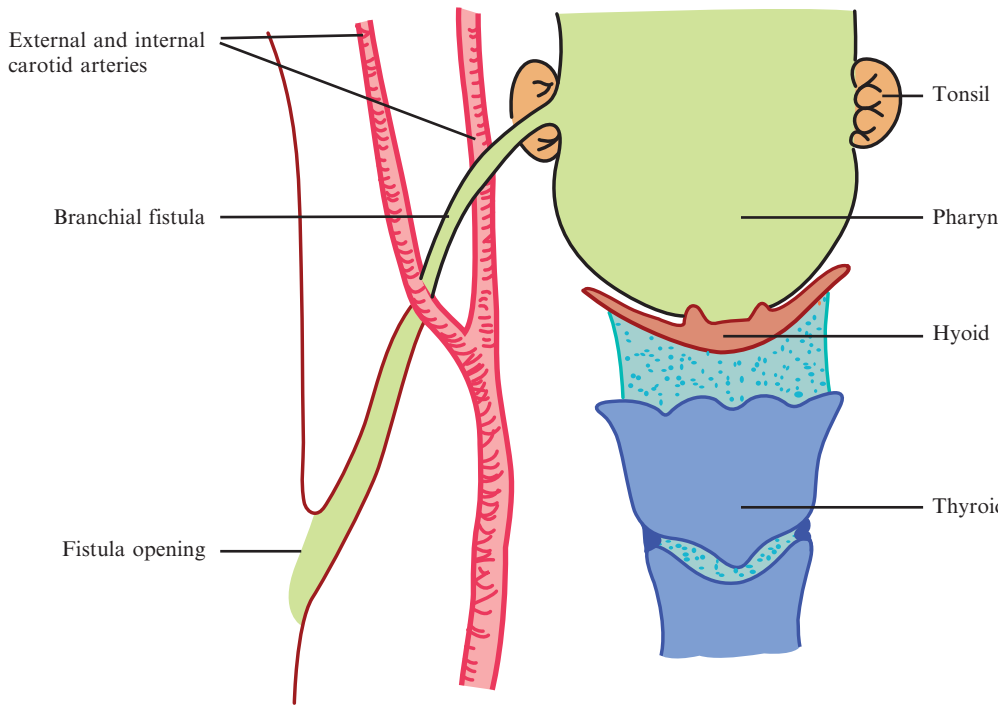

Figure 18.5. Branchial fistula tract.

on the anterior border of sternocleidomastoid muscle in the inferior third of neck. Internal branchial sinuses are uncommon, and if present, they open in the tonsillar fossa.

Branchial fistula – This results from the persistence of parts of the second branchial groove and second pharyngeal pouch (Figure 18.5).

Branchial cysts – The remnants of parts of the cervical sinus, the second pharyngeal groove, or both may persist and form a cyst.

Branchial vestiges – These are persisting parts of the pharyngeal cartilages on the side of the neck, usually found anterior to the inferior third of the sternocleidomastoid muscle.

2. First Arch Syndrome:

This is due to deficient migration of neural crest cells into the first arch during the fourth week. Treacher Collins and Pierre Robin are examples of this syndrome.

Development of Thyroid and Tongue

Thyroid: The thyroid gland appears during the fourth week in the floor of the pharynx and descends to its final position in front of the trachea by the seventh week. During its descent, it maintains its connection to the tongue by a narrow canal thyroglossal duct, which is normally obliterated by the seventh week. Remnants of the thyroglossal duct can form a cyst or a fistula. Aberrant thyroid tissue can be found anywhere along the course of its descent.

Tongue: Development starts around the fourth week from the following structures (Figures 18.6 and 18.7).

1. The anterior two-thirds of the tongue develops from two lateral swellings and one medial swelling that originates from the first pharyngeal arch. Because the mucosa covering this part arises from the first arch, the sensory nerve supply is by the mandibular branch of the trigeminal nerve.
2. The posterior one-third or root of the tongue develops from the second median swelling formed by the mesoderm of the second, third, and part of the fourth arches. This is innervated by the glossopharyngeal and vagus nerves.

The tongue muscles are derived from occipital somites and are innervated by the hypoglossal nerve.

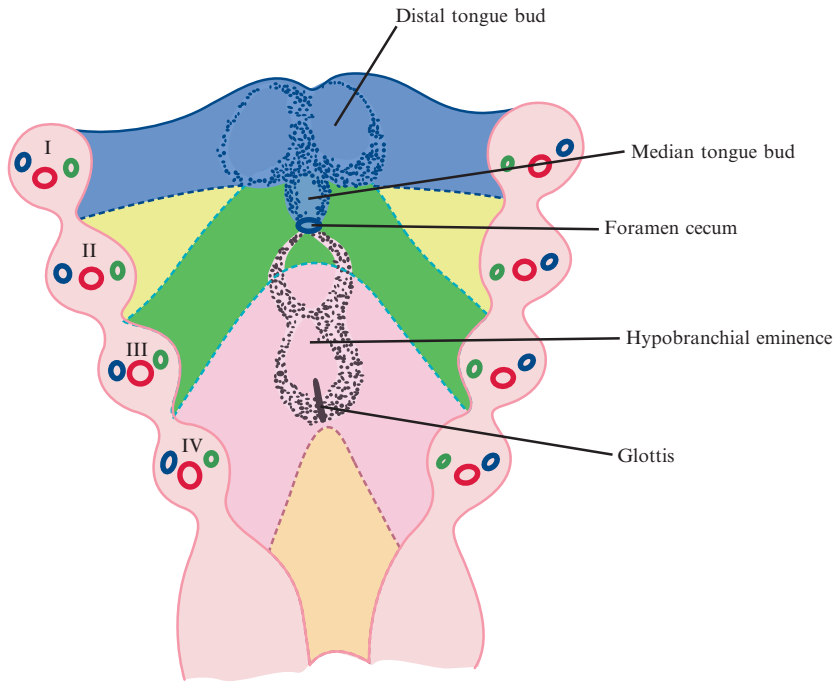

Figure 18.6. Development of tongue showing pharyngeal arch derivatives.

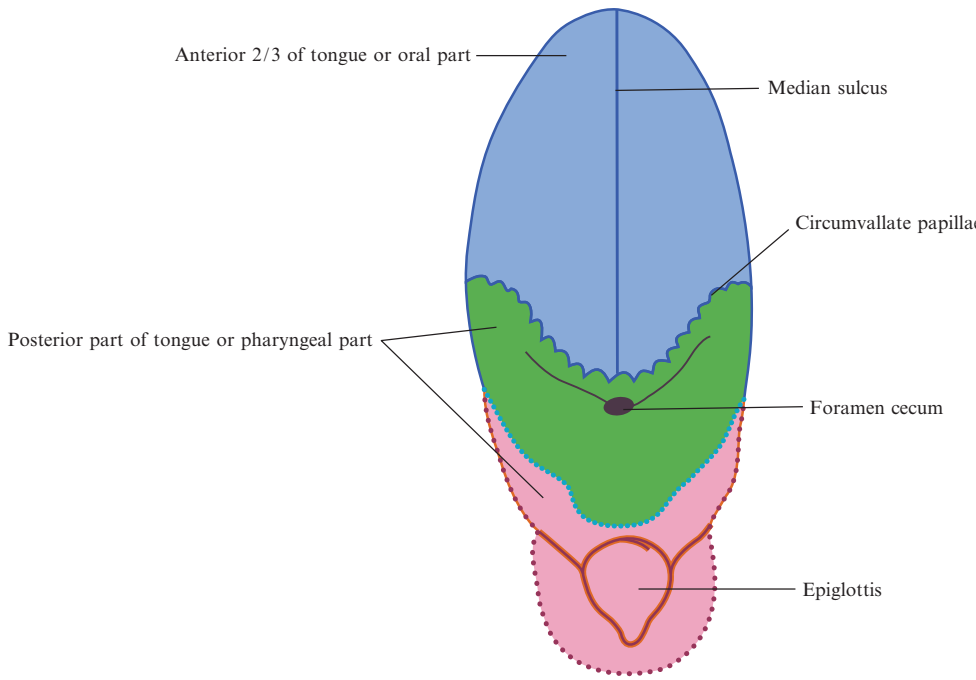

Figure 18.7. Adult tongue, branchial arch derivatives.

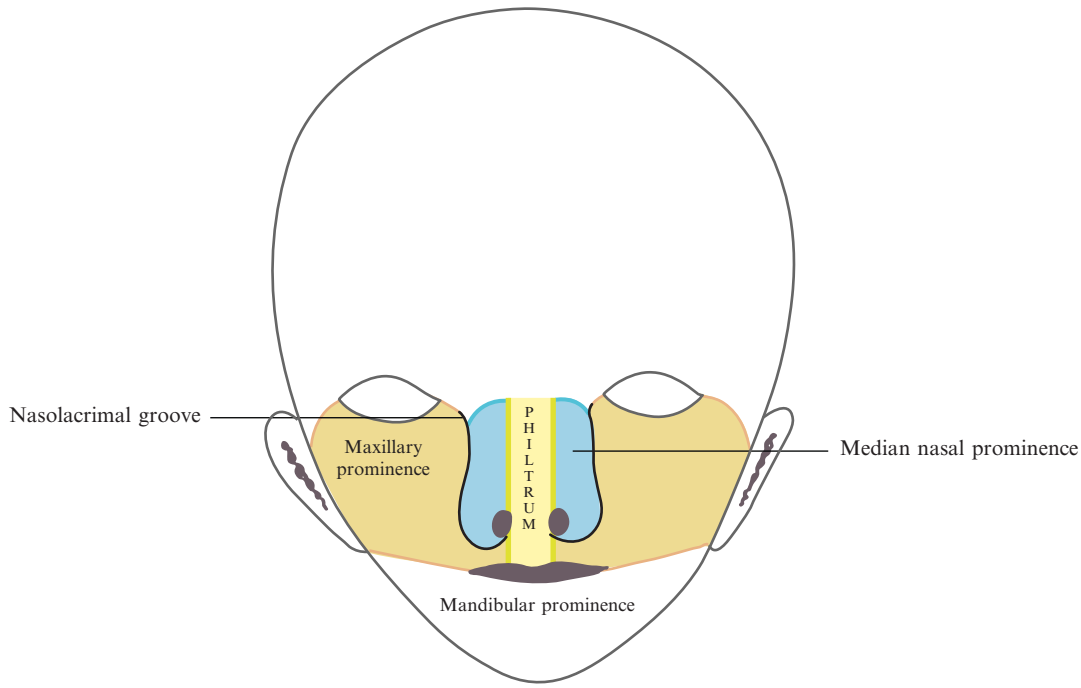

Figure 18.8. Embryo face at 10 weeks.

Table 18.4. Development of the face.

Primordia	Part of face
Frontonasal (single)	Forehead, bridge of nose, and medial and lateral nasal prominence
Maxillary (paired)	Cheeks, lateral portion of upper lip
Medial nasal (paired)	Philtrum of upper lip, crest, and tip of nose
Lateral nasal (paired)	Alae of nose
Mandibular (paired)	Lower lip

Development of the Face

The face is formed by the five primordia (frontonasal prominences and paired maxillary and mandibular prominences) that appear around the stomodeum early in the fourth week (Figure 18.8 and Table 18.4). A recognizable human face develops by the eighth week, and from there onward, facial proportions develop.

Development of the Palate

Palatal development begins at the end of the fifth week and is completed by the 12th week.

The palate develops from the primary and secondary palates. The primary palate develops from the intermaxillary segment. The secondary palate is formed by the fusion of two internal shelve-like outgrowths from the maxillary prominences called the palatine shelves (Figure 18.9). Fusion of the palatine shelves begins from the anterior to the posterior. At the same time as the palatine shelves coalesce, the primary palate and nasal septum also fuse with the secondary palate (Figure 18.10). The incisive foramen marks the midline fusion point of the primary and secondary palates.

Clinical Correlates

Cleft lips and cleft palates are common malformations that result in an abnormal facial appearance and speech problems. Although often associated, cleft lips and cleft palates have different etiology and malformation sequences.

Clefts anterior to the incisive foramen are caused by lack of partial or complete fusion between the maxillary prominence and the medial nasal prominence on one or both sides. Clefts posterior to the incisive foramen result from failure of fusion of the palatine shelves.

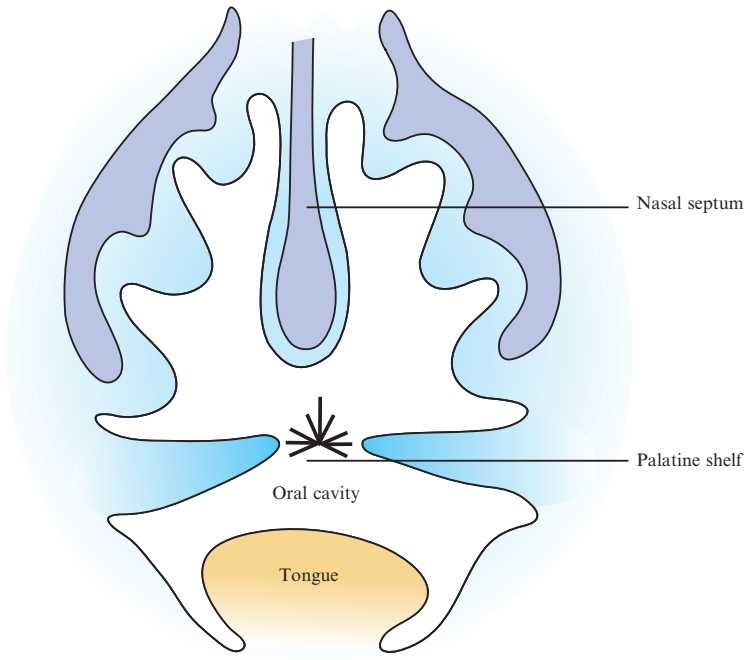

Figure 18.9. Coronal section showing formation of palate.

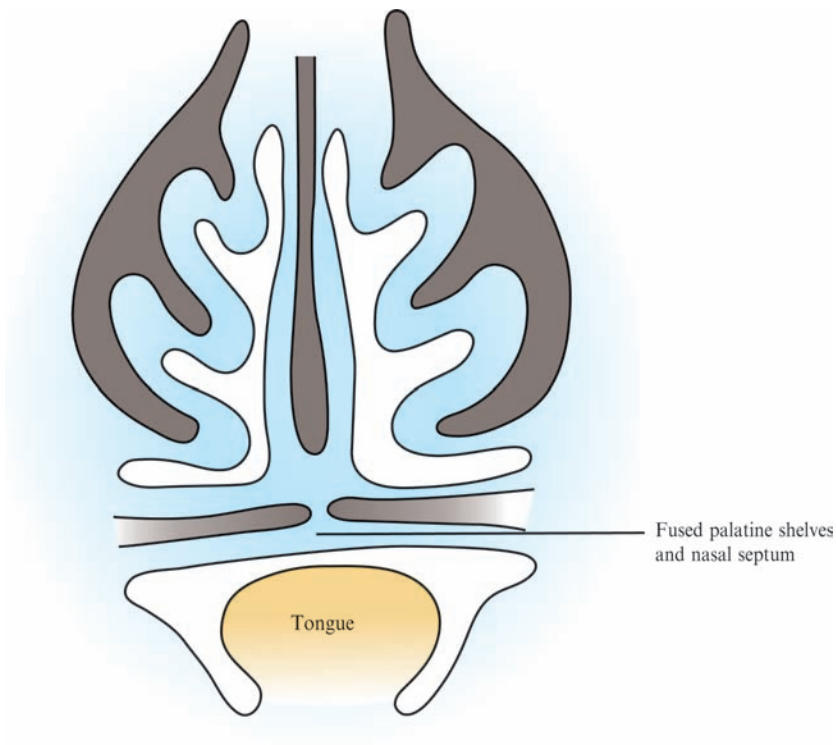

Figure 18.10. Fused palatine shelves and nasal septum.

Finally, clefts may occur in a combination of both anterior and posterior fusion defects.

Other rare facial clefts include the following:

1. Oblique facial cleft – The maxillary prominence does not merge with its corresponding lateral prominence, leading to exposure of the nasolacrimal duct to the surface. It is often bilateral and extends from the upper lip to the medial margin of the eye.
2. Transverse facial cleft – This extends from the mouth to the ear.
3. Median cleft of the lower lip and mandible – This is caused by the failure of the mandibular prominences of the first arch to fuse in the midline.
4. Bifid nose – This is caused by the failure of the medial nasal prominences to merge.

Anatomy of the Head and Neck

The development and morphology of the human body are largely determined in utero as described in the preceding section. This pattern can be modified to some extent during the growth period and sometimes even afterward. Whatever modification is desired, the plastic surgeon must have at the back of his mind the various anatomical landmarks and indices upon which any intervention must be based. He must also be aware of variations that exist based on race, sex, and age.

The head must be viewed from the frontal, lateral, and anteroposterior aspects. In doing this, certain facts typical of human anatomical contours must be borne in mind. This includes facial height, width, and symmetry and cranial size, shape, and contour. In the frontal view, the face height is divisible into three equal thirds; hairline to the glabella, glabella to the subnasale, and subnasale to the menton. The width-to-height ratio of 3:4 is fairly typical. The horizontal width of the nose at its ala bases should correspond approximately to the distance between the medial canthi, the width of one eye, and it also equals one fifth of the widest diameter of the face.[7,15]

From the lateral perspective, the general profile of all faces is one of three types – the straight, the convex, or the concave.[2] These are a few important anatomical considerations in aesthetic facial surgery; more pertinent information can be derived from appropriate soft tissues and hard tissue cephalometric analysis of the skull and jaws.

The neck is simply designed as a nearly cylindrical part of the human body connecting the head to the thorax. From a gross anatomical perspective, the differential proportion of the neck relative to the head is perhaps the most important consideration.

For the purpose of the current discussion, we review the basic anatomy of the head and neck under two headings: "Hard Tissue Framework" and "Soft Tissue Envelope."

Hard Tissue Framework

The overall size, shape, and contour of the head are a reflection of the hard tissue framework, whereas the soft tissue of the neck conceals the skeletal anatomy to a large extent. The framework of the head consists of the *Cranium* and the *facial skeleton*, whereas the *neck* is made up of an axial arrangement of cervical vertebrae joined to one another at intervertebral joints and to the cranium at the atlanto-occipital joints. In the posterior aspect of the neck, the spinous process of the seventh cervical vertebra is prominent and palpable under the skin.

The Cranium

The *cranial vault* (Figure 18.11) consists of bones derived from intramembranous ossification viz the frontal bone, the paired parietal bones, and the occipital bone. On the lateral aspect, the temporal bone lies posterior to the greater wing of the sphenoid bone bilaterally. The bones are united at immobile joints called sutures reinforced by thick fibrous connective tissues – the sutural ligaments.[14] During infancy and early childhood, these joints are mobile and can be molded to alter the shape and contour of the head. The *cranial base* on the other hand consists of a complex architecture of endochondrally ossified bones with foramina of various sizes between them. These foramina transmit delicate vascular and neural structures between the intracranial and extracranial compartments. The bones of the cranial base include the body, lesser and part of the greater wing of the sphenoid bone, petrous part of the temporal bone, and the basiocciput. The clivus is the central posteroinferiorly inclined platform on which the bones of the mid-facial skeleton abut. Architecturally, the bones of the cranium are made up of an external and internal table of

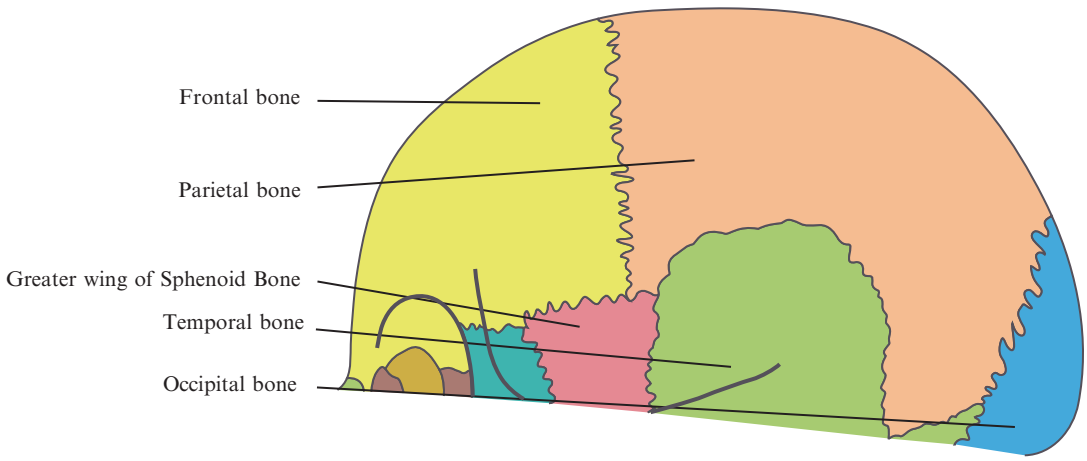

Figure 18.11. The cranial vault.

compact bone separated by a layer of coarse spongy bone called diploe. The inner table is thinner and more brittle. There is periosteum on both sides of the cranial bones but the inner periosteum is fused with the dura. The cranial cavity contains the brain and its surrounding meninges, portions of the cranial nerves, arteries, veins, and venous sinuses. The bone of the cranial vault is a versatile resource for calvarial bone grafts for reconstructive purposes in the head and neck region.[5]

The Facial Skeleton

The *facial skeleton* can be divided into the cranial third (upper third), middle third, and the mandible (lower thirds) (Figure 18.12) to provide a simple basis for anatomical and clinical study of this region. The *orbital cavity* is formed between the cranial third and the middle third, whereas the *external ear* is positioned somewhere between the middle third and the lower third. The *upper third* is essentially a part of the cranium, discussed earlier. It comprises mainly the frontal bone, as it curves downward at the forehead to make the thickened upper margin of the orbits. It terminates at the frontonasal and frontomaxillary sutures in the midline and frontozygomatic sutures at the lateral margins of the orbits. The supraorbital notch or foramen is an important anatomical landmark on the superomedial aspect of the orbital rim; the supraorbital nerves and vessels exit from here, and these must be respected when incision or flaps are being made in this region.[6,9] Above the orbital margins, the frontal bone is hollowed out and expanded to form the frontal sinuses. The orbital part of the frontal bone forms most part of the roof of the orbits.

The *orbital cavity* is a vital anatomical part of the facial skeleton; it contains the eye and important neural and vascular elements and connective tissues. It is formed between the cranial third and middle third of the face. The orbital rim is formed superiorly by the frontal bone, medially by the processes of the maxilla and frontal bone, inferiorly by upper margins of the maxilla and zygoma, and laterally by the processes of the zygomatic and frontal bones. The orbital cavity is roughly pyramidal in shape with its apex at the optic foramen and its base formed outwardly by the orbital margins. It has a medial and lateral wall, a roof, and a floor. The medial wall is thin, formed by the orbital plate of ethmoid bone, which contains the ethmoidal sinuses. The floor is extremely thin, particularly in the region of the infraorbital groove, which anteriorly becomes the infraorbital canal. The orbital floor is made up of the orbital part of maxillary and zygomatic bone, which is thin. It is bounded laterally by the inferior orbital fissure; posteriorly, it is made up of the orbital process of the palatine bone. The lateral wall is formed by zygomatic bone and greater wing of sphenoid, whereas the roof is formed by the orbital plate of the frontal bone. Both lateral wall and roof are

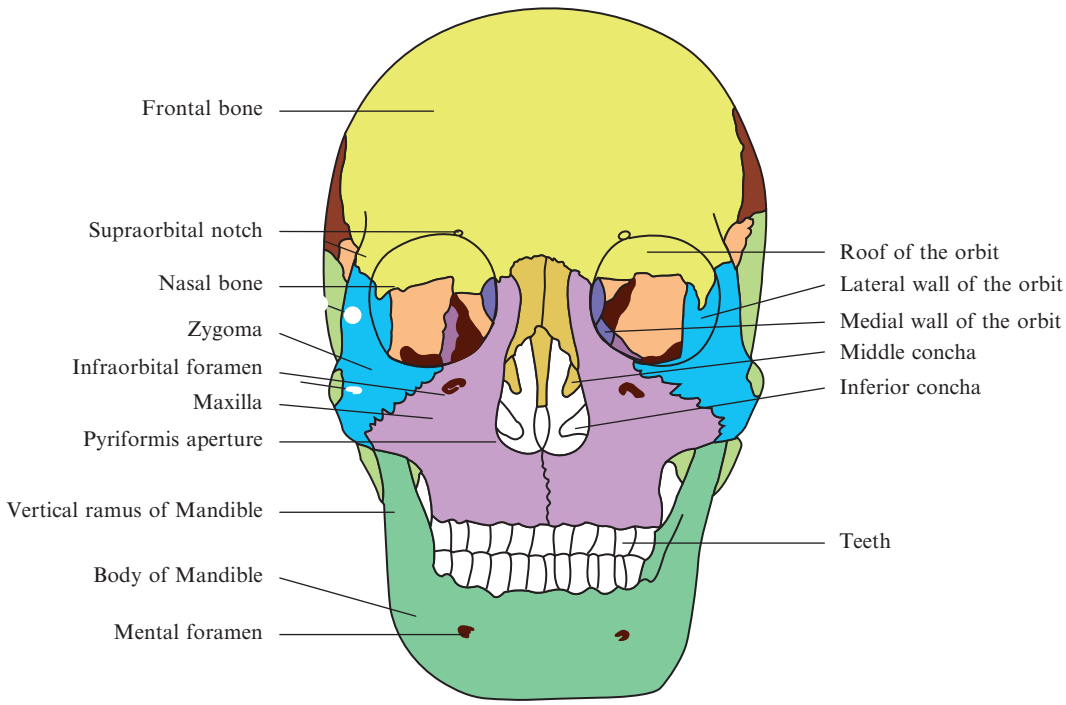

Figure 18.12. The facial skeleton.

relatively thick. The surgical importance of this architecture is that the thick sidewall protects the orbital content from the impact of a direct force lateral, whereas the thick roof protects against fracture and intrusion into the anterior cranial fossa. The thin floor and medial wall fracture easily in response to a direct compressive force on the eyeball and thus the orbit, to prevent significant damage to vital intraorbital structures.[1]

The *middle third* constitutes the central part of the facial skeleton. It involves a complex articulation of fragile bones. The area is defined superiorly by a horizontal line drawn across the skull from the frontozygomatic suture across the frontonasal and frontomaxillary suture to the frontozygomatic suture on the opposite side and inferiorly by the occlusal plane of the upper teeth. It extends as far backward as the pterygoid plates of the sphenoid.[1] The composite structure of this complex of bones is so designed that it will withstand the forces of mastication from below and provides protection in certain vital areas when these are traumatized.[3] This region is occupied mainly by the paired maxillary bones, with the anterior nasal aperture lying between them. The maxilla is roughly pyramidal in shape, with a hollowed region constituting mainly of the maxillary sinuses. It contributes to the upper jaw, bridge of the nose, the nasal aperture, and the inferior margin and floor of the orbit. The infraorbital foramen lies in the upper aspect of the maxilla about 1 cm below the inferior orbital margin and transmits the infraorbital neurovascular bundle. The maxillary alveolar process contains sockets for the upper teeth, whereas the medial wall of the maxillae contributes to the lateral nasal wall. Laterally the maxilla articulates with the zygomatic bone and further laterally this articulates with the zygomatic process of the temporal bone to complete the zygomatic arch.

At the inferior aspect of the maxilla, the two palatine bones fuse at the median palatal suture, forming the roof of the oral cavity. Posterior to this, the horizontal plate of the palatine bone completes the hard palate. The lesser and greater palatine nerves exit from small foramina in the posterior aspect of the hard palate. The maxillae articulate with the pterygoid at its posterior margin, forming the pterygomaxillary suture.

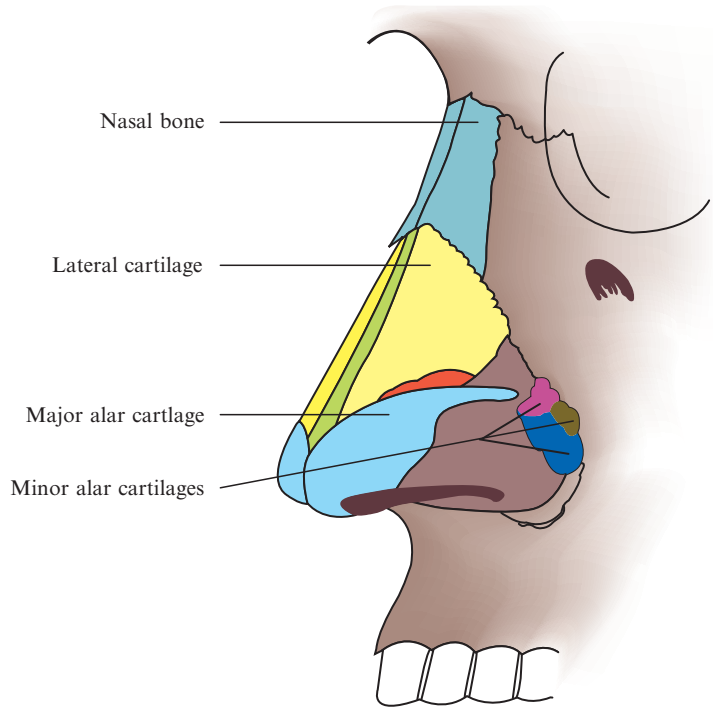

Figure 18.13. The nasal skeleton.

The nasal skeleton is an important part of the mid face. The supporting framework of the external nose is composed of bone and fibroelastic cartilages (Figure 18.13). The paired nasal bones, the frontal processes of the maxillae, and the nasal processes of the frontal bones complete the nasal bridge. The ethmoid bone with its attachment to the superior and middle turbinate bones forms part of the lateral nasal walls, whereas the cribriform plate of the ethmoid forms the nasal roof. The inferior turbinates are separate bones attached to the maxillary aspect of the lateral nasal wall, and they are important anatomical structures that must be considered in nasal surgery. The bony part of the nasal septum is formed by the perpendicular plate of the ethmoid bone at the posterosuperior aspect and the vomer bone posteroinferiorly. The cartilaginous framework consists of the median quadrilateral septal cartilage sandwiched between the perpendicular plate of the ethmoid and vomer bone (Figure 18.14) and the paired upper lateral and alar nasal cartilages, and these are connected to each other and to the nearby bones by the continuity of the perichondrium and periosteum.

The *mandible* is the bone of the lower third of the facial skeleton. It consists of a horseshoe-shaped body and a pair of vertical rami (Figure 18.12). It has outer and inner cortical plates that are thicker anteriorly and along its inferior border, and it articulates with the cranial base at the temporomandibular joints bilaterally. The mandible is a unique structure in several respects: the teeth and their occlusion are important in correction of bony facial trauma and in aesthetic surgery. The synchronous movement of the condyle in relation to the base of the skull and the complex muscle attachments around it determines the biomechanics of traumatic injuries to the bone and cranial base. This provides a challenge when trauma is sustained in this region.

The symphysis menti appears as a faint midline ridge on the outer surface, and the mental foramen can be seen below the second premolar tooth. This is the exit for the mental nerve that becomes susceptible in trauma or surgery in this region. Medially, the genial tubercles are formed on the inner surface by attachments of the genial muscles. Bilateral parasymphyseal fractures can cause a backward and downward pull of these

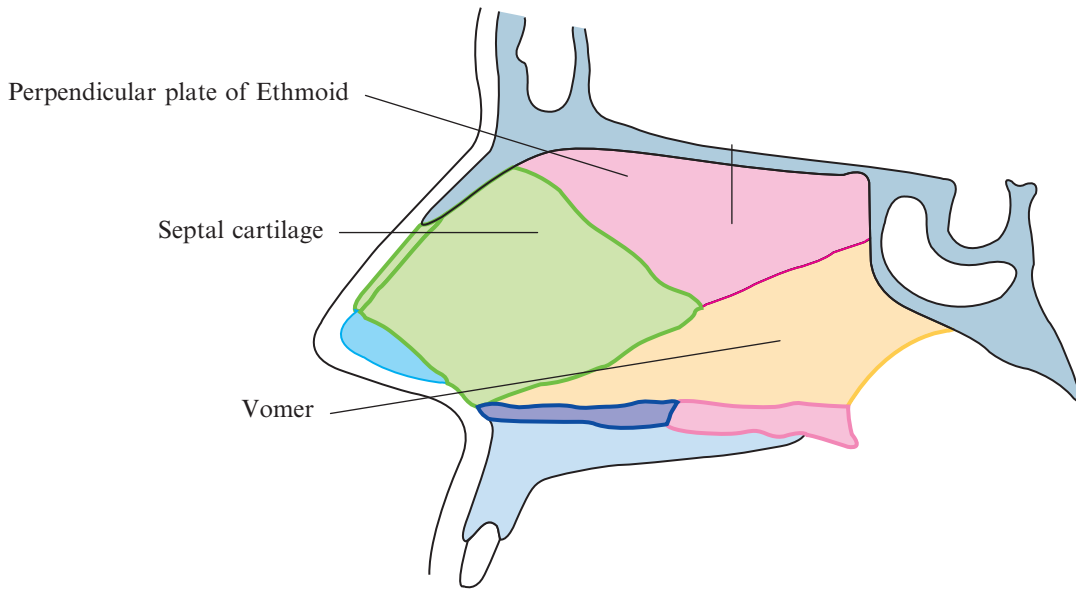

Figure 18.14. The nasal septum.

muscles on the median segment, causing the tongue to obstruct the airway in an unconscious patient. Lateral to these tubercles, the mylohyoid line runs posteriorly as an oblique ridge to an area behind the third molar; here it gives attachment to the muscles of the floor of the mouth. The vertical ramus has an anterior coronoid process and posterior condylar process; these have ligamentous and muscular connections with the cranium. The condylar neck is the weakest part of the mandible and is most susceptible to fracture – this mechanism protects against transmission of significant force to the cranial base. On the lateral surface of the vertical ramus, the markings for the attachment of the masseter muscle are seen, whereas the mandibular foramen for the inferior alveolar neurovascular bundle is abutted on the medial surface by a bony projection called the lingula, to which the sphenomandibular ligament is attached. The mandibular foramen leads into the mandibular canal, which continues to the mental foramen from which the mental nerve exits. Further from this area, the canal persists as an incisive canal transmitting nerve supply to the roots of the incisors and canine teeth. The upper border of the body of the mandible is termed the "alveolus" and contains the lower teeth sockets.

The framework of the auricular part of the *external ear* is entirely cartilaginous. It consists of a thin plate of fibroelastic cartilage molded by eminences and depressions (Figure 18.15). There is no cartilage in the lobule or between the tragus and the crus of helix. Anteriorly, where the helix curves upward, there is a small cartilaginous projection, the spine of the helix. Its other extremity projects inferiorly as the tail of the helix. The cranial aspect of the cartilage bears the eminentia conchae and eminentia scaphae.

Soft Tissue Envelope

The soft tissue envelope of the head and neck consists of the skin, subcutaneous connective tissue, muscles, vascular, and nerve distributions. The basic structures of these tissues are essentially the same throughout the region of the neck, but there are significant topographical modifications in the head region. The neck is draped in a smooth stretch of skin with a variable natural line of cleavage that runs almost horizontally around it. The subcutaneous fat determines its shape.[13]

The Scalp and Facial skin

The *scalp* is made up of five layers (Figure 18.16), the first three of which are tenaciously bound together. These are the skin, dense connective

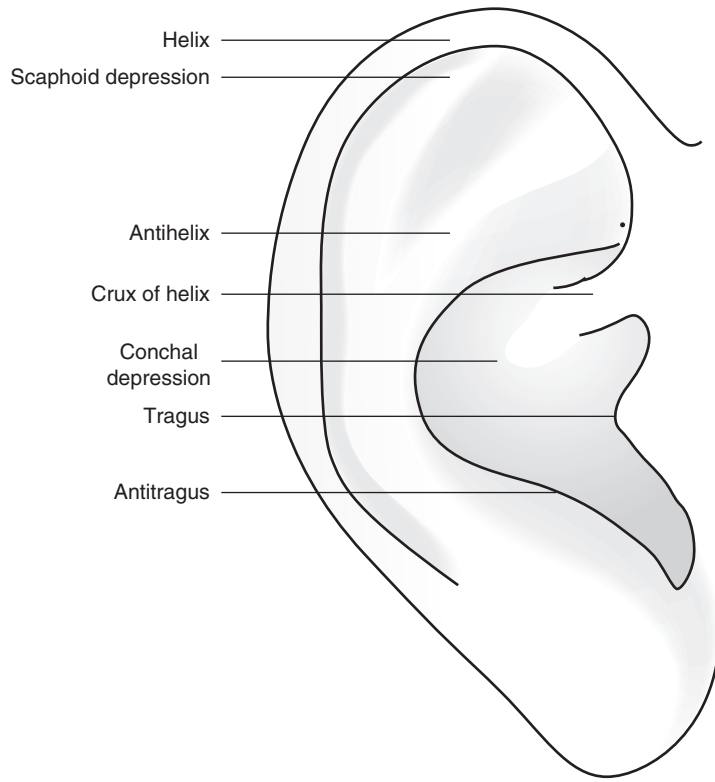

Figure 18.15. The external ear.

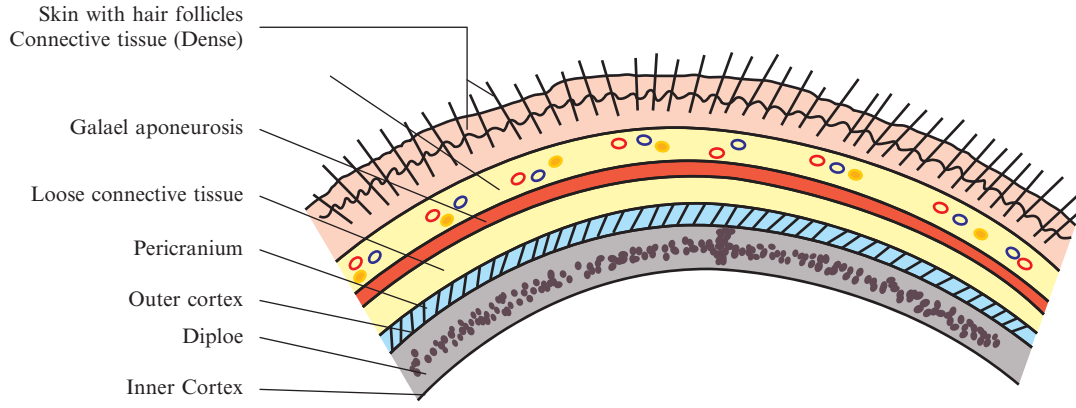

Figure 18.16. The scalp.

tissue, and the galea aponeurosis. Deep to these are the layers of loose connective tissue and the pericranium. The skin is thick and hair bearing, containing numerous sebaceous glands. There are numerous arteries and veins in the dense connective tissue layer, forming free anastomoses between branches of the internal and external carotid arteries and their accompanying veins. The galea aponeurosis is a thin tendinous structure that unites the frontal and occipital belly of the occipitofrontalis muscle. It is attached laterally to the temporalis fascia. The loose

connective tissue layer contains a few small arteries and important emissary veins that connect the scalp veins to the diploe of the calvarial bones and the intracranial venous sinuses.

Because of its rich vascular supply, the scalp provides a rich resource for flaps of varying thickness and designs. In a cadaver study of the vascular anatomy of the anteriorly based galeopericranial flap, Potparic et al.[11] demonstrated that the blood supply of the flap depends entirely on the branches of supratrochlear and supraorbital vessels. The reliability of this supply is said to be predictable to a limited distal extent. Some increase in bulk and vascularity may be achieved if the pericranial and galeal-frontalis myofascial flaps are harvested as a single unit. Similarly, Sharma et al.[12] found that the posteriorly based full-thickness galeo-occipitalis flap can be made of larger volume and richer vascularity when raised below the subperiosteal plane.

The temporal area of the cranium is occupied by the temporalis muscle, which originates along the inferior temporal lines and the floor of the temporal fossa and converges on its tendon beneath the zygomatic arch to attach to the coronoid process of the mandible. The muscle is covered by the temporalis fascia, which is a continuum of the galea aponeurosis, as it attaches superiorly along the superior temporal lines and inferiorly along the upper border of the zygomatic arch.

The *facial skin* can be divided into aesthetic units based on the consistency of color, texture, thickness, mobility, vascular quality, and hair density.[7,10] The aesthetic units include the forehead, the temple, the cheeks, the nose, the periorbital area, the lips, and the chin. Wherever possible, it is desirable that these boundaries are not violated during surgery. The natural cleavage lines of the individual aesthetic unit tend to run in the same direction, hence incisions placed within or between the boundaries tend to heal without significant scarring in most patients.

The skin of the face becomes specialized in the area of the eyelids and the nose. The eyelids are composed of two structural lamellae formed by the orbicularis muscle and its overlying skin and the internal lamella of the tarsal plate and conjunctiva.[4] The skin of the eyelid is extremely thin and delicate containing numerous small lacrima, sweat and sebaceous glands, and hair follicles.[8] The skin of the nose is tightly attached to the lower lateral cartilage in the tip area. In the other area, it is less tightly adhered to the underlying infrastructure. The skin is thin at the nasal root and tip areas and thicker in the supratip region. The skin of the auricle is thin, has no dermal papillae, and adheres to the underlying cartilaginous framework.

Subcutaneous Connective Tissues

Underneath the facial skin is a layer of thin, loose, areolar connective tissue in which are embedded the muscles of mastication. *It must be stressed that no deep fascia exists on the face*.

Immediately deep to the skin of the neck is a thin layer of loose superficial fascia that encloses the platysma muscle and contains superficial veins, lymph nodes, and cutaneous nerves of the neck. The deeper structures of the neck are wrapped around by the investing layer of the deep cervical fascia. This fascia splits to enclose the trapezius and sternocleidomastoid muscle. It is modified on its deep aspect to form the pretracheal and prevertebral fascia and the carotid sheath, which all divide the neck into separate compartments. The investing layer is attached below to the manubrium sterni, clavicle, and scapular and above to the hyoid bone, the inferior border of the mandible, zygomatic arch, mastoid process and the superior nuchal line of the occipital bone. It splits between the angle of the mandible and the mastoid process to enclose the parotid gland. This modification forms the parotid capsule or fascia. The deep fascia supports the muscles, viscera, and vessels of the neck.

Muscles and Viscerae of the Head and Neck

The muscular apparatus of the head and neck is best understood when it is divided into groups. The Cranial vault is covered by a compound muscle comprising two bellies joined together by the galea aponeurosis of the scalp – the occipitofrontalis. The facial muscles consist of two groups: the masticatory muscles and the muscles of facial expression, which derive from the first and second pharyngeal arches, respectively. In the neck, the muscle can be classified as the suprahyoid, infrahyoid (strap muscles), and prevertebral muscle groups, which are also of different embryonic origins. The sternocleidomastoid and trapezius muscles belong to a separate group. Important viscera in the head and neck include the tongue, thyroid and parathyroid glands, thymus, and the laryngeal apparatus.

Discussion of the anatomy of these viscera is beyond the scope of this book.

Nerve Supply

The somatic sensory and motor supply to the head and neck is derived mainly from cranial nerves V, VII, XI, and branches of the cervical plexus from spinal roots C1–5.

The sensory supply to the scalp includes the supraorbital and supratrochlear branches of the V1 division (ophthalmic) of the trigeminal nerve, which supply the forehead up to the vertex; the auriculotemporal branch of the V3 division (mandibular) of the trigeminal nerve, which supplies the temporoparietal region of the face and scalp; and the occipital nerves (greater and lesser), which supply the back of the scalp and are derived from the cutaneous branches of the cervical plexus. In the mid-face region, branches of the V2 division (maxillary) and in the lower face, branches of the V3 division (mandibular) of the trigeminal nerves provide sensory supplies (Figure 18.17).

The cranial nerve VII (facial nerve) provides motor supply to the muscles of facial expression as it emerges between the substance of the superficial and deep lobes of the parotid gland in its characteristic "spread-fingers" fashion (Figure 18.17). The masticatory muscles receive motor supply and proprioception from the motor part of the cranial nerve V3 (mandibular). The muscles in the suprahyoid group are supplied by either the motor part of V3 (e.g., anterior belly of digastric) or cranial nerve VII (e.g., posterior belly of digastric) depending on their embryonic origin. The infrahyoid muscles are supplied by motor branches from the C1, 2, and 3 spinal nerves. The trapezius and sternocleidomastoid muscles are innervated by the cranial nerve XI (accessory nerve), whereas the prevertebral

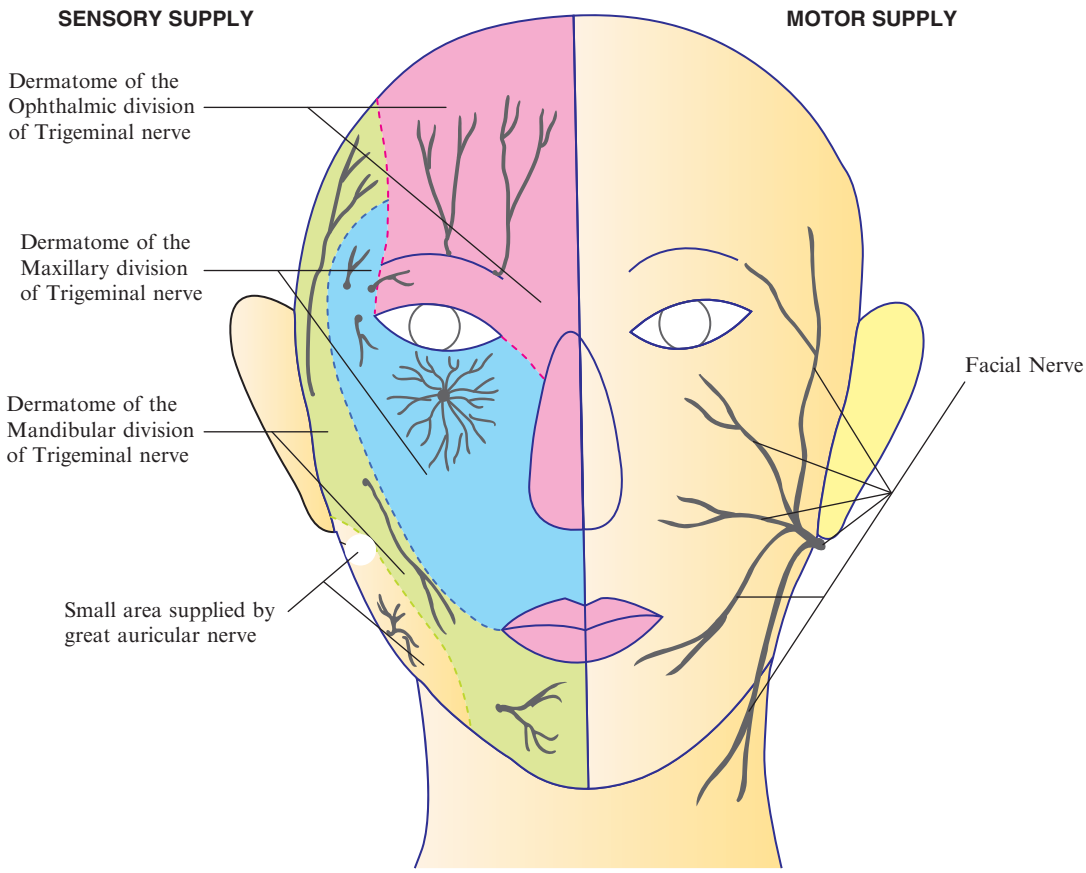

Figure 18.17. Sensory and motor supply to the face.

muscles are innervated by muscular branches of the deep cervical plexus.

Vascular Supply and Drainage

The common carotid arteries via their internal and external carotid branches provide the major source of blood supply to the head and neck (Figure 18.18). Additional arteries arise from the subclavian artery, particularly the vertebral artery. The external carotid artery is essentially extracranial. It gives rise to many branches that supply the head and neck region. Of particular importance to the neck, face, and scalp are the superior thyroid, posterior auricular, facial, and the superficial temporal artery. The extracranial contributions of the internal carotid artery come via the supraorbital and supratrochlear arteries to the forehead and the anastomotic branches within the scalp. The venous drainage of the head and neck follows a similar course (Figure 18.8), the main tributaries being the facial vein, the retromandibular vein, posterior auricular vein, and the anterior jugular and transverse cervical veins, all draining into the main external jugular vein.

Lymphatic Drainage

The lymph nodes of the head and neck are arranged as a regional collar that extends below the chin to the back of the head and as a deep vertical terminal group along the axis of the internal jugular vein (jugulo-digastric and jugulo-omohyoid groups).

Drainage from the center of the forehead above the root of the nose, the pyramidal area of the maxilla and upper and lower lips drain directly or indirectly into the submandibular nodes. The lateral aspects of the forehead and scalp, the eyelids, and the temporal and cheek areas of the face drain into the parotid and buccal lymph nodes. Drainage from these primary nodes as well as other parts of the neck ultimately drains into the deep vertical group of cervical lymph nodes, which eventually empty into the jugular trunk.

The aim of this brief anatomical survey of the head and neck has been to highlight areas that have a complex anatomy; an attempt has been made to simplify the description and to make it more relevant to some of the more complex procedures that have been introduced in recent years.

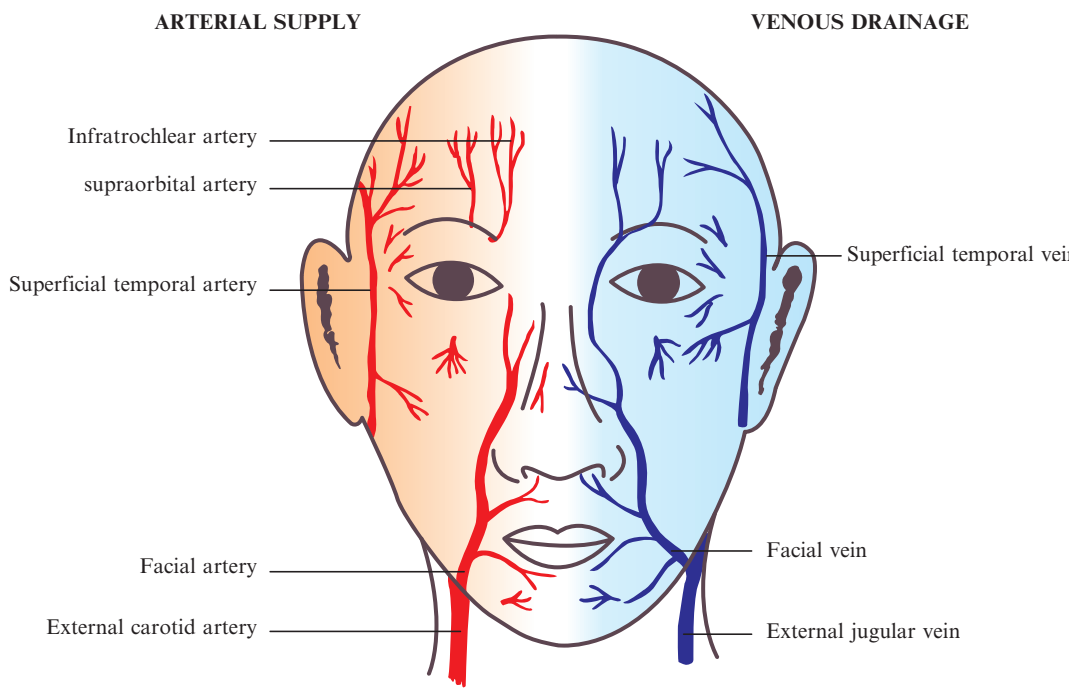

Figure 18.18. The vascular anatomy of the face.

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19

Craniofacial Clefts and Craniofacial Syndromes

Claude-Jean Langevin, Earl Gage, and Frank Papay

Summary

Craniofacial clefts are challenging problems encountered by plastic surgeons. These anomalies embrace a diverse group of abnormalities ranging from simple soft tissue defects to complex craniofacial malformation. They are rare occurrences, and their wide spectrum of presentation makes classification difficult. The treatments of craniofacial clefts require a thorough knowledge of the craniofacial anatomy, the underlying embryological pathology, and the specific characteristic of each entity. In addition, careful surgical planning with a multidisciplinary team is essential to achieve both functional and aesthetic goals.

Abbreviations

OMENS	Orbit asymmetry, Mandibular hypoplasia, Ear deformity, Nerve dysfunction, Soft tissue deficiency
SAT	Skeletal, Auricle, soft Tissue
TMJ	Temporomandibular joint
TNM	Tumor, Node, Metastasis

Introduction

Composed of different aesthetic units, the face represents an outward projection of our inner self, making it the focal point of our social identity. We can only imagine how distressing it would be for parents when their child comes into this world with a severe facial distortion. Improper treatment of the deformities can have a huge impact on the child's social and mental development. Fortunately, craniofacial clefts are rare. However, due to the wide variability of the physical phenomena and the lack of understanding with regard to their embryology, classification remains problematic. Therefore, the surgical management of facial clefts requires a thorough knowledge of the craniofacial anatomy and the specific characteristics of each anomaly. In addition, careful surgical planning with a multidisciplinary team is essential to achieve both functional and aesthetic goals.

Craniofacial Clefts

Craniofacial clefts have a multitude of clinical presentations with different levels of severity. There is a substantial amount of data to report with confidence the incidence of the common clefts of the lip and palate with their associated racial variations. The common cleft is more likely to occur within the Asian population versus Caucasians and African Americans with incidences of 2.1 in 1,000 live births, 1 in 1,000 live births, and 0.4 in 1,000 live births, respectively.[3,18,48] The exact rate of occurrence of atypical clefts is unknown but is estimated at 1.4–4.9 cases per 100,000 live births,[32,33] therefore

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approximately 100 times less frequent than the common clefts. The estimates of prevalence are largely dependent on the examiner's attentiveness to the minor form of craniofacial clefts. Tissue deficiency or tissue excess characterize these malformations.

Embryology and Pathogenesis

The crucial period of organogenesis is defined as the first 12 weeks of gestation.[29,64] The structural development of the cranium and face occurs between the third and eighth weeks, and it is during this period that most of the craniofacial anomalies take place.[2,37,61,64] By the end of the eighth week, the face takes on a recognizable human appearance. The human face is derived from five facial prominences that surround the primitive mouth known as the stomodeum. They consist of a single frontonasal process and two bilateral maxillary and mandibular processes. Both the maxillary and mandibular processes are derived from the first branchial arch.

Two theories exist to explain the formation of facial clefts. The *classic theory* by Dursy[13] and His[24] claims that failure of fusion of the various facial processes would explain the morphogenesis of facial clefting.[13,24] According to this theory, facial processes are thought of as free-end processes, and once epithelial contact is established between them, mesodermal penetration completes the fusion. The *mesodermal penetration theory* proposed by Veau,[75] Warbrick,[77] and Stark[66] is based on the assumption that the embryonic face consists of a continuous bilaminar ectodermal membrane with epithelial seams defining the major facial processes. Mesenchymal migration and penetration within this bilaminar ectoderm smooth out the seams and support the epithelial walls. Failure of mesenchymal penetration would lead to dehiscence and ultimately create a cleft. Consequently, the degree of cleft severity would be inversely proportional to the degree of mesodermal penetration. Furthermore, Johnston demonstrated the prime importance of neuroectodermal cells, a group of cells arising from the dorsal lateral ectoderm, in the mesenchymal development of craniofacial structures.[28] Failure in the formation, migration, or differentiation of these cranial neural crest cells leads to abnormal bone, cartilage, and connective tissue development.

The etiology of craniofacial clefting is believed to be multifactorial. Genetics seem to play a minor role if we make abstraction of Treacher Collins syndrome.[17,20] The major etiologic causes include viral infections (rubella, cytomegalovirus, toxoplasmosis), maternal metabolic abnormalities (phenylketonuria), drugs (Isotretinoin), and large-dose radiation exposure.[14,26,32]

Classification

In 1976, Paul Tessier, the father of craniofacial surgery, fashioned a simple system of classification for craniofacial clefts based on skeletal and soft tissue landmarks.[72] With the orbit serving as the reference point, the clefts are divided into a cranial and a facial component. Each cleft is assigned a number, from 0 to 14, relative to its position from the sagittal midline (Figure 19.1). The sum of the facial and cranial clefts typically adds up to 14. Craniofacial clefts rarely occur in a pure isolated form; they may have a unilateral or bilateral presentation with different possible clefting patterns on each side.

In 1983, van der Meulen et al.[74] proposed an embryological classification based on the development of the craniofacial skeleton along a helical course symbolized by the letter S (Figure 19.2). The term "dysplasia" is preferred over "cleft" to describe an arrest in skin, muscle, or bone development. The ultimate craniofacial malformations will depend on the localization and the time of disturbance. However, the simpler and more descriptive Tessier's classification still prevails and allows for easy and effective communication between physicians.

No. 0 Cleft

A broad range of expression has been reported from its minor form, represented as a subtle midline notch of the upper lip, to a true median cleft lip with a broad columella, bifid nasal tip, broad and flattened nasal bridge, alveolar cleft between the central incisors, and hyperteleorbitism. The nasal septum can be thickened, duplicated, or absent. It is important to note the differences between hyperteleorbitism, the increased distance between the medial orbital walls, and telecanthus, which is the lateral displacement of the medial canthi commonly seen in blepharophimosis. Cleft No. 0 may be expressed as either a central tissue deficiency

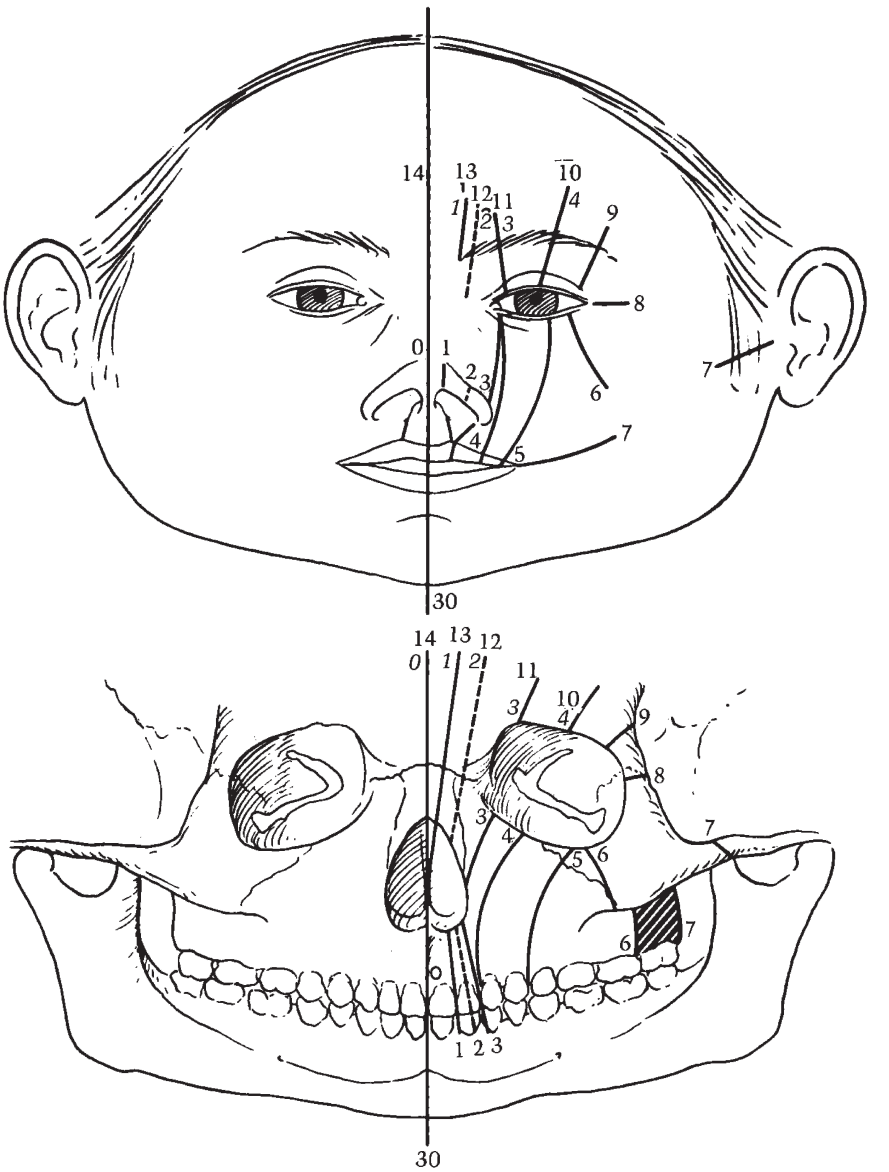

Figure 19.1. Tessier's classification of facial clefts. (Reprinted from Tessier[72], Copyright 1976, with permission from Elsevier.)

(holoprosencephaly) or tissue excess (median cleft face dysmorphism). Cleft No. 14 represents the cranial extension.[8,10,20,32,51,72]

No. 1 Cleft

This cleft originates in the cupid's bow area analogous to a common cleft lip and travels cephalad through the alar dome, parasagittal nasal dorsum, medial aspect of the eyebrow, and with possible extension into the cranium as a cleft No. 13. A notch of the alar dome is specific to this cleft. An alveolar cleft can be present between the central and lateral incisors extending to the piriform aperture lateral to the anterior nasal spine.[8,10,20,32,51,72]

No. 2 Cleft

The cleft begins at the cupid's bow area and typically exhibits hypoplasia of the middle third of

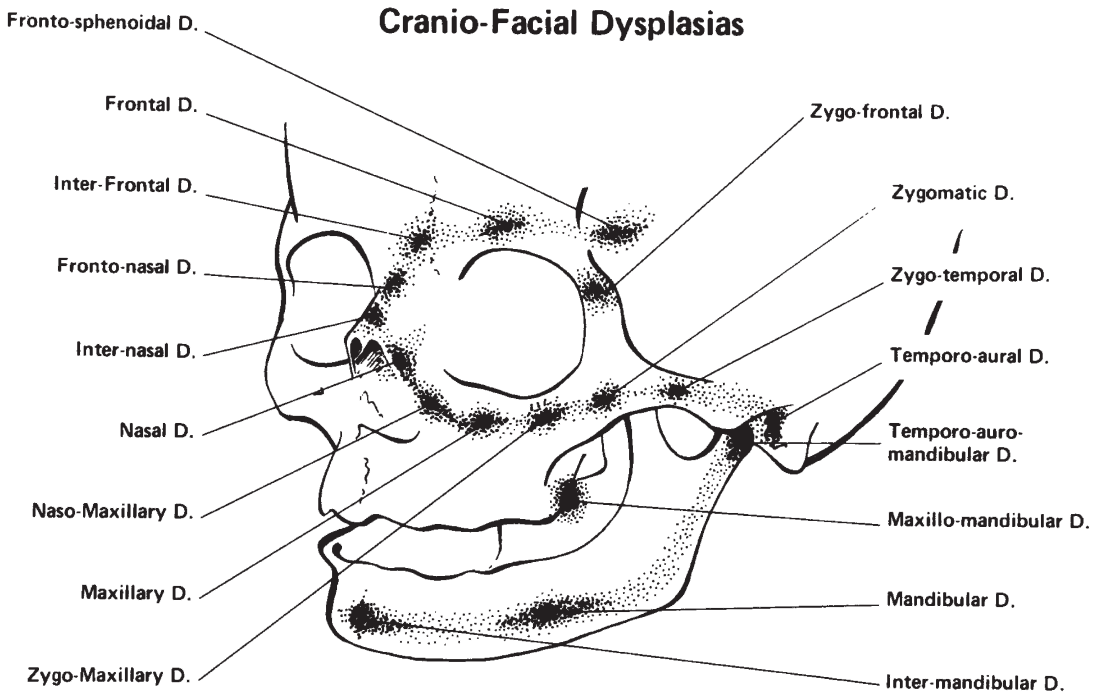

Figure 19.2. van der Meulen's morphogenetic classification of craniofacial malformations. (Reprinted with permission from van der Meulen et al.[74])

the alar rim, which gives a flattened appearance to the lateral aspect of the nose. Hypoplasia, not a true notch of the middle third of the ala, is typical. Hyperteleorbitism is noted; however, the nasolacrimal system, eyelid, and palpebral fissure are not involved. Alveolar clefting may be present at the lateral incisor position. Distortion on the medial brow is seen in cleft No. 12.[8,10,20,32,51,72]

No. 3 Cleft (Oronasal-Ocular Cleft)

Similar to clefts No. 1 and 2, it originates at the cupid's bow area but extends cephalad across the alar base and continues superiorly between the medial canthus and the inferior lacrimal punctum, resulting in an inferiorly displaced medial canthus, coloboma, and nasolacrimal disruption. The alveolar cleft is usually present between the lateral incisor and the canine and terminates in the lacrimal groove. The oral, nasal, maxillary sinus and orbital cavities are contiguous. Dystopia is present and microphthalmia may be noted. Cranial extension represents a cleft No. 11.[8,10,20,32,51,72]

No. 4 Cleft

Cleft No. 4 originates lateral to cupid's bow and terminates at the lower eyelid medial to the punctum without affecting the nose. The medial canthus remains intact and the nasolacrimal system functional except for the inferior canaliculus that is disturbed by the cleft. The alveolar cleft begins between the lateral incisor and the cuspid and travels medial to the infraorbital foramen. The cleft creates communication between the oral, maxillary sinus and orbital cavities excluding the nasal cavity. Dystopia and microphthalmia may be noted. Cleft No. 10 represents this cranial extension.[8,10,20,32,51,72]

No. 5 Cleft

Extremely rare, it begins just medial to the oral commissure and extends obliquely across the cheek to end at the lateral third of the lower eyelid. Dystopia and microphthalmia may be present. Alveolar clefting occurs in the bicuspid region, courses lateral to the infraorbital foramen, and ends at the orbital rim and floor. Cleft No. 9 is regarded as its cranial extension.[8,10,20,32,51,72]

No. 6 Cleft

Often referred to as an incomplete form of Treacher Collins syndrome, this cleft is characterized by a channel along the zygomaticomaxillary suture with a hypoplastic malar bone and an intact zygomatic arch. There is no alveolar cleft. Soft tissue defect is minimal in this cleft. It is mainly characterized by a vertical groove extending from the lateral lower eyelid toward the angle of the mandible, contributing to the antimongoloid slant and coloboma. Hearing impairment is often present, although external ear deformities are rare.[8,10,20,32,51,72]

No. 7 Cleft

Cleft No. 7 is the most common cleft and the most lateral craniofacial cleft.[5,20] It is found in both Treacher Collins syndrome, and craniofacial microsomia, discussed later in detail. Its clinical presentation varies widely from a mild lengthening of the oral commissure with preauricular skin tag to complete macrostomia extending to the anterior border of the masseter with microtic ear, absence of the parotid gland and duct, and paresis of the cranial nerves V and VII. The skeletal cleft is located at the pterygomaxillary junction with various degrees of hypoplasia affecting the middle ear, maxilla, zygoma and mandible.[8,10,20,32,51,72]

No. 8 Cleft

Commonly associated with other cleft abnormalities, it originates from the lateral canthus and extends into the temporal region. It divides the facial clefts from the cranial clefts and is considered the cranial extension of cleft No. 6. The skeletal involvement occurs at the frontozygomatic suture. A dermatocele, a true lateral commissure coloboma with absence of the lateral canthus, is often observed in Goldenhar syndrome in conjunction with epibulbar dermoids. The complete from of Treacher Collins syndrome is best described by the bilateral manifestation of clefts No. 6, 7, and 8, the hallmark being an absent zygoma.[8,10,20,32,51,72]

No. 9 Cleft

This cleft occurs at the superolateral orbit, creating abnormalities of the lateral third of the upper eyelid and eyebrow. The lateral canthus is distorted and the superolateral orbital bone deficiency generates a lateral displacement of the globe. Microphthalmia may be present in severe cases. The temporal hairline projects anteriorly.[8,10,20,32,51,72]

No. 10 Cleft

Cleft No. 10 begins at the middle third of the upper eyelid and eyebrow and extends into the frontal bone. Possible ocular abnormalities include elongated palpebral fissure, ablepharia, and coloboma. Frontal hair projection, encephalocele, and orbital hypertelorism may be present. It is a cranial extension of facial cleft No. 4.[8,10,20,32,51,72]

No. 11 Cleft

It connects the medial third of the upper eyelid and eyebrow to the frontal hairline. It corresponds to the cranial branch of cleft No. 3. If the ethmoidal labyrinth is disrupted medially, hyperteleorbitism will be noted. However, a path lateral to the ethmoid will result in coloboma of the medial third of the upper eyelid with disruption of the corresponding supraorbital rim and frontal hairline.[8,10,20,32,51,72]

No. 12 Cleft

Cleft No. 12 is located medial to the medial canthus with superior extension within the medial eyebrow margin. It is a continuation of cleft No. 2. The cleft travels through the frontal process of the maxilla and the ethmoid superiorly, increasing the transverse dimension of the ethmoid labyrinth, which leads to hyperteleorbitism. However, the cribriform plate remains intact.[8,10,20,32,51,72]

No. 13 Cleft

Cleft No. 13 extends through the olfactory groove with widening of the cribriform plate in the transverse dimension, creating hyperteleorbitism and dystopia. A paramedian frontal encephalocele would be located between the nasal bone and the frontal process of the maxilla. It represents the extension of cleft No. 1.[8,10,20,32,51,72]

No. 14 Cleft

Cleft No. 14 is a midline cranial cleft equivalent to that of facial cleft No. 0 accompanied by abnormalities within the central nervous system. Both of these clefts can be associated with tissue excess or deficiency. Hypoteleorbitism and microcephalic cranium occur with tissue deficiency commonly

seen with the holoprosencephalic disorders, which encompass cyclopia, ethmocephaly, and cebocephaly. Holoprosencephaly results from the incomplete septation of the anterior portion of the neural tube into the cerebral hemispheres, thereby creating a single forebrain.[10] Forebrain anomalies are typically proportional to the degree of facial malformations. In general, due to severe brain abnormalities, holoprosencephaly is incompatible with life. In the more commonly seen cases of tissue excess, hypertelorbitism and flattening of the frontal region, especially the glabella, are produced by a variety of midline protuberances, such as the median frontal, frontonasal, or frontoethmoidal encephalocele. The crista galli may be widened, duplicated, or absent.[8,10,20,32,51,72]

No. 30 Cleft

The caudal extension of the clefts No. 0 and 14, this median cleft of the lower jaw is located between the central incisors, extending into the mandibular symphysis. Similar to the other clefts, a wide spectrum of severity is seen from a small notch of the lower lip to a true cleft involving the entire mandible, with malformation of neck structures. Several tongue anomalies have been reported, such as agenesis, bifidity, and ankyloglossia.[8,10,20,32,51,72]

Treatment

Specific details regarding the complex reconstruction of these rare facial clefts are beyond the scope of this chapter. However, surgical objectives include the following: (1) functional reconstruction of the macrostomia; (2) reconstruction of the eyelid soft tissue to prevent globe exposure; (3) separation of the confluent oral, nasal, and orbital cavities; and (4) aesthetic correction of the deformity.[26]

Attention should be directed first to soft tissue closure and cranial defect correction during the first year of life.[49] The scar within the cleft should be excised up to normal tissue followed by layered closure of the soft tissue. Emergent procedure in the neonatal period should be reserved for functional problems such as globe exposure to prevent corneal ulceration. The facial and cranial skeleton frequently requires reconstruction and grafting, which is best performed once the child is older, approximately 6–9 years of age.[49] Correction of maxillomandibular anomalies with orthognathic surgery should be initiated once skeletal maturity has been achieved; approximately 15 years of age in female and 17 years of age in male.[58] Cessation of growth of the craniofacial structures can be correlated with axial skeletal growth, by either hand films to determine epiphyseal plate closure or serial cephalometric analysis. The preferred method involves serial cephalometric radiograph at 6-month intervals to assess the relative movement of the mid face or mandible relative to the cranial base.

Craniofacial Microsomia (Hemifacial Microsomia)

Craniofacial microsomia, also known as first and second branchial arch syndrome, refers to a wide spectrum of complex skeletal and soft tissue anomalies derived from the embryonic first and second branchial arches.[22,65] Gorlin and Pindborg[21] popularized the term hemifacial microsomia; however, this term implies that the disorder is unilateral and limited to the face. Its bilaterality has been noted in 5–30% of cases.[52,62] The incidence has been reported as approximately 1 in 5,000 live births.[22,54] It is the second most common facial birth defect after cleft lip and palate.[47] The etiology is believed to be a vascular insult to the stapedial artery, resulting in hemorrhage and hematoma in the developing first and second branchial arches.[53,54] The majority of cases are sporadic, although an autosomal dominant transmission has been observed in first-degree relatives.[23,50,57,60,63,67–70] Patients with autosomal dominant inheritance are more often bilaterally affected than patients with sporadic occurrence.[71]

The broad clinical manifestation of craniofacial microsomia includes varying degrees of underdevelopment in the mandible, zygoma, maxilla, temporal bone, external and middle ear, muscles of facial expression, muscles of mastication (masseter, temporalis, medial, and lateral pterygoids), palatal muscles, tongue, parotid gland, and cranial nerves, especially the facial nerve.[22] Also contributing to the overall cheek hypoplasia is the commonly observed macrostomia or clefting through the oral commissure that correlates to cleft No. 7.

Varying degrees of hypoplasia of the mandible observed in craniofacial microsomia proportionally

affect the maxillary growth on the distorted side.[30,31] This ipsilateral maxillomandibular hypoplasia results in dental malocclusion and upward occlusal cant (rotation of the occlusal plane in the frontal view) and contributes to deviation of the chin to the affected side (Figure 19.3).

Several classification systems have been elaborated in attempts to standardize the reporting of craniofacial microsomia to facilitate diagnosis, clinical analysis, and treatment planning. However, investigators are still faced with a challenging task considering the complexity and heterogenecity of this disorder.

Pruzansky[55] proposed a classification that was later modified by Mulliken and Kaban[30,44] to describe and determine treatment protocols for mandibular deficiency. The subdivision of type II relates to the functionality of the temporomandibular joint (TMJ) (Table 19.1).

The auricular deformity of craniofacial microsomia was graded by Meurman.[41] In grade I, a hypoplastic auricle with all components present; grade II is characterized by the absence of the external auditory canal and varying hypoplasia of the concha; and in grade III an absent auricle is seen with an abnormally shaped and malpositioned lobule.

More inclusive classifications include the SAT, a multisystem classification. The acronym stands for S = skeletal; A = auricle; and T = soft tissue.[8] The physical manifestations are graded according to five levels of skeletal deformity (S1–S5), four levels of auricular deformity (A0–A3), and three levels of soft tissue deformity (T1–T3). OMENS, later revised to OMENS-Plus (indicates presence of extracranial anomalies – skeletal, cardiac, central nervous system, pulmonary, gastrointestinal, and renal), offers another multisystem classification attempting to grade this disorder according to the dysmorphic severity of these five clinical features on a scale from 0 to 3: *o*rbit asymmetry, *m*andibular hypoplasia, *e*ar deformity, *n*erve dysfunction, and *s*oft tissue deficiency.[25,76] These two classifications include elements of the previously described Pruzansky (Kaban modification) and Meurman classifications with minor modifications.

Surgical correction should be individualized to the patient and ideally be performed in stages.[6,30]

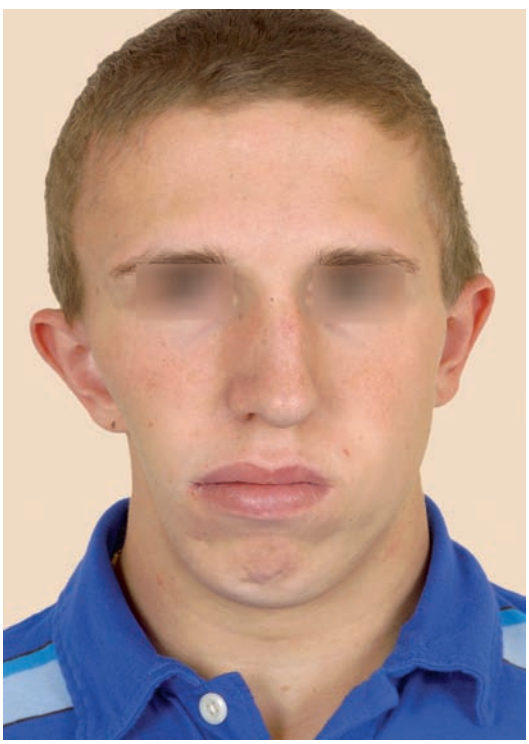

Figure 19.3. Facial asymmetry and chin deviation secondary to right mandibular hypoplasia in an 18-year-old boy with craniofacial microsomia.

Table 19.1 Mulliken and Kaban skeletal classification of hemifacial classification.

Type I	Small mandible and glenoid fossa with mild hypoplasia of the ramus (mini mandible)	
Type II	Short and abnormally shaped mandible	A Glenoid fossa-condyle relationship is maintained (functional TMJ) B Abnormal glenoid fossa-condyle relationship (nonfunctional TMJ)
Type III	Complete absence of the ramus, glenoid fossa, and TMJ	

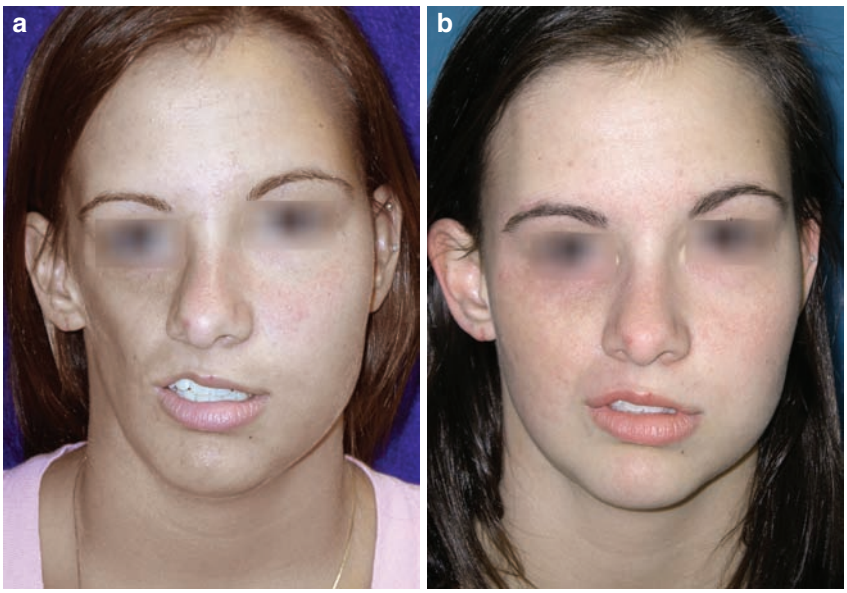

Figure 19.4. (a) 18-year-old female with craniofacial microsomia. (**b**) One-year postoperative view following autologous fat injection to improve right facial soft tissue contours. (Courtesy of Dr. D. Medalie.)

Macrostomia repair by commissuroplasty and preauricular skin tags excision are performed in the first 2 years of life. Distraction osteogenesis of the severely hypoplastic mandible may be necessary in cases of airway compromise.[40]

The mandible is reconstructed according to the Kaban modification classification. In children with mild type I deformity, observation during growth and orthognathic surgery at skeletal maturity are recommended. Distraction osteogenesis may be considered in patients older than 2 years of age with obvious facial deformity secondary to a more pronounced hypoplasia of the ramus such as that in types I and IIA.[42,44] In patients with a type IIB and III mandible,[45,47] the absent ramus, condyle, zygoma, glenoid fossa, and TMJ are reconstructed with a costochondral rib graft usually before the age of 5 years. Generally, craniofacial microsomia patients would require orthodontic treatment to control eruption and malocclusion during their adolescent years. This is usually followed by bimaxillary surgery at skeletal maturity to correct skeletal asymmetry and bone grafting to the deficient portions of the craniofacial skeleton.[46]

Auricular reconstruction is preferably delayed until 8 years of age when the ear has reached more than 85% of its full size.[15] This is especially true in the case of microtia where a mature costochondral graft of sufficient size is to be used as an ear cartilage framework. Although growth of the reconstructed microtic ears has been documented eliminating this concern,[1,69] very few patients show evidence of significant psychological issues regarding the abnormal appearance of their ears before 5 or 6 years of age.

Following skeletal reconstruction, soft tissue augmentation to improve form is accomplished either with autologous fat grafting or microsurgical free tissue transfer[9,36,39,43,73] (Figure 19.4a and b).

Goldenhar Syndrome (Oculoauriculovertebral Dysplasia)

In 1952, Goldenhar described three cases of mandibulofacial dysostosis associated with epibulbar dermoids, auricular appendages, and pretragal fistulas.[19] Goldenhar syndrome has features similar to those of craniofacial microsomia. The clinical presentation is typically bilateral. In addition, it demonstrates epibulbar dermoids and vertebral anomalies including fused and/or hemivertebrae.[5,21] It is now commonly

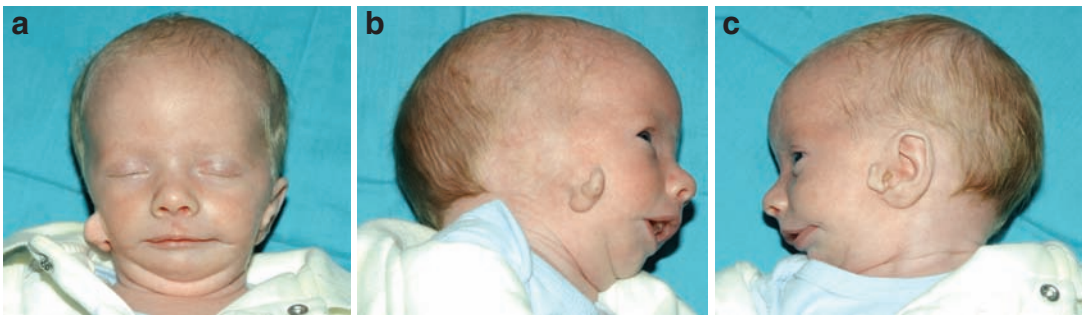

Figure 19.5. (a) Severe bilateral mandibular hypoplasia in this 3-week-old boy with Goldenhar syndrome. (**b**) Right microtia characterized by an abnormally shaped and anteriorly positioned lobule. (**c**) Left preauricular appendage and atresia of the external auditory canal.

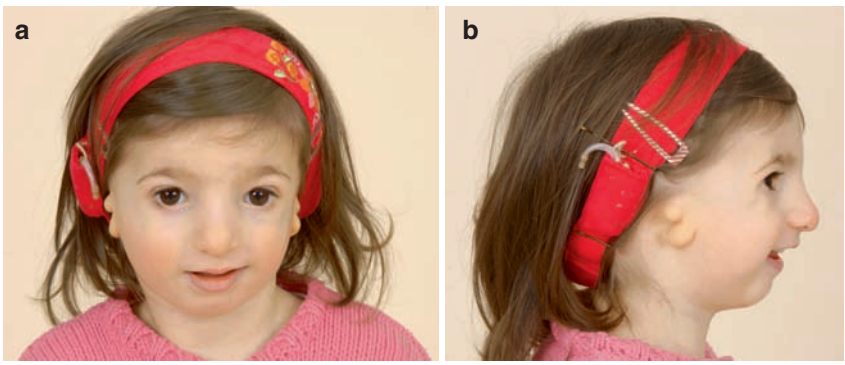

Figure 19.6. (a) A 2-year-old patient with Treacher Collins syndrome. Note the absence of medial lower eyelashes. (**b**) A convex profile is observed secondary to hypoplasia of the zygoma, maxilla, and mandible.

considered to be part of the craniofacial microsomia continuum (Figure 19.5a through c). Its occurrence is usually sporadic, although some genetic tendencies have been reported.[5,34]

Treacher Collins Syndrome (Mandibulofacial Dysostosis)

Originally described by Berry in 1889, Treacher Collins is an autosomal dominant disorder with a variable degree of penetrance. Abnormal bilateral first and second branchial arch development is due to a mutation in the TCOF 1 gene, which has been mapped to the long arm of chromosome 5, more precisely 5q31.3–5q33.3 gene locus.[12,27] Despite its wide phenotypic expression, bilateral and symmetrical presentations are a key feature of this craniofacial anomaly. The incidence is estimated to be 1 in 10,000 live births.[20]

As previously mentioned, the complete form of Treacher Collins[32,72] represents a bilateral occurrence of clefts No. 6, 7, and 8, whereas the incomplete type is equivalent to cleft No. 6.

Clinical features include hypoplastic zygoma and mandible, coloboma, antimongoloid slant, external and middle ear deformities, macrostomia, broad midnasal dorsal hump, convex profile, and low-lying hairline, and one-third have a palatal cleft (Figure 19.6a and b). Intelligence is typically normal.

The Pierre Robin sequence can be associated with Treacher Collins syndrome.[4] This sequence describes an association of micrognathia (hypoplastic mandible), glossoptosis, and cleft palate[16,56,59] (Figure 19.7a and b). These patients may experience feeding and respiratory difficulties

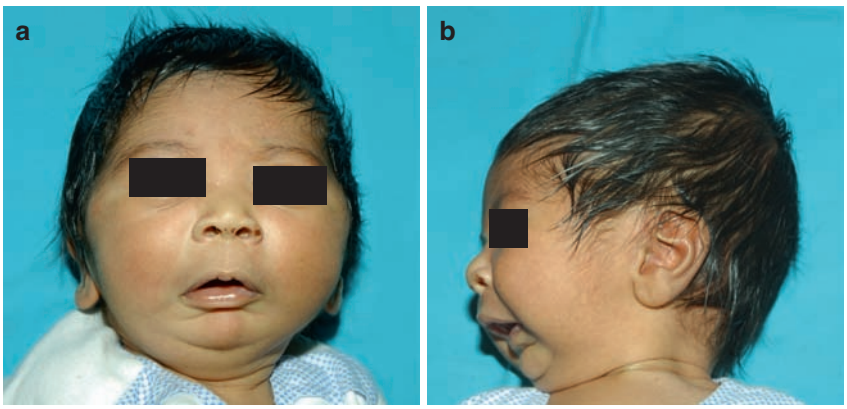

Figure 19.7 (a, b). An 8-day-old baby with Pierre Robin sequence with microretrognathic mandible.

potentially leading to failure to thrive, life-threatening airway obstruction, and cardiac death.

Treacher Collins syndrome shares many characteristics with craniofacial microsomia; however, it can be distinguished on the basis of heredity, colobomas of the eyelids, antimongoloid slant, absence of medial lower eyelashes, and absence of antegonial notching of the mandible.[7,22,38]

The paramount concern with Treacher Collins patients is airway management. Lifesaving tracheostomy is often required secondary to marked reduction of the airway passage and extreme retrusion of the mandible. Other interventions include lip–tongue adhesion, distraction osteogenesis, or conservative measures such as prone positioning, especially during feeding.[11,35]

Reconstruction of Treacher Collins syndrome is focused toward skeletal hypoplasia, involving the maxilla, mandible, and zygoma, as well as the soft tissue defects of the eye and ear. Eyelid coloboma must be corrected early on to prevent exposure keratopathy. Auricular reconstruction is challenging because of the low hairline, with tongue-shaped caudal extensions in the preauricular region. Furthermore, middle ear reconstruction is generally not attempted in this syndrome secondary to the degree of severity. Hearing aids are often necessary to allow normal speech development and production.

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20

Benign and Malignant Tumors of the Head and Neck

Peter C. Neligan

Summary

This chapter provides an overview of the commonest benign and malignant head and neck tumors. It concentrates specifically on tumors that are unique to the head and neck. It includes a description of salivary gland, endocrine gland, upper aerodigestive tract, and mid-facial tumors. The treatment of these lesions is discussed and examples are given.

Abbreviations

AVM	Arteriovenous malformation
DFSP	Dermatofibrosarcoma protuberans
FNA	Fine-needle aspirate
HPV	Human papilloma virus
MFH	Malignant fibrous histiocytoma
MEN	Multiple endocrine neoplasia
NPC	Nasopharyngeal carcinoma
SLNB	Sentinel lymph node biopsy
SES	Socioeconomic status

Introduction: Benign and Malignant Tumors of the Head and Neck

Creating a catalog of all of the potential benign and malignant tumors of the head and neck is an exhaustive exercise and is beyond the scope of this text. However, there are some tumors, in both categories, benign and malignant, that are common and these are discussed in detail. Probably the most common lesions that we come across are nonmelanoma skin cancers and melanoma. Again, these are discussed elsewhere, and unless there is a particular issue that affects the head and neck, these lesions are discussed only in a very superficial way, once more emphasizing issues that directly relate to the head and neck. Similarly, lesions such as arteriovenous malformations (AVMs), giant hairy nevi, and so forth, which can occur anywhere in the body, are not discussed in detail.

Skin Tumors: Nonmelanoma Skin Cancers and Malignant Melanoma

As already mentioned, these are, for the most part, tumors of the skin as opposed to specifically being head and neck tumors. For purposes of this chapter, all of these are common in the head and neck. Nonmelanoma skin cancers are more common in sun-exposed areas, and the head and neck fall into this category. Similarly, melanomas are common in the head and neck. However, treatment of all of these lesions is similar, no matter in which body region they occur, and so they are not discussed further.

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Tumors of the Salivary Glands

The salivary glands are, of course, unique to the head and neck, so a discussion of salivary gland tumors is particularly relevant to this chapter.

Benign Tumors of the Salivary Glands

Tumors of the salivary glands account for less than 5% of head and neck neoplasms. Although we like to categorize lesions as benign or malignant, there is a certain gray zone in many tumors, and salivary gland tumors are no exception. There are three paired major salivary glands, the parotid, the submandibular, and the sublingual glands. There are also numerous minor salivary glands dispersed throughout the whole of the upper aerodigestive submucosa, including the palate, lip, pharynx, nasopharynx, larynx, and parapharyngeal space.

The parotid is the most common site for salivary gland tumors, accounting for 70% of lesions found. The vast majority of these (75%) are benign. The minor salivary glands account for 22% of tumors, whereas the submandibular gland makes up the rest (8%). In contradistinction to tumors of the parotid gland that are mostly benign, more than 50% of submandibular tumors and 80% of minor salivary gland tumors are malignant.

Pleomorphic Adenoma

The pleomorphic adenoma is the most common type of salivary gland tumor, accounting for almost 50% of all neoplasms in these organs. It is also known as the mixed tumor of the salivary glands, because it contains both epithelial and mesenchymal elements. This is a benign tumor and is found in both major and minor salivary glands. However, the vast majority (85%) of these tumors are found in the parotid gland.[61] The remaining are found in the sublingual (10%) and submandibular glands (5%).[61,66] It is probably best considered as a benign tumor with malignant potential.[5] The rate of malignant change has been reported to be only 2–3%, and only a few cases of metastasizing pleomorphic salivary gland adenomas have been described to date.[31,34] The most usual presentation is a painless lump in the parotid. It is important to get an accurate diagnosis before definitive treatment. This may be done with a combination of histopathologic diagnosis, often based on fine-needle aspirate (FNA) biopsy and imaging with CT and/or MRI.[35,65] There is no place for open biopsy unless through a parotidectomy incision. Treatment is surgical excision. Since most of these occur in the superficial lobe of the parotid gland, this, for the most part, translates to a superficial parotidectomy. Pleomorphic adenomas are surrounded by a pseudocapsule, and for this reason, "shelling out" the lesion is not widely practiced, particularly in North America.[13,39] Monomorphic adenomas are also seen, predominantly in the parotid. The type depends on the histologic characteristics of the cell of origin. The most common type is basal cell adenoma.[45]

Warthin's Tumor

Warthin's tumor is also known as papillary cystadenoma lymphomatosum or adenolymphoma. It is rarely encountered outside the parotid gland.[55] Warthin's tumor is a benign neoplasm accounting for 4–15% of salivary gland neoplasms. It has a preponderance in males and is most commonly found in the 60 and 70 years age group. Warthin's tumors generally present as a painless swelling within the parotid. They commonly occur in the tail of the parotid and are found bilaterally in a small but significant number of cases.[18,70] The etiology of Warthin's tumors is controversial. There is considerable argument about whether they represent true neoplasms, developmental malformations, or exogenously generated mutations.[2,37,66] As with the pleomorphic adenoma, treatment is by surgical excision.

Malignant Parotid Neoplasms

Mucoepidermoid Carcinoma

Mucoepidermoid carcinoma accounts for 5% of all salivary gland tumors and is the most prevalent salivary gland malignant neoplasm. The parotid gland is the most common location, with 66% of tumors found here. The remaining one-third are found in the minor salivary glands. There is a female preponderance, and the majority are seen in the fifth decade. When these tumors arise within the oral cavity, the palate is the most common site. Even though it is most commonly seen in the fifth decade, it is also the most common malignant tumor to arise in children and adolescents under 20 years of age.[55]

The tumor is a firm mass and usually asymptomatic. Low-grade tumors have well-defined glandular elements histologically and rarely metastasize. High-grade lesions can be difficult to differentiate from squamous cell carcinoma because of the paucity of glandular elements, and special stains are required to identify mucin-producing cells.[56] Pain is usually associated with high-grade histology tumors, and they also have a high risk of regional metastases.[17] The prognosis of mucoepidermoid carcinoma is based on the clinical stage and histologic grade. Low-grade lesions are associated with a 5-year survival in excess of 75%. This figure drops significantly with higher-grade tumors and is generally in the range of 20–40%.[17,23,24,56]

Adenoid Cystic Carcinoma

Adenoid cystic carcinoma is the most common malignant neoplasm of the submandibular gland. Adenoid cystic carcinoma is known to have a prolonged and protracted clinical course. It has a propensity for local recurrence as well as pulmonary metastases,[60] and disease-related deaths have been recorded as late as 20 years after original presentation.[59]

Adenocarcinoma

The histological appearance of adenocarcinoma can vary to include papillary, ductal, or mucinous subtypes. There is a significant drop in survival rates between 5 and 10 years, indicating the need for long-term follow-up[58] (Figure 20.1).

Acinic Cell Carcinoma

Acinic cell carcinoma is rarely found outside the parotid gland. It is generally a low-grade tumor but does have the potential for local recurrence and regional metastases.[57,61]

Presentation of Salivary Gland Neoplasms

Generally, these lesions, both benign and malignant, present as painless lumps. FNA biopsy is a useful tool in making the diagnosis. Imaging can also be useful, particularly in situations in which regional metastases are suspected.[35]

Treatment of Salivary Gland Neoplasms

Surgery is the mainstay of treatment for both benign and malignant lesions. For malignant lesions, consideration also needs to be given to treatment of the neck. High-grade lesions and those where there is clinical evidence of regional disease are treated with neck dissection. Furthermore, in the case of malignant neoplasms, radiotherapy is also considered especially for high-grade tumors, in the presence of regional metastases and in situations in which clear margins have not been assured.[22] Currently, there is a lack of effective chemotherapeutic agents for treatment of these diseases, so chemotherapy tends to be reserved for palliative purposes.[55]

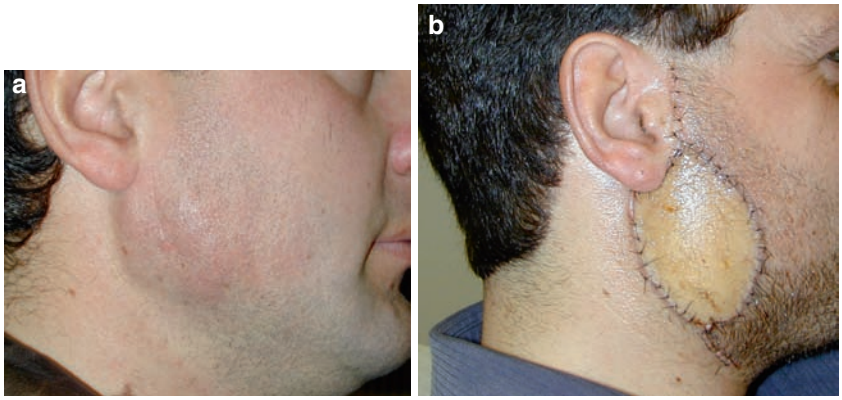

Figure 20.1 (a) Thirty-eight-year-old man with adenocarcinoma ex pleomorphic adenoma of the parotid gland. Note scar from previous pleomorphic adenoma excision directly over parotid (not recommended). (**b**) Early postoperative appearance following parotidectomy with facial nerve preservation and reconstruction with a lateral arm flap. Skin resection necessary because of presence of previous scar and adherence of lesion to overlying skin.

Tumors of the Thyroid and Parathyroid

Benign tumors of the thyroid gland are important only in the need to distinguish them from malignant processes. They have the potential for growth and in rare cases may cause symptoms because of their size and the consequent effect on adjacent structures such as the airway. Rarely, they can undergo malignant transformation.[47] These are all adenomas,[46] and FNA biopsy can be useful in making an accurate diagnosis.[68] Malignant thyroid cancers are relatively uncommon and are particularly uncommon as a cause of death. The risk of developing cancer of the thyroid has been shown to be greater in patients with a history of radiation to the head and neck.[1,14]

Papillary Carcinoma

Papillary carcinoma is the commonest thyroid neoplasm, accounting for 80% of thyroid cancers.[25,65] It is more common in females and tends to occur in the third and fourth decades. It may be multifocal and frequently involves both right and left lobes. There is a 50% incidence of cervical node metastases. Despite this, it is associated with excellent long-term survival with 20-year survival rates quoted as high as 90%.[52]

Follicular Carcinoma

Follicular carcinoma is much less common, accounting for 10% of thyroid neoplasms.[25,68] It also occurs in an older age group than papillary carcinoma, peaking between 40 and 50 years.[30] Like papillary carcinoma, it is more common in women. It is also associated with excellent survival statistics, though not quite as favorable as papillary carcinoma. Ten-year survival is 80–85%, and this drops to 70–75% over the ensuing 10 years.[52]

Medullary Carcinoma

Medullary carcinoma of the thyroid arises from parafollicular cells. These are calcitonin-releasing cells also known as C Cells. Medullary carcinoma accounts for between 5% and 10% of thyroid neoplasms, and it can either arise sporadically or in association with familial multiple endocrine neoplasia (MEN) syndromes.[19]

Anaplastic Carcinoma

As its name implies, anaplastic carcinoma is a highly malignant neoplasm. It generally presents late, often with symptoms of respiratory difficulty, and it is associated with a bleak prognosis.[7,67]

Diagnosis of Malignant Thyroid Neoplasms

As is the case with many malignant lumps, malignant thyroid nodules are frequently hard, fixed, and irregular. Clinical lymphadenopathy is not an unusual finding. Patients may also present with voice symptoms associated with progressive recurrent laryngeal nerve palsy. Ultrasound is a useful tool in the diagnosis of thyroid nodules.[26] The key diagnostic procedure is FNA biopsy.[7,69] It is particularly accurate in differentiating between benign and malignant processes in the thyroid.

Treatment

Thyroidectomy is the mainstay of treatment for thyroid cancers.[68] Neck dissection may also play a role. The role of radioiodine ablation remains an important one.[6,15] External beam radiation also has a role to play and is the mainstay of treatment for anaplastic tumors, where its role is mainly a palliative one. There is currently no role for cytotoxic chemotherapy.[27]

Parathyroid Disease

The vast majority of parathyroid nodules are adenomas and present as hyperparathyroidism. Malignant disease of the parathyroid is extremely rare and is more likely to present as a lump in the neck.

Tumors of the Upper Aerodigestive Tract

Tumors of the aerodigestive tract include those of the oral cavity, the pharynx, and larynx.

Cancer of the Oral Cavity

Carcinoma of the lip can be considered as part of the discussion of oral cavity cancers. Squamous cell carcinoma is the commonest type of cancer seen in the lips. For purposes of

definition, the lips comprise the vermillion, including that portion that contacts the opposite lip. Surgical treatment of lip cancers must consider the function of the lips, and in this instance, restoration of function outweighs aesthetic considerations, although usually it is possible to achieve both.

It is interesting how geography and, more likely, environmental and social circumstances influence the incidence of oral cavity cancer.[16] In the western hemisphere, oral cavity cancer accounts for approximately 5% of all cancers.[30] In addition, in the United States one sees specific trends in the epidemiology of this disease.[32] In the east, and particularly in India, it accounts for 50% of cancers.[16,28] However, going beyond geographic differences, it may be that the common denominator is socioeconomic status (SES), with increased risk of oral cavity cancer being seen in individuals of lower SES regardless of geographic location.[11] However, there are certain recognized risk factors. These include use of tobacco and alcohol as well as various chewing practices including tobacco[42] and betel nuts,[8] not forgetting the practice of reverse cigar smoking in parts of India.[3,8,63] More recently, the role of human papilloma virus (HPV) in the etiology of several cancers, including oral cavity cancers, has been recognized.[67,66]

Most oral cavity cancers arise in the floor of mouth and in the anterior tongue. Cancers can also arise in other areas such as the alveolar ridge, buccal mucosa, and hard palate (Figure 20.2). The vast majority of oral cavity cancers are squamous cell carcinomas. Also, other neoplasms do, however, occur. These include mucosal melanomas, which are rare, as well as neoplasms of the minor salivary glands, as discussed earlier. Other tumors less frequently seen include lymphomas and soft tissue sarcomas. As well, there are a number of premalignant conditions that are important to recognize. These include the leukoplakias, of which there are several varieties. These tend to be very confusing and, depending on the characteristics, can have a higher or lower risk of malignant change. Management includes optimizing oral hygiene, moderating alcohol use, changing diet to reduce exposure to irritants such as spicy foods, and so on.[48] Probably the most important issue is that the presence of leukoplakia demands vigilance and should increase the index of suspicion. In this chapter, the general principles of treatment are discussed rather than getting into the specifics of ablation and reconstruction. The former is beyond the scope of this text, and the latter is discussed elsewhere.

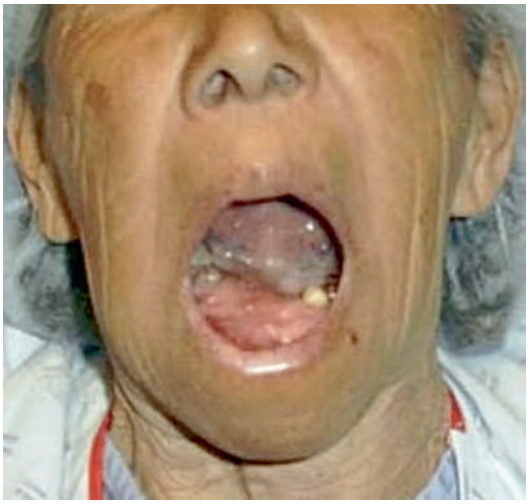

Figure 20.2. Seventy-four-year-old woman presenting with T4 floor of mouth carcinoma arising in the oral mucosa and alveolar ridge. The tumor deeply infiltrates the mandible, requiring segmental mandibulectomy and bilateral neck dissection.

Treatment of Oral Cavity Cancer

Treatment of oral cavity cancer depends on how advanced it is at the time of diagnosis as well as on the location of the primary tumor. Early cancers can easily be ablated by simple surgical excision, whereas more advanced disease may require a combination of surgery, often involving extensive reconstruction, and adjuvant treatment, usually in the form of radiation and/or chemotherapy.

Management of regional disease is always an issue and must always be considered when dealing with oral cavity cancers. Use of sentinel lymph node biopsy (SLNB) is reasonably well established as part of the evaluation of head and neck melanoma.[21] Its use in the staging of squamous cell carcinoma is less well established. However, there is considerable interest in this technique, and it is the subject of large multicenter trials. It is very likely that SLNB will become indicated for T1 and T2 oral cavity squamous cell carcinoma with N0 necks, and it is possible that the indication will extend to all early

stage head and neck squamous cell carcinomas.[12] For established neck disease, neck dissection is indicated. The specific type of neck dissection will be dictated by the extent of clinical disease as well as specific characteristics of the tumor, including T stage, depth of invasion, histological grade, and tumor morphology.

Tumors are classified according to the TNM classification system and staged based on TNM groupings. Five-year survival figures deteriorate with advanced stage and vary between 70% and 80% survival for Stage I disease to 10–20% for Stage IV[48] (see Table 20.1).

Tumors of the Nasopharynx

The majority of tumors arising in the nasopharynx are nasopharyngeal carcinomas (NPCs) NPC is relatively rare in the west; however, it is endemic in Southern China and is also seen in Chinese communities in other parts of the world. It is also seen in other distinct Asian communities as well as in the Arctic.[9] The annual incidence of NPC in western societies is 0.5/100,000 as opposed to an annual incidence of 50/100,000 in Southern China including Hong Kong. Interestingly, Chinese ethnicity seems to also be a positive prognostic indicator in this disease.[44] There is a close association between NPC and Epstein–Barr Virus.[33] The most common presentation is that of a lump in the neck. This illustrates NPC's proclivity for lymphatic spread. The primary tumor may cause nonspecific nasal symptoms, such as intermittent blockage of the airway, discharge, or bleeding. In general, the prognosis is not good. Treatment is not primarily surgical, but NPC is more commonly treated with radiotherapy ± chemotherapy, and there is some controversy with regard to optimal treatment.[29]

Table 20.1. Staging of oral cavity cancer.

Stage grouping			
Stage 0	Tis	N0	M0
Stage I	T1	N0	M0
Stage II	T2	N0	M0
Stage III	T3	N0	M0
	T1	N1	M0
	T2	N1	M0
	T3	N1	M0
Stage IVA	T4a	N0	M0
	T4a	N1	M0
	T1	N2	M0
	T2	N2	M0
	T3	N2	M0
	T4a	N2	M0
Stage IVB	Any T	N3	M0
	T4b	Any N	M0
Stage IVC	Any T	Any N	M1

Source: Used with the permission of the American Joint Committee on Cancer (AJCC), Chicago, IL. The original source for this material is the *AJCC Cancer Staging Manual*, 6th Edition. Springer Science and Business Media LLC; 2002. Available at www.springerlink.com.

Tumors of the Oropharynx

Tumors of the oropharynx are far less frequent than tumors of the oral cavity and, as in the oral cavity, squamous cell carcinoma is the commonest malignant tumor seen. The difficulty with this anatomic region, however, is that it is at the divergence of the digestive tract and the airway so that the functional effects of tumors and their treatment can have far-reaching results, and quality of life can be significantly affected by both disease and treatment.[10] The intrinsic role of the posterior pharynx in the mechanics of swallowing makes this region unique, and tumors in this region can have their effect on swallowing. For the same reason, this is a difficult region for the reconstructive surgeon. The role of HPV in the etiology of head and neck squamous cell carcinomas in general and oropharyngeal and laryngeal carcinoma in particular cannot be ignored.[38,64]

Apart from squamous cell carcinomas, other tumors seen in the oropharynx include lymphomas arising from the lymphoid tissue in the tonsils and tongue base as well as tumors of the minor salivary glands.[40,62]

These tumors are often advanced at the time of presentation, as the early symptoms tend to be vague and are often dismissed. These symptoms include discomfort in swallowing as well as otalgia. Up to 70% of patients present with Stage III or IV disease, with a high incidence (>65%) of nodal metastases.[53] Treatment typically combines surgical resection and radiotherapy.

Tumors of the Larynx

As with the oropharynx, tumors arising in the larynx can have profound effects on swallowing, breathing, and speech. The vast majority of tumors affecting the larynx are squamous cell carcinomas. More rarely other tumors are also seen. These include chondrosarcoma, mucoepidermoid carcinoma, and mucosal melanoma. Major risk factors include alcohol and tobacco. HPV is now also recognized as a potential associative factor.[66] TNM staging is also used for laryngeal cancers, although in the case of the larynx, it is possibly less useful than at other sites (Table 20.2).

Treatment of Laryngeal Tumors

Treatment of early lesions is frequently by radiotherapy, with surgery reserved for radiation failures and more advanced cancers.[40] Endoscopic laser resection also has a valuable role to play in early lesions.[4] Surgery has traditionally meant laryngectomy, but it is being increasingly recognized that laryngeal preservation surgery and reconstruction have a place.[20,36]

Table 20.2. Staging of laryngeal tumors.

Stage grouping			
Stage 0	Tis	N0	M0
Stage I	T1	N0	M0
Stage II	T2	N0	M0
Stage III	T3	N0	M0
	T1	N1	M0
	T2	N1	M0
	T3	N1	M0
Stage IVA	T4a	N0	M0
	T4a	N1	M0
	T1	N2	M0
	T2	N2	M0
	T3	N2	M0
	T4a	N2	M0
Stage IVB	T4b	Any N	M0
	Any T	N3	M0
Stage IVC	Any T	Any N	M1

Source: Used with the permission of the American Joint Committee on Cancer (AJCC), Chicago, IL. The original source for this material is the *AJCC Cancer Staging Manual*, 6th Edition. Springer Science and Business Media LLC; 2002. Available at www.springerlink.com.

Sarcomas of the Head and Neck

Sarcomas account for less than 1% of head and neck malignancies. The fact that they are so uncommon frequently leads to delays in diagnosis. The prognosis for sarcoma varies depending on histology, grade, location, and size of the primary tumor. In one large series, 5-year survival is quoted as 62%, with a local recurrence rate of 41% and a distant recurrence rate of 31%.[34]

Head and neck sarcomas are categorized under two broad headings, those arising from soft tissues and those arising from bone. Staging uses the standard TNM classification system. However, lymph node involvement in sarcomas is relatively rare. Soft tissue sarcomas most commonly seen in plastic surgical practice include dermatofibrosarcoma protuberans (DFSP) and malignant fibrous histiocytoma (MFH), probably because of cutaneous involvement. Diagnosis is by a combination of clinical examination, imaging, and biopsy. FNA biopsy may be helpful, but open biopsy is the gold standard and should be designed in such a way as to facilitate excision of the biopsy scar at the time of definitive resection. Primary treatment is surgical resection. Radiation is used in situations in which surgical clearance is difficult or has been unsuccessful. It is also used in the treatment of recurrent disease.[40] Neoadjuvant chemotherapy is used in the treatment of osteogenic sarcoma.

Other Tumors

Odontogenic Tumors

The commonest odontogenic tumor encountered is the ameloblastoma. These tumors most commonly occur in the mandible but can also be found in the maxilla.[41] Unicystic and multicystic varieties occur, and the multicystic variety is commonest. Although the ameloblastoma is benign, it is a locally aggressive odontogenic neoplasm. It is most commonly seen in the third decade and affects males and females equally. Surgery is the mainstay of treatment, with the optimal treatment being wide en bloc resection. Radical resections, including marginal and segmental mandibulectomy, result in local control

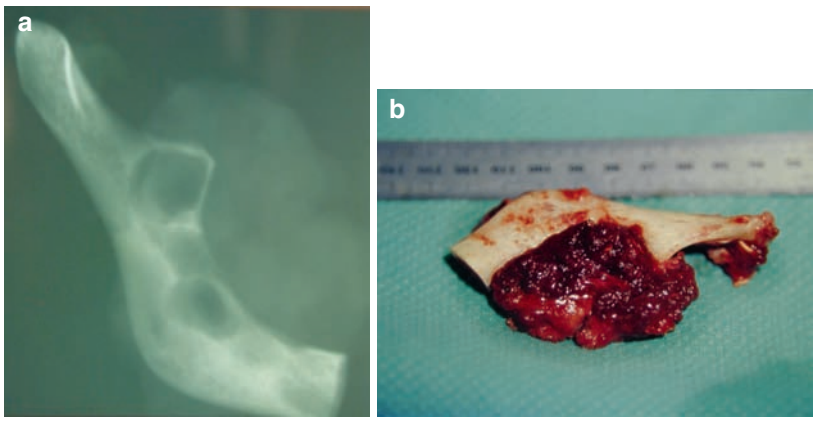

Figure 20.3. (a) X-ray of resected mandibular ramus and condyle containing multicystic ameloblastoma. (**b**) The resected specimen.

rates exceeding 90%. Curettage results in lower control rates of 80% for unicystic lesions and only 50% for multicystic lesions and is not recommended[41,50] (Figure 20.3).

Sinonasal Tumors

Tumors occurring in the sinonasal region are probably the most diverse of all the regions of the head and neck. Although squamous cell carcinoma is the commonest encountered, other tumors, both benign and malignant, are also found here. Most are epithelial or ectodermal in origin, but mesenchymal tumors, both benign and malignant, also occur[39] Some of these have already been discussed. Because of the unique anatomy of this region, the ectodermal lesions include squamous tumors and adenomas as already discussed. They also include neuroectodermal tumors, such as meningiomas, neurofibromas, and gliomas. As already mentioned, the odontogenic tumors are also ectodermal in origin.

Mesenchymal tumors arising in this region include tumors, both benign and malignant, of all tissue origin, including vascular, muscular, cartilaginous, osseous, and lymphoreticular.[39] All of these tumors are rare and are not discussed in further detail.

Conclusion

The head and neck is a complex region. It is unique in having so many vital structures in close proximity. For this reason, the types of tumors encountered here are diverse, and from the plastic surgery perspective, the defects created by resection of these tumors pose significant challenges to our reconstructive abilities.

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21

Craniofacial Trauma and Reconstruction

Chien-Tzung Chen, Ruei-Feng Chen, and Fu-Chan Wei

Summary

The concept of treatment of craniofacial injury has evolved from conservative, delayed, multiple-staged surgery into early, aggressive, and one-stage operation. Adequate exposure, accurate anatomic reduction, rigid fixation, primary bone grafting, and soft tissue suspension remain the gold standard to obtain expected results. A variable plating system applied to specific anatomic areas of the facial skeleton produces three-dimensional (3D) reconstructions, enhances bone healing, and decreases infection. Minimal invasive surgery replaces part of conventional wide incision and achieves competitive results. Microvascular free tissue transplantation makes reconstruction of devastating injury on the face possible, with a more pleasing outcome. Alloplastic implants become more popular especially for orbital reconstruction and produce equivalent results compared with conventional bone grafts. Contemporary computed tomography (CT) scans gradually replace conventional plain films to provide more accurate diagnosis and can be used as a preoperative simulation tool. With the advancement of diagnostic imaging, surgical approaches, and instruments, optimal functional outcome and aesthetic facial appearance can be achieved.

Abbreviations

MMF	Maxillomandibular fixation
NOE	Naso-orbito-ethmoidal
TMJ	Temporomandibular joint

Introduction

Management of craniofacial trauma is a great challenge confronting reconstructive surgeons. Reconstruction of the deformities and defects emphasizes both restoration of optimal function and appearance. Improper treatment of facial injuries results not only in functional problems but also in facial disfiguration, which may lead to serious emotional and social problems. The conventional concepts of delayed surgery, use of small incisions, minimal exposure of bony fragments, nonrigid wiring or external fixation, and minimal attention to primary soft tissue management have dramatically evolved into early one-stage repair, wide exposure of all fracture segments, rigid-plate internal fixation, and definite soft tissue management, after those new craniofacial techniques developed in the late 1970s and in the 1980s.[32,39,59,68] Although these techniques improve the outcome of craniofacial trauma largely, some adverse sequelae such as soft tissue damage and contracture may occur. In recent years, minimal invasive techniques and better surgical instruments that have emerged

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help to minimize surgical complications yet produce equivalent or even superior outcomes.[22]

Mechanism of Injury

The causes of facial injuries include motorcycle accidents, motor vehicle accidents, assaults, athletic injuries, industrial accidents, and falling down accidents. The epidemiology of facial trauma varies with the geographic region, population density, socioeconomic status, and historical year. Motor vehicle accident is the most common cause for all age groups in the United States.[35]

The mechanism of injury usually identifies the probable energy of impact and the likely extent of injury. Details of the nature of the traumatic force, and its direction, will aid in predicting fracture patterns.

General Considerations

Evaluation of the Multiply Injured Patient

While dealing with facial trauma, the first priority is to give an overall picture of the injury and find out life-threatening problems including airway, breathing, and circulation. Keeping airway patency in the setting of significant maxillofacial injuries is mandatory. Airway compromise can occur directly and rapidly as a result of facial injury or secondarily due to soft tissue swelling around oronasal structures. The "horseshoe" configuration of the mandible suspends the tongue, and loss of this suspension in mandible fracture may cause tongue drop and airway obstruction (Figure 21.1). In the event of orotracheal intubation, cervical spine injury should be ruled out before the procedure. Nasotracheal intubation is contraindicated in maxillofacial injuries with associated skull base fracture.

Life-threatening bleeding following maxillofacial trauma is rare, with the incidence around 1.25–9.4%.[71] Internal maxillary artery and its branches are the major origin of bleeding in maxillofacial trauma. Rapid resuscitation followed by anterior and posterior nasal packing is usually sufficient to control bleeding. Angiography with selective embolization of bleeding vessel is the procedure of choice in those who fail with nasal packing.

The incidence of associated brain injury in patients with facial trauma ranges from 5.4% to 55%.[20] Glasgow Coma Scale (GCS) is commonly used in adult patients to evaluate the present conscious status. Internal carotid injuries with subsequent large brain infarction in facial fractures have been reported.[69] Therefore, CT of the brain and facial bone should be taken at the same time in case of facial trauma with abnormal GCS to identify possible lethal brain injury.

Cervical spine injuries occur in 0.9–6.7% of facial trauma.[26] Patients who have penetrating trauma to the neck, disturbances in sensation or motor function, or are unable to move their extremities by order must be assumed to have cervical spine injury. Cervical spine radiography

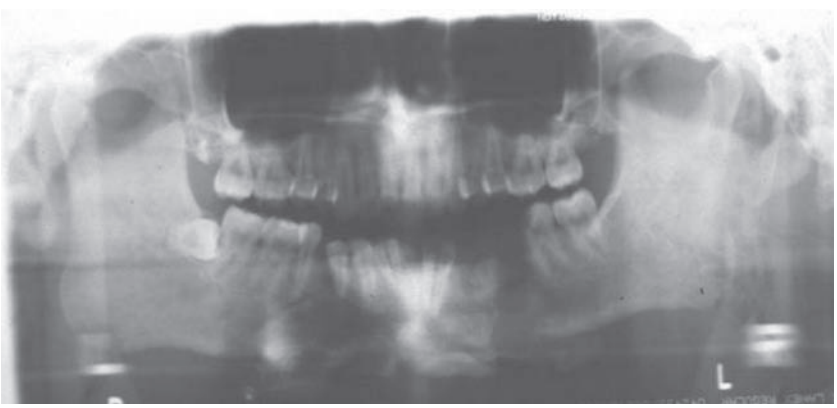

Figure 21.1. Panorex view showing mandible symphysis and body comminuted fractures with collapsed mandible arch.

at lateral and anterior–posterior views can be used as initial screen, but C1–C2 and C6–T1 are the most commonly missed lesions. Spinal CT is more accurate than plain radiographs and requires less neck movement

Face and Facial Skeleton Evaluation

The skull, face, eyes, ears, nose, tongue, and mouth are inspected step by step. Facial open wound should be paid more attention, because the fractured sites are usually just underneath the open wound (Figure 21.2). It is of particular importance that the eyes should be inspected first to exclude the possibility of traumatic optic neuropathy. Damage to facial sensory nerves is identified by light touch perception. Specific regions of interest include the forehead (supratrochlear and supraorbital nerves), the cheek and upper lip (infraorbital nerve), and the lower lip (mental nerve). Facial nerve function can be tested by several actions such as brow elevation, eye closure, smiling, and pursing the lips or whistling. Temporal bone fracture should be considered if facial paralysis comes without an open wound.

Swollen periorbital and ecchymosis are usually the initial symptoms of orbital fracture. Palpation around the orbital rim should be done slowly and carefully. Uneven orbital surface or step deformity indicates orbital rim fractures found frequently at the zygomatic-frontal junction or infraorbital rim. Careful inspection of nasal dorsum and tip may disclose deviation or depression. However, it may be obscured by tissue swollen at early onset of trauma. Palpation of the nasal bones is crucial to elicit stability and mobility of fractured nose. Displacement of the septal cartilage, mucosal tears, and septal hematoma are all assessable by intranasal examination. Integrity of the medial canthal ligament may be perceived by applying lateral traction on the lateral canthus.

The anterior wall of the maxilla is palpated through the buccal sulcus. Abnormal movements of the mid face, indicating Le Fort fracture, can be elicited by grasping the maxillary alveolar ridge and applying right and left movements while holding the forehead with another hand. Intraoral examination, including occlusion status, tooth fracture or missing, and palatal laceration, should also be recorded. The presence of missing

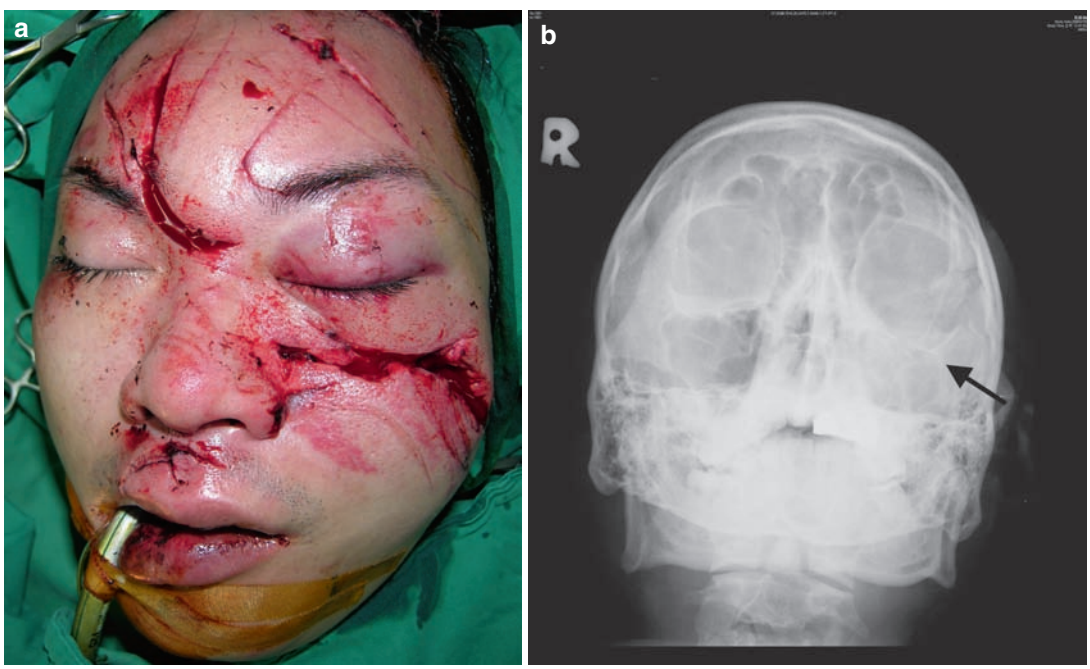

Figure 21.2. (a) Male presenting with multiple facial laceration. (**b**) Waters film revealed left infraorbital rim fracture just beneath the left cheek and lower eyelid open wound.

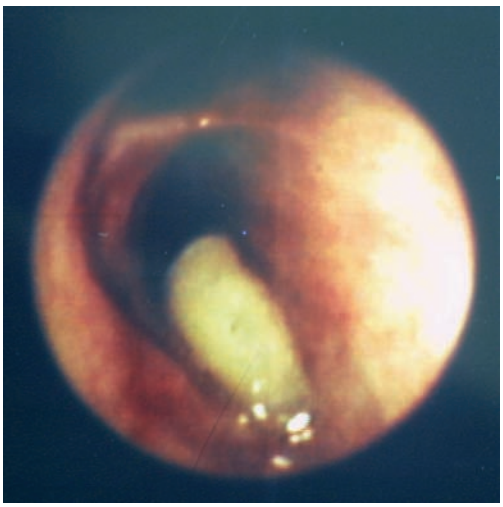

Figure 21.3. Tooth in trachea after severe mandible fracture and careless endotracheal intubation.

teeth needs to be carefully searched in the oral cavity, because they may be pushed into the trachea during careless endotracheal intubations (Figure 21.3). The stability of the mandible may be detected by applying up and down manual pressure on the anterior portion of the mandible. Pain, crepitus, and instability may indicate the possibility of mandible fracture when this maneuver is performed. Loss of condylar head movement, detected by placing an examining finger beneath the zygomatic arch, may imply possible condylar fracture.

Ophthalmologic Examination

The presence of vision is confirmed by covering each eye. The response of the pupil to light is checked both directly and using consensual stimuli with a penlight. The Marcus Gunn sign suggests the paradoxical dilation of the pupil owing to loss of direct light reflex in optic nerve injury. Snellen chart and reading test quantify the severity of visual loss. The visual field can be assessed by confrontation. Eye movements are evaluated by asking the patient to fix on a distant object in different directions. This may elicit diplopia or ocular misalignment. Forced duction test is performed after a topical anesthetic drop to distinguish muscular entrapment from neurological or swollen effect. A positive response suggests entrapment or orbital tissue or restriction of one of the extraocular muscles. Paralytic strabismus may occur after facial trauma that is caused by injury of the third, fourth, or sixth cranial nerve, alone or in combination. Proptosis or enophthalmos should be aware of and measured with exophthalometer if the lateral canthal area is not fractured. Injuries of lacrimal system should be suspected in any laceration around the punctum. If there is unusual eyelid swelling, it is auscultated to detect a bruit. Carotid cavernous sinus fistula may happen immediately or delay several days to weeks after trauma.

Diagnostic Image

Careful clinical examination not only predicts the type of injury but also guides the selection of radiographic studies. Conventional plain films are accessible, cost effective, and provide direction to further CT studies.

Plain Film

The standard radiographic facial series consists of four views: the Waters (occipitomental projection), Caldwell (occipitofrontal projection), lateral, and submentovertex (base) views. The Waters view is perhaps the most valuable of the routine facial series. It provides optimal visualization of the mid-face region, the orbital rim and floor, nasal bones, zygoma, and maxilla. The Caldwell view provides visualization of the superior orbital rim, frontal sinuses, and orbital region, although the orbital floor is often obscured because of bony overlap. The lateral view is useful for evaluation of the frontal sinus and maxillary sinus and detecting fractures through the pterygoid plate, which occur in Le Fort fractures. The submentovertex view provides best visualization of the zygomatic arches. Whenever there is any suspicion of mandibular fractures, panoramic radiography is considered first. The panoramic view is obtained by rotating the x-ray beam and the film around the patient's head to show the entire mandible. The only limitation of panoramic radiography is that anterior fractures can be missed if their fragments override. The Towne's radiograph is a supplement to diagnose the fracture of subcondylar area and assess the direction (medial or lateral) of the condylar process.

Computed Tomography and Magnetic Resonance Image

Although plain film radiography demonstrates absolute evidence of the bone injury, it cannot provide adequate information for precise diagnosis and planning preoperatively. Computed tomography offers superior accuracy compared with plain radiographs in the diagnosis of maxillofacial injury due to the ability of CT scanners to acquire multiple nonsuperimposed, cross-sectional images. The data obtained from a CT scan (Figure 21.4) can be reconstructed in multiplanar and 3D to provide more useful information. Axial, coronal, and sagittal views (2D-CT) are frequently employed to clarify the degree of comminution and displacement of fractures. Although 3D CT images do not provide more detailed information than 2D CT images, they can help surgeons to explain the fracture pattern

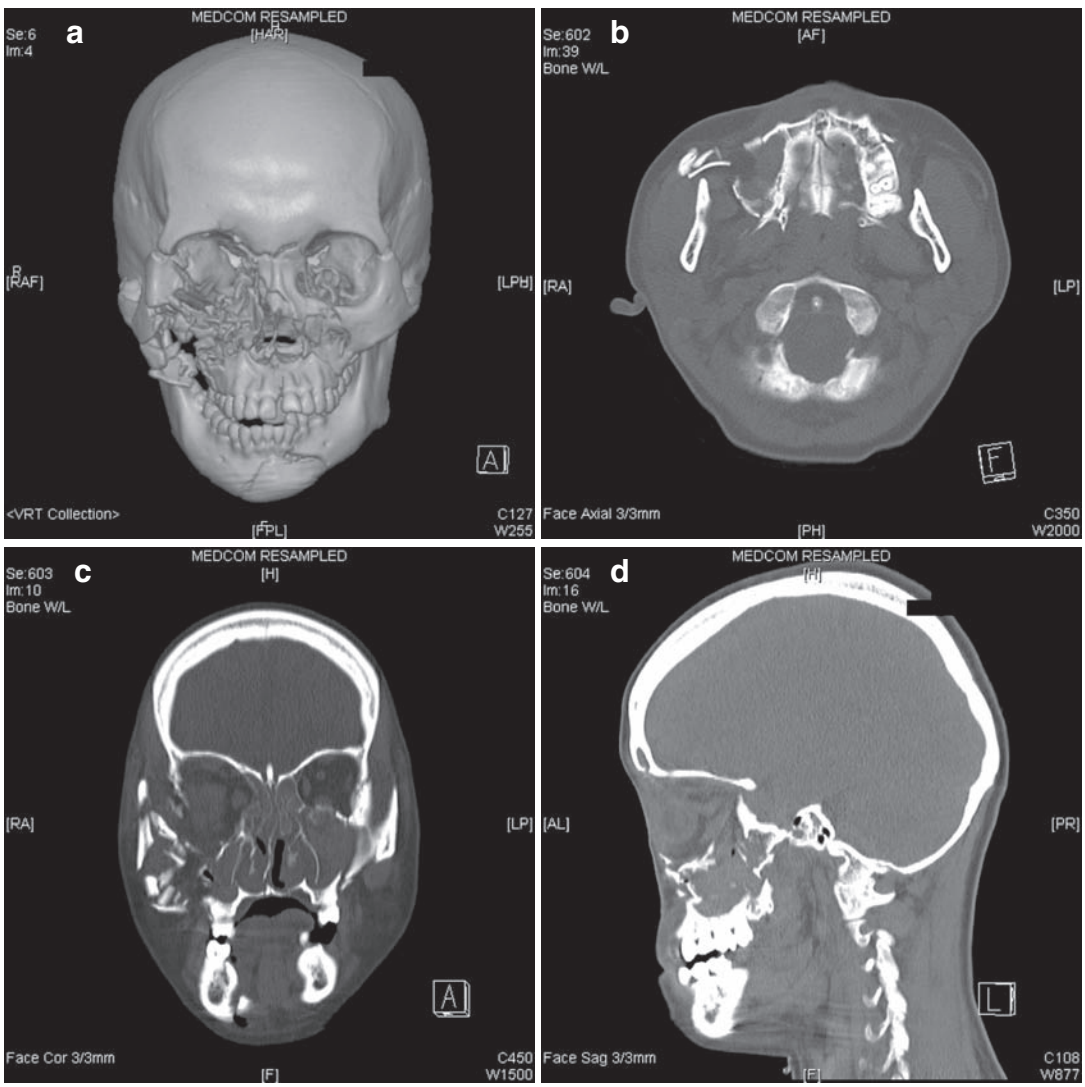

Figure 21.4. CT scan of a complex orbit, zygoma, maxilla, palate, mandible, and naso-orbit-ethmoidal fractures. **(a)** 3D CT scan. (**b**) Axial view of CT scan. (**c**) Coronal view of CT scan. (**d**) Sagittal view of CT scan.

and degree of fracture displacement to patients or their family. Nowadays, CT has become the imaging gold standard for assessing injuries to all regions of the maxillofacial skeleton. In contrast, magnetic resonance image (MRI) has little role in imaging of fracture of maxillofacial skeleton, but it may have an adjunctive role to CT in the assessment of soft tissue injury such as disc of temporomandibular joint (TMJ) in condylar fracture. Choi et al.[18] found that the disc was medially dislocated out of the fossa when the condyle fracture was medially displaced from the fossa.

Soft Tissue Injury and Management

General Principles

The types of soft tissue injuries include abrasions, tattoos, simple or complex lacerations, bites, avulsions, and defects. The principles of wound management consist of cleaning, irrigation, adequate debridement, and minimal tension closure. Any dirt on the wound should be carefully scrubbed out and cared with moist dressing until disappearance of serous discharge from the wound (Figure 21.5). Some wounds will benefit from local flap for closure; some wounds will even require free tissue transfer for complete restoration of function and appearance.

Direct Repair

Lacerations that involve the lid margin require careful closure to avoid lid notching and misalignment. Injuries that involve full-thickness loss of 25% of lid can usually be closed primarily. Any laceration to the medial third of the eyelid should suggest a canalicular injury, which should be carefully identified and repaired. Disruption of either the medial canthal ligament or lateral canthal ligament should be repaired primarily to avoid telecanthus and shortening eye fissure.

When facing a patient with cheek laceration, the primary concern is for possible injury to the underlying structures such as facial nerve, parotid gland and duct, and facial bone. The diagnosis of parotid duct injury can be confirmed by injection of methylene blue into the orifice of parotid duct with evidence of leakage of fluid from the wound. Duct injuries are usually repaired over a stent to allow healing. When there is any deficit in facial motion, facial

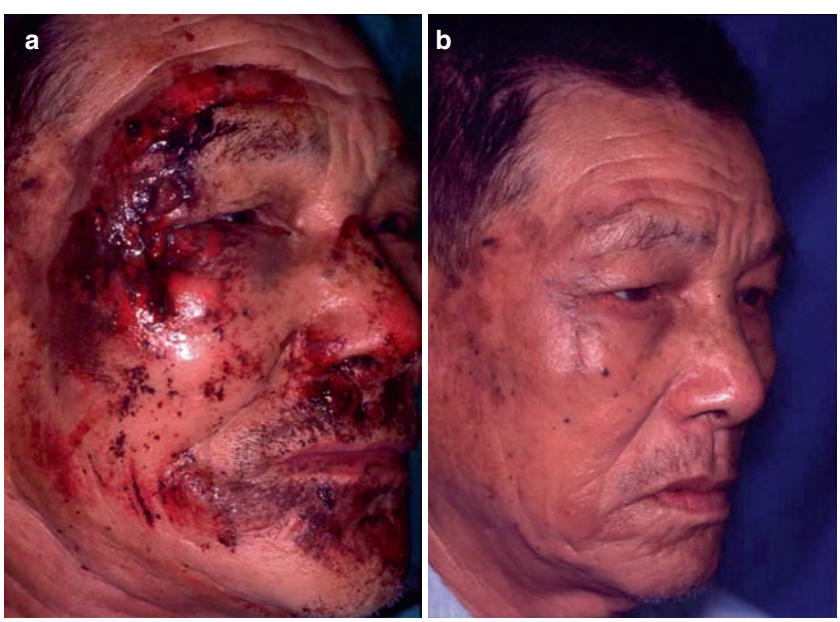

Figure 21.5. Multiple facial laceration and abrasion with dirt and traumatic tattoo. (**a**) Preoperative appearance. (**b**) Postoperative appearance after adequate debridement and meticulous wound repair.

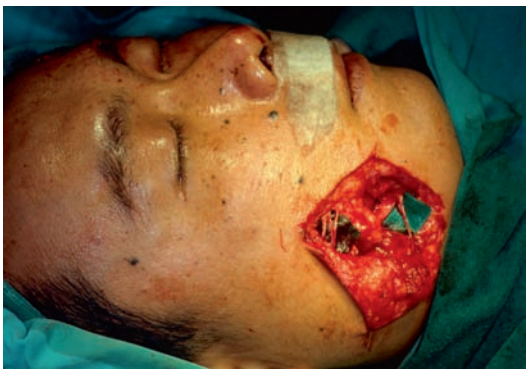

Figure 21.6. Facial nerve branches are identified and repaired in cheek laceration.

nerve should be explored and repaired as early as possible (Figure 21.6).

Auricular hematoma may result in fibrotic change of ear cartilage with subsequent ear deformity. Therefore, the hematoma should be evacuated through surgical procedure as soon as possible. Lacerations of the ear can usually be sutured primarily without placing a separate layer of sutures within the cartilage because of firm adherence between the skin and the cartilage framework.

Nasal lacerations should be repaired primarily when possible, beginning with the nasal lining and then proceeding to external structures. A septal hematoma should be removed to prevent subsequent infection, necrosis, organization of the clot into a calcified, subperichondrial fibrotic mass. Lip repair must focus on oral competence, adequate month opening, sensation, complete skin cover, oral lining, and the appearance of vermilion to avoid prominent cosmetic defects.

Regional Flap

Partial-thickness defects of eyelid can be repaired using full-thickness skin grafts (FTSG). If the defects of upper eyelid are between 25% and 75%, the choice of reconstruction is a sliding tarsoconjunctival flap. The lower-lid switch flap with a cheek rotation-advancement is chosen for reconstruction of upper eyelid defect greater than 75%.[45] Regarding the lower eyelid, full-thickness defects less than 50% of lid length can be closed primarily with cantholysis and local tissue advancement. A sliding tarsoconjunctival flap with a skin graft can also be applied for the defects involving 50–75% of lid length. When the defects are greater than 75%, the choice of restoration of the anterior layer is a cheek flap, and the posterior layer is restored by a composite graft harvested from nasal septal cartilage and inner lining.[54]

A defect along the medial cheek and alar base can be repaired primarily with a V-Y advancement flaps. For those medium defects of cheek, transposition flaps such as banner flap, bilobed flap, and rhomboid flap are ideal choices. Cervicofacial rotation flaps with various designs are useful for reconstruction of moderate to large defects of the upper medial cheek.[2]

When the ear is injured with partial defect, the upper size limit for direct closure after wedge resection is 1.5 cm. Following rim advancement, an Antia–Buch flap is useful for helical rim reconstruction.[3] The Converse's tunnel procedure can be used for helical defects larger than 3 cm. Whenever there is a middle third of auricular defect, the postauricular flap based on the edge of the hairline can be raised, and the free margin of the skin flap is sutured to the anterior edge of the defect. This flap is divided 2–3 weeks later. Defects in the lower third of the auricle and earlobes can be reconstructed using local soft tissue flaps.

Traumatic nasal defect, with underlying cartilage, septum, or bony structure exposure, is better repaired with a local flap. A banner flap is essentially a transpositional flap that can be used for defects less than 1.2 cm in diameter. A bilobed flap is designed at 90°–100° to use the laxity of skin in the upper third of the nose to cover the more caudal defects. A nasolabial flap is a versatile flap for reconstructing portions of the nasal lobule and the sidewall. The donor site can usually be closed primarily. If there is deficiency of useful local tissue for reconstruction, a remote flap such as forehead flaps can be elevated based on either the supratrochlear or supraorbital pedicle. It is a versatile workhorse flap for large tip defects and subtotal or total nasal reconstructions.

The importance of the lip is that it maintains oral secretion to prevent drooling and acts as a dam. Defects up to 30% of the upper or lower lips are still possible to be approximated directly. When the defect involves one-third to two-thirds of upper lip, an Abbe flap with perialar crescentic excision can be used for central defect of the

upper lip.[1] The Estlander flap is usually used for lateral defects of the upper lip. As for lower lip defects at the same degree, a Karapandzic flap is the first choice if the commissure is involved.[33] For lip defects greater than two-thirds of upper lip, the defects can be repaired with Bernard procedures[62] if sufficient cheek tissue is present. On the other hand, a Karapandzic flap is useful for large lower lip up to 80%.

Microsurgical Free Flap

For patients with complex and extensive facial defects, or contour deformities, which cannot be adequately reconstructed with these techniques, a free tissue transfer becomes necessary. The following are the most commonly used flaps for soft tissue coverage.

A radial forearm flap (Figure 21.7) is a good choice for cheek reconstruction because of its thin and pliable nature with long and reliable pedicle. It is a fasciocutaneous flap based on the radial artery. It can be a sensate flap if the lateral antebrachial cutaneous nerve is repaired. The disadvantages of the flap include donor-site morbidity and color mismatch.

A parascapular flap is a fasciocutaneous flap based on the circumflex scapular artery. Compared with the radial forearm flap, it is more suitable for the reconstruction of extensive defects of cheek because of larger dimension and thicker flap.

Latissimus dorsi muscle can be also included on the same pedicle. However, it requires reposition of the patient.

The anterolateral thigh flap is the most popular flap in our practice because of its high applicability. The pedicle is based on the perforating vessels of the descending branch of the lateral circumflex femoral artery. The flap can be thin and pliable or bulky with inclusion of variable amount of vastus lateralis muscle. The advantages of the flap include constant anatomy, adequate pedicle length and vessel diameter, and the versatility of flap design with minimal donor-site morbidity.[64] Color match and hair bearing may be the drawbacks of the flap.

Management of Craniofacial Fracture

General Principles

In general, definite treatment of craniofacial fracture will be scheduled in optimal condition after correction of shock, dehydration, and electrolyte imbalance and stabilization of other associated trunk trauma such as hemopneumothorax, visceral organ injuries, and spine injury. Choice of surgical intervention depends on displaced degree of fracture site, clinical symptoms and signs, patient's dentition, age, and associated medial disease. For instance, minor orbital fracture without enophthalmos or limitation of extraocular muscle movement is treated conservatively. In contrast, a small orbital trapdoor fracture with entrapment of orbital tissue should be managed by open reduction. The detailed

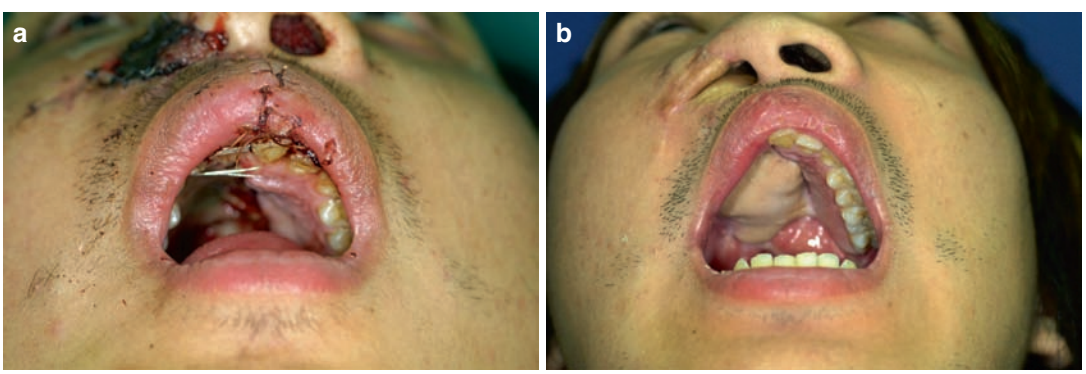

Figure 21.7. Forearm flap for reconstruction of facial defect. (**a**) Facial trauma with right palate, nasal alae, and base soft tissue necrosis. (**b**) Free forearm flap is transferred to reconstruct these soft tissue defects.

surgical plane includes the location of incision placement for direct fracture exposure, selection of proper plate for rigid internal fixation, and the necessity of primary bone graft or alloplastic implant reconstruction. Several incisions based on aesthetics, morbidity, ease of dissection, and extents of exposure have been commonly used to get access to the craniofacial skeleton. The coronal incision commonly exposes the cranium, zygomatic body and arch, orbit and nasoethmoidal area. The subciliary or transconjunctival incision is used to approach the orbital rim, floor, and anterior zygoma. The upper gingivobuccal incision exposes the maxilla and zygomatic body. The lower gingivobuccal incision provides exposure to the mandible. The condylar process fracture is usually approached via pretragal or Risdon incision. Inadequate exposure of the fracture fragments poses the difficulty of assessing the relationship between fracture segment and intact bone and can result in inaccurate reduction with a sequel of secondary deformity.

Plate Selection

The use of screws and plates providing rigid fixation has revolutionized the care of craniofacial trauma. They provide accurate and three-dimensional stability for the bony framework with much decreased secondary relapse rate. Rigid internal fixation facilitates primary bone healing and also assists resolution of infection. It is necessary to have two tight screws in each major bone fragment for rigid internal fixation.

The choice of proper hardware for internal fixation depends on anatomic position of fracture sites, the fracture line, and comminuted status of fracture fragments, and the necessity of primary bone grafts. Each case regarding the fracture classification and the minimal amount of hardware necessary to resist external force must be considered. Of all the facial bones, the mandible has the greatest potential for fracture displacement caused by the exertion of the mastication muscles. When injuries result in comminuted or defect fractures of the mandible with minimal bone contact, a load-bearing fixation device is required to bridge the injured area and resist the forced generated by the masticatory system. The most commonly used load-bearing device is a mandibular reconstruction bone plate accompanied by 2.3-, 2.4-, or 2.7-mm screws. However, minimal three bone screws are required to provide stability of the bone fragments. Besides, the bone plates will usually fail in time if the missing bone is not replaced with bone grafts or the comminuted bony fragments have not consolidated by time.

Fortunately, most mandibular fractures are simple, liner fractures with solid bony fragments on each side of fracture bearing some of the functional load, and a load-sharing device including a variable 2.0-mm miniplate system is applied to achieve adequate stability. Lag screw techniques are also load bearing and provide more compression force across the facture line to facilitate bone healing although technically demanding.

When facing the supramandibular facial and skull fractures, 2.0-mm miniplates are universally used to stabilize the bony mid-facial and upper facial structures. An advantage of miniplate fixation is the ease with which multiple fracture fragments can be aligned within three-dimensional space. The miniplate can be bent first using the normal site as a reference to fit the usual configuration of the region, and the rest of the fragments are aligned serially and fixed to the prefabricated plate. Titanium miniplate has malleability yet would not lose sufficient strength to resist local muscle force and wound contraction. Recently, the microplate system has been developed to assist more rigid fixation for small fracture segments. The low-profile microplate is particularly useful in areas with thin overlying soft tissues such as orbital rim, zygoma arch, and frontal area.

Graft Material

Bone Graft

The gold standard to restore absent bone in facial skeleton is to replace the defect with autogenous bone graft. Primary bone grafts help to prevent soft tissue contracture and subsequent secondary deformity. The common sources of bone grafts include calvarial skull bone (membranous bone origin), rib, and iliac crest (endochondral bone origin). Splitting the intact skull provides bone for small- to medium-size bone defects. In case of extensive craniofacial defects, splitting the craniotomy bone flap provides a large source of cranial bone (Figure 21.8). Clinical observation and experimental study have shown a less resorption rate in grafts of

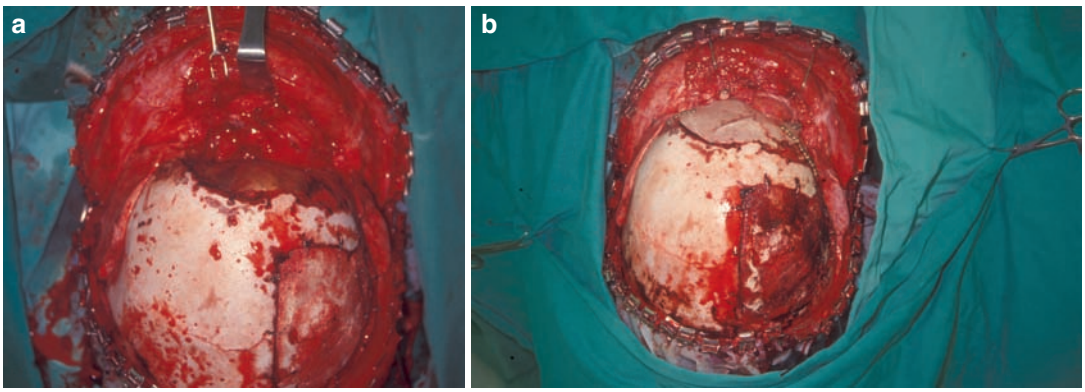

Figure 21.8. Calvarial bone graft used for reconstruction of large frontal bone defect. (**a**) Bone defect at frontal skull bone. (**b**) Outer table of parietal craniotomy bone flap used to repair frontal bone defect with rigid-plate fixation.

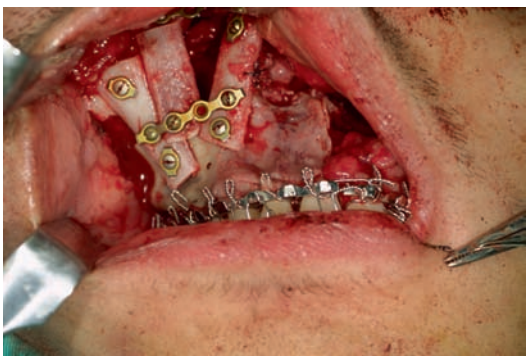

Figure 21.9. Rib grafts used to reconstruct the maxillary buttresses with miniplate rigid fixation.

membranous bone when compared with those of endochondral bone origin. However, calvarial bone is more brittle and hard to be contoured to adapt to the complex structure of the internal orbit. Rib graft is easier to bend and contour and is most commonly used for reconstruction of orbital rim and cavity, zygoma, and maxilla (Figure 21.9). Iliac crest grafts provide enough bone sources but may resorb unpredictably.[56] No matter what kind of bone grafts are used, they should be rigidly fixed with either miniplates or lag screws to ensure correct position and decrease their resorption rate. The main disadvantages are potential donor-site morbidity, longer operation time, and unpredictable graft resorption.

When there is a segmental defect, especially associated with inadequate coverage either in the mandible or in the maxilla, the result of reconstruction with these techniques becomes unreliable. Microsurgical vascularized bone graft provides a good solution for this problem. The donor sites of vascularized bone can be fibula, iliac, scapula, radius, and others depending on the volume, length, and quality of the bone graft required. The pedicle length, size, and possibility of inclusion of a skin flap are also important considerations.[57,58,63,65,66] Among those donor sites, the most commonly used one is fibula. Harvested as an osteoseptocutaneous flap, the fibula provides an ideal source for vascularized bone graft for one-stage total reconstruction of a compound bone and coverage defect (Figure 21.10).

Alloplastic Implant

Alloplastic implants are a useful option for craniofacial skeleton reconstruction in properly selected patients, taking location, quality of recipient site, overlying tissue, the amount of functional stress exertion on implants, and patient's age into consideration. The advantages of alloplastic materials are availability, no donor-site morbidity, and decreased operation time, with a multitude of sizes and shapes. However, all implants will inevitably produce local inflammatory reaction, encapsulation, and requirement of adequate overlying soft tissue coverage. Although most alloplastic implants are fairly well tolerated by the host, complications can occur after placement, such as migration or extrusion of implants, infection, and inappropriate

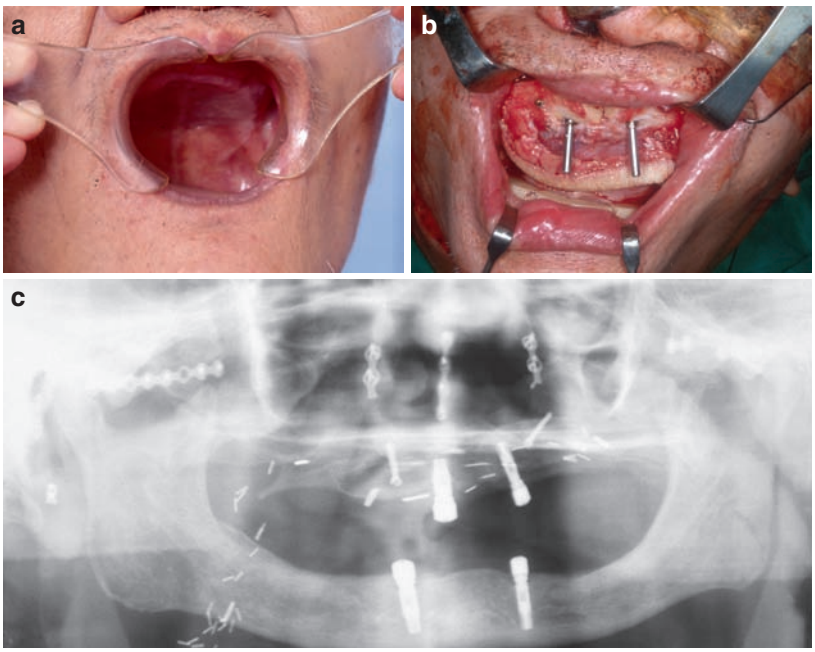

Figure 21.10. Traumatic segmental maxillary defect reconstructed with fibula osteoseptocutaneous flap and immediate osteointegration teeth. (**a**) Traumatic segmental maxillary defect. (**b**) Bone contouring and osteointegration teeth implantation during reconstruction with a fibula osteoseptocutaneous flap. (**c**) Final x-ray appearance.

contour due to improper selection. Alloplastic implants can be either nonresorbable or resorbable. Metallic mesh, hydroxyapatite, polyethylene, silicone, Teflon, and polymethylmethacrylate are nonresorbable implants. Resorbable implants include polydioxanone (PDS), polylactides, polygalactin, and gelatin film. The choice of material for reconstruction is largely determined by the experience of the surgeon and implant cost. Titanium mesh has gained popularity in the management of orbital wall fractures, especially for floor blow out fracture. Because of the characteristic structure, soft tissue is easy to grow through and around the implant, providing further stability. Porous polyethylene such as Medpor (Porex Surgical, College Park, GA) is highly biocompatible with pore size ranging from 100 to 200 μm to permit rapid tissue and bone ingrowth and minimize capsule formation. It is available in many different forms designed for specific area reconstruction on craniofacial skeleton. However, polyethylene is difficult to be visualized on CT scan when compared with titanium alloplastic implants. Resorbable alloplasts are mostly used for orbital wall reconstruction. Complete resorption occurs from 3 months up to a few years after implantation. To date, bioresorbable materials used for orbital reconstruction have manifested an 8.3% incidence of inflammatory reactions.[51]

Frontal Sinus Fracture

Frontal sinus fractures comprise between 5% and 15% of maxillofacial fractures[23] and are most commonly caused by high-velocity impact. The force required to fracture the frontal sinus has been reported to be between 800 and 2,200 lb of force and is usually sufficient to cause significant associated injuries in other maxillofacial regions or brain.[15,34]

The important clinical findings include forehead laceration with palpable bony irregularities or step deformity and possible direct brain debris exposure. Rhinorrhea should be highly suspected with the possibility of cerebrospinal fluid (CSF) leak. The halo test or laboratory confirmation of beta-2 transferrin can be used to

confirm the leakage of CSF. Thin-section axial and coronal axial CT scans provide the essential information regarding the involvement of the anterior and/or posterior table as well as the degree of comminution or displacement of the fractures. Coronal section can elucidate the injury to the frontonasal duct.

The main goals of management are (1) restoration of frontal contour, (2) protection of intracranial structures, (3) cessation of CSF leakage, and (4) prevention of infection. The treatment strategy depends on the degree of frontal sinus injuries with consideration of the involvement of posterior table fracture, injury of frontonasal duct, and presence of CSF leak. Coronal incision is preferred to direct access from laceration wounds to obtain adequate exposure. Early surgical exploration and treatment can reduce the incidence of long-term complications.[36]

Displaced anterior table fractures without frontonasal duct injury require reduction and fixation of fracture segments for aesthetic consideration. If the fracture is not comminuted, endoscope-assisted approach through two slit incisions can be adapted to avoid extensive coronal incision (Figure 21.11).[14] If the frontonasal duct injury is detected from the CT scans or defined intraoperatively, the duct and sinus should be managed to avoid late complications. Some authors attempt restoration of injured duct by cannulation with a stent to prevent late obstruction. However, long-term results show high failure rates up to 30%.[38] Therefore, sinus mucosa should be removed completely with high-speed burrs following by plugging the frontonasal duct with a pericranium graft or bone grafts. Complete sinus obliteration can be performed by variable materials such as fat, muscle, or bone.[44] The use of cancellous bone grafts can achieve complete frontal sinus ossification by osteoconduction but potentially may have donor-site morbidity.[27] An alternative method is spontaneous osteoneogenesis by denuding the wall of the sinus to promote the formation of bone tissue.[50] This technique avoids the donor-site complications, but potential sinus infection may take place during nonobliterated periods. The technique of partial obliteration by bone grafts taken from frontal fractured bone chips was developed, and it produced compatible results without donor-site morbidities.[15] In case of posterior table fracture greater than one table thickness found in CT scan, cranialization has been suggested.[50] However, persistent CSF leakage more than 7 days is the key factor to determine this procedure in the author's experience.[15] This procedure consists of (1) removal of the entire posterior wall of the sinus; (2) repair of torn dura; (3) obliteration of the frontonasal duct by bone grafts and (4) separation of the intracranial cavity form the aerodigestive tract using a galeal-frontalis flap (Figure 21.12) to avoid ascending infection.

Early complications (<6 months) included wound infections, sinusitis, meningitis, and 3–10% of CSF leaks.[60] Most CSF leaks are transient and treated conservatively with bed rest, head elevation, stool softeners, modest fluid

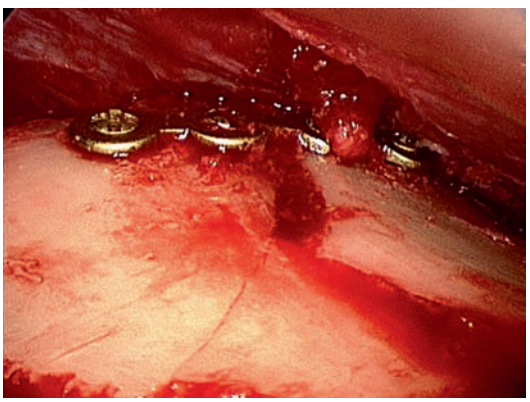

Figure 21.11. Endoscopic view of microplate fixation of frontal sinus fracture.

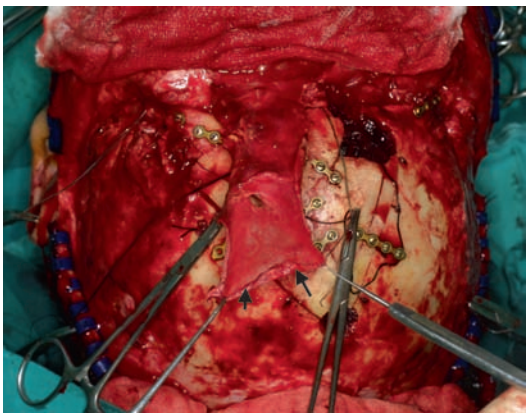

Figure 21.12. Left galeal-frontalis flap is raised (arrows) to separate the frontal sinus base from the nasal cavity.

restriction, diuretics, and lumbar drainage. The incidence of infection is related to the timing of surgical intervention. Early treatment within 24 h did not develop any wound infection in Shen's study.[53] Late complications include mucocele, mucopyocele, brain abscess, and CSF leakage.[46] Mucocele can occur in any postoperative period, even up to 40 years after the initial insult.[29] Regular and long-term follow-up is necessary for early diagnosis of these potential complications.

Nasal Fracture

Fractures of the nose are the commonest facial fracture reported in most published series. The prominent position and fragile element of the nasal bone make it easily injured by a low-energy impact. Lateral force fractures seldom cause serious disruption between the bony and cartilaginous elements; therefore, the aesthetic and functional results are usually better than those of a nasal bone injured by a frontal impact. Nasal dorsum depression or deviation can be inspected before the onset of edema. Palpation of the nasal bones and frontal processes of the maxilla may reveal mobile bony fragment and crepitus. Treatment aims at restoration of nasal projection and airway function. Simple nasal fracture is usually reduced by the closed method, and the adequacy of the reduction is judged by intraoperative inspection and palpation. Reduction is ideally performed either immediately after injury, prior to the development of edema, or a few days after the edema has subsided. Nasal bone is outfractured with Walsham forceps, and septum is straightened with Asch forceps. With the assistance of intraoperative fluoroscan, nasal fracture can be corrected more accurately and objectively (Figure 21.13).[16] Intranasal packing by gauze impregnated with diluted adrenaline solution and external splint are applied to control mucosa bleeding, support the bony fragment, and prevent iatrogenic septal hematoma. Although internal packing is removed 3 days after operation, external splint is left for 7–10 days individually.

Naso-Orbito-Ethmoidal Fracture

While the primary point of injury is the nasal bone complex, a high-energy impact often produces radiation to ethmoid framework, frontal sinus, orbital medial wall, and maxilla. Radiation of fracture extending superiorly may involve the floor of anterior cranial fossa with risks of a cranionasal fistula and possible olfactory nerve disruption. Disruption of lacrimal drainage system may occur with naso-orbito-ethmoidal (NOE) fracture.

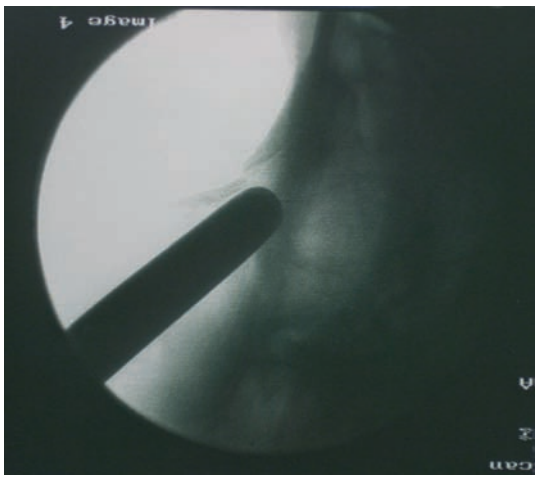

Figure 21.13. Reduction of nasal bon fracture under fluoroscan supervision.

These fractures are characterized by collapse of the NOE complex with or without disruption of medial canthal ligament. The resultant deformity is depressed nasal dorsum, widening of nasal base, obtuse nasolabial angle, shortened palpebral fissure, shallow naso-orbital valley, and telecanthus (intercanthal distance > 40 mm). Disruptions of medial canthal ligament allow an unopposed lateral pull in the suspensory sling and causes telecanthus. A manual examination by placing the thumb and index finger over the canthal-bearing medal orbital rim is performed to assess the motility of the NOE segment.[48] If there is any instability or movement, open reduction with stabilization is indicated. The most popular classification is proposed by Markowitz et al.[43] According to fracture severity and involvement of medial canthal tendon, NOE injuries are divided into three fracture patterns as follows: type I, single segment of NOE fracture; type II comminuted fracture; and type III, comminuted fracture with avulsion of attachment of the medial canthal tendon (Figure 21.14).

The goal of treatment remains the restoration of bony nasal projection, preinjury intercanthal distance, normal naso-orbital valley, and reestablishment of the continuity of the lacrimal system when necessary. There is a high incidence of associated orbital wall fractures; therefore, simultaneous reconstruction of internal orbital wall is crucial to avoid late enophthalmos. Three incisions, including upper buccal sulcus, lower eyelid, and coronal approach, are generally used for complete exposure of NOE skeleton. Coronal incision with subperiosteal dissection allows wide exposure of medial orbital walls, nasal bones, and frontal process of the maxilla. Care is taken to preserve the bony insertion of the medial canthal ligament during dissection. Telecanthus rarely occurs in NOE type I fracture. The isolated single NOE segment can be reduced adequately and fixed with a plate at the infraorbital rim and nasomaxillary buttress without coronal incision. Whenever telecanthus is present, more common in type II and III, the canthal-bearing segment needs to be adequately reduced to maintain the intercanthal distance. Transnasal wires (gauge 26) passing though this segment are placed posterior and superior to the medial canthus and tightened on the medial-supraorbital rim with 2-mm screws (Figure 21.15). Overcorrection should be attempted, because relapse of intercanthal distance frequently occurs after surgery, and secondary correction of telecanthus is much less successful. In extreme comminution

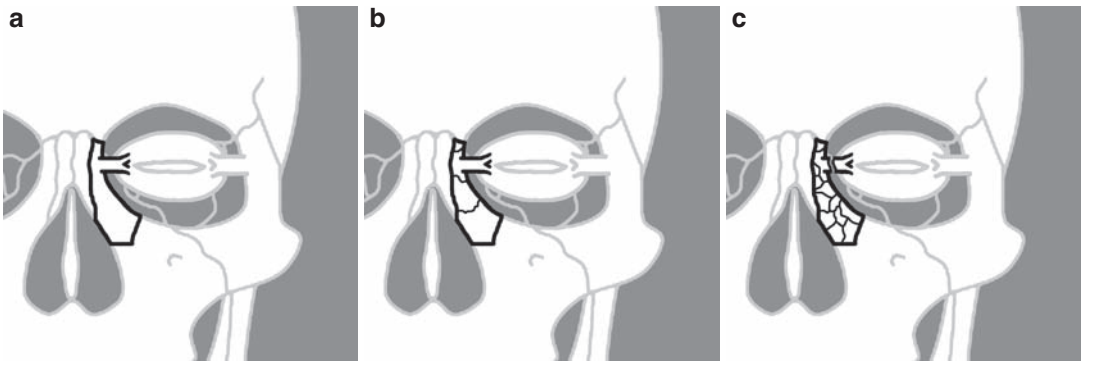

Figure 21.14. Classification of nasoethmoidal-orbital fracture. (**a**) Complete type I injury. (**b**) Type II injury with comminuted fracture. (**c**) Type III injury with medial canthal tendon disruption.

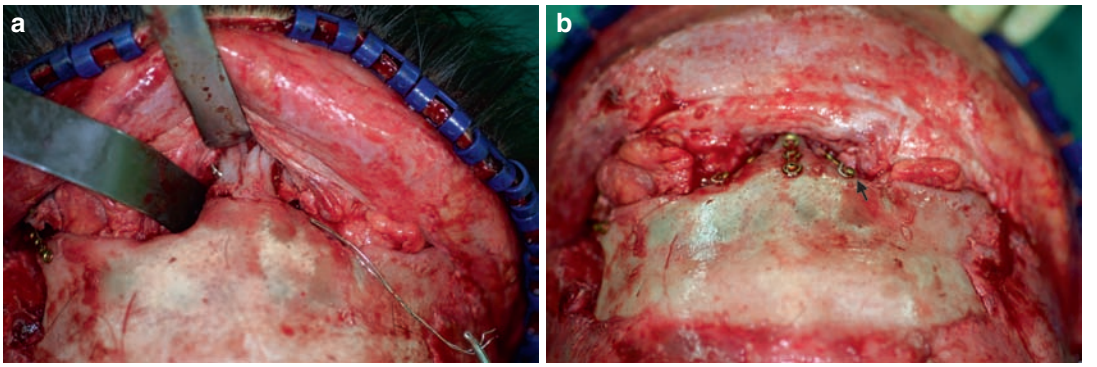

Figure 21.15. Transnasal wiring technique. (**a**) The wire is inserted through left ligament-bearing fragment and placed posterior to the canthus. (**b**) The transnasal wire is pulled out and tightened on a 2-mm screw at the opposite supraorbital rim (*arrow*).

of the central segment (type III) with avulsion of the medial canthal ligament, transnasal medial canthopexy by passing a 2–0 wire suture is indicated to allow anchoring of the medial canthal ligament and reduce intercanthal distance. The depressed nasal dorsum is reconstructed with a contoured bone graft fixed superiorly at the nasofrontal region by a miniplate and screws.

Zygomatic Fracture

Zygoma itself presents as an important structure in facial width, mid-facial height, and projection. The high incidence of zygomatic fractures is related to the prominent position of cheekbones within the facial skeleton. The anterior lateral portion of the orbit is occupied by zygoma; therefore, displacement of the zygoma will change facial contour and the orbital volume. Masseter muscle origin on zygomatic body and arch is usually considered to be the major deforming force in maintaining the displacement of a fractured zygoma.[4] The pull of the muscle must be overcome during fixation for optimal stabilization and prevention of relapse.

Patient may present malar depression when swollen cheek tissue subsides and some degree of difficulty in mouth opening because of muscle spasm or impingement of the coronoid process by the displaced zygomatic arch. Altered sensation in the distribution of the infraorbital nerve implies infraorbital rim fracture around the infraorbital foremen with the possibility of nerve impingement. The ocular symptom such as diplopia or enophthalmos may accompany zygomatic fracture. Waters view and submental view can be used as screen test to reveal evidence of zygomatic fracture.

The isolated zygomatic arch fracture is usually a low-velocity injury, with localized arch contour depression. The mainstay of reduction alone has been the Gillies technique. A 2-cm incision is carried though the temporal hair-bearing area, and then a Dingman elevator is inserted beneath deep temporal fascia to elevate the arch. Traditionally, the assessment of reduction is carried out subjectively by inspection and palpation. To improve the surgical results, intraoperative computed tomography has been used to aid in positioning the fracture.[55] Fluoroscan is another option to assess the adequacy of reduction intraoperatively.[24] In severe comminuted or unstable arch fractures, a coronal approach with rigid-plate fixation should be considered (Figure 21.16). Gruss[25] stressed the importance of restoration of the zygoma arch, which is responsible for determining the width and the anterior projection of the mid face.

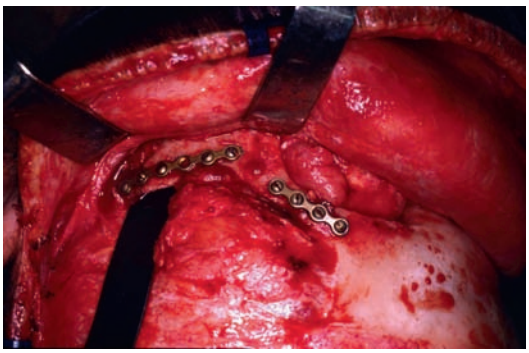

Figure 21.16. Left comminuted zygomatic arch fractures are approached through coronal incision to obtain adequate exposure and reduction.

Nondisplaced malar fractures may be managed conservatively. Patients are instructed to avoid local pressure on the malar prominence, and a soft diet is suggested during the initial bone-healing period. Displaced zygomatic fractures require open reduction and rigid internal fixation after proper alignment of all fracture sites. Two incisions including buccogingival and lower eyelid incision are necessary to accomplish this purpose. The lower eyelid incision with mobilization of the lateral canthus is used for exposure of the zygomaticofrontal suture, and lower and lateral orbit, to avoid upper eyelid incision.[40] The distinct shape of the lateral orbital wall is the thickest portion of the orbit and rarely comminuted. This makes the zygomaticosphenoidal junction a reliable reference point for adequacy of reduction. Rigid miniplate fixation is usually done over the zygomaticomaxillary buttress and infraorbital rim. The additional plating at the zygomaticofrontal suture may be necessary if stability cannot be achieved. When displacement of the zygomatic fractures is not comminuted without bone loss, adequate reduction with optimal outcome can be achieved with one buccal incision. Recently, endoscope-assisted reduction and fixation of the zygomatic complex fracture has been developed to reduce the morbidity of coronal incision.[12]

Finally, suspension of the cheek soft tissue by suturing the periosteum to the infraorbital rim is critical in preventing cheek drooping. Other benefits of suspension are providing support for the lower eyelid and reducing the risk of lid retraction.

Orbital Fracture

The orbital floor is most vulnerable to fracture because of thinness of the maxillary roof, existence of the infraorbital canal, and curvature of the floor. Immediately behind the orbital rim, the floor is concave, whereas further back, it becomes convex and is the called posterior ledge, in which the bony structure becomes thicker and less deformed in the orbital floor fracture. Orbital floor fractures are mainly caused by one of two primary mechanisms: (1) the hydraulic theory,[49] direct transmission of pressure from the globe or intraorbital contents blowing out the floor of the socket, or (2) bone conduction theory,[61] indirect transmission of pressure from the orbital rim along the bone to the floor.

Several clinical symptoms are frequently associated with orbital fractures. Subconjunctival hemorrhage caused by rupture of blood vessels on the conjunctiva usually resolves without treatment within 1–2 weeks. Orbital emphysema may present as an isolated radiographic finding without significant symptoms and is usually self-limited. Occasionally, the intraorbital air may potentially cause central retinal artery occlusion.[31] Diplopia is caused by restricted ocular movement and occurs most commonly in upward gaze. The etiology may be attributed to incarceration of the orbital contents or extraocular muscles, muscle contusion, or damage to the nerves that innervate the extraocular muscles. Enophthalmos is most commonly caused by enlarged orbital cavity after blow out fracture. CT scan in coronal view provides most valuable information to assess the orbital fractures.

Most common consensus for repair of orbital fractures includes enophthalmos greater than 2 mm, evidence of incarceration of orbital contents, significant hypoglobus, or persistent diplopia more than 2 weeks.[7] The orbital floor fracture can be approached through subtarsal, subciliary, or transconjunctival incisions. The main advantage of transconjunctival incision is the elimination of external eyelid scar. Alternatively, the endoscope-assisted approach developed to repair the orbital medial wall and floor defect (Figure 21.17) decreases the incidence of sequelae related to eyelid or periorbital incisions.[7–9] A successful reconstruction of orbital fracture depends on anatomic placement of the grafts across the defect. A common mistake is inadequate dissection and exploration of the defect due to fear of damage to the optic nerve.[10] The defect of orbital fracture can be repaired with either autogenous bone grafts or alloplastic implants. Both materials provide adequate orbital support.[28]

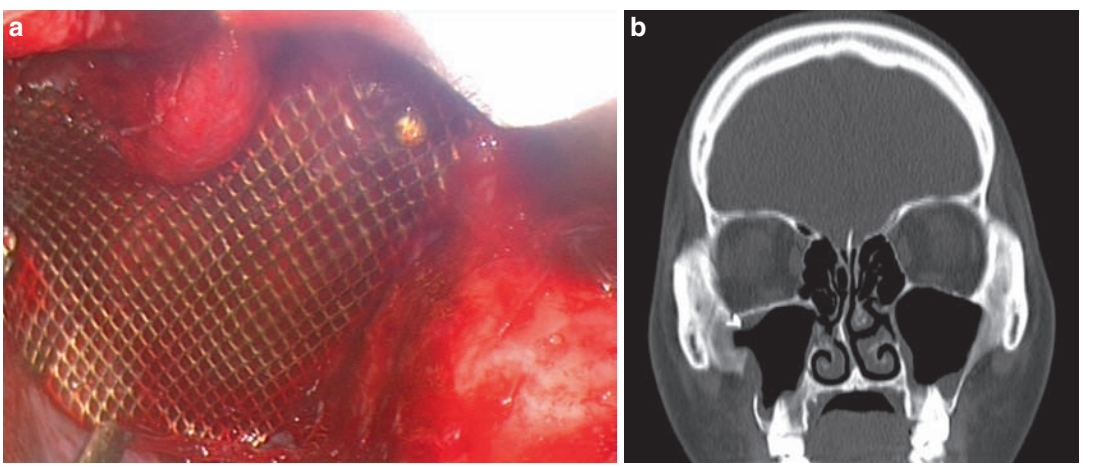

Figure 21.17. Endoscope-assisted reconstruction of orbital floor fracture. (**a**) Endoscopic view of right orbital floor fracture repaired with titanium mesh through transantral approach. (**b**) Postoperative CT scan revealing adequate reconstruction of right orbital floor.

The common postsurgical sequelae are ectropion or entropion, which can be minimized with meticulous surgical dissection and soft tissue resuspension. Transient postoperative diplopia is expected and usually resolved within 2–3 months. Exaggerated overcorrection by grafting should not be attempted to avoid further interference in extraocular muscle movement. A forced duction test should always be done at the end of surgery to minimize the mechanical restriction of orbital contents. Residual enophthalmos is caused by the difficulty in accurately assessing the orbital volume intraoperatively. The most severe but rare complication is retrobulbar hematoma, which may result in retinal ischemia due to elevated intraocular pressure or direct compression of optic nerve.[17] Visual deterioration or blindness may occur subsequently if prompt treatment is not initiated. Therefore, meticulous hemostasis with placement of a small drain tube in the lower eyelid is recommended to avoid this potential disaster.

Maxillary Fracture

The maxilla is the keystone of the bony structure of mid face, with three vertical buttresses including nasomaxillary, zygomaticomaxillary, and pterygomaxillary buttresses.[39] These buttresses protect the maxilla primarily against the forces in the vertical direction. Reconstruction of the pillars of the maxilla in relation to the cranial base superiorly and the mandible inferiorly provides a stable facial contour.

Le Fort identified three great lines of weakness in the mid-facial skeleton that corresponded to the most common sites of fractures (Figure 21.18). Le Fort I fractures with the fracture line crossing transversely along the maxillary wall develop a floating maxilla. Le Fort II fractures

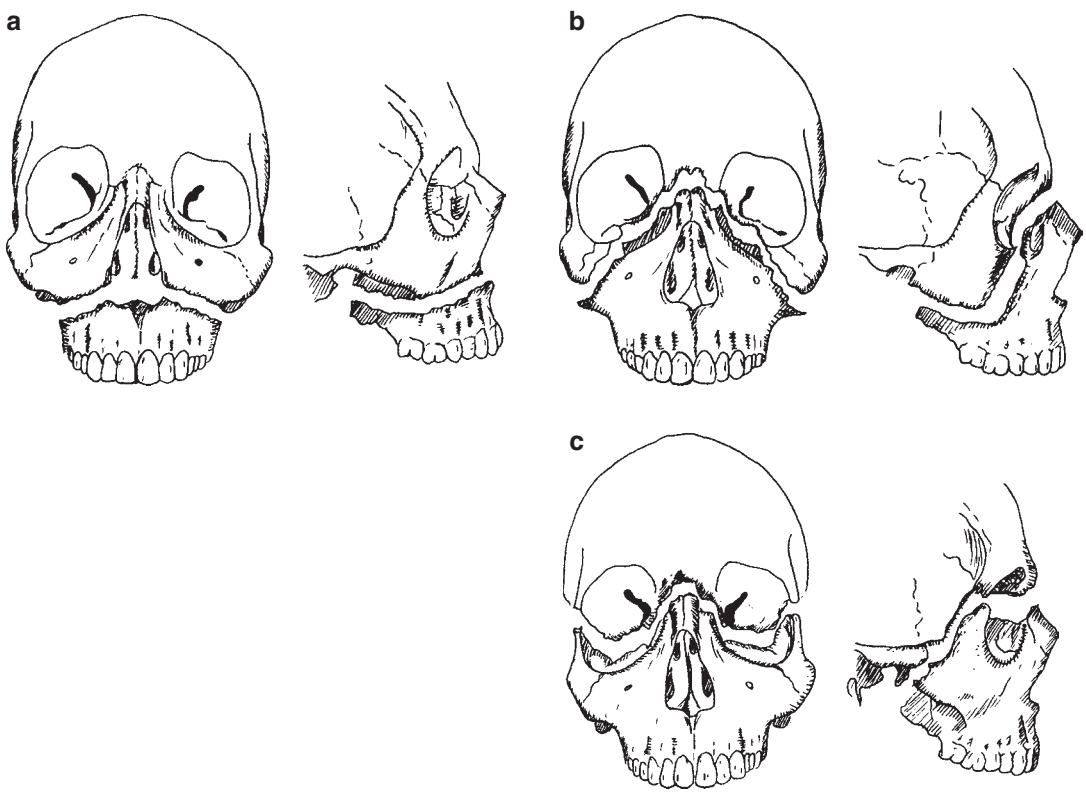

Figure 21.18. Classification of Le Fort fractures. (**a**) Le Fort I maxillary transverse fracture. (**b**) Le Fort II maxillary pyramidal fracture. (**c**) Le Fort III fracture, craniofacial disjunction.

(pyramidal fractures) produce separation and mobility of the mid face extending through the orbits and nasofrontal suture. Le Fort III fractures disconnect the face from the cranium, resulting in a craniofacial disjunction. However, the majority of maxillary fractures are not pure types of Le Fort fractures but a combination of several types.

The common clinical presentations of Le Fort fractures are periorbital and subconjunctival ecchymosis, mid-facial swelling and retrusion, lengthening of the face, and malocclusion. A step deformity at the nasal root can be felt in Le Fort II fractures. Le Fort III fractures may reveal movement at the lateral orbital rim with the upper jaw. Independent mobility of the right or left side of the palate indicates a sagittal fracture of the palate.

The goal is to restore the preinjury occlusion and mastication, facial appearance, and communication capacity. Lower maxillary fractures are approached through upper gingivobuccal sulcus incisions to get access to all the anterior buttresses as well as the infraorbital rim. However, Le Fort II fractures may need additional lower eyelid incision for better exposure at the infraorbital rim. If adequate reduction and stability cannot be achieved, exploration of the nasofrontal area in Le Fort II fracture is indicated through a coronal incision. Mandible is the principal structural pillar of the lower face, upon which Le Fort fractures can be reduced and stabilized. Once proper occlusal relationship is obtained with temporary maxillomandibular fixation (MMF), the maxillary buttresses are fixed rigidly with miniplates.

Palatal fractures frequently accompanied with Le Fort fractures have previously been classified according to either the location of fractures or the treatment plans[30,47] but did not provide a clear treatment algorithm with a given type of fracture. A simpler classification taking into account both the anatomy of fracture sites and corresponding treatment plans has been advocated recently as follows: type I, sagittal fracture; type II, transverse fracture; and type III, comminuted fracture.[6] Manson et al.[41] preferred open reduction and internal fixation for the sagittal fractures of the palate. However, this technique may introduce the possibility of malocclusion and late plate exposure. The authors prefer the method of using intermolar wiring fixation to maintain side-to-side instability in sagittal fractures of the palate (Figure 21.19). Prolonged intermaxillary fixation with a dental splint for 4–6 weeks is required for comminuted palatal fractures.

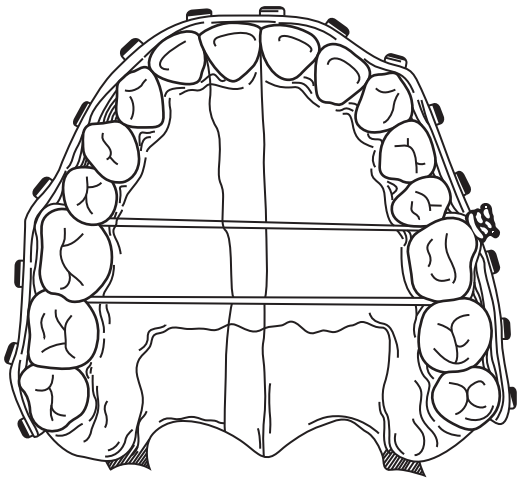

Figure 21.19. Application of intermolar wire to reduce and maintain stability of sagittal fracture of palate.

Mandibular Fracture

The mandible is a U-shaped long bone consisting of tooth-bearing and non-tooth-bearing portions, with unique joints that allow mandible movement. It consists of mandibular symphysis, body, angle, ramus, coronoid process, region of the alveolar process, and condyle. The weakest point is at the condyle region, which makes it susceptible to fracture with the incidence of 20–30% of mandibular fractures.[37] There are two main groups of muscles including masticatory muscles and suprahyoid muscles group acting upon the mandible, which makes it easily displaced at fracture sites and requires larger plate fixation to overcome the force exertion from the muscles.

Malocclusion, open bite or cross bite, trismus, and paresthesia over the lower lip, implying damage to the inferior alveolar nerve, are common complaints. Suspicion of unilateral condylar fracture arises from deviation upon mouth opening, premature teeth contact, and hemorrhage from external auditory meatus. Mandible retrusion, anterior open bite, and premature posterior contact indicate bilateral condylar fractures. Panoramic radiograph (panorex) is

the most common diagnostic tool in suspected mandibular fractures. However, CT scans have a 100% sensitive rate in diagnosing mandibular fractures compared with 86% by a panorex film.[67]

Nondisplaced or incomplete fracture without malocclusion can be treated conservatively with diet restriction. Similarly, coronoid process fractures rarely require surgical treatment. Open reduction is indicated for displaced mandibular fracture with malocclusion. Fractures of the tooth-bearing region of the mandible, which are not comminuted, are readily approached through an intraoral incision with careful protection of the mental nerve. MMF is applied to achieve stable occlusion before application of a plate and can be released immediately after rigid fixation. For the comminuted fractures, extraoral incision is chosen for adequate exposure and fixation (Figure 21.20).

The unique characteristic in mandibular angle is the presence of the third molar tooth, which will increase the incidence of angle fracture due to decrease in the osseous support and weakening of the mandible in this region. However, the third molar should be preserved to maintain the stability during reduction of angle fracture. Extraction of the third molar is indicated when the tooth is damaged, severely diseased, and prevents fracture reduction. The mandibular angle fracture is usually fixed with two 2.0-mm noncompression miniplates at the superior border through intraoral incision.

Proper management of condylar fracture is still controversial. Several studies showed no difference in jaw mobility, joint problems, and occlusion status between open versus closed reduction and favored nonoperative treatment of condylar fractures.[42] In the other hands, open reduction with rigid internal fixation has evolved as a popular method especially for subcondylar fractures because of its superior results when compared with those of closed reduction.[5,21] In Ellis's study of 200 cases of condylar process fractures, he found that patients treated with the open method achieved more consistent occlusal results and quicker postoperative mandibular motility.[21] The most common recognized indications for open reduction of condylar process fractures are malocclusion, with either condylar displacement or ramus height instability.[70,72] Facial nerve injury and facial scarring are the two major concerns of traditional open approaches via the preauricular incision or retromandibular incision. The endoscope-assisted reduction of condylar process fracture through the intraoral incision provides equivalent results and avoids facial scar formation and facial nerve damage.[13,52]

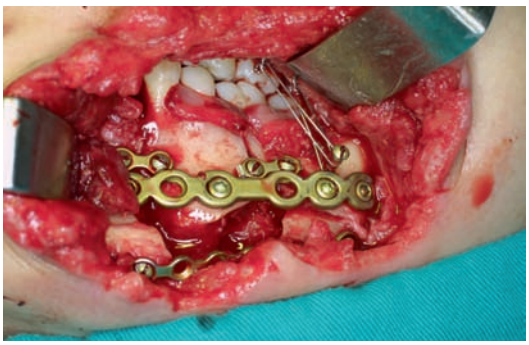

Figure 21.20. Comminuted mandibular fractures stabilized with 2.3 mm plate and multiple small plates.

Postoperative Care

Airway Management

Recently, rigid maxillomandibular fixation (MMF) shifted to elastic band fixation in our practice after rigid fixation with stable occlusion. It helps to guide the patient in correct occlusion during mouth opening exercise, while extubation can be achieved immediately and safely. It is worth emphasizing that swelling of injured orofacial tissue may increase for the first few days, potentially threatening the airway. If there is any concern about the potential airway obstruction after extubation, the endotracheal tube should be left for few days. Wire cutters should be available by the bedside for immediate opening of the jaws.

Postoperative Bleeding

Hypotensive anesthesia may mask the small bleeder, and unexpected bleeding can occur after recovery from anesthesia. Pain relief, cold packing, and control of blood pressure are effective in most situations, and nasal packing is particularly used after reduction of nasal bone fractures. Cold packing starts immediately after surgery around the surgical field, with a duration of 10–15 min

every 2 h for 1 week until the swollen phase subsides. Coagulation status should be checked if bleeding continues after loss of one unit of blood volume. Persistent oozing of blood into the pharynx may cause unpleasant vomiting if swallowed. Special attention is given to those with potential bleeding after periorbital surgery, because retrobulbar hematoma may cause optic nerve compression. The authors prefer to place one open drainage tube at the eyelid wound in most cases of orbital reconstructive surgery.

Antibiotics

In our routine management of maxillary or mandibular fractures with approach through the mouth, postoperative intravenous antibiotics are continued for 72 h. First-generation cephalosporin and clindamycin are our first-line choice. Prolonged administration of broad-spectrum antibiotics may promote the possibility of resistant strain growth and does not decrease the incidence of postoperative infection.[19] However, antibiotic therapy for established osteomyelitis has to be maintained until the infection is under control.

Steroid Medication

Steroid is not routinely used for alleviating post-traumatic swelling in the facial tissues, as it may impair wound healing. In certain special cases, such as traumatic optic neuropathy or orbital apex syndrome, steroid is prescribed after an optic nerve decompression procedure for reducing edema.[11]

Oral Hygiene

Oral hygiene needs to be maintained with an oral suction tube, tooth brush, and tooth irrigator, when the fracture is approached from an intraoral incision. Mouth washing with 0.2% chlorhexidine gargle every 2 h is used to remove any blood clot from the suture line and arch bar.

Diet

Oral feeding starts as soon as possible if there is no contraindication such as gastrointestinal disease. Cool water is given initially in small amounts as test, and then appropriate diet begins. In surgery involving fracture of the upper or lower jaw, liquid diet is preferable in the first 2 weeks after surgery according to the stability of occlusion. Nonchewing food can be eaten without producing stress or compression forces at recently stabilized fracture sites, and the food will not tend to be collected in areas of the mouth that are insensitive.

Rehabilitation

Depending on the nature of the injuries, specific physical therapy will be arranged to improve the result. For those with upper or lower jaw fractures, mouth opening training can resume soon once the rigid MMF is released or changed to elastic rubber bands. Ocular excursion exercise is urged for those receiving orbital wall reconstructions.

Summary

A well-organized team approach to a craniofacial trauma patient is critical in reducing mortality and disability. Once life-threatening problems have been resolved, secondary survey is initiated to exclude possible dental, ophthalmologic, and otolaryngologic problems via specialist consultation. Definite diagnosis is made through careful physical examination and fine-cut CT scans to form a thoughtful preoperative plan. Although the optimal time for surgical repair relies on the nature of the associated injuries, most craniofacial injury can be readily repaired within a 2-week period after injury to achieve better results.

These approaches have emphasized primary definite treatment of both bone and soft tissue injury with adherence to the principles of direct wide fracture exposure, accurate anatomic reduction with rigid-plate fixation, and resuspension of soft tissue envelop. Significant soft and bone tissue loss in devastating injury can be replaced with microsurgical free tissue transfers. Application of a minimal invasive technique permits adequate visualization through the smallest exposure to achieve competitive results in selected cases. With modern surgical techniques and standard care, good outcome of reconstruction of craniofacial trauma can be expected both functionally and aesthetically.

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22

Eyelid and Periorbital Aesthetic Surgery

Colin M. Morrison, Claude-Jean Langevin, and James E. Zins

Summary

The eyelids and periorbital area are important aesthetic facial subunits as well as sensitive projectors of facial aging. Brow ptosis, eyelid ptosis, dermatochalasis, fat herniation or protrusion, tumors and trauma can critically alter the anatomical relationships in this region. Aging may also convey an inaccurate message of tiredness, anger, or sadness, diminishing the overall aesthetic appearance of the face.

This chapter describes the relevant eyelid and periorbital anatomy, techniques, and potential complications resulting from cosmetic surgical procedures performed in this area.

Abbreviations

MRD	Margin reflex distance
ROOF	Retro-orbicularis oculi fat
SOOF	Suborbicularis oculi fat
SMAS	Superficial muscular aponeurosis system

Introduction

The eyelids and periorbital area are a common focal point during human interaction.[2] This area is also frequently the one that first demonstrates facial aging. Laugh lines, at first, present only with animation but are ultimately visible at rest. This specific manifestation is particularly noted in fair-skinned (Fitzpatrick Type I and II) females. With increasing age, the lateral brows descend more rapidly than the medial brows because of a lack of lateral muscular support. With age, the orbital septum, a distensible anatomical layer of the eyelid, weakens. The orbicularis muscle and the supporting ligaments of the eyelid also lose elasticity. The contents of the orbit produce a downward and anterior displacement of the orbital fat because of the loss of this septal and muscular support of the fat pads.

There is also adjacent loss of fat over the medial and central orbital rim. These changes result in a "dark circle below the eye" commonly referred to as the nasojugal groove or tear trough deformity. Dehiscence or weakness of the levator aponeurosis may also cause an involutional upper eyelid ptosis associated with dermatochalasis (Figure 22.1).

The eyelids themselves act to protect the anterior surface of the globe and aid in the regulation of light reaching the eye. Additionally, they distribute the protective and optically important tear film over the cornea during blinking and maintain tear flow by a pumping action on the conjunctival and lacrimal sacs. Over-resection of upper eyelid skin may cause the patient with mild or asymptomatic dry eye to become symptomatic. This may result in discomfort or excessive tearing. Lid malposition, the most common complication following lower eyelid surgery may also result in eye irritation or excessive tearing.[15]

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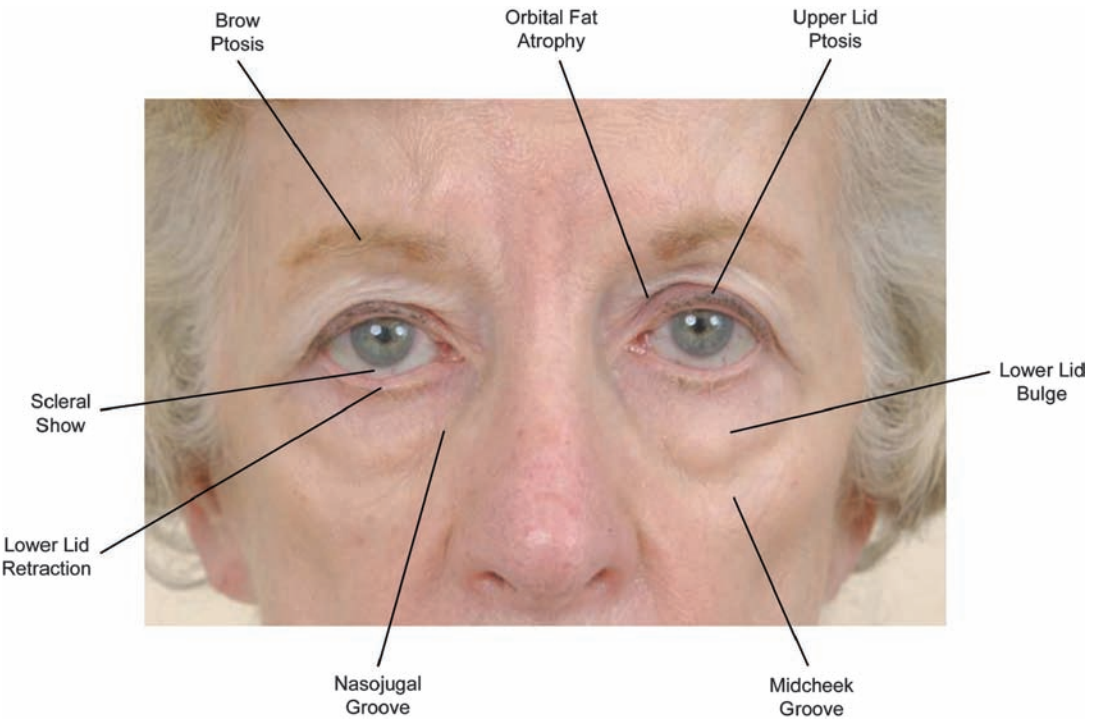

Figure 22.1. Aging eyelids and periorbital region.

Surgical Anatomy

Surface Landmarks

The soft tissue cephalometric dimensions of the periorbital area are different in men and women. Typically, male brows are lower and less arched. The medial brow in both sexes begins at the level of the supraorbital rim, and the eyebrow peaks at the lateral two-thirds, going from medial to lateral. Ideally, the lateral end of the eyebrow finishes at a higher level than the medial brow.[35]

The orbit is a transverse oval shape, with a slight 2–4° upward lateral tilt to the palpebral fissure.[1,32] Aesthetically appealing eyes have an almond shape with associated supratarsal fullness (Figure 22.2). The ideal canthal angle is 2 mm higher than the medial canthus in Europeans and 3 mm higher in Asians.

Globe position (hypoglobus, hyperglobus) and globe protrusion (proptosis, exophthalmos) should be evaluated. Asymmetry of globe position may alter the appearance of the superior sulcus, and surgery alone may not necessarily correct this.

The upper eyelid skin crease is formed by the attachment of the superficial insertion of levator aponeurotic fibers into the dermis. In Caucasian women, the crease is usually 8–11 mm above the lid margin, and in Caucasian men, usually 6–9 mm. Some asymmetry in margin crease distance may result from disinsertion of the levator aponeurosis, and this should be noted before surgery.

In contrast, the Asian eyelid has more fullness of the upper eyelid, narrower palpebral fissures, and a lower lid crease. The lower lid crease is due to the insertion of orbital septum into the levator at or over the anterior surface of the tarsus and a lower insertion or absence of levator fiber insertion into the dermis.[7] A medial epicanthal fold may also be present.

The lower lid extends below the inferior orbital rim to join the cheek. However, the definition of the lid–cheek junction is controversial. It may be defined either by the junction of eyelid skin and cheek skin[18] or by surface contour change.[9,25] In young people, the change at the lower lid cheek junction is at or above the infraorbital rim, whereas it falls below this level with advancing age.

Figure 22.2. The female brow slopes superiorly from medial to lateral paralleling the palpebral fissure. There is at least 1 cm between upper eyelid crease and lower brow lashes.

The nasojugal fold located at the lower lid–cheek junction runs inferiorly and laterally from the medial canthal region, forming the tear trough. The malar fold runs inferiorly and medially from the outer canthus toward the inferior aspect of the nasojugal fold.

In adults, the upper lid margin rests 1.5 mm below the limbus, and the lower eyelid margin rests at the level of the lower limbus.

Structures

The anatomy of the connective tissues of the temporal and periorbital areas has been previously described in detail in several studies. These articles describe the relationship of the connective tissue planes to local nerves and vessels.[17,23]

The eyelids themselves are composed of skin, orbicularis oculi muscle, the orbital septum, preaponeurotic fat, the tarsi, lid retractors of the upper and lower eyelids, and the conjunctiva. The upper and lower lids, however, may be considered analogous structures, with differences only in the arrangement of the lid retractors.

In eyelid reconstruction, it is more useful to consider the repair of the anterior and posterior lamellae, with the anterior lamella being the skin and orbicularis and the posterior lamella being the tarsus and conjunctiva. The middle lamella consists of the orbital septum.

Orbicularis Oculi Muscle

The orbicularis oculi muscle is one of the superficial muscles of facial expression. It is invested by the superficial musculoaponeurotic system (SMAS), and muscle contraction is translated into movement of the overlying skin by fibrous septa extending from the SMAS into the dermis. The muscle may be divided into an orbital and palpebral part, with the latter being further subdivided into preseptal and pretarsal components. The orbital portion is used in forced closure, whereas the palpebral portion is used in blinking and voluntary winking (Figure 22.3).

Orbicularis muscle fibers extend superiorly to interdigitate with the frontalis and corrugator supercilii muscles. Innervation is from the temporal and zygomatic branches of the facial nerve. The nerves are orientated horizontally and innervate the muscle from its deep surface.

The lateral canthus is the tendinous insertion of the orbicularis oculi muscle into the lateral orbital rim. The canthus is composed of an inferior retinaculum that is in continuity with the lower lid and an upper retinaculum that is in continuity with the upper lid. The superior and inferior canthal extensions fuse to form a common band that inserts into Whitnall's tubercle inside the lateral orbital rim (Figure 22.4).

Submuscular Areolar Tissue

Submuscular areolar tissue consists of variable loose connective tissue below the orbicularis oculi muscle. This submuscular plane continues superiorly and terminates at the retro-orbicularis oculi fat (ROOF), which is most pronounced in the eyebrow region. The suborbicularis oculi fat (SOOF) found in the lower lid is the continuance of this plane inferiorly.

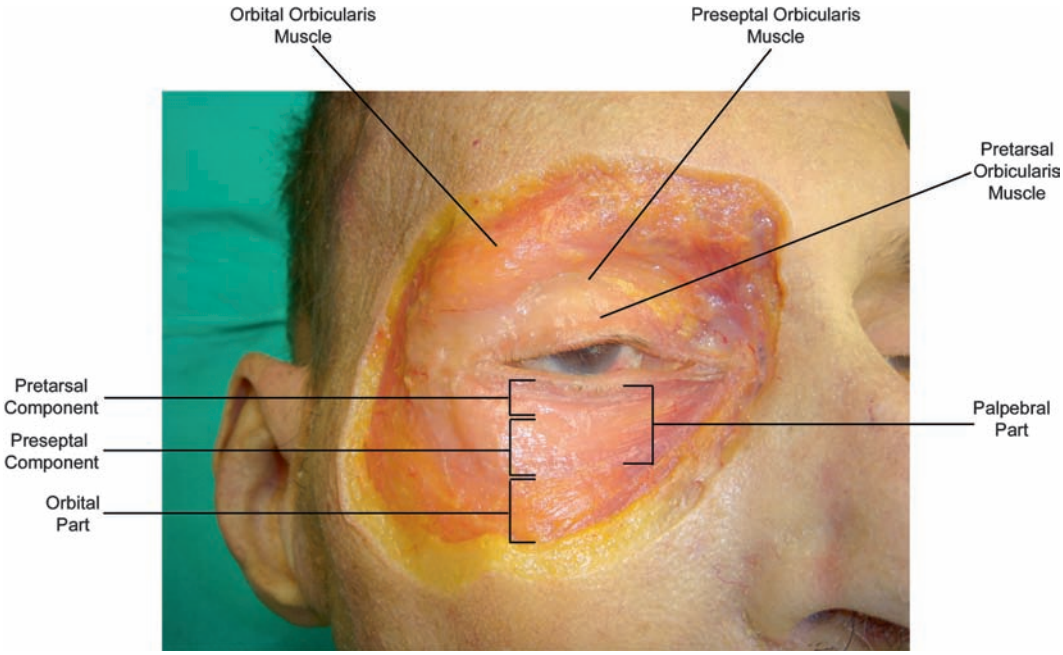

Figure 22.3. Anatomic dissection of the eyelid. The skin and malar fat pad were removed exposing the orbital and palpebral orbicularis oculi muscle. The temporalis and zygomatic branches of the facial nerves innervate this muscle.

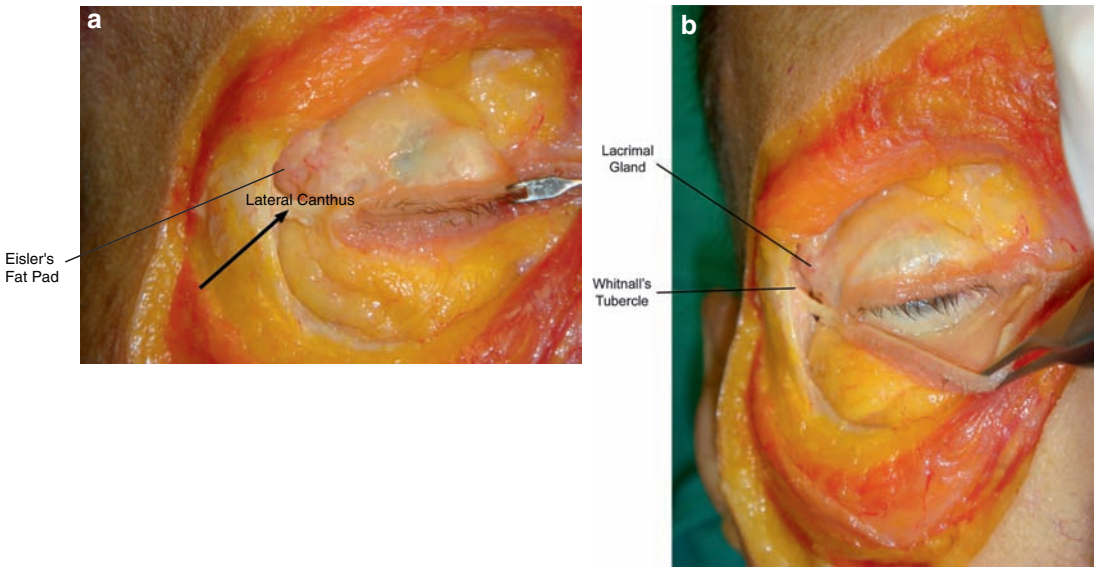

Figure 22.4. Lateral canthus. (**a**) Superior and inferior canthal extensions form a common band that inserts into Whitnall's tubercle located 1.5–3 mm posterior to the lateral orbital rim. (**b**) Lower lid distraction test: Distraction greater than 6 mm from the globe indicates lid laxity and is an indication for lid support if lower eyelid surgery is planned.

Tarsal Plates

The tarsal plates are responsible for the structural integrity of the eyelids. They are composed of fibrous tissue and sebaceous meibomian glands. Each tarsus is approximately 29 mm long and 1 mm thick. The crescentric superior tarsus is 10 mm high centrally, narrowing medially and laterally. The smaller rectangular inferior tarsus is 3.5–5 mm high. The medial and lateral ends of the tarsi are attached to the orbital rims by the medial and lateral canthal tendons (Figure 22.5).

From an aesthetic and reconstructive viewpoint, the lateral canthal tendon is a vital structure. It passes deep to the septum orbitale to insert into the lateral orbital tubercle 1.5–3 mm posterior to the orbital rim and 10 mm inferior to the frontozygomatic suture. The Eisler fat pocket lies between the orbital septum and the lateral canthal tendon (Figure 22.4).

Orbital Septum

The orbital septum is a fibroelastic structure that attaches at the periphery of the entire orbital rim. The arcus marginalis represents the confluence of the orbital septum and periosteum. Centrally the orbital septum fuses with the lid retractor structures near the lid margins, acting as a diaphragm to retain the orbital contents. In the lower lid, the upper septum is reinforced by capsulopalpebral fascia. Conjoined septum and capsulopalpebral fascia then attach to the inferolateral orbital rim as the arcuate expansion. The lower part of the orbital septum has no such reinforcement and is consequently weaker.

Upper Lid Retractors

The levator palpebra superioris arises from the under surface of the lesser wing of the sphenoid bone. It passes anteriorly for 40 mm and then continues as an aponeurosis, changing to a more vertical direction at the superior transverse (Whitnall's) ligament (Figure 22.6).

The aponeurosis fuses with the orbital septum before reaching the superior border of the tarsal plate. Some aponeurotic fibers descend to insert into the lower third of the anterior surface of the tarsal plate. An anterior extension from this fusion inserts into the pretarsal orbicularis oculi muscle and overlying skin, forming the upper lid skin crease.

Müller's muscle is smooth muscle innervated by the sympathetic nervous system. Fibers originate from the under surface of the levator in the region of the aponeurotic–muscle junction, travel inferiorly between the levator aponeurosis and conjunctiva, and insert into the superior margin of the tarsus.

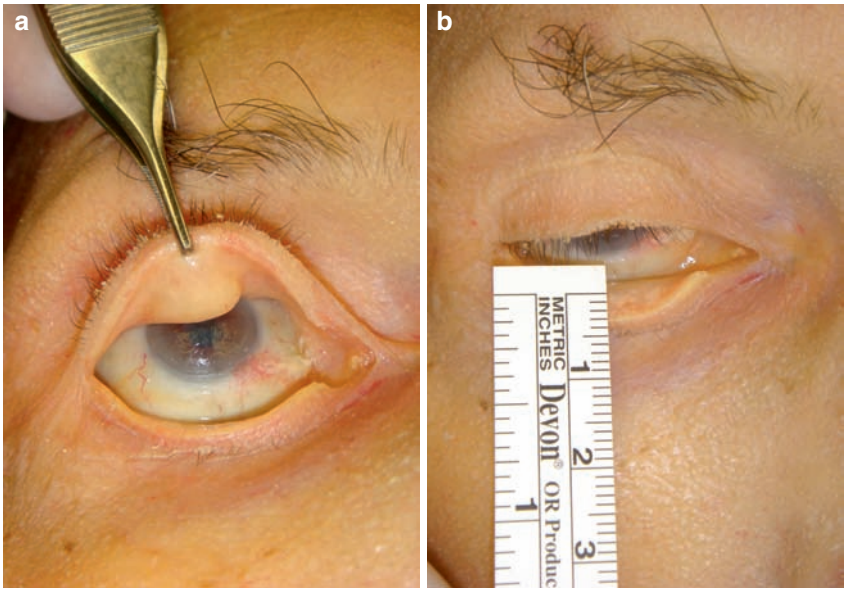

Figure 22.5. (**a**) The tarsal plate of the upper eyelid is crescentric in shape and approximately 10 mm in height. (**b**) The inferior tarsus has a rectangular configuration and is 3.5–5 mm high. Note the lower lacrimal punctum.

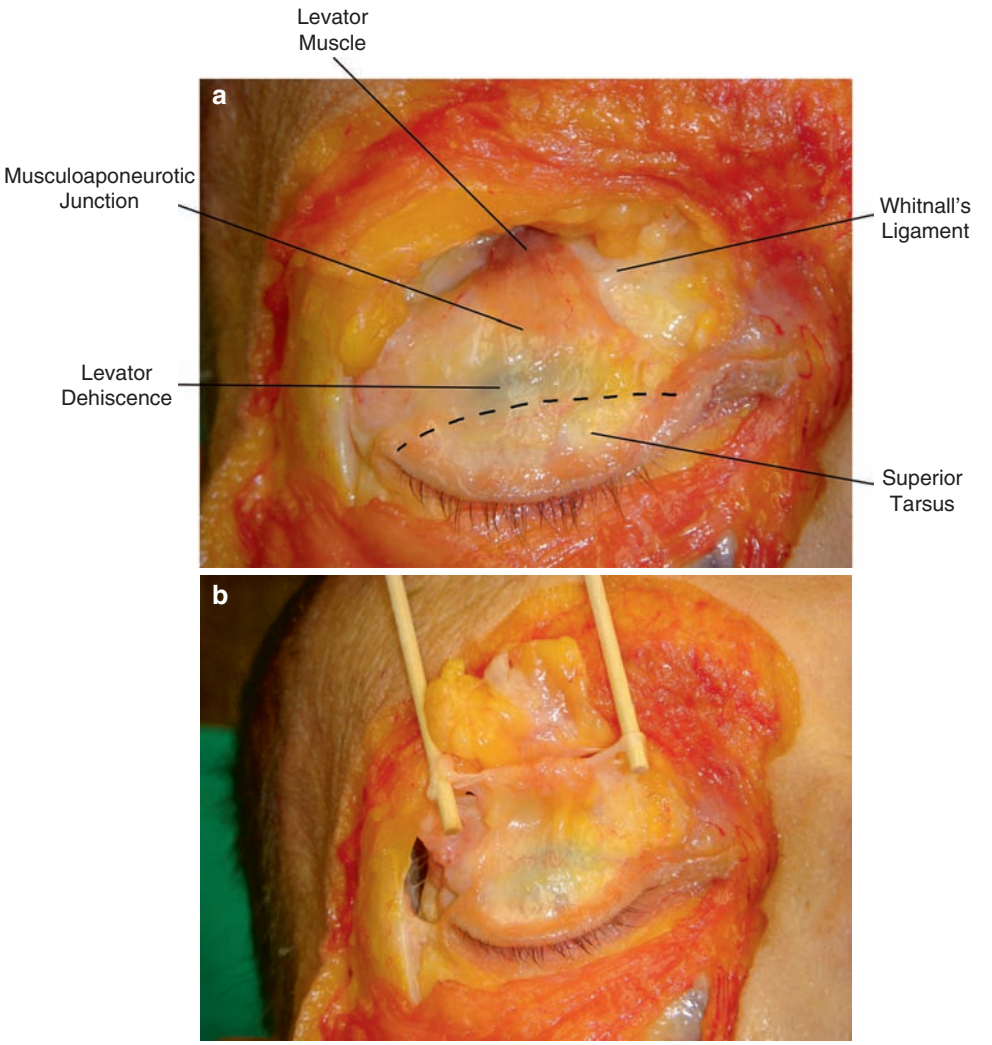

Figure 22.6. (**a**) The levator palpebra superioris muscle with the levator aponeurosis inserting into superior tarsal plate. Note the levator aponeurosis dehiscence between the levator muscle and the superior tarsus. (**b**) The Whitnall's ligament forms a fibrous connective tissue sleeve surrounding the levator muscle. It acts as a fulcrum for upper eyelid function.

Lower Lid Retractors

The lower eyelid retractor is a fascial extension of the inferior rectus muscle. It splits to envelop the inferior oblique muscle and reunites as the inferior transverse (Lockwood's) ligament. The fascial tissue then passes anterosuperiorly as the capsulopalpebral fascia and inserts into the inferior border of the inferior tarsus. The orbital septum fuses with the capsulopalpebral fascia approximately 5 mm below the inferior tarsal border. Sympathetically innervated smooth muscle fibers are also noted in the lower eyelid and constitute the inferior tarsal muscle.

Fat Pads

In the upper eyelid, preaponeurotic fat is found immediately deep to the orbital septum and anterior to the levator aponeurosis. A central fat pad and a medial fat pad are described.[13] The medial fat pad is pale yellow or white, whereas the central fat pad is yellow and broad. A portion of the lateral end of the central fat pad surrounds the medial aspect of the lacrimal gland.

The lacrimal gland has a pinkish lobulated firm structure. The gland's anterior border is normally just behind the orbital margin, but involutional changes may lead to prolapse anteroinferiorly,

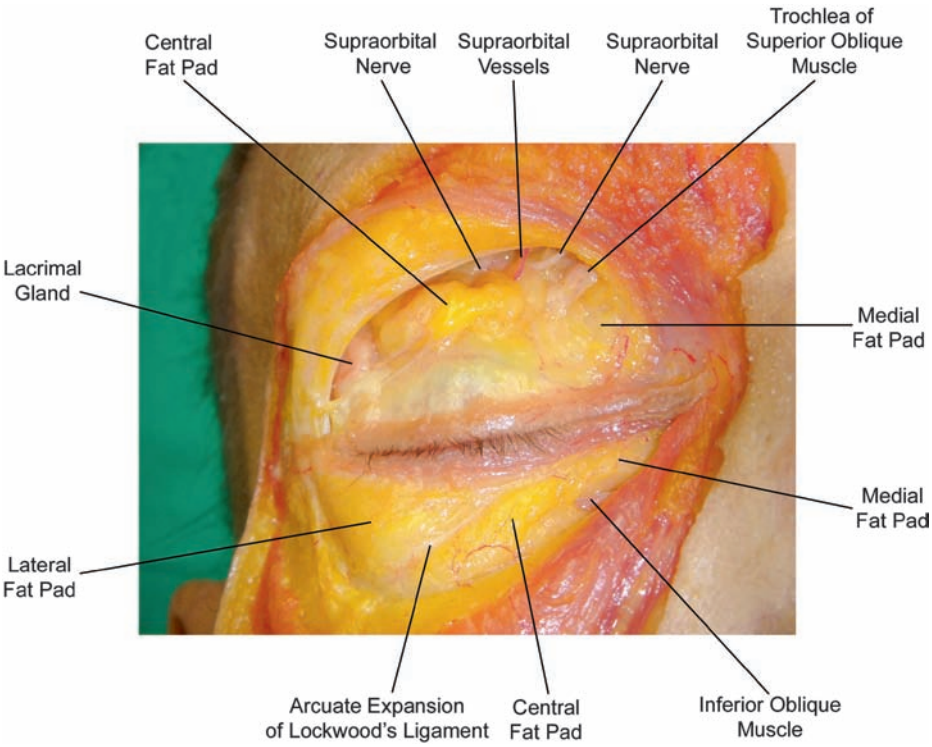

Figure 22.7. Upper and lower orbital fat pads.

which is prominent on external lid examination. Three retroseptal fat pads are found in the lower eyelid. The medial and central fat pads are separated by the inferior oblique muscle. The muscle's location makes it susceptible to injury during surgical dissection of the surrounding fat pads. The middle and lateral fat pads are separated by the arcuate expansion, the conjoined septum, and capsulopalpebral fascia extending to the inferolateral orbital rim (Figure 22.7).

Clinical Examination

Clinical examination includes visual acuity, ocular motility, visual field testing, and basic tear secretion testing using the Schirmer's test. The Schirmer's test is performed by placing a strip of filter paper over the lateral third of the lower lid conjunctiva and measuring the wetting on the strip after 5 min. If the measurement is less then 10 mm, the patient may have difficulty producing tears, predicting postoperative dry-eye problems. The value of the Schirmer's test is, however, controversial.

Examination of the patient should include an evaluation of specific landmarks, including palpebral fissure distance; margin reflex distance-1 (MRD1), which is the distance between the center of the pupil in primary position and the central margin of the upper eyelid; and margin reflex distance-2 (MRD2), which is the distance between the center of the pupil in primary position and the central margin of the lower eyelid. Ptosis of the upper eyelid should be suspected when the palpebral distance is less than 10 mm and MRD1 is less than 4 mm (Figure 22.8).

The individual components of the periorbital region are thoroughly assessed before surgery. The relationship of the brow position to the upper lid determines whether an isolated upper lid blepharoplasty is sufficient or whether brow position adjustment is necessary to achieve the desired results. This is done with the patient in an upright position and with the patient looking in a mirror to help judge how brow position affects the upper eyelid.

Once brow position has been determined, the surgeon assesses the components of excess skin,

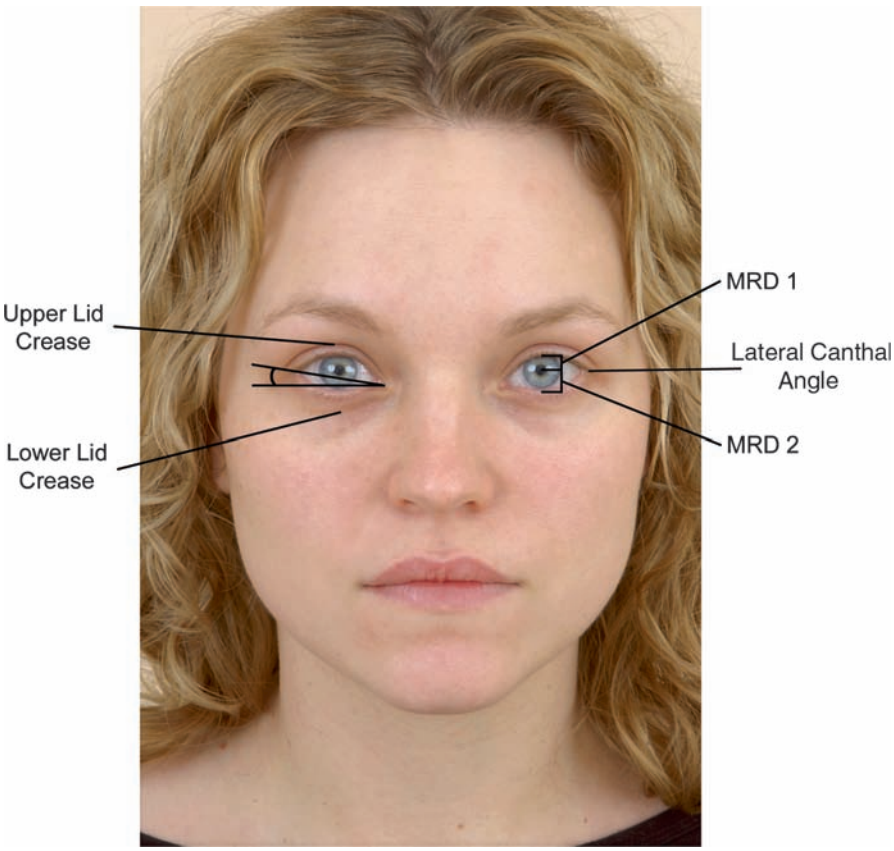

Figure 22.8. Margin reflex distance 1 and 2. An MRD1 less than 4 mm is indicative of upper eyelid ptosis.

skin laxity, and fat herniation in the eyelids. The most effective means of assessing excess lower eyelid skin is by asking the patient to look upward without moving their head. This stretches the skin and gives the surgeon an idea of how much redundant skin is present. Herniation of all the fat pads is also best tested with the patient's eyes in an upward gaze. Excess or herniated fat causes a protrusion or convex contour. Finally, careful assessment of lower lid tone is essential in all patients. A lax lower lid is an indication for lateral canthopexy, canthoplasty or wedge excision in some patients[14,16,20] (Figure 22.4).

The patient with a negative vector on clinical examination should be approached with care as such patients are at significant risk for the postoperative complications of scleral show or ectropion (Figure 22.9).

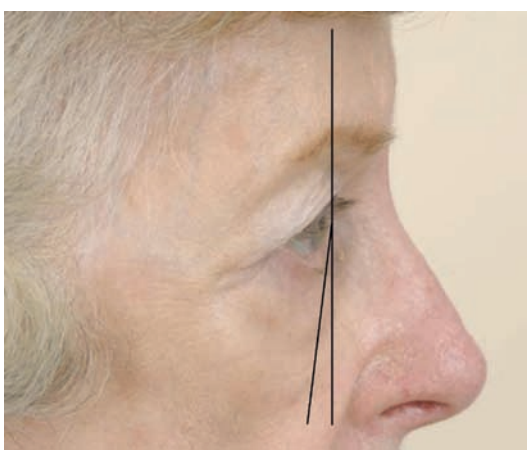

Figure 22.9. A negative vector is present when the corner of the globe is anterior to the anterior-most surface of the malar soft tissue prominence.

Surgical Techniques

Browlift

Brow position may be corrected by either endoscopic or open techniques. The endoscopic forehead lift has gained significant popularity, because it avoids the long coronal or hairline incision associated with coronal and hairline browlifts, respectively. Although it has many enthusiastic proponents,[4,10,24] it has also been criticized by others for its lack of correction and longevity.[33,34] There are those who have given up this technique entirely.

Endoscopic Browlift

The endoscopic browlift can be performed in the subperiosteal or the subgaleal plane. Our preference is to use the subperiosteal plane for both the ease of dissection and the enhanced endoscopic visualization. Because the skull is light in color, it reflects light from the endoscope and provides greater illumination during the procedure (Figure 22.10).

The operation is begun with a temporal incision made within the hairline approximately 1.5 cm in length. This incision is located on a line from the alar base to the lateral canthus. Once an optical cavity is developed under direct vision, the 5-mm endoscope is introduced, and dissection is continued to the temporalis fascia proper and then either to the intermediate fascia or just deep to the intermediate fascia, to the lateral orbital rim. Significant subperiosteal release needs to be performed along the lateral orbital rim to the lateral canthus and along the proximal portion of the zygomatic arch. An identical dissection is performed on the opposite side. Four scalp incisions are then made in the hairline corresponding to the medial and lateral orbital brow on each side. The subperiosteal dissection is continued to the arcus marginalis and the arcus marginalis is released in its entirety. Care is taken to protect the deep branch of the supraorbital nerve.[22] Muscle modification is performed by partial or total resection of the corrugator and, if desired, procerus muscles.

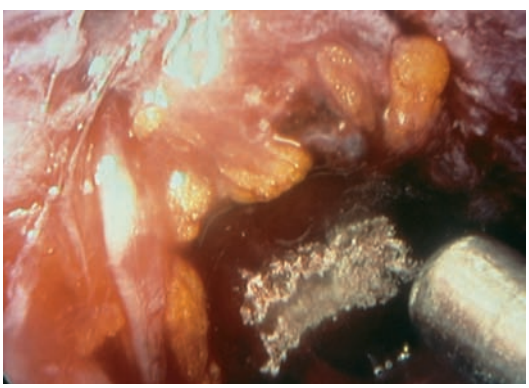

Figure 22.10. Endoscopic view of corrugator muscle using a 5-mm, 30° endoscope during endoscopic browlift. CO_2 laser was used to partially ablate the corrugator.

Although the need for bony fixation is controversial, we do fix the brow through the lateral incisions using a cortical tunnel technique, suturing the under surface of the brow flap through the cortical tunnel. Fixation of the temporalis fascia proper on the right and left sides is also performed, suturing superficial temporalis fascia superiorly and posteriorly through the temporal incisions on the right and left sides, respectively (Figure 22.11).

Open Browlift

In the case of the high forehead (7 cm or greater), the hairline browlift is preferred, since the brow can be elevated and the forehead lowered simultaneously. This is done through a hairline incision in the forehead area with extensions into the hair-bearing temporal region. Dissection can be performed in the subgaleal or in the subcutaneous plane over the non-hair-bearing skin. Subgaleal dissection is carried out in the non-hair-bearing areas. Injury to hair follicles is possible in the subcutaneous plane (Figure 22.12).

Upper Lid Blepharoplasty

Transcutaneous

Planned skin excisions are marked preoperatively with the patient in the upright position. The lid crease incision is marked first, just below the eyelid crease in the upper lid. The crease is curvilinear, with the arc of the incision peaking just medial to the central point of the eyelid. Nasally, the incision extends no further than an imaginary line projected upward from the medial punctum, to avoid potentially unsatisfactory

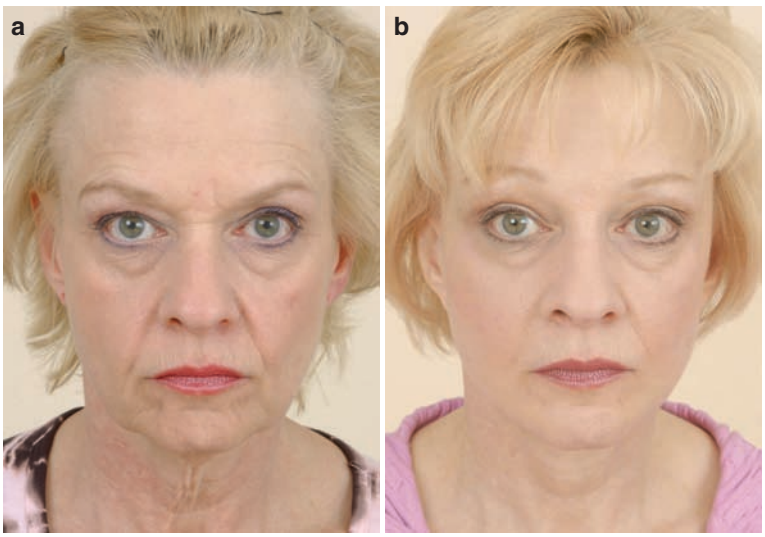

Figure 22.11. (**a**) Preoperative view of 57-year-old female with eyebrow ptosis and facial aging. (**b**) One-year postoperative view following endoscopic browlift, facelift with extended SMAS, and fat injections to cheeks.

Figure 22.12. (**a**) Preoperative view of 63-year-old patient with facial aging, eyebrow ptosis, deep corrugator rhytids, and a high forehead. (**b**) One-year postoperative view following hairline browlift, lower lid blepharoplasty, and facelift with extended SMAS dissection and autologous fat injections to cheek and infraorbital areas.

scarring in the medial canthal region. The temporal aspect of incision is then curved gently upward in a natural skin crease, being careful not to extend this mark beyond the orbital rim. Lateral extension may also result in a more prominent and visible scar.

One then gets the patient to gently close the eyelids. A smooth forceps is used to grasp the

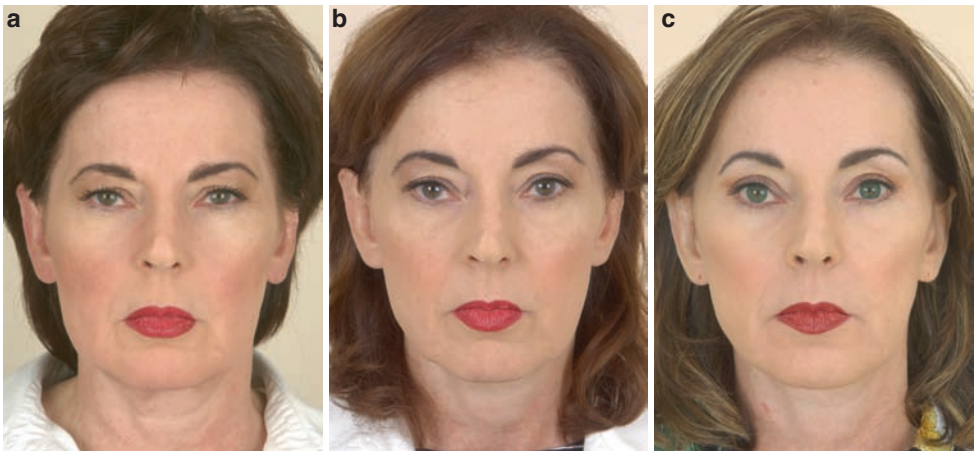

Figure 22.13. (**a**) Preoperative 56-year-old patient with skin excess of the upper lids and eyebrow ptosis. (**b**) One year following endoscopic browlift. (**c**) Two years following endoscopic browlift and 1 year following upper lid blepharoplasty and facelift with extended SMAS dissection.

excess skin above the eyelid crease incision until the eyelashes begin to rotate upward. This point is marked as the maximum amount of skin that may be safely removed. It is important that the superior border of the incision should pass no closer than 1 cm from the inferior border of the brow hairs. This prevents excess skin removal that may cause lagophthalmos and also prevents the blepharoplasty excision from causing downward traction on the brow position.

Next, one presses on the globe to observe protrusion of the fat pockets. Protrusion or prolapse of the lacrimal glands is also noted. The location and amount of sub-brow fat is assessed and considered for surgical contouring. This is especially relevant in the absence of a browlift procedure.

Upper lid blepharoplasty is performed using a scalpel or by CO_2 laser incisional techniques.[5,26] Local infiltration with lidocaine and adrenaline placed subcutaneously provides sufficient anesthesia. In addition, a protective scleral shell may be placed over the surface of the eye after topical administration of tetracaine. The excess skin is removed, either alone or with part of the underlying orbicularis muscle.

Removal of eyelid skin, sub-brow fat, reconstruction of the eyelid crease, eyelid ptosis correction, brow ptosis correction, modification of the glabellar wrinkles, or resuspension of the lacrimal gland may all be performed through this lid crease incision.[36]

After exposure or excision of part of the orbicularis muscle, one identifies the orbital septum. When necessary, the orbital septum is opened to expose the preaponeurotic fat. In the upper eyelid, two fat pockets are present: one central and the other medial. When gentle pressure is placed on the globe, the fat tends to protrude through the open septum. The medial fat pad has a pale white color, distinct from the deeper yellow of the central fat pad. Fat is excised as necessary to achieve the desired correction in contour.

If the lacrimal gland is found to be protruding, suturing it back in position inside the orbital rim prevents postoperative fullness in the lateral aspect of the upper eyelid.

To alter or emphasize the eyelid crease, a supratarsal fixation suturing technique is used to create adherence between the skin and underlying tissue. This is accomplished by attaching the subcutaneous tissue at the lower aspect of the eyelid crease incision to the levator aponeurosis just above the tarsus.

Commonly used materials for skin closure include nonabsorbable sutures, such as 6-0 nylon or 6-0 polypropylene, in a running subcuticular fashion, interrupted fashion, or in an external running fashion. Alternatively, 6-0 plain catgut may be used as an absorbable suture. All sutures are removed in 3–5 days (Figure 22.13).

Lower Lid Blepharoplasty

Lower lid blepharoplasty alone does not eradicate fine skin wrinkling, regardless of which technique is used. This issue is better addressed

when the blepharoplasty is combined with either CO_2 laser resurfacing or phenol-croton oil peeling. Numerous publications have described the combined use of CO_2 laser and transconjunctival blepharoplasty. Although Seckel has described as much as 30% skin contraction with CO_2 laser resurfacing, our experience is that this skin contraction is short lived.[27] However, the improvement in hyperpigmentation can be dramatic and longstanding. The other attractive alternative is the combination of transconjunctival blepharoplasty and phenol-croton oil peeling, pinch blepharoplasty and phenol-croton oil peeling, or secondary phenol-croton oil peeling after transcutaneous lower lid blepharoplasty. This leads to significant effacement of lower lid rhytides and improvement in hyperpigmentation[8,39] (Figure 22.14). However, when any of these alternatives are used in the presence of lower lid laxity, lower lid support should be provided in the form of a lateral canthopexy to prevent temporary lower lid malposition.

The presence of excess orbital fat is best assessed by examining the patient preoperatively, while he or she is in the upright position and by studying preoperative photographs. Once the patient is supine, judgment regarding excess fat is much more difficult. Gentle pressure on the globe with the eyelids closed causes excess fat to bulge anterior to the orbital rim. For bulging lower eyelid fat, the choices are to excise the fat, push the fat back into the orbit, or transfer the fat into the infraorbital rim hollow (tear trough deformity or nasojugular groove). Fat excision may be accomplished using a transcutaneous or transconjunctival approach.

Transcutaneous

The planed incision is marked approximately 2 mm below the ciliary margin in a natural skin crease below the lash line. The incision should not extend laterally past the orbital rim.

A skin only flap is elevated by first scoring the incision across the lower lid, taking care to protect the lashes. A No. 15 scalpel blade combined with a small hook for retraction is then used to elevate the flap off the underlying orbicularis muscle down to the orbital rim. If a skin muscle flap is chosen, the original incision is the same. The flap is raised preserving 4 mm of the attachment of the pretarsal orbicularis muscle. Once the orbicularis muscle is divided, the retroorbicular plane is readily identifiable and is again raised down to the orbital rim.

If a skin flap is elevated, the orbicularis muscle is opened over the medial, central, and lateral

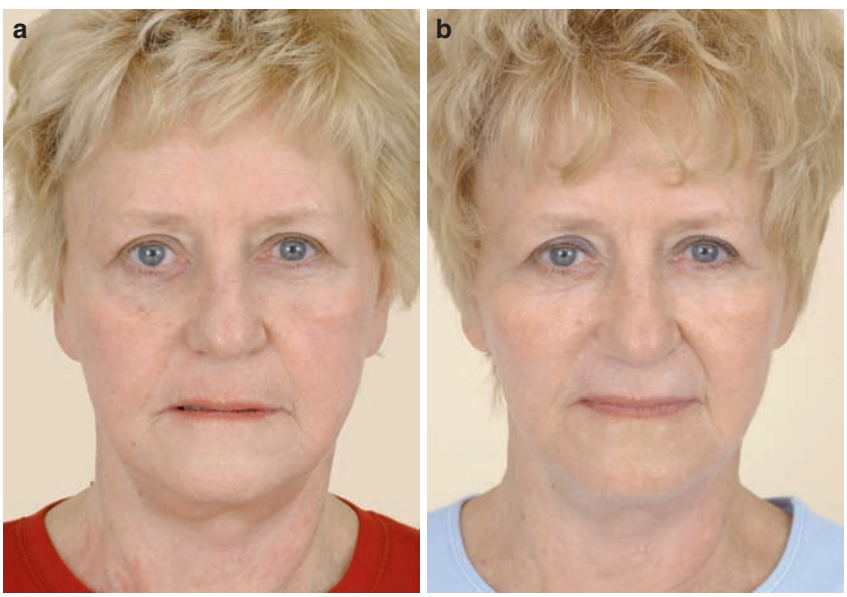

Figure 22.14. (**a**) Preoperative view of 69-year-old female with perioral and periocular rhytids and cheek ptosis. (**b**) Eight months postoperative view following endoscopically assisted cheeklift, periocular, and perioral phenol-croton oil peel.

compartments. Using a skin muscle flap, the compartments are readily visible. The inferior oblique muscle, separating the medial and central components, is identified and protected. (Figure 22.7) The orbital fat is teased out with a combination of gentle pressure on the globe and a small cotton-tip applicator.

To complete the procedure, the skin is redraped over the underlying lower lid structures. Skin excision should be performed conservatively, as excess skin removal is the most common cause of lower lid malposition following transcutaneous lower lid blepharoplasty. If the surgery is under local anesthesia, have the patient look up with the mouth open, to aid in conservative resection. Excess skin is trimmed, and absorbable or nonabsorbable sutures are used to reapproximate the existing edges.

Transconjunctival

Transconjunctival blepharoplasty is useful in patients with fat excess and fine skin wrinkling and in those with fat excess in whom fat excision alone allows for redraping of the lower lid skin.[3,37]

Transcutaneous blepharoplasty has been associated with the round eye appearance, inferior scleral show, and frank ectropion as a consequence of overgenerous skin resection.[38] Since lower lid malposition is obviated using this technique, the transconjunctival approach can also be used in the elderly patient with significant lower lid laxity simplifying the lower lid procedure.[28,38]

To anesthetize the conjunctiva and cornea, drops of tetracaine hydrochloride ophthalmic solution are instilled into the lower fornix of each eye. This is followed by transconjunctival injection of lidocaine with epinephrine injected into the orbital floor.

An incision is made on the lower lid conjunctiva using the Colorado needle 2–3 mm below the inferior tarsal plate. The incision is taken through conjunctiva and capsulopalpebral fascia exposing the fat pads.[30,31] Fat is trimmed judiciously as required. The quantity of fat resection is more difficult to assess via the transconjunctival approach than with a transcutaneous technique. Gentle pressure on the globe provides a guide to the level of fat resection. No sutures are used to close the incisions (Figure 22.15).

Options for pushing fat back into the orbit include septal plication[12] or capsulopalpebral fascia and conjunctiva plication to the orbital rim via a transconjunctival[6] or transcutaneous approach.[21]

Fat transfer has been described by both Loeb[19] and Hamra.[11] Loeb used suture fixation of transferred fat to the periosteum and to the origin of levator labii superioris. The septum was not advanced. Hamra's technique also included pulling down the septum with the fat, calling it a "septal inset." To have a free edge of septum to pull down with the fat, it had to be divided along the length of the orbital rim. Hamra termed this the "arcus marginalis release."[11]

Drawing the septum down with the fat (septal inset) has the advantages of creating a smoother layer filling in the depression and also tightening the septum, thus inhibiting further bulging of fat from the orbit, into the lower lid.

The planes of dissection for fat transfer into the tear trough deformity include both subperiosteal and supraperiosteal. The transferred fat can be sutured down to the periosteum and/or SOOF, sutured up to the underside of the orbicularis oculi, or fixated using transcutaneous bolster stitches.

It is important to note that these procedures require extensive dissection in the middle lamella of the lower lid in comparison with simple excisional techniques. These procedures induce more edema and more potential scar than those in simple excisional procedures. Therefore, postoperative support of lower lid position is imperative.

Complications of Blepharoplasty

Bleeding and infection are uncommon but serious complications. To help prevent a hematoma, hypertension is controlled, and medications that predispose to bleeding are discontinued 2 weeks before surgery. Careful hemostasis at the time of the procedure is also vital.

Retro-orbital hemorrhage and visual loss are rare.[29] Retro-orbital hemorrhage most frequently occurs following lower eyelid blepharoplasty, with an incidence of 1 per 22,000.

Infections after blepharoplasty are unusual because of the rich vascularity of the eyelids. However, prompt attention and treatment with appropriate antibiotics are required, when they do occur. The wound is opened, drained, and cultured, and any necrotic tissue is debrided.

Diplopia results from extraocular muscle imbalance, due to inadvertent damage to the superior or

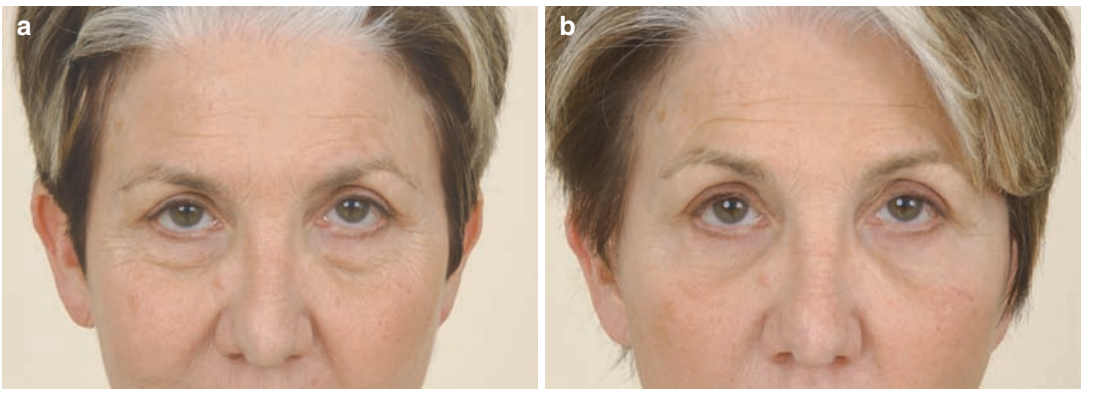

Figure 22.15. (**a**) Preoperative view of 60-year-old patient with lower lid bags. (**b**) Six-month postoperative view of the same patient following transconjunctival lower lid blepharoplasty and facelift with extended SMAS. Note the apparent skin tightening of the lower eyelids without worsening of the scleral show.

inferior oblique muscles during fat excision. A sound knowledge of eyelid anatomy, adequate hemostasis, and meticulous surgical technique make this complication readily preventable.

Blepharoptosis can occur secondary to inadvertent levator injury during the procedure. Transitory mechanical ptosis is occasionally found secondary to eyelid edema or hematoma, but if the ptosis persists following complete resolution of edema and swelling, repair of the levator aponeurosis will be required.

Excessive skin removal may result in lagophthalmos with exposure keratitis, ectropion, or downward traction of the brow position. Mild lagophthalmos is common in the immediate postoperative period and is treated with reassurance, lubricant eye drops, and ointment. Extension of incisions may lead to a web over the medial canthal angle or an unsatisfactory visible scar past the lateral orbital rim.

Ectropion is due to tissue deficiency of the anterior or middle lamellae. If ectropion does occur, the surgeon must determine the location of the pathology and address it. If the anterior lamella is deficient, skin may be replaced with grafting, but the best treatment is avoiding overresection. If ectropion is due to early formation of cicatrix in the middle lamella, injection with low-dose steroids may be used. If middle lamellar scarring and lower lid malposition or retraction are persistent, surgical release of middle lamellar scarring and space grafting may be required. Middle lamellar scarring should be suspected if the involved lower lid cannot be manually elevated to the top of the limbus.

Hollowing results from excessive fat removal. However, lower lid malposition and residual excess skin or fat are probably the two most common adverse sequelae following upper or lower eyelid surgery. Asymmetry of eyelid creases is the result of poor preoperative planning or a surgeon's attempt to alter the crease position. In patients with a preexisting unilateral ptosis, the asymmetry may appear more prominent following removal of the overlying skin folds.

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23

Nasal Reconstruction and Aesthetic Rhinoplasty

Devra Becker and Bahman Guyuron

Summary

The nose is one of the most prominent features on the human face, and rhinoplasty has preoccupied surgeons for centuries. The nose is often divided into thirds, each third has its own features. Nasal analysis begins with a frank discussion with the patient and includes a thorough history taking, including prior surgeries and any drug use. Analysis of the nose consists of the relationship of the nose to the face in the facial horizontal thirds, vertical fifths, and facial angles. It also includes an assessment of harmony between the nasal segments, nasal length, tip shape, projection, and rotation, and the alar–columellar relationship. Deformities of the upper vault include a dorsal hump, which is treated with rasping and occasionally block resection, or dorsal deficiency, which is treated with grafts. The tip can be modified by grafts, resection of alar cartilages, or suture placement. Nasal deviation must be localized to the upper, middle, or lower vault and is treated with either osteotomies for the upper vault or cartilaginous repositioning for the middle and lower vaults. All rhinoplasties require thoughtful planning and preoperative customization.

A nose which varies from the ideal of straightness to a hook or stub may still be of good shape and agreeable to the eye
—Aristotle

Abbreviations

N-AG	Nasion to the alar groove
SMAS	Subcutaneous musculoaponeurotic system
Sn	Subnasale
SON	Supraorbital notch

Introduction

Ideals of nasal aesthetics have preoccupied men for millennia. Indeed, descriptions of rhinoplasties date back to Sushruta (circa sixth century BCE), and some of his techniques endure today. Multivolume textbooks, and journals, concern themselves with the subtleties of nasal anatomy and techniques of rhinoplasty. It is the goal of this chapter to familiarize the reader with the fundamental principles of rhinoplasty. An understanding of nasal anatomy is necessary to understanding the logic of specific rhinoplasty plans. We will begin with a review of anatomy and discuss aesthetic and reconstructive principles in the context of specific nasal zones.

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Throughout this chapter, the assumption is made of an unoperated nose. Secondary rhinoplasties have unique challenges (often requiring slight modification of techniques), and analysis and management of the previously operated nose are beyond the scope of this chapter.

Anatomy

Most authors divide the nose into thirds: the upper bony vault (the upper third), the middle cartilaginous vault (the middle third), and the lower cartilaginous vault (the lower third).[22,23] Each region has its own anatomic features, from soft-tissue envelope to blood supply. Conceptualizing the nose in these three distinct regions makes the nose easier to analyze and facilitates planning surgery.

Skin

The quality of nasal skin will ultimately influence any aesthetic and reconstructive outcome.[21] The skin is slightly thick on the upper third of the nose.[20] It is also mobile and loosely attached to the underlying bony framework.[22] The skin becomes thinner in the middle third, and rethickens and increases in sebaceous glands in the lower third.[20] The subcutaneous fat is thickest at the supratip area.[17] In the lower third, the skin is also attached to the underlying cartilage. It is these differences in skin quality, and relationships to underlying structures, that explain why healing by secondary intention can be successful in the upper third but usually results in deformity in the lower third.[22]

The skin is also a tip-support mechanism, though small.[20] As we age, changes in the skin contribute to overall aesthetics of the nose. The skin becomes attenuated, and there is a loss of subcutaneous tissue.[21] Skin elasticity is diminished, and the tip sebaceous glands become denser. The aging nose, then, can have the appearance of being longer, with a nasal-tip ptosis, due to changes in the skin quality and weakening of the suspensory mechanisms.[12]

Muscles

Muscles of the nose serve two functions: animation and nasal airflow. The muscles themselves reside within the sheath of the nasal subcutaneous musculoaponeurotic system (SMAS). Continuous with the facial SMAS, the nasal SMAS holds the muscles in place, smoothly distributes the tensile force, and acts as "sling" during contracture.[20]

The eight nasal muscles are often categorized in four groups according to function[20,23]: the elevators, which act to shorten the nose and elevate the nostril (procerus, levator labii superioris alaeque nasi, and musculus anomalous nasi); the depressors, which lengthen the nose and dilate the nostrils (alar nasalis, depressor septi nasi); the compressors, which lengthen the nose and narrow the nostril (transverse nasalis and compressor naris minor); and the dilators (dilator naris anterior). Of these muscles, two are considered to have clinical import.[25] Inappropriate contraction of the depressor septi muscle can produce a downward deviation of the nasal tip during a smile. Hypofunction due to – for example – a facial nerve palsy, of the levator labii alaeque nasi, which keeps the external nasal valve open, can lead to nasal obstruction.[20]

Blood Supply

The blood supply to the nose derives from the internal and external carotid arteries. The internal carotid artery branches primarily supply the cephalic nose, and the external carotid artery primarily supplies the caudal nose.

Externally, the blood supply to the nose arises primarily from the angular artery, a terminal branch of the facial artery. Additional contributions are made to the dorsum and sidewalls from the infraorbital artery (a branch of the internal maxillary artery) and the ophthalmic artery.

Toriumi[30] showed that the blood supply to the external nose runs superficial to the musculoaponeurotic layer. This makes the safest plane for dissection below the musculoaponeurotic layer. Because the transcolumellar incision used in open rhinoplasty severs the columellar vessels, Rohrich studied the blood supply to the nasal tip to identify the impact of the incision on that blood supply. The study, which looked at 31 cadavers, showed that the lateral nasal vessels were 2–3 mm above the alar groove and that the columellar and lateral nasal arteries arose deep at the nasal bone and ended at the tip. The clinical conclusion of the study is that a columellar incision is safe, because the lateral nasal artery provides blood supply, but defatting compromises blood supply.[26]

Internally, the nose is supplied by the anterior and posterior ethmoidal arteries and the sphenopalatine artery. The anterior middle turbinate and septum are supplied by the lateral internal nasal branch, which is a branch of the anterior ethmoid artery (arising from the ophthalmic branch of the internal carotid artery). The superior turbinate and posterior septum are supplied by the posterior ethmoid artery (also arising from the ophthalmic branch of the superior ethmoid artery). Anteriorly, the nasal septum is supplied by the superior labial artery (a terminal branch of the facial artery). Additionally supplying the turbinates, lateral walls, sinuses, and septum is the sphenopalatine artery, a branch of the internal maxillary artery, after it divides into posterior lateral and posterior septal branches.[21]

Kesselbach's plexus (also known as Little's area) is an area of arterial anastomoses between the greater palatine, superior labial, sphenopalatine, and anterior ethmoidal arteries located in the anterior nasal septum. Trauma to this area can cause epistaxis.[21]

Nerve Supply

Sensation to the external nose is supplied by branches of the ophthalmic and maxillary divisions of Cranial Nerve V. The ophthalmic division provides sensation to the root and dorsum of the nose, whereas the maxillary division provides sensation to the nasal alae. The anterior ethmoidal nerve emerges from between the nasal bones and upper lateral cartilages and provides sensation to the nasal tip.

The sensory terminal branch of Cranial Nerve V2 is the infraorbital nerve, which emerges from its foramen and gives branches to the lower lid, upper lip, and nasal alae and columella.[23] The infratrochlear branch emerges from under the eyebrow and divides into a superior branch, which supplies the nasal root, and an inferior branch, which supplies the inner canthus and lacrimal apparatus.

Internally, the nose is innervated by the nasociliary nerve and sphenopalatine ganglion.

Osteocutaneous Framework

Upper Bony Vault

The upper third of the nose is made up of the paired nasal bones. They articulate with the frontal bone superiorly and the ascending maxillary processes – which make up the upper lateral sidewalls of the nose – laterally. The nasal bones are thick cephalomedially but taper and become thin inferiorly where they join the upper lateral cartilages. This meeting point between the nasal bones and upper lateral cartilages is known as the *keystone area*. The relationship between the nasal bones and the upper lateral cartilages is one that should not be disturbed[20,23] – an interruption can damage the internal nasal valve or can cause an inverted V deformity (depression of the nasal side wall).

The nasal bones themselves may be relatively short or long. An examination of the nasal bones is necessary during physical examination. In the case of short nasal bones, the cartilaginous portion contributes a greater proportion of the nasal skeleton, and the nasal bones themselves become less structurally significant. Osteotomies on short nasal bones to narrow the nose must be performed with caution[20,23] and must be performed as low as possible to avoid overnarrowing or an inverted V deformity.

Upper Cartilaginous Vault

The middle third of the nose is made up of the upper lateral cartilages (ULCs) and the underlying septum. Medially, the ULCs fuse with the septum – the angle made between the upper lateral cartilages and the septum is usually 10–15° – and constitute the internal nasal valve. Cephalically, the ULCs articulate with the nasal bones at the keystone area as described earlier. Laterally, the ULCs articulate with the piriform aperture, and inferiorly the ULCs articulate with the lower lateral cartilages in the *scroll area*. The scroll area has several different configurations, with slightly over half interdigitating[20] and with an overlap of 1–3 mm.[23] This is also a means of tip support.

Lower Cartilaginous Vault

The lower third of the nose, the nasal tip, receives its structural support from the alar cartilages (also referred to as lower lateral cartilages [LLCs]) and the septum. The alar cartilages themselves are shaped like arches with a medial crus, a middle (or intermediate) crus, and a lateral crus. There is variability in the thickness of the alar cartilages. The stability of the lower vault is dependent on the thickness and the strength of the alar cartilages. In addition, tip stability is dependent on the domal suspensory ligaments

at the scroll area, the suspensory ligament between the medial crura, and the attachments of the alar cartilages to the caudal septum.

Angle of Divergence

The relationship between the alar cartilages affects tip appearance. If the two domes are far apart from each other (a widened angle of divergence), the tip has a bulbous or boxy appearance. If the individual dome arcs are wide, the tip appears bulbous.

Tip-Defining Points

The supratip breakpoint occurs just cephalad to the superior alar cartilages. It is created by the relationship of the lateral crura, which slant posteriorly, and the dorsal septum. The tip-defining points are the points at which the medial and lateral domes meet with a thickening of cartilage and can be further identified by a light reflex.[7] The pronasale is the most anterior point of the nasal tip on a lateral view. The terminology becomes confusing, as some authors refer to the pronasale as a tip-defining point.[4]. The columella breakpoint is the point at which the columellar segment of the medial crus becomes the lobular segment of the middle crus.

The tripod theory of nasal tip stability was originated by Anderson.[1] In this model, the caudal leg is the medial crura, and the cephalic legs are the lateral crural complexes. Tip position can be modified by altering the relationship between the legs or by altering their absolute position. If the caudal leg is shortened relative to the others, the nasal tip is turned caudally and posteriorly. If the upper legs are shortened, the tip is displaced cephalad and posteriorly. If all three legs are shortened, the tip is displaced upward and backward, and if the caudal legs are lengthened, the tip moves cephalad and anteriorly.

Function

The nose has several physiologic functions. It humidifies inspired air, participates in heat exchange, provides speech resonation and olfaction, and filters particulate matter. In addition, it provides a first-contact immunological defense; most of the protein content of nasal mucous is immunoglobulins.[21]

The nasal airways have a twofold higher resistance than the oral airway. This comes in large part from two sources: the nasal valves and the *nasal cycle* of mucosal swelling. The main portion of the nasal airway[21] is 130 mm^2, and at the level of the internal nasal valve, it is 20–40 mm^2. This drop in surface area at the nasal valves provides resistance to incoming air. It also causes turbulence of the inspired air, which results in more contact between the air and the nasal mucosa.[21] The nasal cycle is a process by which the capillaries and vessels become engorged. The cycles occur over a time period of 30 min to 4 h. Although at any one point in time, the nasal resistance differs between sides, the total airway resistance is kept constant by this cycle.

Approximately, 85% of adults are preferentially nasal breathers.[21]

Nasal Analysis

Thorough nasal analysis always begins with a frank discussion regarding the patient's current perception of his or her nose and personal goals for rhinoplasty. Unrealistic expectations for surgery should be identified and noted. A full nasal history should be obtained, including history of trauma or prior nasal or facial surgery and use of prescription and over-the-counter medications, as well as use of illicit drugs and tobacco. The quality of skin should be noted, including general texture and thickness. This has importance for surgery, as thick skin tends to disguise operative intervention but can result in more postoperative edema.[19] The remainder of nasal analysis proceeds in a systematic way to identify problems.

Vertical Fifths

The face is first divided into vertical fifths. The fifths are calculated from the edges of the ears to the lateral canthi, from the outer canthi to the inner canthi, and one nose width. These fifths should be approximately equal. The width of the alar base should be, in this measurement, 2 mm wider than the width of the intercanthal distance. In addition, the bony base of the nose should be assessed and should be approximately 80% of the alar base. A ratio larger than this can indicate a need for osteotomies.[19]

Horizontal Thirds

The face is then divided into horizontal thirds, from the hairline to the supraorbital notch (SON), from the SON to the subnasale (Sn), and finally from the Sn to the menton. The lowest third is then again divided, from the oral commissure to the Sn (which represents one-third) and from the oral commissure to the menton (which represents two-thirds).

If the components do not represent their expected ratios, it may be evidence of an underlying bony dysmorphology.[19]

Nasal Length

The dorsal aesthetic lines should then be examined for symmetry, and any deviation should be noted. Nasal length should be one-third of the total facial height, and the width should be 70% of the length. Byrd and Hobar describe measuring nasal length by calculating the radix to tip length, which should be the same length as that from the stomion to menton.[5]

Nasal Tip

The nasal tip is then assessed for projection and rotation. First, the supratip break and tip-defining points are noted. Tip projection is calculated using Goode's ratio, in which a vertical line is drawn from the nasion to the alar groove (N-AG) on a lateral view. A line is then drawn from the nasion to tip, and a line perpendicular to N-AG is drawn from the alar groove to the tip. The ratio of the length of the alar groove-tip line:nasion-tip line should be 0.55–0.6:2.

Facial Angles

Farkas[8] measured distances between landmarks and the angles they formed in his work *Anthropometry of the Head and Face in Medicine*. Those measurements relevant here characterize the nose in relation to the rest of the face. Facial angles allow us to deduce three-dimensional relationships from two-dimensional analysis. There are four measurements that are most useful in nasal analysis. The first three measurements, nasofrontal, nasofacial, and nasomental angles, use a common line: a line along the dorsum of the nose that intersects the nasion. This line is created ignoring localized abnormalities of the nasal dorsum. The nasofrontal angle takes this nasal dorsal line and a line intersecting the nasion and tangential to the glabella. It is therefore the angle formed between the nose and the forehead. A normal nasofrontal angle is 115–120°. Patients with oxycephaly have a sloping forehead and an extremely obtuse nasofrontal angle. Often, these patients will present for rhinoplasty, but correction of the nasofrontal angle through forehead advancement is what is needed.

The nasofacial angle takes the nasal dorsal line and forms an angle with the anterior facial plane, which is defined as a line between the glabella and the pogonion. This angle should be between 30 and 40° (ideally 36°). The nasomental angle takes the line along the dorsum of the nose through the nasion and intersects it with a line from the pronasale to the pogonion. The normal angle for this is 120–132°.

The last measurement is the columella-labial angle. This measurement describes the relationship of the nose to the upper lip. The first line is taken from the Sn to the labrale superius, which defines the cant of the upper lip, and forms an angle with a line from the Sn tangential through the caudal-most portion of the columella. Normal measurements for men are between 90 and 95° and for women between 95 and 110° (Figure 23.1).

It should be noted that authors and practitioners may vary in their techniques for determining these angles. Anatomic points can be used instead of tangential lines. Though anatomic points have greater inter- and intrarater reliability than tangential lines,[18] tangential lines may be more useful for individual operative planning.

Alar–Columellar Relationship

Gunter classified the alar–columellar relationship in six types.[10] To determine the relationship, first a line is drawn through the long axis of the nostril, the anterior and posterior-most points of the oval-shaped nostril on lateral view. The distance from this line to the alar rim cephalically or the columella caudally should be 1–2 mm. A distance too large or too small in either of these directions classifies a deformity, as does a combination of the two. Type I is hanging columella, Type II is retracted ala, Type III is a combination of I and II, Type IV is hanging ala, Type V

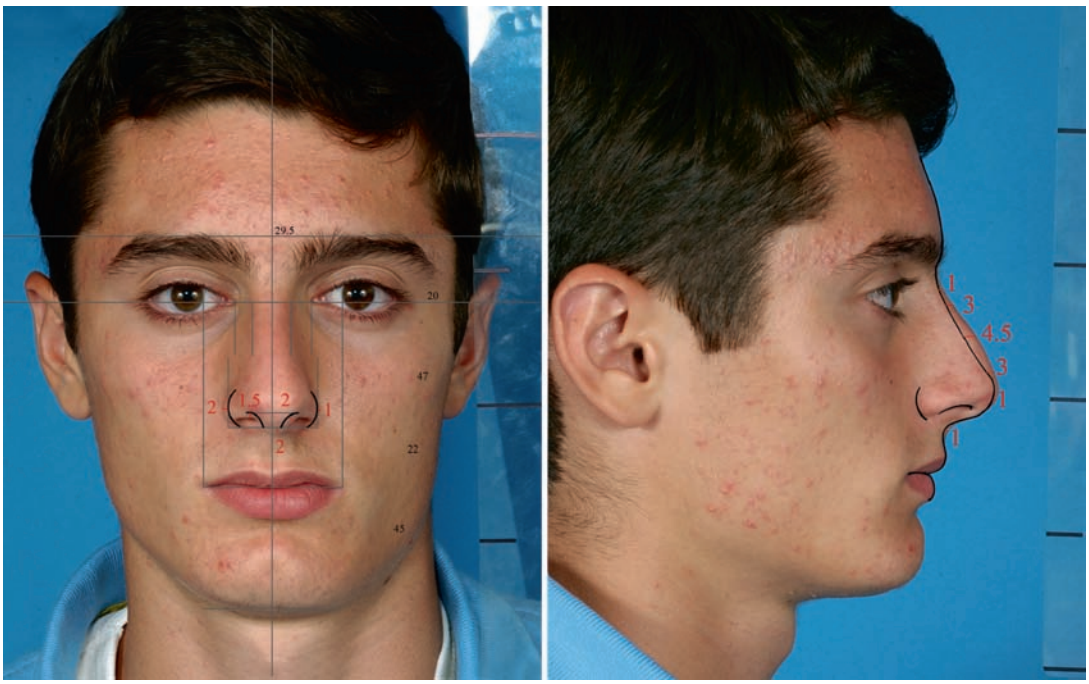

Figure 23.1. Facial analysis is performed from standardized life-sized photographs with a transparent overlay. The overlay allows the surgeon to view ideal angles and visualize the deviation from the ideal present in the photographs.

is retracted columella, and Type VI is a combination of Types IV and V.

A hanging columella is generally treated with resection of the membranous septum and the caudal margin of the medial crura. A retracted ala can be treated with a composite graft (septal cartilage and mucosa or conchal cartilage and skin) or an alar strut graft placed parallel to the nostril rim.

A hanging ala, as in Types IV and VI, is generally treated with excision of the vestibular skin above the most hanging component of the ala. A retracted columella can be treated with a cartilaginous columellar strut graft.

Deformities of the Upper Vault

In an unoperated nose, deformities of the upper vault are relatively easy to identify, although aesthetic correction of defects can be challenging and elusive. At its simplest, the upper vault can be either too big, as in the case of a dorsal hump, or too small. A dorsal hump will require reduction, whereas upper vault deficiency will require augmentation.

Dorsal Hump

The general principles of dorsal hump correction, as described by Ortiz-Monasterio, are as follows: (1) do not overcorrect, (2) limit undermining to preserve blood flow and maintain stability, and (3) preserve the integrity of the mucosal lining. There are two basic methods of correction. One can perform the entire resection in one piece with an osteotome, or one can undertake progressive resection with a rasp (Figure 23.2).[23]

One of the most significant complications from resection of a dorsal hump is over-resection[23] and subsequent iatrogenic internal nasal valve narrowing (and collapse) and the so-called *inverted V deformity*, which occurs when the attachments between the nasal bones and upper lateral cartilages become disrupted, and the upper lateral cartilages become relatively short.

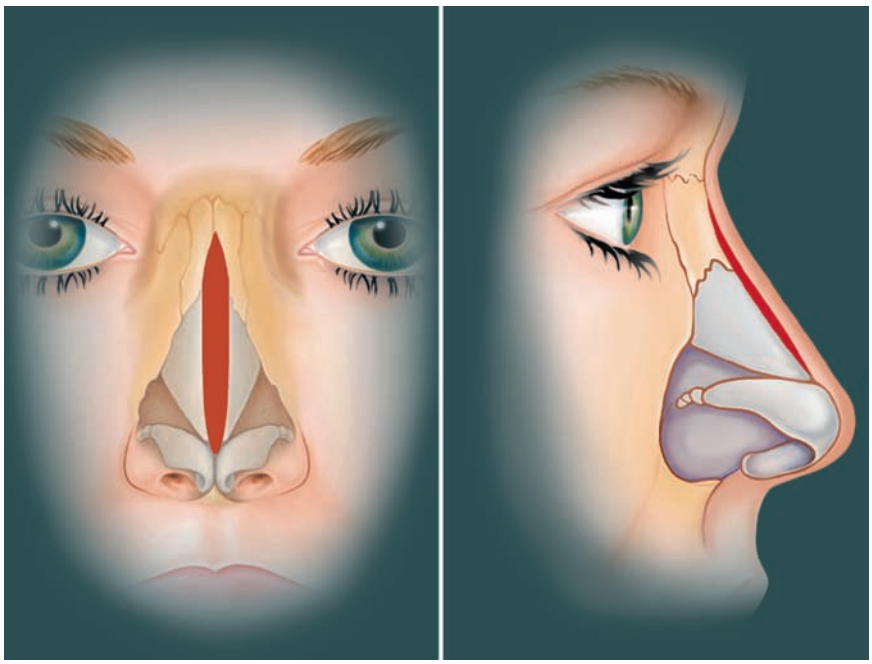

Figure 23.2. Dorsal hump correction.

The caudal edge of the nasal bones become visible, and the resulting soft tissue envelope looks like a ribbon placed transversely across the nose in the shape of an inverted V. Risk factors for this deformity include short nasal bones, because the ULCs are easily resected as part of the nasal hump.[23]

Because of such complications, progressive resection gained favor as a means of managing the dorsal hump. This technique allows finer control of the ultimate amount of resection. Handheld rasps are commonly employed for this purpose, and Rohrich describes the use of a scalpel blade or septal scissors.[27] The senior author (B.G.) first described the use of a power-assisted, guarded burr for deepening the nasion in 1989 (Figure 23.3).[11,14] Nine years later, in 1998, Becker reported using shielded cutting burr with good results in a series of 30 patients.[2] In 2003, Davis reported on 92 patients who had dorsal hump reduction using power-assisted rasps. He lists seven indications for using power-assisted rasps: (1) a small deformity, (2) a shallow nasofrontal angle, (3) thin or brittle nasal bones, (4) unstable nasal bones, (5) a narrow nasal vault, (6) modest bony asymmetry, and (7) bone smoothing before augmentation.[6]

Dorsal Deficiency

Augmentation of the nasal dorsum is indicated when the dorsal line on lateral view is more than 2 mm posterior to a line from the nasofrontal angle to the pronasale on a female. Options for augmentation include cartilage graft (septal, conchal, or costal) or alloplastic onlay grafts (such as alloderm). The length of the graft depends on the size of the nose but usually varies between 25 and 40 mm.

Nasal Tip

The nasal tip is usually described in three ways: by shape, projection, and rotation. Changes to each of these require different interventions.

Tip Shape

The tip shape is defined by the shape of the lower lateral cartilages and their relationship to each other, as well as the soft tissue envelope. Malformed nasal tips develop in large part because of malformed cartilage. Columellar lengths and nostril shape are to a large degree determined by the

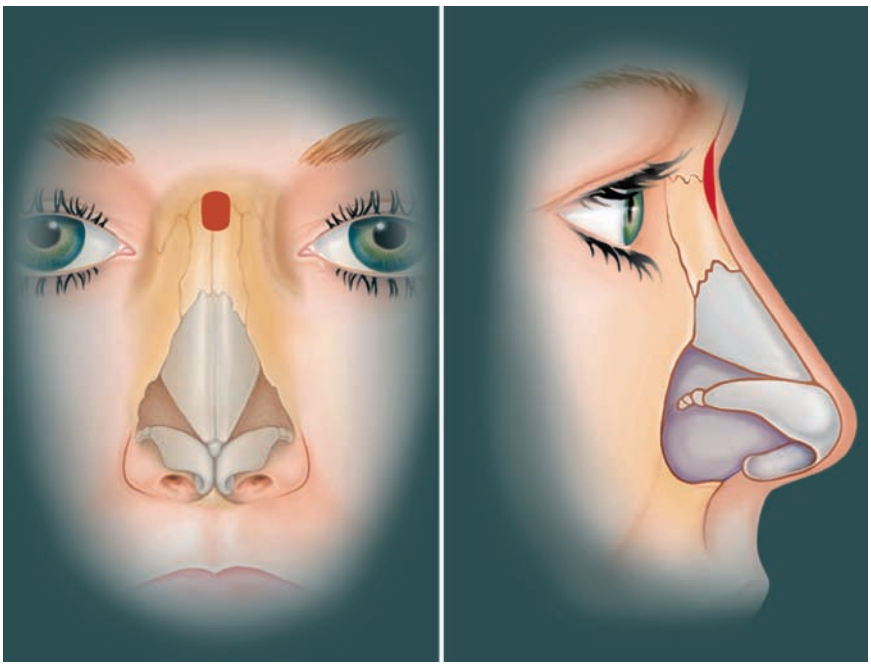

Figure 23.3. Nasion reduction. We perform nasion reduction with a guarded burr.

footplates of the medial crura.[13] A boxy nose is usually so because the lower lateral cartilages are far away from each other.

Historically, tip correction was achieved by resection of these cartilages. The long-term results were poor, however, and about the same time advances were being made in cleft lip and palate surgery through the use of sutures. Over the course of the twentieth century, rhinoplasty techniques evolved to stress reshaping cartilage rather than resecting it and incorporated suture techniques (Figure 23.4).[3] The medial crural suture is used to narrow and increase lobular size in a wide nasal tip. The middle crural suture, placed slightly more anterior than the medial crural suture, is used for a wider nasal tip, since it results in a more significant reduction of the interdomal distance. The anterior-most stitch to reduce wide domal arches is the transdomal suture.

The soft tissue envelope can be thinned by trimming excess soft tissue between and covering the domes.

Tip Projection

Tip projection relies on the integrity and support of the lower lateral cartilages, as well as the soft tissue structures such as the interdomal ligament. Thus, increasing projection requires augmenting these structures. Several suture techniques increase projection. These include *transdomal* sutures, which are useful in a bulbous tip; *columellar-septal* sutures, which can be used in place of a graft; and *medial crural* sutures, which increase tip projection by 1–2 mm. *Interdomal* sutures will also provide minimal increases in tip projection. Posteriorly, because the medial crural footplates act as pillars of the tip,[13] a *U-shaped* stitch in the footplates can have the effect of increasing tip projection. This stitch also narrows the columellar base and improves nostril shape.

In cases where tip projection needs to be augmented more than sutures can provide, grafts are appropriate. Though grafts have many variables that can change the ultimate result,[29] they can be very useful. The Peck onlay graft[24] can be used for tip projection; a variation of this graft is the anatomic graft described by Gruber.[9] Sheen described customized grafts and infralobular grafts to achieve tip projection.[28] The infralobular graft can be beveled or morselized. An advantage of tip grafts, in general, is that they can be stacked to achieve the desired projection. The senior author uses special punch devices to harvest cartilage graft.[15]

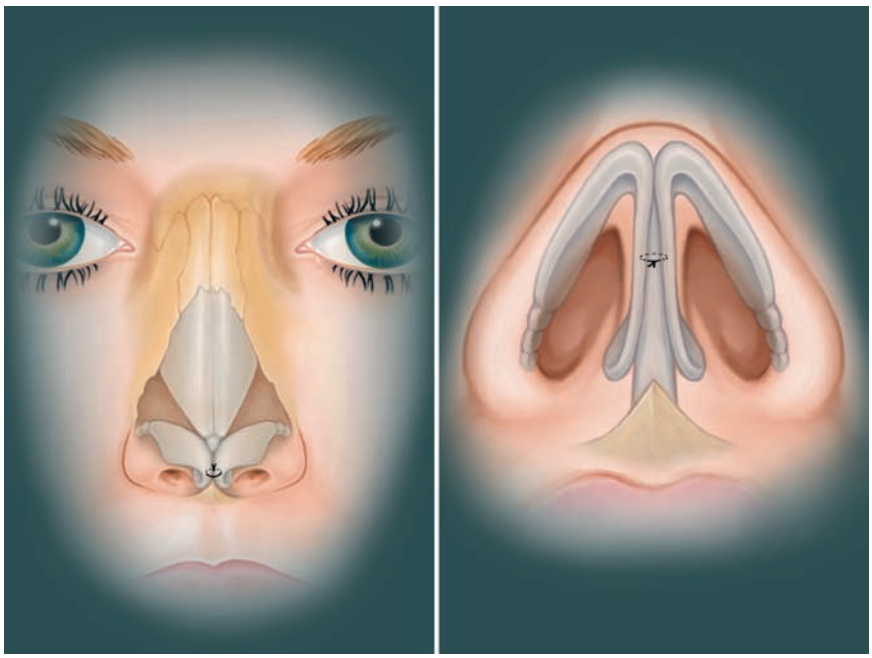

Figure 23.4. Medial crural suture placement. This suture narrows and increases lobular size in a wide nasal tip. We increasingly use sutures, rather than resection alone, to perform tip correction.

To decrease tip projection, the medial crura can be shortened, depending on the aesthetic goals. If the nose needs to be shortened and the projection needs to be reduced, the lateral crura are transected and overlapped. If the nose is short, the medial crura are adjusted. If the nasal length is optimal, both medial and lateral crura are adjusted.

Tip Rotation

Tip rotation can be changed by cartilaginous resection, columellar strut grafts, or by suture placement.

Resection of the cephalic portion of the lower lateral cartilages (a cephalic trim) causes slight cephalic rotation of the nasal tip. At least 5 mm of the lower lateral cartilages must be left to maintain structural integrity. In addition to rotation, a cephalic trim will help to accentuate the tip-defining points, decrease tip projection, and decrease tip fullness. Resection of the caudal septum will also rotate the tip.

Columellar strut grafts help rotate the tip by providing support along the arc (Figure 23.5).

A tip rotation suture acts as a harness for the lower lateral cartilages and increases tip rotation. The suture is placed caudal to or through the medial genu bilaterally and then through the anterocaudal septum (Figure 23.6).

Nasal Deviation

The nose can be deviated cephalically, which is due to bony deformation, or caudally, which is due to cartilaginous – usually septal – deviation. Bony deviation can be corrected by osteotomies, and there are four types of osteotomies: transverse, low to high, low to low, and double. Osteotomies can also be used to narrow the bony nasal vault. A transverse osteotomy is placed caudal to the nasal radix. It usually requires a lateral osteotomy to be effective. The remaining osteotomies are chosen based on the size of the open-roof deformity. A low-to-high osteotomy is used for small open-roof deformities and has the advantage of a minimal effect on the internal nasal valve. A low-to-low osteotomy is used for larger open-roof deformities, and double osteotomies (in which the medial osteotomy is performed first) are used for the largest open-roof deformities. To prevent narrowing of the nasal valve due to infracture, spreader grafts, pieces of

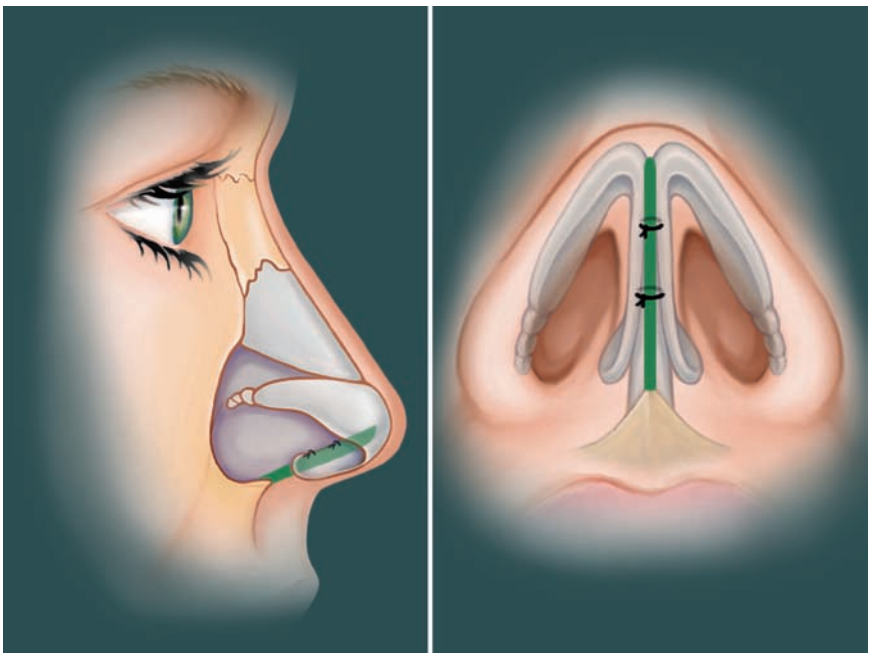

Figure 23.5. Columellar strut graft. These grafts provide stability and also help rotate the tip.

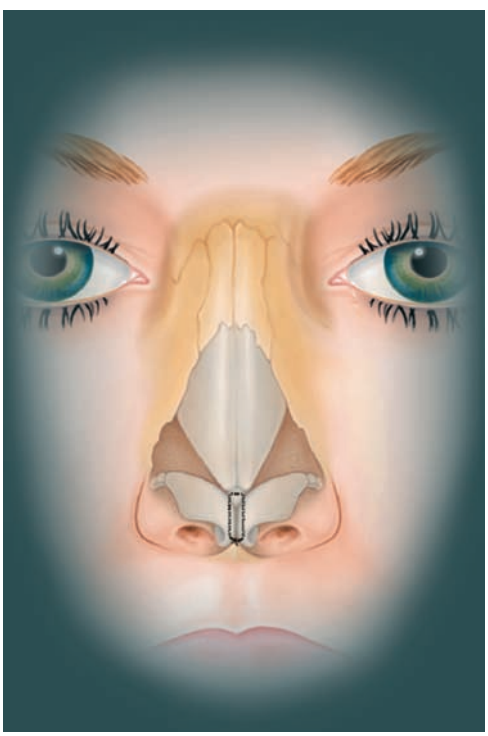

Figure 23.6. A tip rotation suture acts as a harness for the lower lateral cartilages.

cartilage graft placed between the upper lateral cartilages, are used to stent them open.

Septal deviation can be classified based on the types of septal curvature. Six types of septal deviation have been described[16]: septal tilt, in which there is no curve, but is one side anterior and another posterior, C-shaped AP deviation, C-shaped cephalocaudal deviation, S-shaped AP deviation, S-shaped cephalocaudal deviation, and localized deviation.

Rhinoplasty Dynamics

The concept of rhinoplasty dynamics is a useful one. Because the defects can be so varied, and the small technical details make the operation a success or failure, rhinoplasty is not a single operation that is modified based on the type of nose; rather, rhinoplasty consists of techniques that are chosen individually for the procedure based on the defect and desired effect. Therefore, it is useful to know what each procedure will do.

The reduction of the nasion will lengthen or shorten the nose, depending on where it is placed, and will increase or decrease intercanthal distance.

Dorsal augmentation will narrow the nose, whereas dorsal reduction will widen it. Osteotomies decrease intercanthal distance, and transdomal sutures increase nasal tip projection by 1–2 mm. A columellar strut will widen and lengthen the columella, widen the nasolabial angle, and advance the Sn. Approximation of the footplates results in a gain in projection, though small. A cephalic trim will result in a decrease in nasal tip projection. A small tip graft will narrow the tip, as will interdomal sutures.

Our Operative Technique

A full discussion of operative risks and benefits is undertaken with the patient in the office setting. Two weeks before the surgery, the preoperative visit takes place, and the patient is offered an opportunity to ask any questions he or she may have. Our general rhinoplasty sequence is as follows: exposure, radix work, upper vault correction, septoplasty, turbinectomy, middle vault/spreader graft placement, tip work (including columellar strut grafts, tip rotation sutures, and domal sutures), alar rim grafts, and finally skin closure.

Once in the operating room, the patient is placed supine on the operating room table, and general anesthesia is induced. Gauze saturated with 4% cocaine is packed posteriorly and cephalically in each nostril. The external nose is then injected with 0.5% xylocaine with 1:200,000 epinephrine using a 25 ga needle, and the turbinates are injected as well if a turbinectomy is planned. The posterocaudal septum and columella are injected. After several minutes, the injection process is repeated using 1:100,000 epinephrine.

A step incision is made on the columella and traced along the caudal edge of the medial and lateral crura of the alar cartilages. The medial crura are exposed, and dissection takes place using baby Metzenbaum scissors in this plane, exposing the lateral crura. The nasal dorsum is then exposed to the level of the nasal bones. A periosteal elevator is then used to continue the dissection in the subperiosteal plane.

At this point, attention is paid to the radix. If necessary, the radix is deepened using a guarded burr from left to right. The goal of radix reduction is to have the deepest part be at the level of the upper tarsal crease. (DALLAS Rhinoplasty).

The dorsal hump is then reduced with a push rasp, with the sides of the nose protected by the surgeon's fingers.

The upper lateral cartilages are divided in the midline in those patients who have dorsal bumps or deviation.

A septoplasty is performed first by making a left-sided mucoperichondreal flap. The septum is dissected out with a periosteal elevator. The correct plane is identified by the gray color of the cartilage and the relative ease of cephalic and posterior dissection.

At this point, the medial crura can be separated to increase exposure, though we perform this if separation of the medial crura is needed for other reasons. A portion of the soft tissue between the medial crura and the footplates is removed, and the depressor is partially resected. Septal cartilage is then harvested with a swivel blade. A 1-cm strut is left anteriorly and caudally to provide support. A one-sided septal perforation, if small, may be left alone. If the perforation transverses both sides, a small piece of septal cartilage is replaced.

If the maxillary crest of the vomer bone is deviated, this portion is removed using a rongeur. The anterior nasal spine is assessed for position – if deviated, it is placed in the midline using osteotomies. The anterocaudal septum is released from its attachments and replaced in the midline and secured to the anterior nasal spine (ANS) using 5-0 PDS suture.

The inferior turbinates are resected using turbinate scissors, and the raw area is cauterized.

Doyle stents are then placed and secured using 5-0 prolene sutures. We place the Doyle stents prior to the osteotomies to ensure patency of the nasal airway after osteotomies.

We perform a medial osteotomy using a 4- or 6-mm osteotome first. A 2-mm osteotome is then placed percutaneously for a vertical osteotomy. A low-to-low osteotomy is then performed.

Spreader grafts are then designed using the harvested septal cartilage. They are fixed to the middle vault using a 5-0 vicryl stitch. The septum is then resecured to the ULCs.

To rotate the tip, we use a tip rotation suture after placement of the columellar strut graft. The columellar strut graft is secured with two sutures, including a medial crural suture on the caudal margin. For cephalic rotation, a triangular piece of caudal septum, along with soft tissue, is removed.

If there is excess to the medial footplates, they are now excised and sutured together through the membranous septum.

The Doyle stents are further fixated with 4-0 prolene.

We then place a lateral crura-spanning suture, and a transdomal suture is used if the domal arches are wide. If the interdomal distance is large, we place an interdomal suture. A subdomal graft is placed for asymmetric domes or domes that are too close.

The columellar incision is then repaired with 6-0 fast. Alar rim grafts are placed.[31] Then, 6-0 fast sutures are used to repair the alar bases, and steristrips are applied over mastisol. An aquaplast splint is placed over the steristrips, and metal splints are used to guide the aquaplast.

The external and internal splints remain in place for 8 days. The Doyle splints are removed before the external splint. All patients receive cephalosporin antibiotics as long as the Doyle splints are in place; patients without contraindication receive a Medrol dose pack.

Conclusions

Rhinoplasty is a challenging and rewarding facet of plastic surgery. The best results are achieved through careful preoperative assessment and planning and meticulous execution of operative plans.

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24

Lip and Cheek Reconstruction

Matthew J. Carty and Julian J. Pribaz

Summary

The lip and cheek serve key functional and aesthetic roles with regard to the face. Due to their specialized structure, the lips and cheek pose special challenges when they are in need of reconstruction. Reparative strategies for both regions are reviewed in detail in the context of historical advances, current understanding of underlying anatomy, functional concerns, and overriding goals and principles. Reconstructive options are discussed in terms of graduated complexity, including linear closure, local tissue advancement, regional flap reconstruction, and microvascular free tissue transfer reconstruction, supplemented by a review of the potential role of techniques including tissue expansion, flap prelamination, and flap prefabrication. Finally, the special challenge of reconstructing combined lip and cheek defects is discussed.

Abbreviations

FAMM	Facial artery musculomucosal
SMAS	Superficial musculoaponeurotic system

Lip Reconstruction

The lips serve a critical functional and cosmetic role in the overall landscape of the human countenance. In addition to assisting with sustenance and speech, they provide an aesthetic transition from the outer world to the inner alimentary environment and are inherently linked to intimate physical contact in many cultures. Simultaneously, the lips demonstrate an exceedingly complex and delicate structure punctuated by tissues with unique features such as the vermilion that are both prone to injury and relatively unforgiving to distortion.

In general, the etiology of lip defects may be broadly grouped into congenital and acquired categories. The focus of this chapter will be on lip loss related to acquired processes; surgical strategies to meet the challenges of congenital processes will be discussed elsewhere. Among the acquired causes of lip injury, cutaneous malignancy is by far the most prevalent, with squamous cell carcinoma being more common in the lower lip due to increased exposure and basal cell carcinoma witnessed more frequently in the upper lip. Other common mechanisms of acquired lip loss include trauma and burn injury.

Historical Context

Reconstructive lip surgery has a rich history seeded both in recent as well as ancient times. Passages from the *Sushruta Samhita*, for example, provide evidence of the employment of local and regional flaps for the reconstruction of lip and nasal defects as long ago as 600 BC. Other early reports of lip reconstruction can be found

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in *De Medicina* by Celsus, as well as in later renaissance works by Branca, Antonius, and Tagliacozzi.[1]

The subsequent historical record of advances in lip reconstruction is sparse until the early 1800s, when began the veritable pageant of technical and philosophical advances that continues to the present day. In 1829, Dieffenbach described bilateral cheek advancement flaps for lower lip reconstruction and later advocated perialar tissue excision to limit tissue redundancy.[2] His contemporary, Sabattini, described the first cross-lip flap with transfer of a lower lip segment for reconstruction of a philtral defect; although often attributed to Abbe or Estlander, Sabattini's description of this procedure in 1838 remains the earliest in the modern medical literature.[3] A decade later, Stein provided the first description of bilateral central cross-lip flaps from the upper to lower lip.[4] Shortly thereafter, in 1853, Bernard espoused his technique of full-thickness wedge excision paired with cheek advancement flaps for the repair of lower lip lesions. Of note, Bernard's technique included the first description of the technique of excising triangular wedges at the base of an advancement flap to assist with tissue mobility and limit redundancy, although this maneuver was later credited to Burow.[5] In 1857, von Bruns provided the first clear description of the use of caudally based nasolabial flaps for lower lip reconstruction, later elaborated on by Denonvilliers in 1863.[6,7] Estlander's description of upper to lower lip transfer at the commissure in 1872 represented an extension of Sabattini's original design, as did Abbe's recapitulation of the central cross-lip flap in its application for repair of cleft lip defects in 1898.[8,9] Interspersed between these two reports was von Esmarch et al.'s[10] initial description of the mucosal advancement flap as a means to effect repair of vermilion defects in 1892.

The advent of the twentieth century witnessed an explosion of advances in lip reconstruction through a combination of modifications to prior techniques and the introduction of new surgical approaches. In 1920, Gilles described the cheek fan rotation flap, emphasizing the importance of reconstructing "like with like" tissues.[11] In 1934, Esser proposed the use of the forehead flap for lip reconstruction.[12] In 1954, Kazanjian described a modification to Stein's approach to cross-lip repair in which the philtrum could be preserved between wedge flaps recruited from the lateral elements.[13] Owens depicted the use of bilateral rectangular advancement flaps for closure of lip defects in 1955, followed, thereafter, by Webster's modification of the Bernard cheek advancement flap in 1960.[14,15] In 1964, Spira and Hardy elaborated on von Esmarch's notions of vermilion reconstruction to propose the first musculomucosal advancement flap to achieve this purpose, followed shortly by Isaksson and Johanson's description of the stairstep approach to lower lip reconstruction.[16,17] In the ensuing years, bot Karapandzic and McGregor offered substantial modifications to Gilles' fan flap design, thereby further broadening the range of cheek advancement options available to the reconstructive surgeon.[18,19] In addition, significant advances in the design of mucosal flaps for vermilion reconstruction were evidenced in the buccal mucosal flaps described by Rayner and Arscott and the facial artery musculomucosal (FAMM) flap described by Pribaz et al.[20,21] The utilization of microvascular free tissue transfer for total lip reconstruction was first reported by Harii and Ohmari in 1974, a notion further explored by Sadove et al. in their depiction of multifaceted microvascular free tissue transfer for lip reconstruction published in 1991.[22,23]

Further elaboration on the potential capabilities of free tissue transfer has given rise to the notion of increasingly complex reconstructive options such as functional gracilis transfers, as well as composite radial forearm-palmaris longus and anterolateral thigh-fascia lata microvascular free flaps to meet the requirements inherent to total lip reconstruction.[24–26] Although composite tissue allograft repair of total lip defects remains very much in its evolutionary infancy, the recent successful partial facial transplantation of a patient in France renders this technique an increasingly more likely option for reconstruction in the coming years.[27]

Anatomic Overview

Due to their central location, the lips serve as an aesthetic anchor point for the lower third of the face. The boundaries of the upper lip extend from the columellar base and nasal sills superiorly to the nasolabial folds laterally and the vermilion inferiorly. The lower lip's borders are defined inferiorly by the labiomental crease, laterally by the extensions of the nasolabial folds, and superiorly by the vermilion. Intraorally, both

lips may be defined as extending from vermilion to gingivolabial sulcus.

Structurally, the lips exhibit a trilaminar architecture consisting of an outer layer of facial skin and vermilion, an intermediate layer of interdigitated muscle, and an inner layer of mucosa. Each of these layers will be discussed briefly in turn.

Facial Skin and Vermilion

The keratinized facial skin of the lips is histologically similar to that of the rest of the face in its distribution of sebaceous glands and hair follicles, although significant differences in patterns of hair distribution are noted when comparing males and females, in general. With the transition to the nonkeratinized epithelium of the vermilion, these glands and follicles are quickly replaced by a rich capillary network that gives this tissue its characteristic rosy hue. The surface anatomy of the upper lip is punctuated by the central philtrim bordered inferiorly by the Cupid's bow and laterally by the philtral columns – landmarks that also provide the boundaries for the aesthetic subunits of the upper lip (Figure 24.1).[28] Alternatively, the facial skin of the lower lip exhibits a fairly uniform contour as it slopes inferiorly until it intersects with the skin of the chin at the labiomental crease, which represents the external translation of the gingivolabial sulcus and forms the lower boundary of the paired lower lip rhomboidal aesthetic subunits. The transition from facial skin to vermilion is marked by a subtle fold of skin termed the white roll that runs circumferentially along the skin/vermilion junction. The central portions of both the upper and lower lip vermilion demonstrate a notable fullness that provide them with a characteristic pouting appearance and facilitate apposition. As the surface of both lips curves inwards, the external dry portion of the vermilion gives way to the internal wet portion via a transition zone known as the red line. Of note, the red line approximates the margin of lip closure.

Musculature

The muscular support structure underlying the lips' outer layer of facial skin and vermilion includes 21 total muscles: five pairs of evenly distributed elevators, two pairs of lip depressors, paired lip protruders, and two pairs of lateral

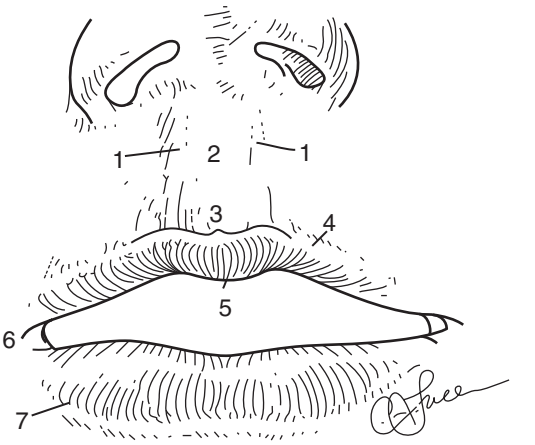

Figure 24.1. Topographic anatomy of the lips. (1) Philtral columns, (2) philtral groove, (3) cupid's bow, (4) white roll, (5) median tubercle, (6) commissure, (7) vermilion. (Published in Zide.[39]. Copyright Elsevier 1990. Reprinted with permission.)

retractors, all interdigitating with the dominant circumferential orbicularis oris. The orbicularis serves as the primary mediator of oral competence, a function it provides through its actions via two separate axes: (1) in the coronal plane, it acts as a sphincter due to its circumferential orientation about the oral stoma; and (2) in the axial plane, it presses the lips against the underlying gingival and teeth as a component of a continuous muscular ring including the buccinators and pharyngeal constrictors (Figure 24.2).[29] An understanding of the second axis of action of the orbicularis oris is critical to assessing the dimensions of lip defects, as disruption of the axial ring of oral competence tends to distract the edges of lip defects laterally, thereby exaggerating the degree of actual tissue loss. The upper lip elevators include the central paired levator labii superioris alaeque nasi, as well as the lateral paired levator labii superioris, levator anguli oris, zygomaticus major, and zygomaticus minor muscles. Protrusion of the central lower lip is mediated by the paired mentalis muscles, whereas depression is powered by the paired lateral depressor labii inferioris and depressor anguli oris muscles. Lip retraction is performed by not only the paired buccinator and risorius muscles but also by the antagonistic actions of the elevators and depressors acting on a point of confluent muscle insertion situated lateral to each oral commissure, termed the modiolus.[30]

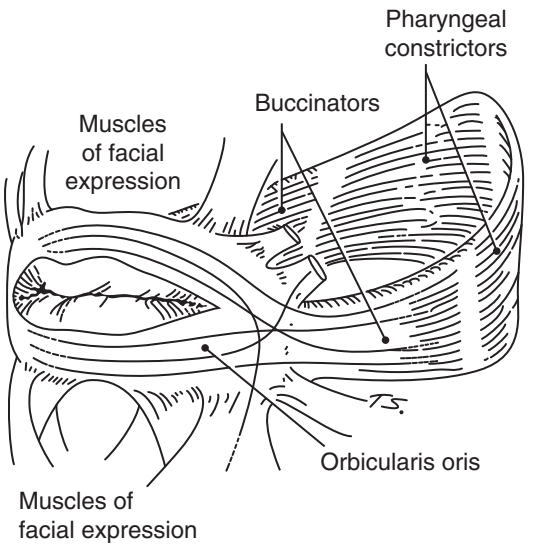

Figure 24.2. The circular ring concept of oral balance.[128]

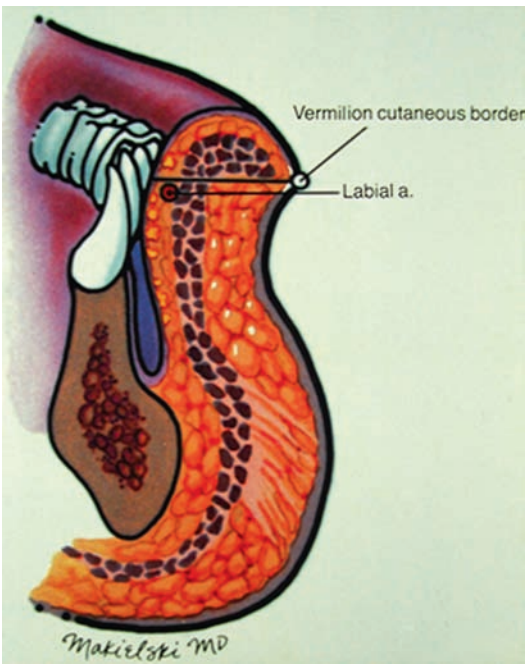

Figure 24.3. Anatomic position of labial artery relative to vermilion cutaneous border. (Reprinted with permission from Larrabee and Makielski.[127])

Mucosa

Similar to the vermilion, the mucosa is composed of nonkeratinized epithelium and serves to line the oral cavity. The transition from vermilion to mucosa usually occurs just posterior to the line of lip closure.[31] The mucosa harbors many deep mucous and serous glands that aid in the maintenance of a moist intraoral environment. In addition, the presence of abundant lymph follicles likely contributes to the relative immunologic resilience of the mucosa and augments its barrier function.[32]

Nervous, Vascular, and Lymphatic Supply

The motor input to the orbicularis oris and muscles of facial expression is provided by the three lower rami of the facial nerve (VII). In general, the lip elevators are supplied by the zygomatic and buccal branches, the retractors by the buccal branches, and the depressors by the marginal mandibular branches. Sensation to the lips is mediated by the trigeminal nerve (V), with the upper lip deriving sensibility from the infraorbital division (V2) and the lower lip from the mental nerve (V3).

Arterial inflow to the tissues of the lips courses via the superior and inferior labial arteries, distal branches of the facial artery. The labial arteries arise from the facial segment of the facial artery just lateral and deep to the modiolus and subsequently course posterior to the orbicularis oris muscle, just deep to the oral mucosa. The axial position of the labial arteries is usually equivalent to that of a posterior translation of the white roll, an anatomic pearl that often assists in the identification of the arteries in the surgical milieu (Figure 24.3). In contrast, the venous system supplying the lips is composed of a diffuse network of much smaller interconnecting channels that eventually coalesces into the facial vein. This anatomic configuration poses a unique challenge when considering replantation of central facial elements such as the lips, since the general absence of sizable outflow channels in this region often provides no option for venous anastomoses.

Lymphatic drainage of the lips is carried out by a rich anastomotic network. Although the entirety of the upper lip and the lateral portions of the lower lip drain to the submandibular nodes directly, the central lower lip drains initially into the nodes of the submental triangle and subsequently to both the submandibular and jugular nodes.[30]

Functional Review

Consideration of potential reconstructive options for the repair of lip defects must not only be informed by a thorough understanding of normal lip anatomy but also by a deep knowledge of the four key aspects of normal lip function. The primary function of the lips is to maintain oral competence, a capacity that is vital to containing oral secretions and providing a seal for feeding activities including suckling and mastication. Second, the lips provide vital assistance with phonation – particularly with articulation of the letters *B, F, M, N, P*, and *V*.[33] Third, they play a fundamental role in the facial expression of emotions including joy, sadness, and distress. Finally, they serve as an erogenous zone due to the exquisite sensitivity of the vermilion and the culturally informed practice of kissing as a form of intimate expression.

Reconstructive Options

Implicit in any algorithm for lip reconstruction are the underlying goals of restoring native function, optimizing aesthetic outcome, and limiting negative operative sequellae. The guiding principles are to restore anatomy using like tissue, plan surgical incisions that respect relaxed skin tension lines whenever possible, adhere to the notion of facial subunits, and minimize donor-site morbidity.[34–37]

In practical terms, consideration of reconstructive options for defects of the lip must begin with a thoughtful assessment of the defect itself. Any such assessment must consider the following factors:

1. **Thickness**: As described previously, the lip exhibits a trilaminar structure; the extent of injury to each of these layers must be considered in the initial wound assessment. The distinction between a partial-thickness and full-thickness injury must be made as soon as possible, as specific reconstructive options are typically associated with each pattern of injury.
2. **Location and Orientation**: Whether a defect involves the upper lip, lower lip, or both lips is a crucial factor in selecting the appropriate reconstructive modality due to the fact that the lower lip generally lacks any definitive central anatomic structures and may therefore be recruited more extensively for reconstructive purposes than upper lip tissues.[38] Other important aspects of defect location include whether it lies centrally or laterally, whether it includes either or both oral commissures, and whether it is primarily oriented in a horizontal or vertical direction.
3. **Dimensions**: The size of the lip defect is a key factor in the consideration as to whether a defect is amenable to linear closure versus complex reconstruction. An accurate estimation of the degree of tissue loss from either or both lips is therefore crucial. Such an estimation may be confounded, however, by the resting tone of the axially oriented muscular ring that includes the orbicularis oris, buccinators, and pharyngeal constrictors; violation of this ring results in its contraction with subsequent splaying of wound edges, resulting in an exaggerated degree of wound widening. For this reason, an assessment of wound size should be performed while applying axial traction on the wound edges. In addition, the involvement of neighboring facial structures including the nose, cheeks, and chin must be noted, as reconstructive efforts should be defined to include these injured zones, as well.
4. **Mechanism**: The selection of the appropriate reconstructive option must take into account the nature and degree of native tissue injury. Certain mechanisms of lip injury are associated with broader zones of tissue damage; for example, lacerations tend to exhibit a more focal region of tissue destruction than do avulsions. In addition, the cleanliness of the wound based on the underlying mechanism of injury should be taken into account when considering operative timing, as dirty wounds such as human or dog bites should generally be permitted to heal by secondary intention before reconstruction, whereas clean wounds may be amenable to immediate repair.
5. **Patient Status**: The overall health status of the patient must be considered before the development of an operative plan for lip repair.
6. **Segment Condition**: In the special circumstance of lip amputation, careful

consideration must be paid to the condition of the amputated segment when weighing various reconstructive options. Although the attachment of an amputated lip segment as a composite unit represents the ideal reconstruction, this is rarely achievable due to a lack of suitable venous targets. In this scenario, attention should be paid to the potential for using the segment as a reservoir of full-thickness grafts.

As referenced earlier, a multitude of techniques for lip reconstruction have been described to date in the medical literature. Although many rational approaches to framing such techniques have been elucidated, the authors find it most intuitive to classify options by the site, size, and orientation of the lip defect in question.

Vermilion-Only Defects

The vermilion serves as a key aesthetic transition point in the anatomy of the lower face due to its distinct hue and texture. Defects involving the vermilion require meticulous repair, particularly if they violate the white roll; as noted by Zide,[39] a 1-mm discrepancy in the continuity of the white roll is readily visible at a distance of 3 feet. Toward this end, multiple methods of reconstructing vermilion defects have been described with varying levels of complexity. Whenever possible, vermilion restoration should be performed, using adjacent vermilion as an advancement flap as first described by von Esmarch et al. in 1892. This method was later elaborated upon by Spira and Hardy, who advocated the advancement of a true musculomucosal flap in order to provide greater soft tissue support.[16] Wilson and Walker further modified the advancement flap strategy in their description of a bipedicled flap design in which the raw harvest area at the sulcus was permitted to heal by secondary intention.[40] For larger vermilion defects, Goldstein devised the technique of turning the adjacent ipsilateral vermilion into an arterialized myocutaneous flap that is subsequently stretched to span the deficient area.[41] This method was further modified by Sawada et al.[42] through the elaboration of bilateral adjacent vermilion advancement flap closure of central vermilion defects.

When ipsilateral vermilion is not amenable to transfer, recruitment of opposite lip tissue is the next preferred option; Kawamoto describes a two-stage, medially based, cross-vermilion lip switch flap technique in which the vermilion donor site is closed in a linear fashion. Division of the vermilion pedicle is performed 2–3 weeks following the initial transposition procedure.[43]

Local options for vermilion reconstruction generally hinge on utilization of neighboring oral mucosa. Rayner and Arscott's description of the buccal mucosal transposition flap provides a sensate option for vermilion reconstruction based on adjacent intraoral tissue.[20] Pribaz et al.'s facial artery musculomucosal (FAMM) flap provides yet another local option for vermilion restoration; depending on its design to incorporate either a superior or inferior pedicle, this technique permits reparation of either the upper or lower vermilion, respectively (Figure 24.4).[21]

The tongue, palate, rectal mucosa, and labia have also been utilized for vermilion reconstruction.[44,45] Recently Serletti et al.[46] reported the use of the ventral tongue to remedy total vermilion loss. Although the color match is not perfect, the texture and appearance is generally a better match than what is achievable using facial skin. Tongue flap division is usually performed 2 weeks following initial inset.

Small-Sized Defects

In general, defects involving up to one quarter of the upper lip or one-half of the lower lip (due to its laxity and lack of distinctive landmarks) may be repaired in a layered fashion via a vertically oriented closure. For such defects, the principal goals of reconstruction include the restoration of orbicularis continuity to facilitate oral competence and the fastidious realignment of key surface landmarks including the white roll and red line.

Many patterns of wedge resection have been described for small lesions of the lip, including the V, W, flared W, canted Y, single-barrel, and double-barrel techniques.[47] Although selection of one technique over another may be based in part on the geometry of the lesion in question, surgeon comfort and familiarity generally represent greater contributing factors (Figure 24.5). In all patterns, the mucosal, muscular, and cutaneous layers are reapproximated individually with meticulous realignment of the vermilion white roll. In addition, in cases in which redundancy is noted along the alar creases following closure of

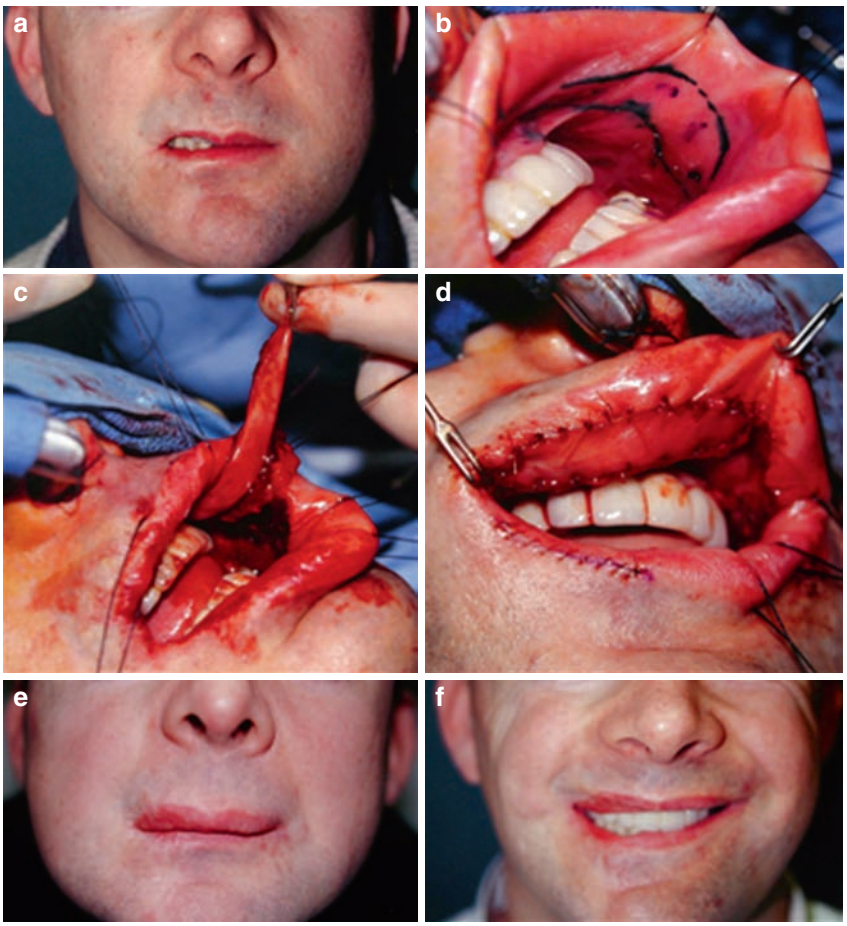

Figure 24.4. FAMM flap vermilion reconstruction in patient with hemifacial microsomia and associated upper lip deficiency. (**a**) Preoperative appearance. (**b**) Flap design. (**c**) Flap elevation. (**d**) Flap inset. (**e, f**) Postoperative appearance in repose and with active smile.

upper lip defects, the surgeon may consider limited perialar excisions to supplement the reconstructive effort. In the special case of a small defect involving the philtrum, linear closure often results in distortion or even frank destruction of the Cupid's bow. For hair-bearing individuals, this feature is readily concealed by a moustache; for others, however, the loss of this central aesthetic unit often warrants consideration of a central cross-lip (Abbe) flap, as described below.

Moderate to Large-Sized Defects

Defects that involve greater than one quarter of the upper lip or one-half of the lower lip generally require the employment of more complex reconstructive strategies.

Partial Thickness

Large partial-thickness defects involving either lip are usually amenable to coverage via a combination of lateral lip advancement, single or double V-Y advancement, and/or nasolabial transposition flap closure (Figure 24.6). Restoration of hair-bearing skin of the lip is especially important in the male, and an adjacent hair-bearing V-Y flap is generally preferable to a nasolabial transposition flap. A key point in such combination flap closures is the potential need for excision of areas of skin redundancy that may be prone to dog-ear formation – areas that may include the vermilion, which tolerates wedge excision well, provided subsequent realignment is performed in a meticulous fashion.[48]

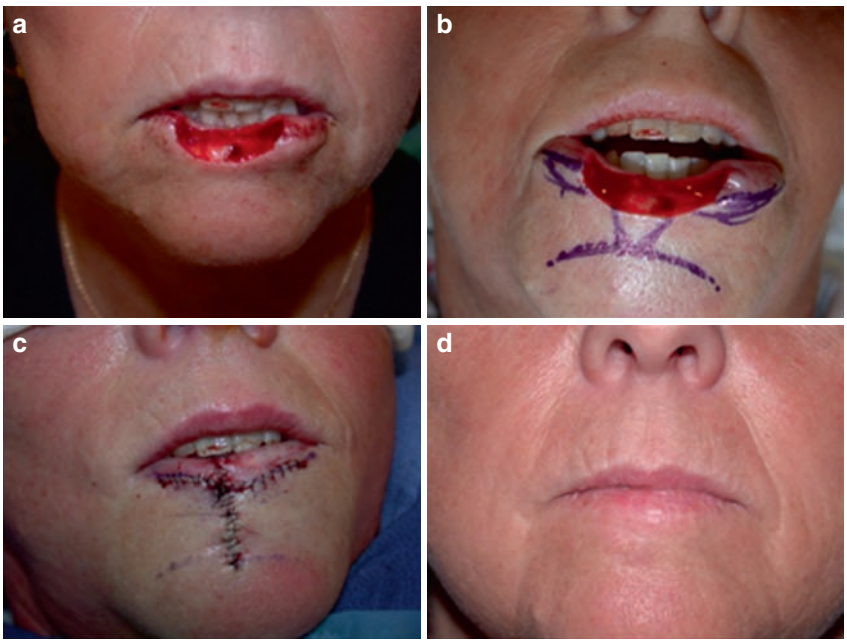

Figure 24.5. Lower lip wedge resection and linear closure. (**a**) Defect. (**b**) Further wedge design to remedy inferior dog-ear and permit lateral tissue advancement. (**c**) Completed closure. (**d**) Postoperative appearance.

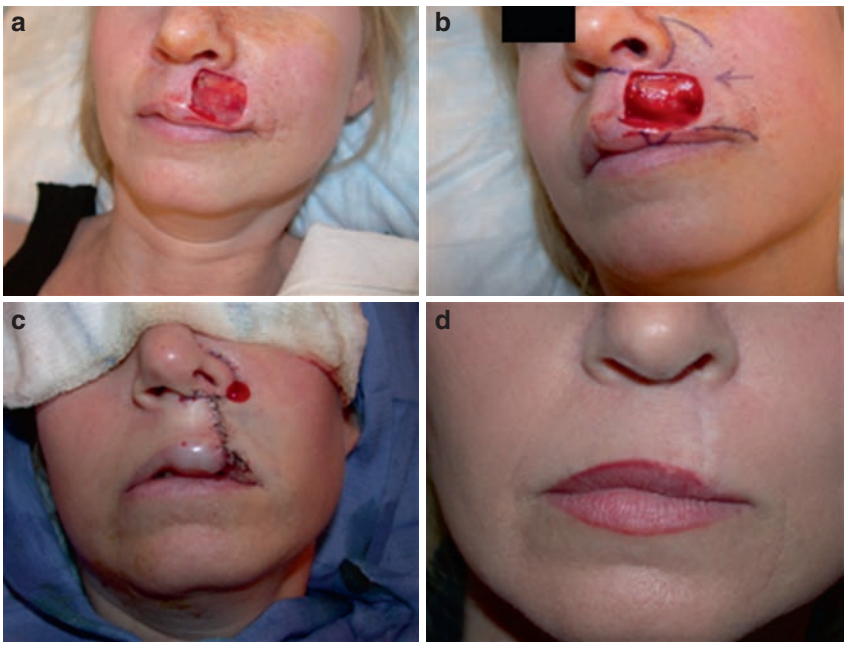

Figure 24.6. Closure of partial-thickness upper lip defect via local tissue advancement and vermilion wedge excision. (**a**) Defect. (**b**) Advancement flap design including vermilion wedge excision. (**c**) Completed inset. (**d**) Postoperative appearance.

Full-Thickness Upper Lip Defects

Options for reconstructing full-thickness defects involving greater than 25% of the upper lip depend in large part on location. Defects involving the central portion of the upper lip, including those incorporating the philtrum and its related surface structures, generally require closure with a central cross-lip (Abbe) flap, which is a full-thickness flap from the central lower lip based on the labial artery and a thin strip of vermilion and mucosa. Division of the flap pedicle is generally performed 2–3 weeks following inset. As noted by Millard, the central cross-lip flap provides an excellent option for philtral reconstruction[49]; in addition, this method may be used in isolation to reconstruct up to 50% of the upper lip.

Large defects situated in the lateral portion of the upper lip are generally amenable to reconstruction with the cross-lip flap or nasolabial flap. As described by Burget and Menick, the Abbe flap design may be accurately tailored so as to rotate about a laterally based pedicle and thereby provide a suitable reconstructive substrate for upper lip defects.[37] Alternately, the lateral cross-lip (Estlander) flap – although originally described for reconstruction of lower lip defects – may be reversed to restore the upper lateral lip. Similar to the Abbe flap, the Estlander flap requires an intact labial artery to serve as a pedicle for a full-thickness lip segment harvested from the lateral component of the lip opposite the defect. Care should be taken to preserve the commissure if still present. Pedicle division is performed several weeks later. In the case of particularly large full-thickness upper lip defects that cross aesthetic units, the reconstructive potential of the cross-lip flap may be augmented through the excision of perialar crescents. This maneuver permits medial advancement of remaining lateral lip elements, essentially diminishing the size of the reconstructive defect (Figure 24.7).

Cross-lip flaps meet Gilles' dictum of reconstructing "like with like" in as much as they provide a true trilaminar replacement for missing tissue, thereby permitting reconstitution of the orbicularis architecture and even potential reinnervation of the transferred muscle. However, lip switch flaps result in a reduction of oral circumference and may, thus, predispose patients to microstomia.

The nasolabial flap represents another option for the reconstruction of large defects of the upper lateral lip and may be particularly indicated for more horizontally oriented defects – especially in females, where moustache restoration is not needed. The flap is based on a rich subdermal vascular plexus and is generally designed according to a template of the defect as a long, caudally based flap with its medial border situated in the nasolabial fold. The donor site is usually amenable to linear closure with local cheek advancement. With aggressive flap design, up to 80% of the upper lip may be amenable to repair with a unilateral nasolabial flap, whereas bilateral flap harvest enables total lip reconstruction. With proper planning and design, nasolabial reconstruction of significant lip defects can be achieved in a single stage.[50]

Full-thickness Lower Lip Defects

Over time, three types of reconstructive flaps have evolved to become the workhorses for the repair of large full-thickness defects of the lower lip: (1) the cross-lip flap; (2) the nasolabial flap; and (3) the innervated composite flap. Modifications of the Abbe and Estlander flaps described previously have been espoused by many authors, a point that illustrates the reconstructive versatility of the cross-lip flap in lip repair[51–53]; notable permutations include double, quadrilateral, forked, or winged variations on the original design. In short, the potential for tailoring cross-lip flaps for lower lip reconstruction is limited only by the surgeon's imagination and the harvest capacity of the upper lip, which is approximately 2 cm (or one quarter of the average upper lip length) (Figure 24.8).

Utilization of the nasolabial flap in the reconstruction of large full-thickness lower lip defects has recently been reviewed and is espoused by some as the technique of choice. Using a unilateral inferiorly based subcutaneous nasolabial flap, Rudkin et al. have described effective functional reconstruction of up to 75% of lower lip transverse length and total lip reconstruction using simultaneous bilateral flaps. Despite lack of muscle inclusion in their muscle design, this group also noted good oral competence without evidence of oral constriction.[54]

Various modifications of the composite lip/cheek flap have been described over the last 150 years. The initial description of composite tissue transfer by Bernard, von Bruns, Gilles, and Webster were noninnervated. In 1974,

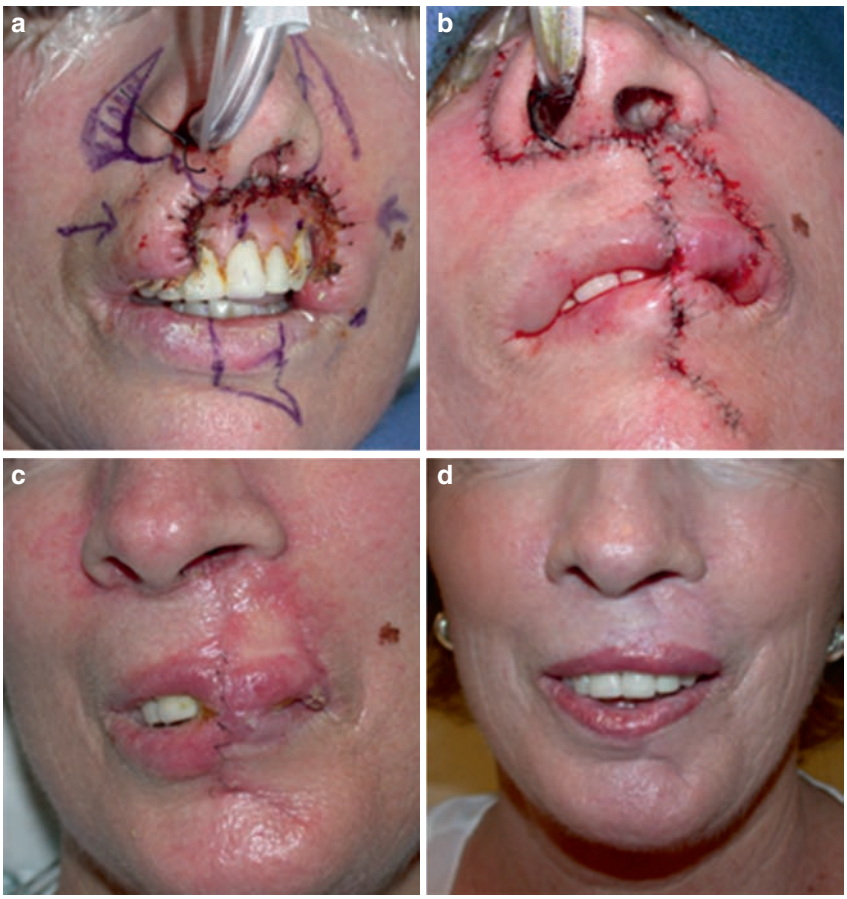

Figure 24.7. Repair of large upper lip full-thickness defect with combination lip advancement, nasolabial flap, perialar excision, and Abbe flap. (**a**) Defect and combination flap design. (**b**) Completed inset. (**c**) Appearance before pedicle division. (**d**) Postoperative appearance.

Karapandzic described the first example of a truly functional, innervated composite flap for lower lip reconstruction. Despite the subsequent modification of Karapandzic's design by several authors over the last 30 years, orbicularis oris composite flaps as a whole continue to bear his eponym. Although other facial muscles such as the levator anguli oris and depressor anguli oris have served as the basis for additional innervated composite flap designs for lip reconstruction, the orbicularis oris composite innervated flap continues to be regarded by most as the first choice for complex lower lip restoration.[55,56]

The innervated orbicularis oris flap is mobilized via a skin incision that approximates the outer edge of the orbicularis muscle. Subsequent dissection of the subcutaneous fat and lateral muscle edge is performed using a blunt technique so as to preserve the neurovascular contributions provided by the facial and trigeminal nerves and the facial and angular arteries. The mucosal translation of the skin incision is then sharply inscribed, and the flap transposed and inset. Donor-site/inset-length discrepancies are generally resolved through the use of radially oriented wedge excisions (Figures 24.9 and 24.10).

Proponents of the innervated orbicularis oris flap cite its relative technical ease of execution and reliability, as well as the teleological virtues of tissue parity and an innervated muscle foundation. Disadvantages of this technique, however, include extensive scarring; the tendency for creation of an underprojected and tightened lower lip; and the near-certain production of

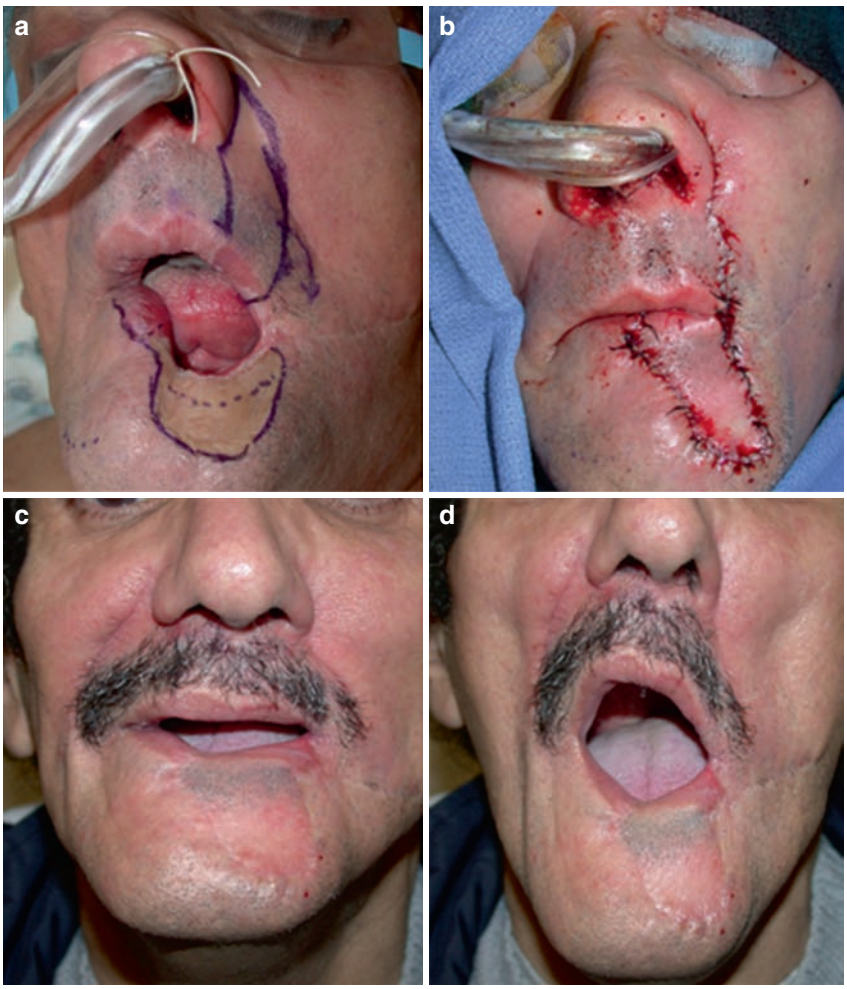

Figure 24.8. Estlander flap reconstruction of large full-thickness lower lip defect. (**a**) Planned excision and flap design. (**b**) Completed inset. (**c**, **d**) Postoperative appearance in repose and with active oral opening.

microstomia that requires secondary commissurotomy and commissuroplasty in up to one quarter of cases. These disadvantages are augmented with repairs in excess of 80% of total lip length; as such, the Karapandzic technique is generally not recommended for total lip reconstruction.[57]

Total Lip Loss

Although the nasolabial flap remains an option for total lip reconstruction using local tissues as described above, the standard approach for local total lip reconstruction remains the Webster cheek advancement technique. Based on the designs espoused by Bernard and Burow, the Webster modification is essentially an innervated composite flap technique based on remaining orbicularis oris muscle and neighboring cheek tissue. The design involves bilateral advancement of lower cheek composite flaps, enhanced through the excision of partial-thickness Burow's triangles in the nasolabial folds and paramental regions (Figures 24.11 and 24.12). With aggressive advancement, total lip reconstruction may be achieved using the Webster technique alone or may be supplemented with cross-lip flaps to assist with restoration of the central lip elements.

Although various authors have suggested several modifications to the Webster technique over

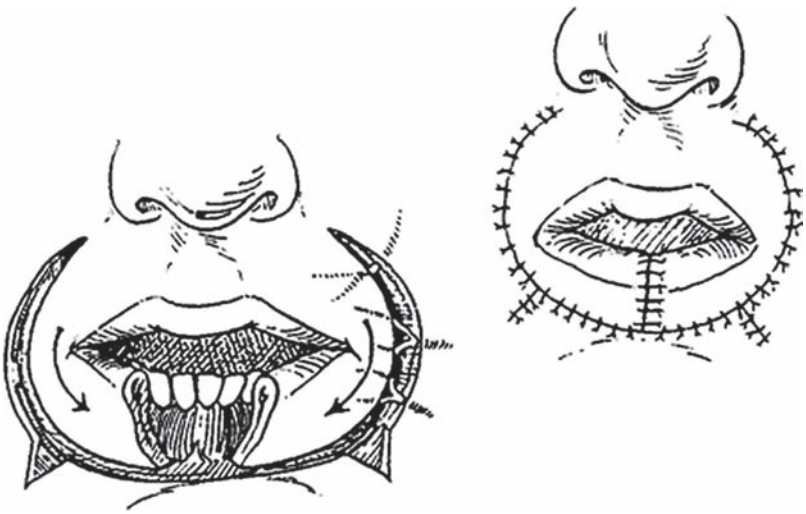

Figure 24.9. Schematic overview of Karapandzic flap design. (Reprinted from Janis JE, Leedy JF, Beran SJ (2006) Lip, cheek, and scalp reconstruction and hair restoration. *Selected Read Plast Surg* 10(13):1–47.)

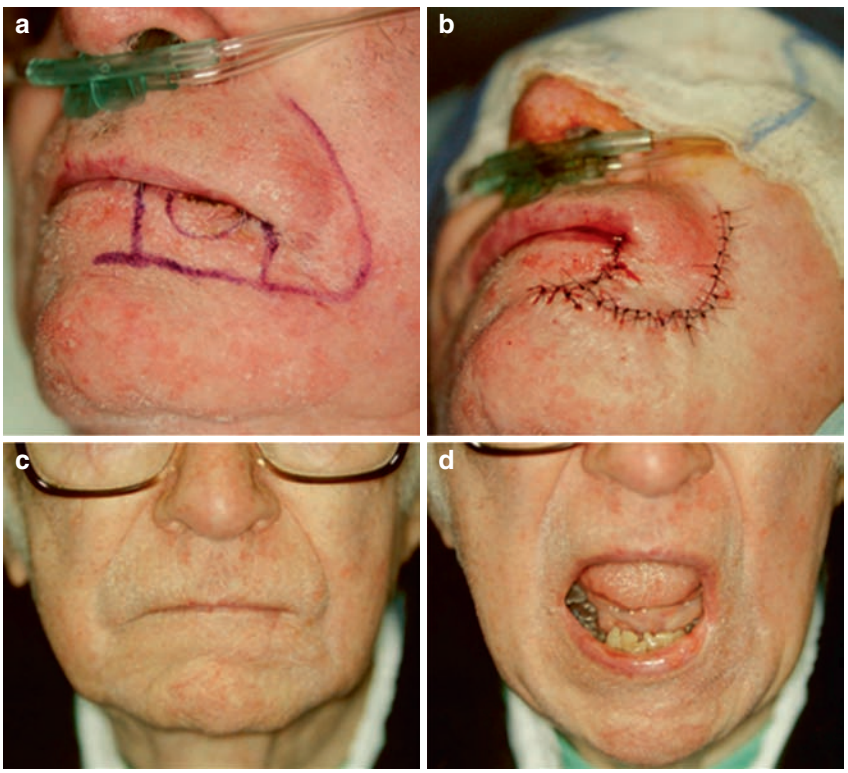

Figure 24.10. Karapandzic flap reconstruction of full-thickness lower lip defect. (**a**) Planned excision and flap design. (**b**) Flap inset. (**c**, **d**) Postoperative appearance in repose and with active oral opening.

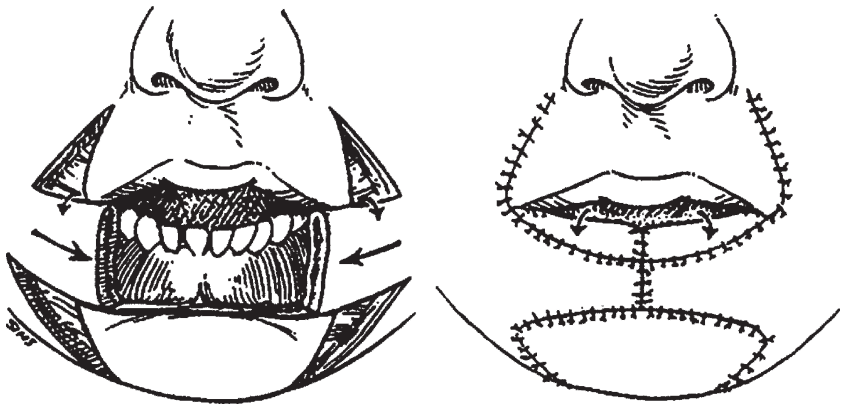

Figure 24.11. Schematic overview of Webster flap design. (Reprinted from Janis JE, Leedy JF, Beran SJ (2006) Lip, cheek, and scalp reconstruction and hair restoration. *Selected Read Plast Surg* 10(13):1–47.)

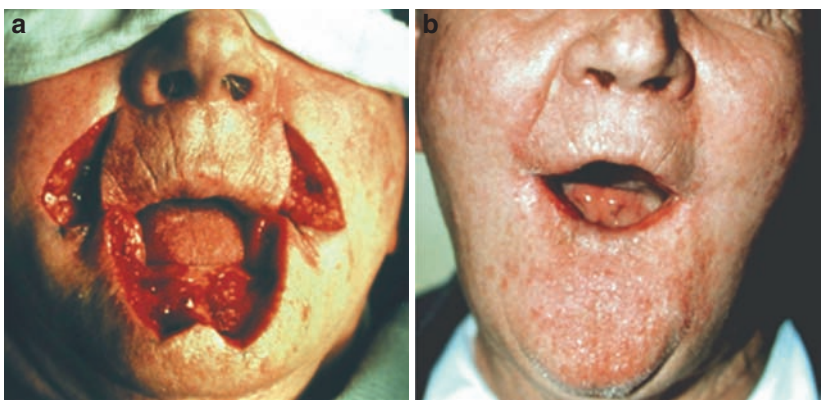

Figure 24.12. Webster flap reconstruction of near-total lower lip defect. (**a**) Defect and excision of nasolabial Burow's triangles. (**b**) Postoperative appearance.

the past half century, the fundamental design remains the same.[58–60] Advocates of the technique point to its reliability, provision of a sensate and innervated construct, and its intrinsic positioning of scars along aesthetic unit borders; for these reasons, it was recently declared the first choice in total lip reconstruction.[56] However, disadvantages of the technique include the blunting of facial creases such as the nasolabial and commissural folds, as well as a tendency for lower lip collapse into the oral cavity.[51]

In general, local tissue recruitment for total lip reconstruction results in superior color match, tissue parity, and contour potential; however, a neighboring tissue reservoir is not always available for reparative purposes – particularly in cases of extensive trauma or oncological involvement. In such situations, regional flaps such as the submental flap have become increasingly useful. This may be used for extensive partial-thicknesss defects, especially in males, to restore hair-bearing lip tissue (both upper and lower). For full-thickness defects, prelamination of a composite submental flap has also proven to be a very useful method of reconstruction. In this technique, the underside of a submental myocutaneous flap is skin grafted with a full-thickness skin graft, and a tissue expander is inserted to not only bolster the graft but also assist in flap delay, promote flap thinning, and allow for easier closure of the secondary defect (Figure 24.13).

When neither local nor regional options exist, the reconstructive surgeon has few alternatives

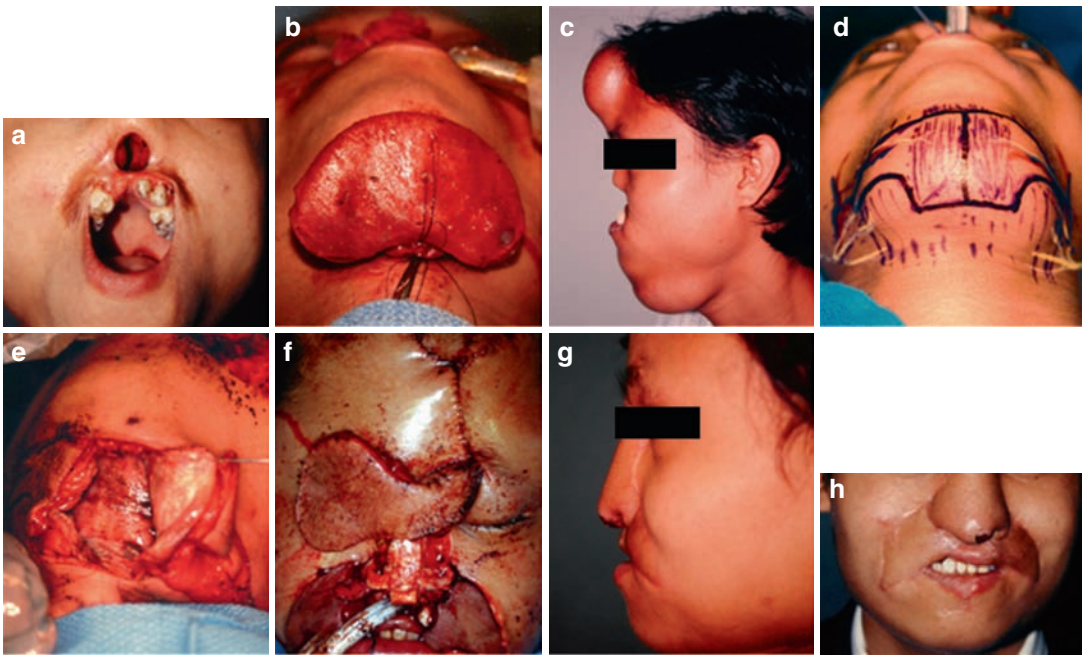

Figure 24.13. Reconstruction of total upper lip and partial nasal tip defect secondary to NOMA infection with prelaminated and expanded submental myocutaneous flap and forehead flap. (**a**) Preoperative appearance. (**b**) Tissue expander enveloped in full-thickness skin graft before placement in submental region. (**c**) Simultaneous expansion of forehead and submental flaps. (**d**) Bilateral submental flap design. (**e**) Submental flap elevation demonstrating successful prelamination. (**f**) Concomitant elevation and inset of submental and forehead flaps. (**g**, **h**) Postoperative appearance.

for total lip reconstruction beyond microvascular free tissue transfer. Since Harii and Ohmari's first description of free flap total lip reconstruction in 1974, multiple different free tissue substrates have been described for this purpose, including the radial forearm, anterolateral thigh, and temporal scalp.[23,61–63] Prefabricated hair-bearing flaps have also been described for lip reconstruction.[64] More recently, the use of functional muscle transfers and composite free flaps has been described to augment expressivity and oral competence.[24–26] With the availability of a multitude of potential donor sites for free tissue transfer, the future reconstructive surgeon may be limited only by his or her imagination when considering options for total lip reconstruction (Figure 24.14).

A recent 18-month follow-up of the first partial facial transplantation patient suggests that satisfactory functional and aesthetic outcomes may be achieved by using this technique in the future. Indeed, Dubenard et al.[65] document the restoration of normal sensibility in the transplanted tissues at 6 months and recovery of motor function as manifest by successful active labial contact at 10 months, coupled with a high degree of aesthetic satisfaction as reported by the patient herself (Figure 24.15). With few post-transplantation complications reported, the relative success of the first partial facial allograft will likely serve as the impetus for a new era of complex reparative procedures based on the notion of replacement rather than reconstruction. Given the highly specialized structure of the lips, regular reconstruction of labial components via allograft-based techniques is likely to become a reality in the not-too-distant future.

Lip Amputation

Traumatic lip amputation represents a rare indication for lip reconstruction and is most often due to either human or dog bites to the face. In cases in which the lip remnant is recovered and in suitable condition (including reasonable ischemia time), every effort should be made to microsurgically replant the recovered segment, since

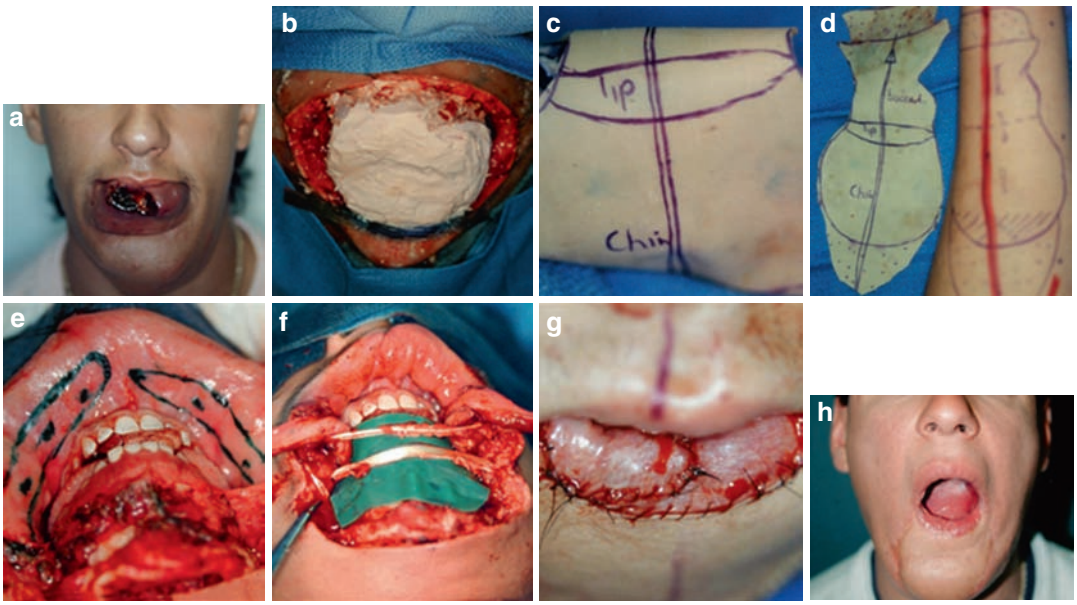

Figure 24.14. Reconstruction of total lower lip and chin via multifaceted radial forearm free flap, bilateral FAMM flaps, and palmaris longus tendon sling following excision of large vascular malformation. (**a**) Preoperative appearance. (**b**) Alginate model of defect created intraoperatively to define required geometry of reconstruction. (**c**) Design of two-dimensional reconstructive template based on three-dimensional alginate model. (**d**) Translation of template to radial forearm flap design. (**e**) Bilateral FAMM flap design. (**f**) Palmaris longus tendon inset to provide appropriate suspension and support. (**g**) FAMM flap inset. (**h**) Postoperative appearance with active oral opening.

this represents the optimal reconstructive option from both an aesthetic and functional perspective and does not compromise subsequent potential reconstructive options (Figure 24.16). In 1998, Walton et al. described a multi-institutional experience in which 13 cases of microsurgical lip replantation were retrospectively reviewed to characterize the variables and assess the outcomes relevant to this reconstructive procedure. In general, it was found that the identification of suitable recipient veins was the most significant technical challenge and that nearly 85% of patients required transient leech therapy to relieve venous congestion in the replanted segment. Notable postoperative complications included an average transfusion requirement of 6.2 units of packed red blood cells per patient, prolonged segment edema, a high revision rate, and a nearly 50% rate of hypertrophic scarring. Despite these complications, the color match, muscle function, and sensibility of the replanted segment were rated as good to excellent, and all patients were reportedly uniformly pleased with their final results.[66] These findings provide further credence to the notion that microvascular lip replantation should be attempted as the first-line reconstructive intervention for significant lip defects whenever possible.

Complications

Potential complications of significant lip reconstruction include impaired oral competence manifest as lip droop and/or drooling, vermilion notching, altered sensibility, impaired motor activity, and scarring, among others. Many of these sequellae may be improved by subsequent revision procedures specific to the presenting complication. By far the most potentially morbid complication with major lip reconstruction is microstomia, which may impair denture utilization and, at worst, interfere with adequate nutritional intake and speech. Multiple approaches to commisuroplasty have been described to remedy this situation, most of which involve advancement or transposition of the vermilion as a full-thickness flap or require the recruitment of oral mucosa.[13,67–69]

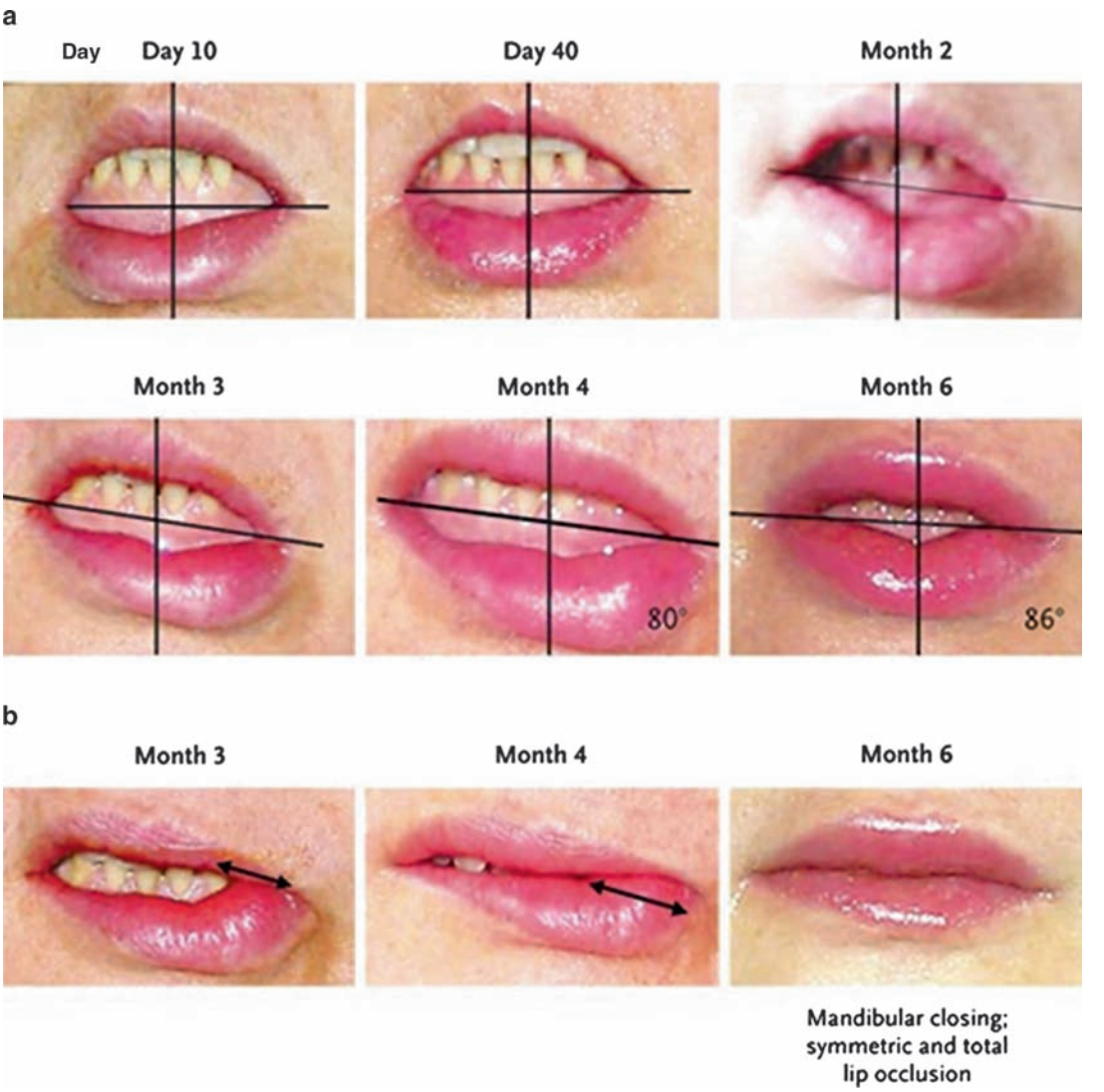

Figure 24.15. Recovery of lip occlusion functionality following partial facial composite tissue allotransplantation. (**a**) Recovery of passive lip occlusion. (**b**) Recovery of active lip occlusion. Symmetric and total passive and active occlusion was attained at 6 months post-transplantation. (Reprinted with permission from Dubenard JM et al.[28] Copyright © 2007 Massachusetts Medical Society. All rights reserved.)

Cheek Reconstruction

Along with the forehead, the cheeks constitute the facial periphery and, thus, serve to frame the primary structures of the countenance. The contour of the cheeks aids in the establishment of facial balance; as such, alterations in cheek shape often have a profound influence on facial aesthetics. In addition, the cheeks play a key role in the maintenance of oral competence and mastication, the facial manifestation of human emotion, and the support of neighboring primary structures. The repair of defects involving the cheeks therefore seeks to achieve both aesthetic and functional ends – both of which must be carefully considered by the reconstructive surgeon.

Taken collectively, the cheeks comprise the largest uninterrupted expanses of skin on the

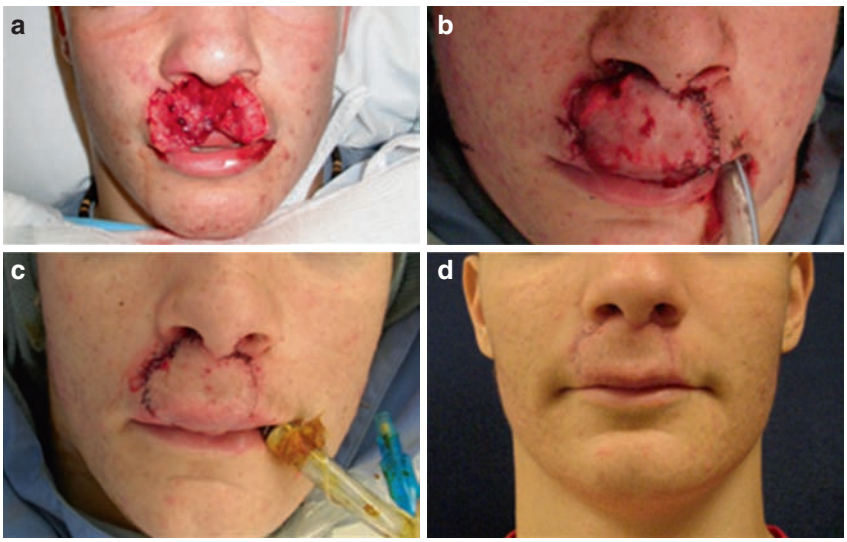

Figure 24.16. Replantation of upper lip segment following dog bite injury. (**a**) Defect. (**b**) Flap appearance at time of inset. (**c**) Flap appearance 7 days following inset. (**d**) Postoperative appearance at 6 weeks. (Photographs courtesy of Helena Taylor, MD, DPhil.)

face and, thus, are statistically the most likely facial feature to sustain injury, regardless of mechanism. The most frequently encountered causes of acquired cheek insults include neoplasia, burns, and trauma, whereas congenital alterations in cheek contour may be due to facial clefts, vascular anomalies, or facial wasting syndromes. The majority of this discussion will focus on the management of cheek defects due to acquired etiologies.

Historical Context

As with the history of lip reconstruction, the earliest recorded examples of surgical techniques to repair the cheek appeared in the *Sushruta Samhita* and mainly detailed strategies for linear closure.[70] Additional perspectives concerning cheek reconstruction were subsequently included in treatises on general facial reconstruction by authors including Oribasius, who described many of the basic principles underlying advancement flaps, and Tagliacozzi, who espoused the employment of staged facial reconstruction through the use of pedicled distant flaps.[71,72]

The evolution of techniques for reconstruction of the cheek in modern times essentially parallels the evolution of our understanding regarding flap physiology and vascular anatomy, as noted by Bunkis et al.[73] Although relatively few techniques have been developed specifically for the sole purpose of cheek reconstruction over the past two centuries, the application of increasingly sophisticated principles regarding flap design during this time has resulted in a continuously expanding palette of options for cheek repair being available to the reconstructive surgeon.[74–77] Although the elaboration of the inferomedially based cervicofacial flap by Esser, the lateral forehead flap by Blair and Brown, and the sternocleidomastoid compound flap by Owens all represented specific advances in cheek reconstruction, none of these early strategies were based on a thorough understanding of underlying flap vascular anatomy.[14,78,79] Indeed, it was not until the advent of the concept of axially based flaps that the principles of design for cheek reconstruction could become further refined. Both McGregor and Bakamjian applied these principles in their elaboration of the axially based temporalis and deltopectoral flaps for cheek reconstruction, respectively.[80,81] The further development of the notion of musculocutaneous flap design by Orticochea and the description of discrete musculocutaneous territories by McGraw et al. enabled the advocacy of new flaps for facial reconstruction based on muscular substrates, including the pectoralis major, sternocleidomastoid, trapezius, and

latissimus dorsi.[82–87] The advent of microvascular free tissue transfer has subsequently broadened the palette for cheek reconstruction even further as have the notions of functional muscle transfer, flap prefabrication, and composite tissue design. A partial list of well-described substrates for cheek reconstruction via free tissue transfer includes the latissimus dorsi, omentum, radial forearm, anterolateral thigh, and functional gracilis.[88–92]

Anatomic Overview

The cheeks serve to frame those structures of the face that are seen in primary gaze – namely, the nose, lips and eyelids – and thus, along with forehead, constitute what Menick has termed the facial periphery.[93] The cheeks are defined medially by the nasofacial sulcus and melolabial folds, laterally by the preauricular crease, superiorly by the infraorbital crease, and inferolaterally by the mandibular border.

As with the lips, the cheeks generally exhibit a trilaminar structure consisting of skin and subcutaneous fat, muscle, and mucosa. The specific attributes of each layer are reviewed in detail.

Facial Skin

The skin of the cheek is relatively homogeneous, keratinized epithelium rich in fine hair follicles and numerous sebaceous glands.[94] As with the lips, the pattern and density of hair distribution in the cheek vary significantly between males and females. The surface anatomy is unremarkable and represents a smooth, generally curving expanse that is generally devoid of any interruptions in contour. The reasonably uniform nature of the cheek led Gonzales-Ulloa to categorize it as a single aesthetic unit in his original treatise in 1957[95]; since that time, however, this designation has been further refined by Zide to describe the cheek in terms of three overlapping subunits: suborbital, preauricular, and buccomandibular.[40] Based more on geographic proximity to potential cervical rotation flap reconstruction than on discrete anatomic boundaries, Zide's subunit schema has since undergone multiple iterations by others as reparative options have continued to evolve. At present, it is uncertain to what extent the notion of aesthetic subunits of the cheek serves as an asset to the reconstructive surgeon, especially as strategies for reconstruction continue to mature.

Musculature

Deep to the skin and subcutaneous fat lies the broad, fanlike superficial musculoaponeurotic system (SMAS). The SMAS represents the fascial continuation of the most superficial components of the forehead and neck musculature; as such, it is generally regarded as extending from the frontalis (zygomatic arch) to the platysma (mandibular border) and from the tragal cartilage periosteum to the central muscles of facial expression.[96] Deep to the SMAS, the muscular infrastructure of each cheek consists of seven distinct components grouped into three anatomic regions. The superior maxillary region includes the levator labii superioris, levator anguli oris, zygomaticus major, and zygomaticus minor, all of which essentially serve as lip elevators via their interdigitation with the orbicularis oris. The intermaxillary region consists of the risorius, which retracts the lips, as well as the deep buccinator, which compresses the cheeks and acts in continuity with the orbicularis oris and pharyngeal constrictors to maintain axial oral competence. Finally, the temporomandibular region features the powerful masseter muscle, which assists with jaw contraction and mastication.[30]

Mucosa

As the mucosal lining of the oral cavity transitions from the lips to the cheeks, it gradually doubles in thickness. The main constituents continue to include glandular and connective tissue with many lymphatic follicles and associated secretory ducts.[97] The most pronounced of the latter is the parotid duct, which terminates in the buccal mucosa via a papilla that is typically situated opposite the second maxillary molar.

Nervous, Vascular, and Lymphatic Supply

The most important components of the cheek's neural anatomy comprise the trigeminal (V) and facial nerves (VII). The trigeminal nerve provides sensation to the cheek via the maxillary (V2) and mandibular (V3) divisions. The maxillary division includes the infraorbital, zygomaticofacial, and zygomatictemporal branches, which collectively provide sensation to the cornea, lower eyelid, infraorbital region, upper lip, and malar eminence; the mandibular division similarly includes the mental, buccal, and

auriculotemporal branches, which serve the lower lip, mandibular border, and temporal regions. In addition, the masseter receives its motor innervation via the masseteric branch of V3, thus representing the only muscle within the cheek infrastructure that does not receive its motor input via the facial nerve.

The facial nerve is the primary motor source to all of the muscles of facial expression. The facial nerve emerges from the cranium and into the facial soft tissue via the stylomastoid foramen, located on the lower surface of the temporal bone. In the cheek, it passes between the deep and superficial parts of the parotid gland, splitting into two main divisions at the pes anserinus, which further arborize into the temporal, zygomatic, buccal, marginal mandibular, and cervical branches. In general, these branches run below the SMAS layer, yet innervate the muscles of facial expression on their undersurfaces via deep motor branches; the buccinator, levator anguli oris, and mentalis muscles serve as notable exceptions to this rule. Although significant variations in the arborization pattern of the facial nerve have been described, the temporal and marginal mandibular nerve segments tend to represent terminal branches and are therefore less forgiving to injury given their lack of cross innervation with neighboring branches.

The arterial blood supply to the cheek is derived primarily from the external carotid system via the facial and transverse facial arteries and their associated distal branches. The facial artery arises in the neck, courses along the body of the mandible in a plane deep to the platysma and fascia, and gives off the submental artery. It then curves over the body of the mandible and enters the cheek just deep to the platysma/SMAS, resting on the anterior surface of the buccinator and giving off multiple small muscular perforators. Just lateral and deep to the modiolus, the artery again arborizes, giving rise to the labial systems and, more distally, the lateral nasal and angular arteries. The transverse facial artery supplies the superior portion of the cheek and generally arises as a branch of the superficial temporal artery or the external carotid itself within the body of the parotid gland. The artery then courses medially between the parotid duct and the zygomatic arch, immediately superficial to the masseter, and subsequently branches extensively. In their distal arbors, the facial and transverse facial arteries anastomose extensively with one another, as with secondary arterial systems, including the infraorbital and dorsal nasal arteries, to form a robust vascular network to support cheek tissues. As in the lips, the venous system of the cheek is composed of a diffuse network of small vessels that coalesce into larger channels. These channels are typically separated spatially from the main arterial branches in the upper cheek but ultimately approximate the path of the facial artery in the lower cheek. Lymphatic egress occurs via an extensively arborized network that drains to the intraparotid, submandibular, and submandibular lymph nodes.[30]

Functional Review

As with the lip, consideration of reconstructive strategies for the cheek must be informed by a thorough understanding of its native functions. Essentially, the uninjured cheek serves six principal roles:

- Participation in the formation of a sealed oral cavity to contain oral secretions
- Assistance in the concerted actions related to intraoral food manipulation[98]
- Execution of the complex motions of facial expression
- Provision of mechanical support to the mobile tissues of the perioral and periorbital regions
- Satisfaction of conduit requirement for neurovascular support structures to central facial soft tissues
- Establishment of an aesthetic frame for central facial features

Reconstructive Options

The primary goals of cheek reconstruction include the restoration of native function, maximization of aesthetic outcome, and limitation of repair-related morbidity. Implicit in this statement is the intent to reestablish both internal and external coverage, expressivity, masticatory function, and aesthetic contour and quality. As with any reconstructive effort, the core principles guiding efforts to repair the cheek include the emphasis on replacing damaged tissues with healthy like tissues, honoring relaxed skin tension lines, and minimizing the distortion of uninjured areas. Unlike some other regions, however, the cheek's relative uniformity of color

and contour render reconstructive options prone to excessive contracture (e.g., healing by secondary intention, skin grafting) less desirable due to high visibility and the potential for distortion of neighboring systems, such as the lips and eyelid. As such, the reparation of cheek defects lends itself less to the notion of the reconstructive ladder and more to the notion of the reconstructive elevator, in which the simplest option may not always be the best one, and the reconstructive plan must consider the strategy in which both form and function will be maximally optimized.[99]

Paramount to the formulation of a reconstructive strategy for cheek defects is a disciplined assessment of the defect itself. Notable factors worthy of consideration include the following:

1. **Thickness:** A thorough understanding of the anatomic layers that are involved in the defect is vital to planning reparative measures. The key factor to ascertain is whether or not the defect represents a partial-thickness versus a through-and-through injury, as this distinction will inform decisions concerning the need for multifaceted reconstruction.
2. **Location:** Although the value of the notion of aesthetic subunits of the cheek may be in question, the location of the defect should be carefully considered in terms of its amenability to certain reconstructive options. In addition, given the cheek's role as a conduit for neurovascular and exocrine structures that support features of the central face (e.g., the facial nerve and parotid duct), the location of the defect must also be assessed in terms of the likelihood for concomitant injury to these underlying structures.
3. **Dimensions and Orientation:** The size and orientation of the defect play a significant role in the consideration of potential reconstructive options; therefore, the transverse, longitudinal, and anteroposterior dimensions of the injury must be fully appreciated before undertaking reparative planning.
4. **Mechanism:** As with all tissue injuries, the mechanism that contributed to the creation of the cheek defect is an important factor in determining the optimal strategy for operative repair. Focal injuries (e.g., stab wounds) are typically associated with a narrow field of tissue damage, whereas broad-based (e.g., ballistic wounds) injuries may involve far more substrate compromise than is initially apparent. The effective field of injury is critical to ascertain, as it may require the enlargement of the initial defect to establish definitively viable wound edges and may preclude the employment of local or regional reconstructive options.
5. **Patient Status:** As always, a clear understanding of the general status of the patient must also figure in the reconstructive algorithm.

Although prior authorities have advocated reconstructive algorithms based on the notion of anatomic subunits of the cheek, the authors favor a framework informed primarily by the relative size of the defect in question.

Small-Sized Defects

Focal defects of the cheek, whether partial or full thickness, can generally be closed in a layered, linear fashion with minimal associated tissue distortion. Ideally, wound closure in this situation can be planned in a fashion so as to appropriately orient the final repair in a direction that honors the relaxed skin tension lines of the cheek; at times, the accomplishment of this goal requires conversion of the defect to a favorably oriented ellipse through the excision of additional tissue. One alternative to this strategy is to perform a staged purse-string closure, which requires two to three total procedures but may reduce final scar length by as much as 69% (Figure 24.17).[100]

Moderate-Sized Defects

Larger partial-thickness cheek defects are typically not amenable to linear or staged purse string closure due to potential distortion of neighboring structures, such as the eyelid and oral commissure; however, the possibility of capitalizing on the inherent laxity of local tissues generally remains an option in the majority of cases. Recruitment of adjacent or proximal reconstructive substrate can be performed through the execution of a number of local advancement, rotation, or transposition flap designs, including rhomboid, V-Y/extended V-Y, horizontal advancement, oblique advancement,

and crescentic advancement at the nasolabial fold (Figure 24.18). In general, advancement flaps are preferred to transposition flaps, as the former usually cause less distortion. As always, flap design should endeavor to honor the directionality of the cheek's inherent relaxed skin tension lines. In addition, the creation of dog-ears may be minimized via the excision of Burow's triangles at appropriate advancement points.

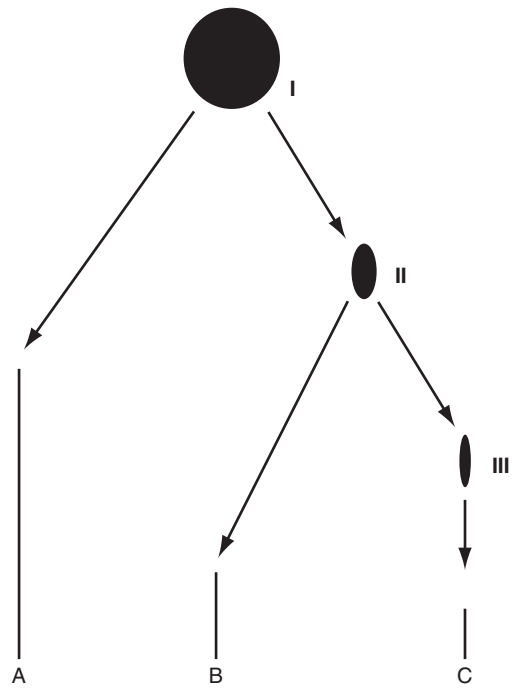

Figure 24.17. Theoretical reduction in scar length achieved through sequential purse string closure of surgical defect. (From Mulliken JB et al.[100])

Large-Sized Defects

Very large partial-thickness defects require more tissue coverage than what is available through the mobilization of immediately local substrates. In such circumstances, therefore, the reconstructive surgeon is generally obligated to consider extended local and regional options, either in isolation or in combination.

Extended Local Options

Over time, the cervicofacial flap has emerged as the workhorse for extended local tissue reconstruction for partial-thickness defects of the cheek. The cervicofacial flap uses the random blood supply of the subdermal plexus to permit simultaneous rotation and advancement of a large segment of cheek and neck skin for coverage purposes. Currently, two primary strategies for cervicofacial flap design are favored: inferomedially based and laterally based. Inferomedially based designs, referred to as anteriorly based designs by some, recruit skin from the remaining

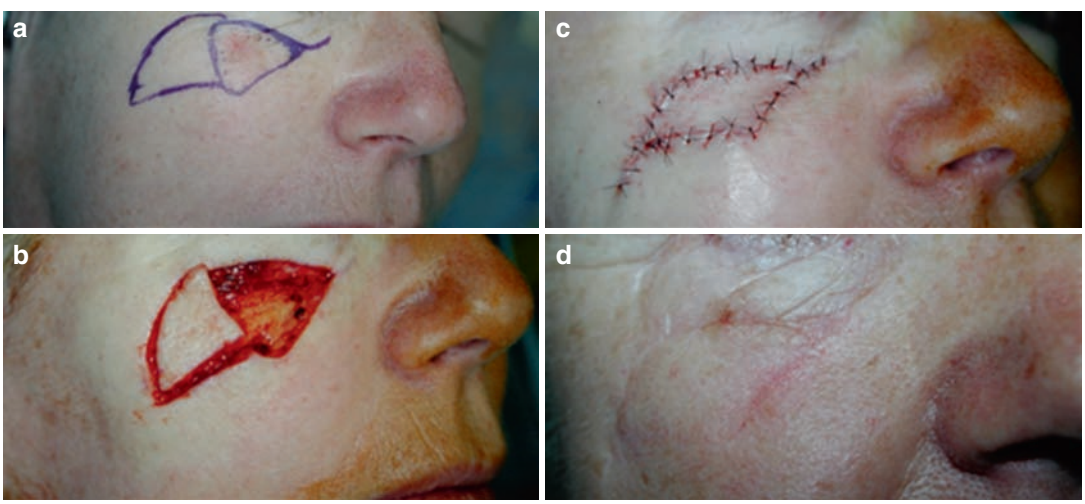

Figure 24.18. V-Y advancement closure of medial cheek defect. (**a**) Planned excision and flap design. (**b**) Defect with elevated flap. (**c**) Flap inset. (**d**) Postoperative appearance.

cheek, neck, and anterior chest.[93] Because the net vector of flap movement is in a transverse (medial) direction, these flaps are best suited for cheek defects that are oriented vertically. Although many iterations on inferomedially based cervicofacial flap design have been proposed over the past century, the authors favor the angle rotation schema. Originally described by Schrudde and Beinhoff and recently reintroduced by Boutros and Zide, the angle rotation design represents an elaboration of Juri and Juri's cervicofacial flap conception in its inclusion of a retroauricular extension.[101,102] Flap construction begins at the posterior edge of the cheek defect, from which a line is drawn laterally to the hairline and then vertically to the inferior edge of the lobule, following the sideburn and tragus. The advancement "angle" is then defined behind the ear, ranging from 60° to 90°. The flap is elevated just above the SMAS and is rotated medially; the retroauricular component is transposed medially and superiorly. The retroauricular donor site is closed first and the flap is then inset. Meticulous flap design usually obviates the need for bone anchors or other supplementary suspension support measures. Relative to other single-stage inferomedially based cervicofacial designs, the angle approach offers the advantages of unobtrusive scarring, obviation of the need for extension into the inferior neck and chest, relative ease of dissection, and the potential for flap redeployment (Figure 24.19).

Laterally based cervicofacial flap designs, such as those described by Al-Shunnar and Manson,[103] tend to transpose in a vertical direction and are, thus, better suited for transversely oriented partial-thickness cheek defects. These flaps recruit tissue from the jowl and neck for reconstructive purposes and result in inset lines placed along the nasolabial fold and orbital rim. The design begins by drawing a line inferiorly, in or parallel to the nasolabial fold and past the oral commissure, then over the curve of the mandible and into the submental region, where the angle advancement is defined. Dissection is carried out above the muscles of facial expression. Inset is generally straightforward and may be supplemented by bone anchor suspension. Elimination of dog-ears may require triangular tissue excision at the inset pivot point and is usually well tolerated (Figure 24.20).

Regional Options

When local tissues are insufficient or unavailable due to anatomic or pathological reasons, a host of regional reconstructive options remain available for reparation of large partial-thickness cheek defects. Although rarely employed

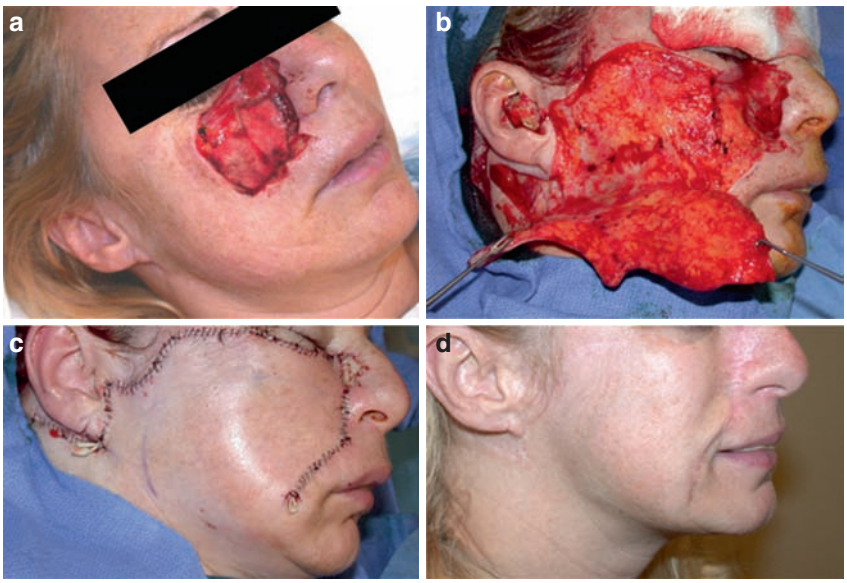

Figure 24.19. Angle rotation flap reconstruction of medial cheek defect. (**a**) Defect. (**b**) Flap elevation with incorporation of retroauricular component. (**c**) Flap inset. (**d**) Postoperative appearance.

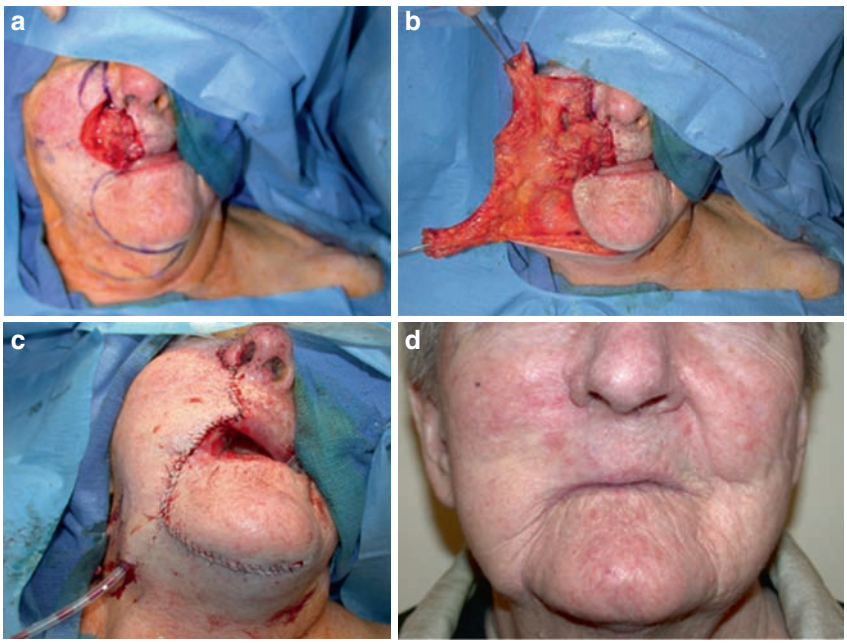

Figure 24.20. Laterally based cervicofacial advancement flap closure of medial cheek and upper lip defect. (**a**) Defect with flap design. (**b**) Flap elevation. (**c**) Flap inset. (**d**) Postoperative appearance.

for cheek reconstruction, the paramedian forehead flap can serve as an adequate donor site for the restoration of soft tissue coverage for the superomedial cheek. In addition, the submental platysmal myocutaneous flap can serve as another regional option for cheek reconstruction that is particular well suited for males, since it not only offers the advantage of excellent color and skin quality matching but also provides a hair-bearing substrate to enable continued beard growth. One additional functional advantage offered by the submental flap is the potential incorporation of innervated platysma, which may be oriented in series with residual lip elevators and retractors to restore active facial expression (Figure 24.21). Other regional options for reconstruction of large cheek defects include the cervicopectoral, deltopectoral, pectoralis major, cervicohumeral, trapezius, and latissimus dorsi all of which have been well described.[81,84,104–108]

Tissue Expansion

An adjunct to the utilization of local and regional substrates for cheek reconstruction is the option of tissue expansion. The value of tissue expansion comes from its assistance in effectively increasing net skin surface area and providing skin that has the same texture, color, and hair-bearing qualities as that missing in the defect. In a summary based on six years of experience in head and neck tissue expansion, Wieslander provides the following recommendations:

- Incisions should be kept small and away from the defect, pocket, and future flap.
- Expansion should be designed so as to achieve a flap that is 30–50% longer and wider than needed to compensate for inherent flap contraction.
- Intraoperative filling of the expander to a volume sufficient to fill the pocket with minimal tension prevents hematoma and seroma formation and reduces the formation of envelope folds.
- The optimal location for the expander valve is in a region at least 7 cm remote from the expander pocket.
- Expander filling should commence 10–14 days following placement and should then proceed at a rate of 1–2 fills per week.[109]

The use of tissue expanders in the reconstruction of cheek defects has been described as a

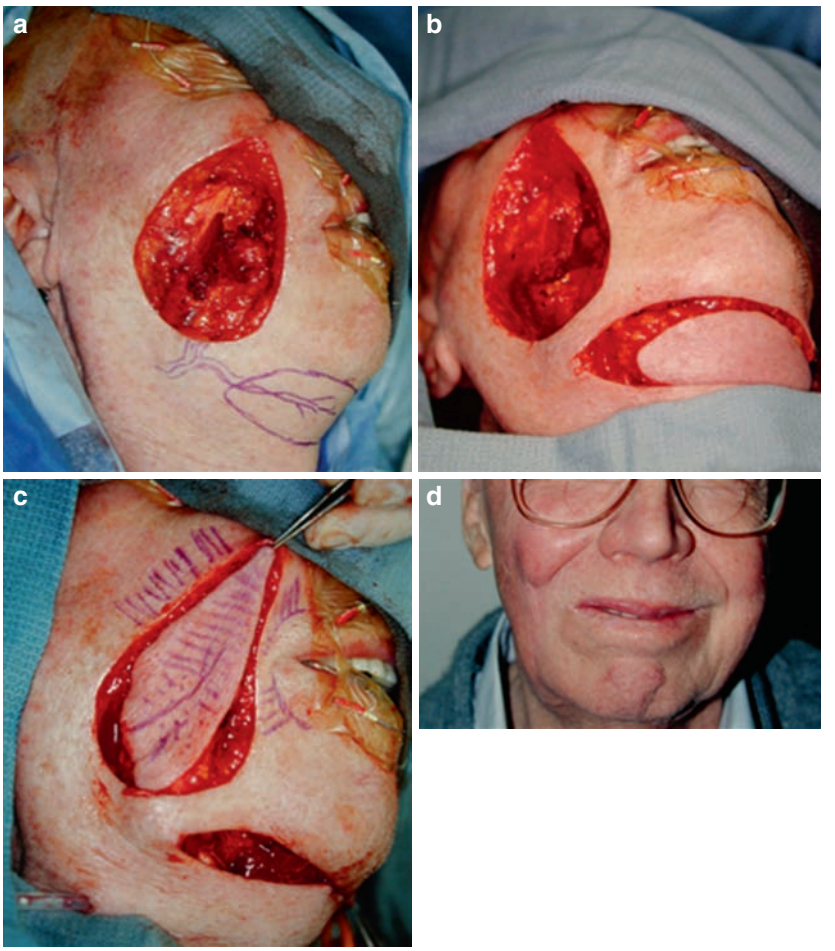

Figure 24.21. Innervated submental myocutaneous flap closure of large central cheek defect. (**a**) Defect extending through muscles of facial expression with flap design. (**b**) Flap elevation. (**c**) Flap inset illustrating polarity of platysma fibers in relation to surrounding facial musculature. (**d**) Postoperative appearance demonstrating functional smile.

means to augment local rotation advancement flaps, prelaminated regional flaps, and staged free flaps.[110–112] In general, positioning of the expander is recommended in a plane superficial to the SMAS/platysma when placed directly adjacent to the defect. Recommendations concerning port positioning vary; notably, Jackson et al.[113] describe the use of an exteriorized port that allows pain-free injection – an option that is particularly well suited to the pediatric population. Final closure under minimal tension has been reported to yield optimal results.

Through-and-Through Defects and Total Cheek Loss

Defects that include the loss of mucosa in addition to skin and muscle pose the additional challenge of lining reconstruction. Often, this scenario arises in the context of very large defects or total cheek loss – in short, situations that require the recruitment of substantial amounts of tissue for reconstructive purposes. When local or regional substrates are sufficient to meet such needs, options for lining reconstruction include

flap prelamination of the submental region or folded-flap approaches to reparation. When local tissues are insufficient, as is more often the case, reconstructive efforts generally rely on free tissue transfer.

Since the advent of microvascular free tissue transfer in the 1970s – when there were very few available flaps and the emphasis was on flap survival – there has been a substantial increase in the number of flap options and a concomitant shift in emphasis toward better flap selection and refinement. Although many flaps have been described for cheek reconstruction, the most commonly used options include the radial forearm, scapular, parascapular, groin, and anterolateral thigh flaps.[114–119] In burn patients, where these common donor sites may have been damaged or destroyed, recently introduced techniques of flap prefabrication may be used. This process involves the introduction of a distally ligated vascular pedicle beneath the skin to be neovascularized. After a period of 8 weeks – during which time inosculation occurs – the flap may be transferred based on its newly acquired blood supply. Prefabrication may be performed at a distance – requiring microvascular transfer – or preferably in the neck, where the flap will have better color and texture for cheek reconstruction. This process is generally performed in conjunction with the use of a tissue expander placed beneath the implanted pedicle that serves to thin the overlying flap, assist with closure of the donor site, and mechanically enhance the neovascular response (Figure 24.22).

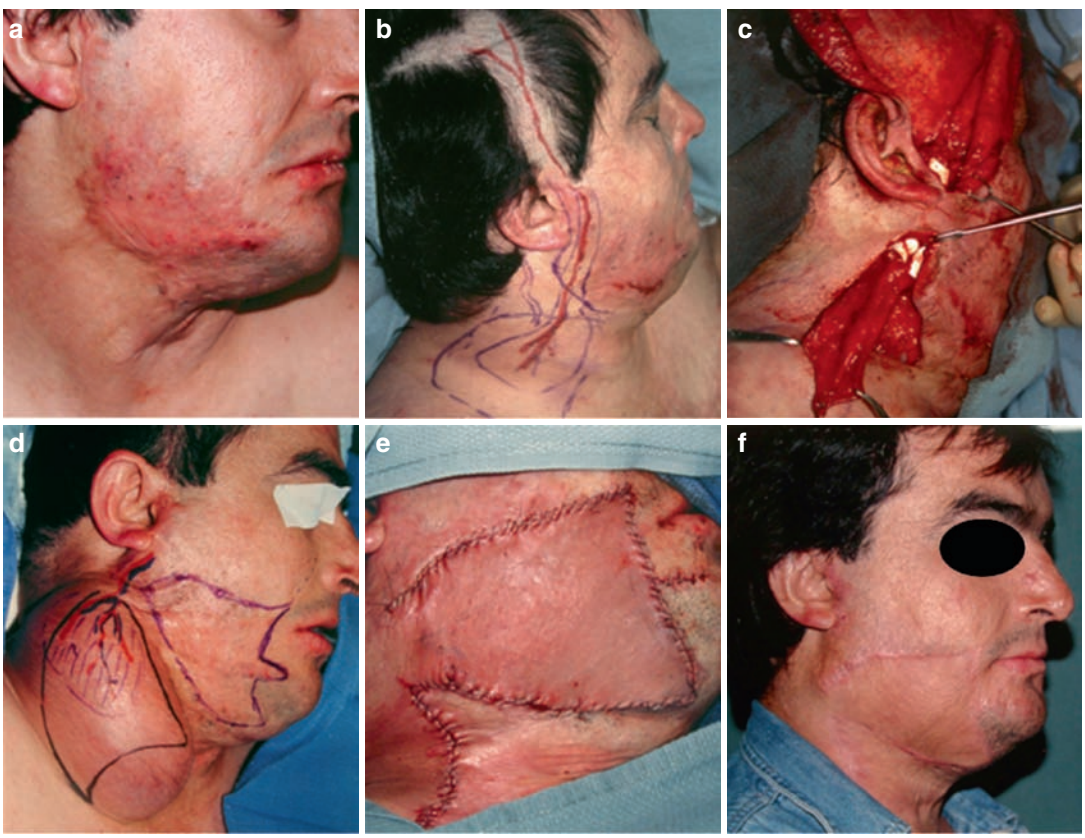

Figure 24.22. Reconstruction of cheek defect with expanded prefabricated flap. (**a**) Preoperative appearance demonstrating region of chronic induration and pain following thermal injury. (**b**) Temporoparietal fascial flap designed for transposition into neck to enable neovascularization of local tissue. (**c**) TPF flap transposition. Tissue expander subsequently placed deep to transposed flap to permit flap thinning. (**d**) Expanded flap and excision design. (**e**) Flap inset. (**f**) Postoperative appearance.

A critical factor in flap selection for microvascular free tissue transfer reconstruction of the cheek is making an accurate diagnosis and assessment of the defect. The geometry of large cheek defects is often difficult to appreciate fully, as is the extent of injury to both the mucosal and facial skin surfaces. Toward this end, the authors have found intraoperative use of alginate models of the operative defect to be an invaluable tool in guiding the design of free tissue transfer substrates. In permitting the three-dimensional rendering of the reconstructive defect, such modeling allows for the precise tailoring of free flaps by facilitating design translation onto a two-dimensional surface. In this manner, customized multifaceted flaps that simultaneously satisfy lining and external coverage needs may be constructed with relative ease. In addition, defect modeling may also facilitate operative planning for prefabricated designs when standard free flap options are insufficient.

Contour-Only Defects

Occasionally the sequellae of traumatic injury, subcutaneous contour defects of the cheek are more often the result of progressive soft tissue disorders, such as Romberg's disease, scleroderma, or facial lipodystrophy syndromes (e.g., HIV-related lipoatrophy, Barraquer-Simons syndrome). As originally outlined by Adams et al., the surgical options for management of these defects include dermal and dermis fat grafts, autologous fat grafts, local deepithelialized flaps, collagen injections, and free tissue transfer. Although nonautologous and avascular autologous techniques are prone to gradual resorption, their simplicity and the ease of "touch up" augmentation have contributed to a recent resurgence in the use of lipofilling and dermal fat grafting.[120] However, free tissue transfer has generally proven more reliable and has emerged as the treatment of choice for most patients suffering from cheek contour defects, with suitable substrates for reconstruction including the groin, deltopectoral, scapular/parascapular, transverse rectus abdominus, and anterolateral thigh free flaps.[115,116,121–123] Notably, most methods of contour reconstruction require revisions; although nonvascularized grafts often need subsequent augmentation, free tissue transfers generally require later debulking procedures (Figure 24.23).

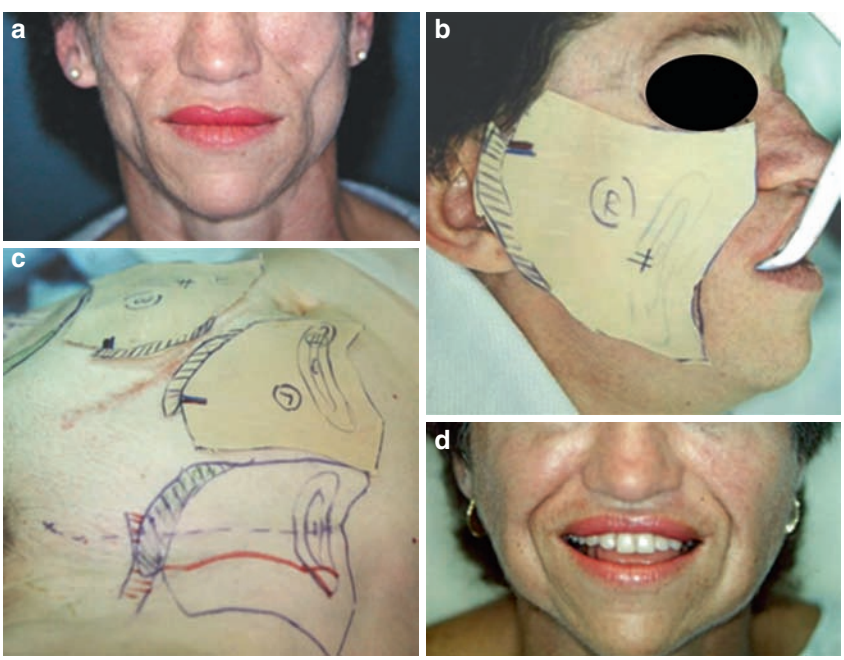

Figure 24.23. Bilateral groin free flap reconstruction for cheek contour deficit secondary to lipodystrophy. (**a**) Preoperative appearance. Note pronounced bilateral cheek hollowing. (**b**) Intraoperative template design. (**c**) Flap design. (**d**) Postoperative appearance.

Complications

Potential adverse sequellae of cheek reconstruction include prolonged edema of the delicate tissues of the lower eyelid, cicatricial or mechanical ectropion, distortion of the oral aperture, abnormal hair distribution, and secondary soft tissue defects. For the most part, derangements of neighboring soft tissue structures may be ameliorated by scar revision techniques, including Z-plasty and local tissue rearrangement. Significant abnormal hair growth occasionally requires ablation or ongoing manual maintenance. Secondary soft tissue defects are managed via secondary reconstructive procedures, including local flaps and free tissue transfer.

Combined Lip and Cheek Reconstruction

Severe injuries to the face – whether secondary to oncologic, traumatic, or thermal mechanisms – are often extensive and rarely take heed of anatomic boundaries. Combined defects of the lip and cheek are not uncommonly demonstrated in such clinical circumstances and often represent a significant challenge to the reconstructive surgeon. When possible, the individual reconstructive techniques described here should be used in combination to repair or diminish these particularly complex defects; however, distinct reconstructive strategies are often required to meet the challenges presented by such injuries.

The critical factor in the repair of defects involving both the cheek and lip is having a full and accurate appreciation of the extent and nature of tissue loss. In the setting of severe trauma or extensive resection, resting anatomy may be significantly distorted so as to render this task less straightforward than might be expected. Under such circumstances, a comprehensive assessment of the reconstructive defect may require the patient to be under general anesthesia, assuming intubation has not already been required for appropriate patient management. As described earlier, reconstructive tissue surface area requirements may be deceivingly greater or lesser than initial visual inspection would suggest; an inexpensive and accurate assessment of tissue needs is therefore assisted by the employment of three-dimensional alginate constructs from which precise two-dimensional flap templates may be fashioned.

In a recent review of reconstructive strategies for the repair of composite defects involving the nose, lip, and cheek, Menick advises surgeons to look beyond the borders of the defect and to approach repair in terms of defined anatomic subunits. Such an approach endorses the notion of altering the wound in site, size, shape, and depth as necessary to recreate the expected units. In addition, surgeons are recommended to use the contralateral normal to fashion exact reconstructive templates and to recognize the fact that the optimal reconstructive strategy may require multiple flaps and/or staged repair.[124] As such, efforts should be made to use local tissues to diminish the overall size of the defect through the employment of substrates that exhibit qualities similar to those lost. Once local reconstructive options have been exhausted, the majority of patients require microvascular free tissue transfer for full defect reparation, as has already been described.

The design of multifaceted free flaps and the employment of prelamination strategies have served as means to satisfy lining requirements for composite repairs, and the incorporation of innervated muscle in free tissue transfer has provided the potential to ameliorate significant motor impairment.[125] When free tissue transfer options are limited, as in the case of patients suffering from severe thermal injuries, flap prefabrication has been advocated as a means of creating a customized, axialized construct for subsequent transfer.[126] Reconstruction in these situations often involves a multistaged approach, where the initial free flaps repair the complex defect and then subsequent procedures are needed to repair and restore the specialized subunits. This may be done by sculpting the free flap or often by using local flap options that may be advanced to provide the best aesthetic result (Figure 24.24).

In the end, the ideal reconstructive strategy for reparation of composite defects involving the lip and cheek will likely be composite tissue allografting – particularly given the specialized structure of many of the facial tissues involved and the difficulty in providing a functional and aesthetic substitution. Until the notion of facial transplantation is more widely embraced, the reconstructive surgeon has a wide array of acceptable options on which to rely for the repair

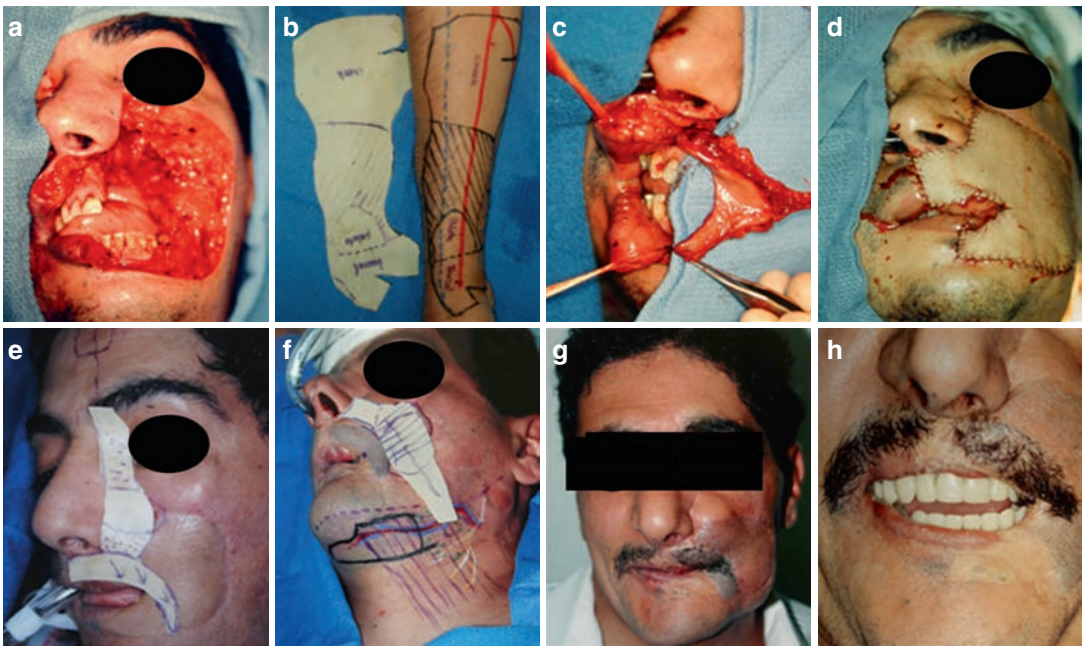

Figure 24.24. Multistaged free and regional flap reconstruction of combined lip and cheek defect secondary to excision of vascular malformation. (**a**) Defect. (**b**) Multifaceted radial forearm free flap design based on template created from alginate model of defect. (**c**) Palmaris longus oral sling. (**d**) Flap inset. (**e**, **f**) Subsequent forehead flap to repair residual malar defect and restore hair-bearing skin in moustache region, followed by innervated submental myocutaneous flap to restore smile functionality. (**g**, **h**) Postoperative appearance in repose and with active smile.

of such complex defects and is constrained only by the limits of the imagination and the tolerance of the patient in question.

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25

Auricular Reconstruction for Microtia

Robert L. Walton and Elisabeth K. Beahm

Summary

Reconstruction of the external ear for microtia remains one of the most challenging clinical problems in reconstructive surgery. Current techniques use an autologous or alloplastic framework covered by a soft tissue envelope. The degree to which the reconstructive effort replicates a normal ear relates to the technical precision and the artistry of the surgeon. In this chapter, the anatomy, embryology, and topography of the external ear and the common forms of ear malformation are overviewed. Two clinical approaches for reconstruction of microtia deformities of the external ear (Brent and Nagata techniques) are presented, including a critique on the advantages and disadvantages and anticipated outcome of each technique. The rationale and timing of middle ear surgery for restoration of hearing in the middle ear in concert with reconstruction of the external ear are explored. Alternatives to autologous external ear reconstruction such as prosthetic rehabilitation and future innovations are discussed.

Introduction

Reconstruction of the external ear is perhaps the most challenging of all reconstructions. This is largely due to the demands imposed by the ear's prominent position on the head and the requirements for ultrarefined, three-dimensional surgical rendering. Numerous techniques for ear reconstruction have been proposed over the years, and these have largely involved the draping of the periauricular skin or fascia over a carved framework, usually cartilage.[15,41,65] Surgical results often fall short of expectations owing to the technical shortcomings of inexperience compounded by the unpredictability of wound repair. In this chapter, we review current approaches to auricular reconstruction for microtia.

Anatomy

The external ear is composed of three parts: the helix-antihelix-scapha complex, the conchal complex, and the lobule (Figure 25.1). Viewed

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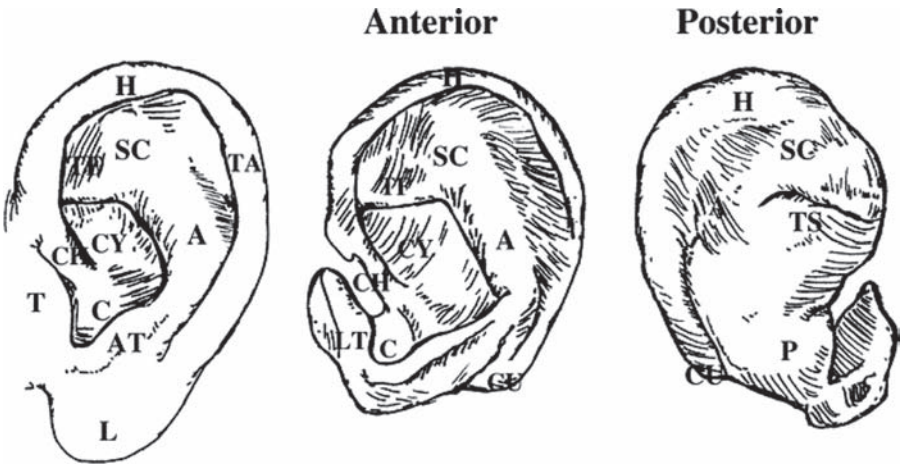

Figure 25.1. External ear anatomy. (*Left*) External ear anatomy. *CH*, crus helicis; *H*, helix; *T*, tragus; *AT*, antitragus; *L*, lobule; *A*, antihelix; *TF*, triangular fossa; *SC*, superior crus; *C*, cavum conchae; *CY*, cymba conchae; *TA*, tuberculum auriculae. (*Center*) Ear elastic cartilage anatomy, anterior surface. *LT*, lamina tragi; *CU*, cauda helicis. (*Right*) Ear elastic cartilage anatomy, posterior surface. *TS*, transverse sulcus; *P*, ponticulus. (Reprinted from Beahm and Walton.[8] Copyright 2002, with the permission of Lippincott Williams & Wilkins.)

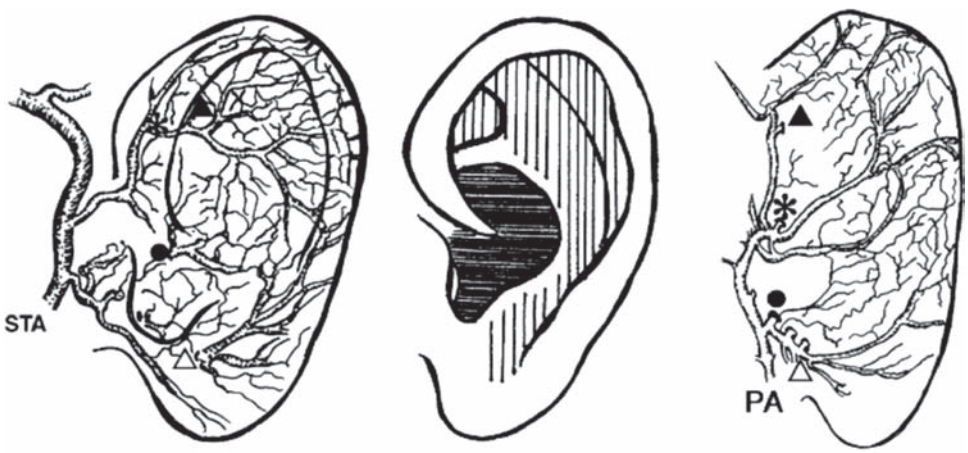

Figure 25.2. Ear blood supply. (Reprinted from Beahm and Walton.[8] Copyright 2002, with the permission of Lippincott Williams & Wilkins.). Ear Blood Supply. () Arterial supply of the anterior auricular surface. The superficial temporal artery () has upper, middle, and lower terminal branches, the most superior (*) of which provides an anastomotic network to the anteroauricular surface with branches of the posterior auricular artery that penetrate anteriorly in the region of the triangular fossa (Δ), concha (•), helical margin, and lobule (Δ). () Arterial networks of the anterior auricular surface. The superficial temporal and posterior auricular arteries contribute to the triangular fossa-scapha network () and the conchal network (). () Arterial supply of the posterior auricular surface. Branches of the posterior auricular artery () penetrate the cartilage at the triangular fossa (Δ), cymba conchae, helical root (*), cavum conchae (•), and lobule (Δ) to anastomose with branches of the superficial temporal artery. (Adapted from[79]. Used with permission.)

from the perspective of elevation, the helix-antihelix-scapha complex articulates with the conchal bowl, and this relationship can be a great advantage when performing ear reconstruction.

Blood Supply

The external ear derives its blood supply from two intercommunicating arterial networks arising from the superficial temporal artery and the postauricular artery (Figure 25.2).

Venous drainage is primarily through the posterior auricular veins to the external jugular vein. Although lymphatic drainage from the ear can be quite unpredictable, it is felt to parallel embryologic development. The concha and meatus drain primarily into the parotid and infraclavicular lymph nodes, and the superior auricle and external auditory canal drain into the mastoid and superior cervical lymph nodes.

Nerve Supply

Sensibility to the external ear is provided by several cranial and extracranial nerves. The great auricular nerve (C2 and C3) and the lesser occipital nerve (C2) supply the posterior ear and lobule (Figure 25.3). The posterior ear surface is primarily supplied by the lesser occipital nerve branches, whereas the inferior ear, the lobule, and a portion of the preauricular area are primarily supplied by the great auricular nerve. The anterior surface of the ear and tragus are supplied by the V3 branch of the trigeminal nerve – the auriculotemporal nerve. The external auditory meatus derives its sensibility from the auricular branch of the vagus nerve (Arnold's nerve).

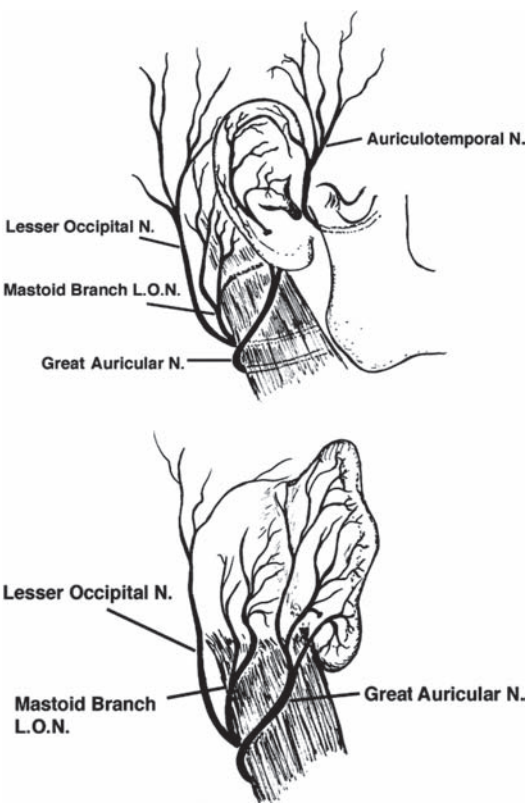

Figure 25.3. Ear nerve supply. (*Above*) Sensory innervation of anterior surface of the external ear. (*Below*) Sensory innervation of posterior surface of the external ear. (Reprinted from Beahm and Walton.[8] Copyright 2002, with the permission of Lippincott Williams & Wilkins.)

Intrinsic Muscles

The ear is surrounded by extrinsic and intrinsic muscles that are generally vestigial in nature. Of these, the posterior auricularis muscle is the most important, as it is commonly encountered during posterior approaches to otoplasty or conchal cartilage graft harvest.

Topography of the External Ear

The size of an adult external ear varies considerably according to race, sex, and familial inheritance patterns.[68] In general, ear width is approximately 55% of its length. The long axis of the ear tilts posteriorly approximately 20° from the vertical axis of the face. Contrary to popular belief, the axis of the ear does not parallel that of the dorsum of the nose – the longitudinal axis of the ear is approximately 15° more vertical. The helical rim projects 1–2 cm from the mastoid. From the superior helix to the lobule, the projection of the ear gradually increases. At the level of the superior helix, the rim projects about 10–12 mm above the mastoid; at mid helix, the projection is 16–18 mm; and at the lobule, the projection is 20–22 mm.

Embryology

The first (mandibular) and the second (hyoid) branchial arches contribute to auricular development.[7] The external ear forms by the fusion of hillocks that arise on the branchial arches between the third to sixth weeks of embryonic life. The tragus, helical root, and the superior helix are derived from the anterior three hillocks arising from the first branchial arch (Figure 25.4). The posterior hillocks derive from the second branchial arch and form the antihelix, the

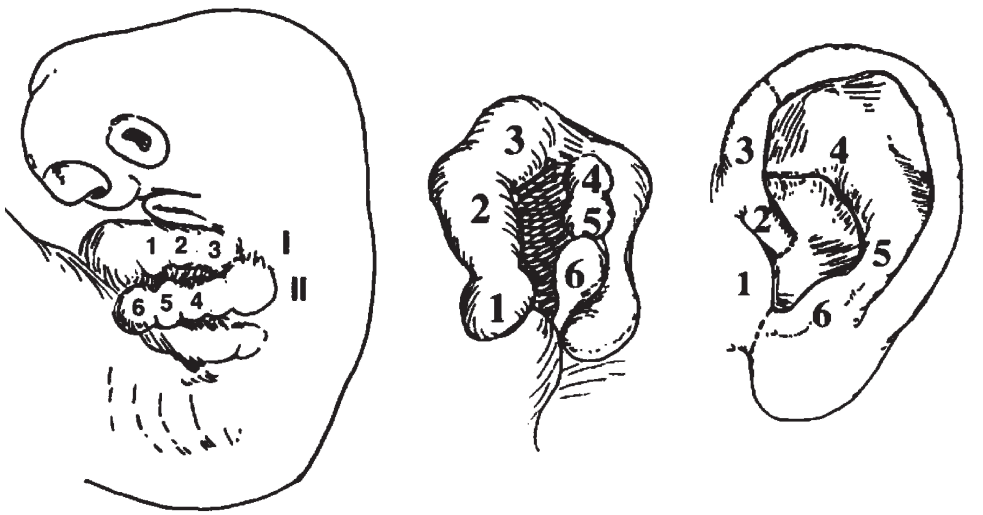

Figure 25.4. Embryology of the ear. () Hillock formation in an 11-mm human embryo. () Hillock configuration in a 15-mm embryo at 6 weeks' gestation. () Adult auricle depicting the hillock derivations. (Reprinted from Beahm and Walton.[8] Copyright 2002, with the permission of Lippincott Williams & Wilkins.)

antitragus, and lobule. The external ear forms around the external auditory meatus, which begins to canalize at the 28th week of gestation. The middle ear cavity begins formation in the first pharyngeal arch at 4 weeks and is complete by week 30. The malleus and incus arise from the first arch cartilage at 8 weeks and begin to ossify at 4 months. The stapes is derived from the second arch cartilage during the same time frame. The mastoid air cells develop after birth.

Microtia is felt to be the consequence of failure of development or adverse events that occur in the embryo during the sixth through eighth week of gestation. Adverse events that occur later in embryonic development result in less deformity. Auricular deformities may be accompanied by facial nerve, middle ear, and mandibular abnormalities, as well as associated cleft lip and palate. In 60–70% of cases, microtia presents as an isolated entity without other readily identifiable clinical anomalies.[37] In these cases, however, radiologic examination will often reveal subclinical deformities in the mandible (condyle), temporal bone, and cervical vertebrae. Isolated microtia is felt by most to represent the mildest form of hemifacial microsomia.[9,24,51]

Epidemiology

Deformities of the external ear demonstrate a range of clinical manifestations, and the variable degrees of penetrance of the gene or genes that are responsible for the hypoplasia of the ear appear to account for the differences in the sizes of the microtic remnants that are seen on clinical presentation. The causes of external ear malformations, including microtias, are felt to be heterogeneous, including a vast array of genetic abnormalities, teratogenic effects, and vascular abnormalities.[9,24,30,38,51] External ear abnormalities occur as isolated deformities or deformities associated with multiple congenital abnormalities such as clefting, neural tube defects, and limb malformations. In the United States, the reported prevalence for microtia ranges from two to three births per 10,000, with a notable increase in Hispanic and Asians, specifically those of Japanese decent.[26,78] The incidence of isolated microtia from large multinational registries of congenital malformations suggests that the prevalence of microtia is from 0.70 to 2.50 per 10,000 births. It is generally held that there is

a difference in the racial occurrence of the disease, with microtia having a lower incidence among Whites and Blacks than that in Hispanics and Asians. The occurrence of microtia in Navajo Indians has been reported as high as 1 in 1,200.[1] It is generally accepted that nonisolated cases of microtia are more common (1.53 to 10,000) than isolated cases (0.63 to 10,000).[57] A male preponderance in microtia is seen in isolated forms of microtia in North America, although a recent study from China suggested that there was no male predominance. This difference appears to be unique if one considers that the Chinese population has a generally higher incidence of males than females (1.2 to 1).

Microtia deformities range from modest defects to those of complete absence of ear development, or anotia.[8] Anotia presents in different proportions among the races with the lowest proportion seen in Caucasians. In unilateral cases, the right side appears to be more frequently involved than the left, especially when the ear malformation occurs as an isolated deformity. The cause of microtia is thought to be multifactoral and most causes are sporadic, with fewer than 15% having a positive family history. In a study of 1.6 million consecutive births in Latin America, it was observed that there was a fivefold increase in microtia in Quito, Equador, compared with the rest of South America. Explanations for this unusual incidence was linked to prenatal drug exposure, high birth order, as well as increased paternal age, although this incidence has not been fully explained. Interestingly, the high topographic elevation of Quito, which is greater than 3,000 feet, might also be considered a risk factor.[16,17] Maternal parity with four or more pregnancies has been suggested as an increased risk for microtia. In this association, anotia is seen to be the more prominent deformity than microtia.[32]

Anotia and microtia are associated with other congenital malformations. Facial clefting and cardiac defects are the most commonly associated with microtia (30%). Anophthalmia or microphthalmia (14%), limb reduction defects or severe renal malformations (11%), and holoprosencephaly (7%) are also commonly associated abnormalities.[8] These abnormalities are present to a comparable degree throughout the spectrum of external ear malformation.

Although microtia has been long felt to represent one portion of the spectrum of the entity considered to be hemifacial microsomia, there are a number continuing disagreements in the literature. Increasing evidence suggests that the commonality of isolated microtia and hemifacial microsomia in the spectrum of diseases such as Goldhar syndrome (oculoauriculovertebral syndrome) is variance to the same condition, exhibiting progressive degrees of auricle malformation.[9,27,51]

Types of Microtia

Many classifications for microtia have been proposed over the years. A widely adapted system originally described by Weerda and then simplified by Aguilar assigns a grade from I to III, based on the severity of the deformity.[6] Grade I depicts a slightly smaller than normal ear with an essentially normal features, Grade II represents an auricle that is rudimentary and malformed but contains some recognizable components. Grade III includes a classic severely attenuated ear manifested with a small nubbin of deformed tissue and anotia considered to be Grade IV.

The most comprehensive and yet simplest classification is that of Nagata, which directly correlates with surgical correction techniques.[41–45] The Nagata classification categorizes microtia into lobule type, concha type, and small-concha type (Figure 25.5). Deformities with a remnant ear and lobule without a concha, acoustic meatus, and tragus are categorized as lobule-type microtia. Auricular deformities with the aforementioned elements of concha, tragus, incisura tragica, and acoustic meatus to a variable degree are categorized as concha-type microtia. Small-concha-type deformities classically exhibit a remnant ear, lobule, and a small cartilaginous indentation representing the concha. Auricular anomalies presenting as hypoplasia of the middle third of the ear are generally classified as small-concha-type deformities. Complete absence of the external ear is termed anotia.

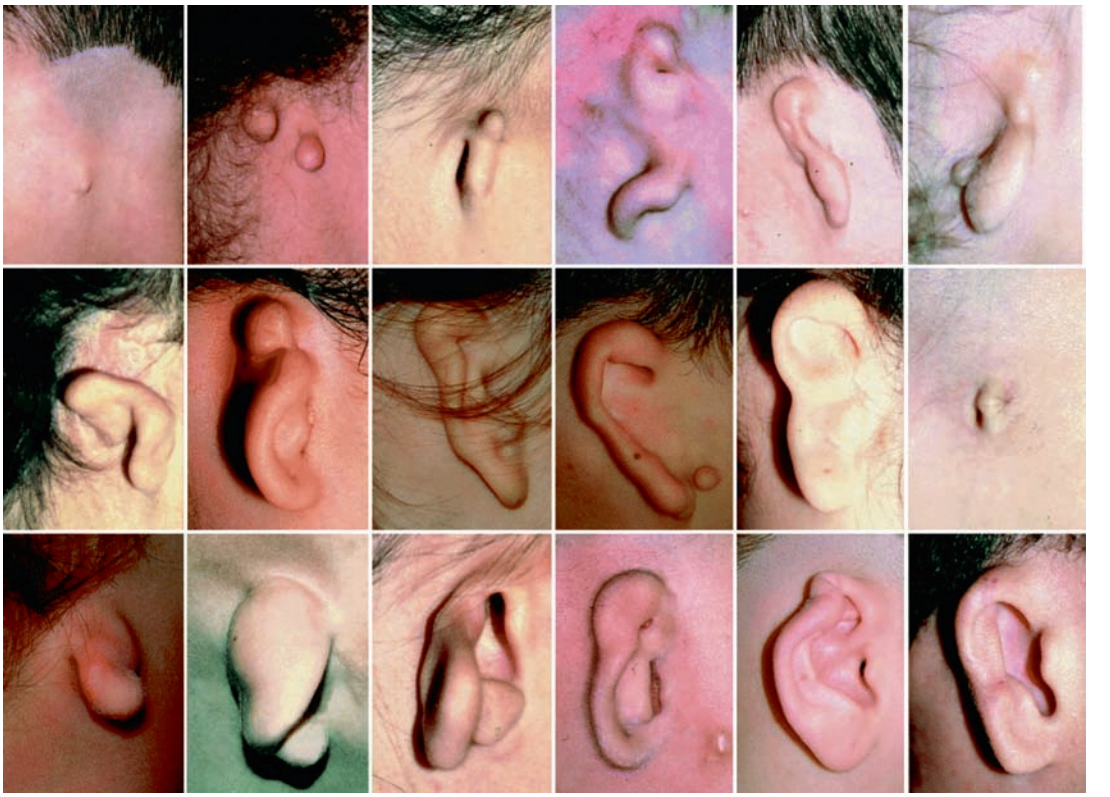

Figure 25.5. Types of microtia. Anotia (*above*, *left*); lobular-type microtia (*above*); intermediate deformities with elements of both lobule and helix (*center*); isolated tragal element with external auditory meatus (*center*, *right*); deformities with conchal, tragal, ear canal, lobule, and helical elements (including cup ear, lop ear, crumpled ear, and conchal and small conchal microtia) (*below*). (Reprinted from Beahm and Walton.[8] Copyright 2002, with the permission of Lippincott Williams & Wilkins.)

Timing of the Reconstruction

The determinants of when to proceed with ear reconstruction for microtia depend largely on the degree of physiologic development of the ear, the size and development of the costal cartilages, and the potential for peer pressure. In general, 85% of ear development is reached by the age of 3 years.[3,23] Ear width will continue to increase through the age of 10 years. The costal cartilages are relatively small until the age of 5–6 years making them impractical for use in the fabrication of an ear framework prior to that time. The growth of the costal cartilages usually parallels the rate of growth of ear cartilage through adolescence. In fabricating a costal cartilage framework for total ear reconstruction, therefore, the size of the framework should be the same as that of the normal ear in older patients or slightly larger in younger patients.[27,64,66] In the United States and Europe, most surgeons prefer to perform the ear reconstruction between the ages of 4 and 6 years, during preschool, completing the reconstruction prior to the child entering first grade. In Japan, Nagata prefers to defer the ear reconstruction until the age of 10 years or later, when the chest wall is nearly fully developed and a trans-xiphoid circumference of 60 cm or greater has been achieved.[25,41–45]

Current Techniques in Auricular Reconstruction for Microtia

Current techniques for reconstruction of the external ear evolved from the work of Tanzer who advocated a carved autologous rib cartilage framework as the basis of the reconstruction.[65]

Two particular techniques championed by Burton Brent and Satoru Nagata have gained wide acceptance in the plastic surgery community and are summarized in the next section.[15,41]

The Brent Technique

First Stage

The Brent technique is a 3–4 stage technique that is similar to that proposed by Tanzer although the sequence is different.[71] In the first stage, a template of the normal ear is traced on celluloid and is then drawn 2–3 mm smaller to accommodate the draped skin envelope (Figure 25.6). The lobular element of the framework is adjusted to account for the variable development of the lobule remnant of the microtic ear. The completed template is then positioned at the microtic site to align symmetrically with the opposite ear using the nose, lateral canthus, and lobule as reference points. In hemifacial microsomia, positioning of the new ear can be problematic because of the asymmetry of the cranial base and foreshortening of the hemifacial landmarks. In these instances, Brent recommends positioning the new ear relative to the superior pole of the contralateral ear,

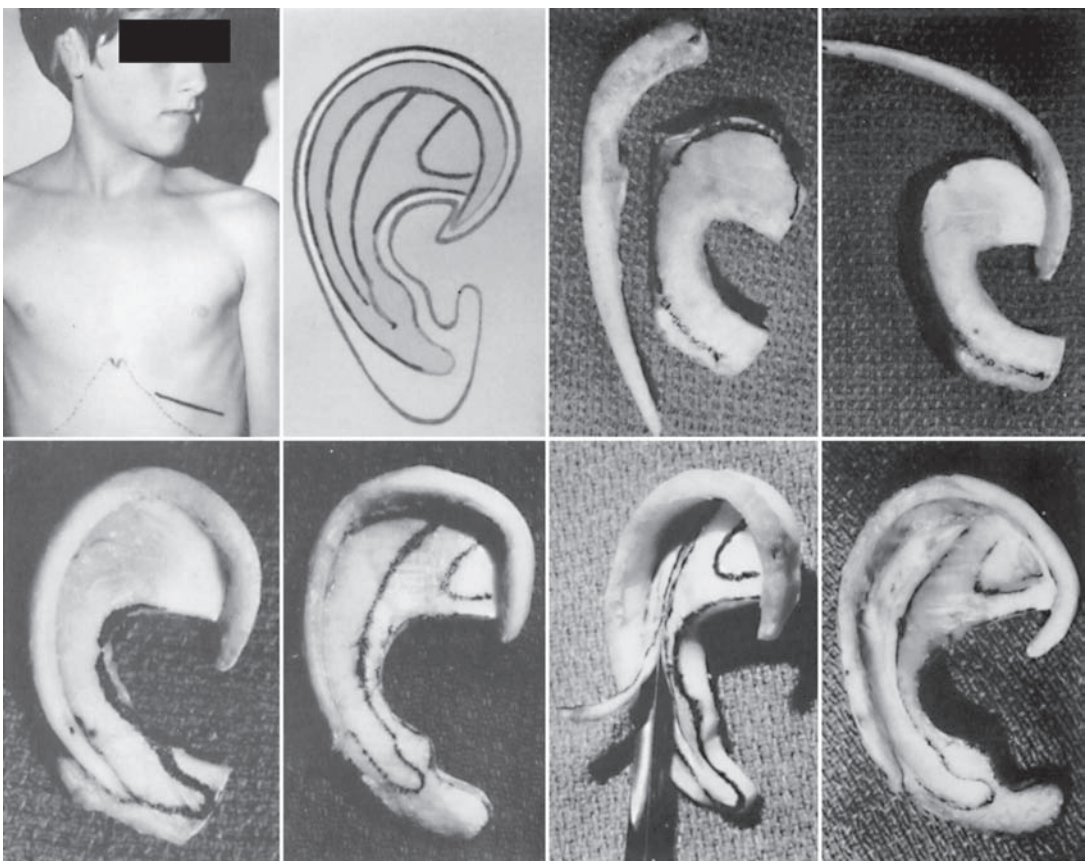

Figure 25.6. Brent technique, first stage. Ear framework fabrication with sculpted rib cartilage. (,) The chest incision is made contralateral to the ear being constructed. (,) Film pattern. Note that the proposed framework is smaller than the reversed ear tracing, which allows for added thickness of covering skin; the framework tip is made substantially smaller to accommodate the lobule when it is transposed. (,) The rib cartilage specimens. The tapered, "floating" cartilage serves as a helix. (,) The thinned helix, attached at its crus. (,) The helix is affixed to the main block; outline of the inferior crus-posterior conchal wall contour line. (,) Carving in progress; auricular details are outlined with ink. (,) Sculpting details with chisel. (,) The completed framework. To best demonstrate the fabrication process, several frameworks are used for this figure. (Courtesy Dr. Burt Brent.) (Reprinted from Walton and Beahm.[71] Copyright 2002, with the permission of Lippincott Williams & Wilkins.)

because the lateral canthus is not a reliable reference point. It is also important to appreciate that the lobular remnant of the microtic ear may be positioned too far medial on the face, necessitating a compromise in ear positioning between the measured distance and that of the lobule.

The framework is derived from the contralateral 6th–8th costal cartilages. The base element of the framework is derived from the synchondrosis of the 6th and 7th cartilages, and the "floating" 8th rib is used to construct the helix element. The framework is assembled with clear nylon suture. The salient anatomic elements of the helix, antihelix, and superior and inferior crus are carved in exaggerated fashion with high relief to accommodate for the loss of definition caused by draping of the skin envelope.

After the framework has been rendered, a pocket is dissected at the microtic ear site to receive the construct. An incision is made on the posterior inferior edge of the vestigial ear, and all remnant cartilages are removed. The skin is dissected in the subcutaneous plane to create a pocket wide enough to both accommodate the framework and to allow enough laxity of the overlying skin envelope to allow draping of the framework without undue tension. The framework is checked for correct positioning and then secured to the deep fascia with an absorbable monofilament suture. Fine suction drains placed beneath and adjacent to the framework are used to occlude the overlying skin flap to the framework to achieve optimal definition and delineation of the carved, three-dimensional relief. It is felt that this technique carries less risk for compromise to the skin flap circulation compared with pressure or bolster dressings. A light head dressing is placed to protect the reconstructed ear from the probing hands and fingers of both parent and child.

In the first stage of the Brent Technique, the lobule is not repositioned. By placing a high-profile construct beneath a virgin, well-vascularized, unsullied skin envelope, the Brent approach is thought to maximize definition while minimizing compromise of the soft tissue cover. It is this technical aspect that sets the Brent method apart from that of Tanzer's, which transposes the lobule in the first stage.

Second Stage

The second stage involves transposition of the lobule (Figure 25.7). This is usually performed 3–4 months following creation and insertion of the cartilage framework. In this stage, the remnant lobule is repositioned anatomically. It is felt that transposing the lobule to articulate with an existing framework is technically much easier and more accurate than transposing the lobule without a structural reference. Depending on the size and extent of development of the lobule, this

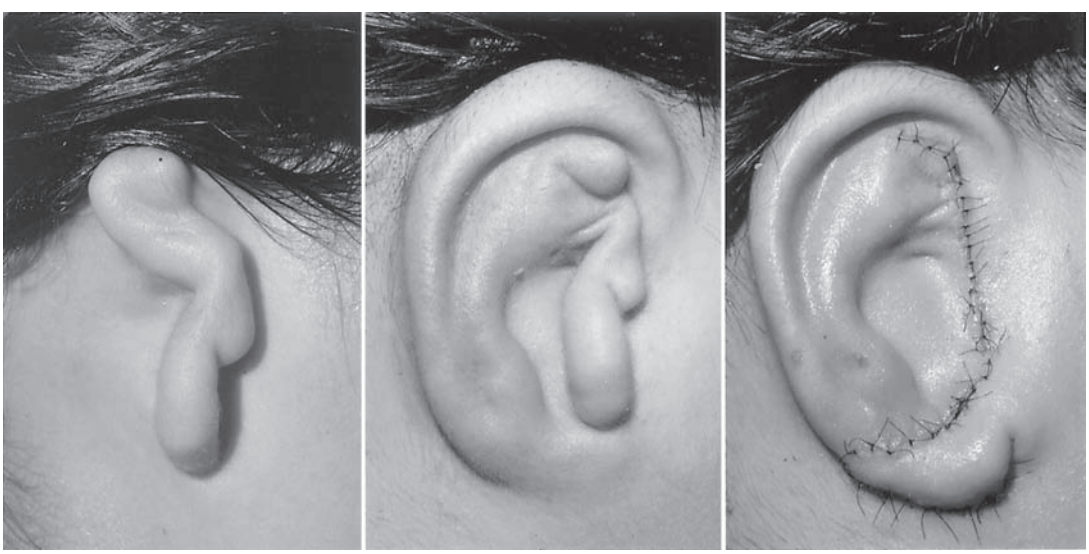

Figure 25.7. Brent technique. Lobule transposition. (Reprinted from Walton and Beahm.[71] Copyright 2002, with the permission of Lippincott Williams & Wilkins.)

can vary from a simple transposition to filleting and insetting of the lobular remnant into the tail of the framework.

Third Stage

The third stage involves elevation of the construct to produce projection of the helical rim (Figure 25.8). In the several months that have transpired since insertion of the cartilage framework, a vascular fibrous capsule has developed on the surfaces of the construct. The cartilage framework with its attached fibrous capsule anchored anteriorly can be dissected from the surrounding tissues without compromise to the blood supply. For elevation of the construct, an incision is made several millimeters from the perimeter of the helical rim, and the posterior surface of the framework is dissected superficial to the fibrous capsule until sufficient projection is achieved. The projection is maintained by placing a wedge of carved rib cartilage (banked in the subcutaneous tissues following the First Stage) behind the raised framework in a fascial pocket. The retroauricular skin is then advanced to the posterior sulcus and secured with monofilament PDS sutures. The exposed capsular surface of the posterior side of the construct is closed with a medium-thickness split skin graft from the hip secured with a tie-over, stent-type dressing.

At 7 days, the stent dressing is removed. Graft take is usually quite good. The elevated ear will remain slightly overprojected for several weeks until the skin graft has healed sufficiently and contracted. During this time, the ear is protected with a foam doughnut dressing secured with a headband to avoid inadvertent injury.

Fourth Stage

In the fourth Stage, the tragus is reconstructed and the concha is excavated to achieve optimal depth and definition (Figure 25.9). Further adjustments may be made on ear positioning as well as matching the projection of the opposite ear with a setback procedure as required. The Brent method for tragus reconstruction uses a composite skin cartilage graft from the opposite ear conchal bowl via an anterior approach. A J-shaped incision is made along the proposed tragus margin, and a subcutaneous pocket is dissected anteriorly to receive the conchal graft. The size and configuration of the composite graft are determined by fashioning and fitting foil templates to achieve projection of the tragus and cavitation of the retrotragal hollow. The conchal bowl is then excavated. An alternative

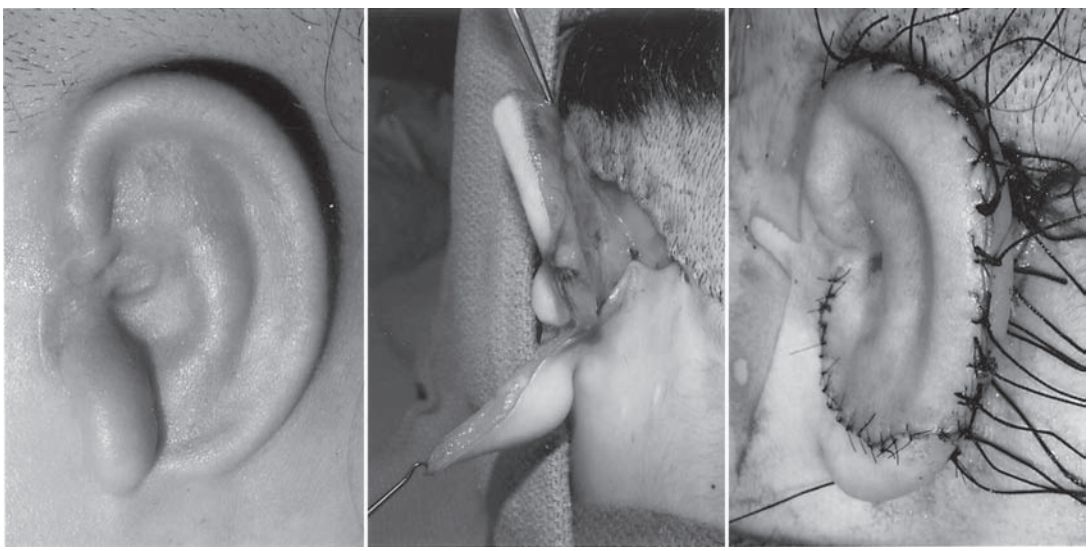

Figure 25.8. Brent technique. Elevation of framework. The lobe transposition combined with elevation procedure, which was safe because the skin bridge above the short lobule carries circulation across to the auricle. (Courtesy Dr. Burt Brent.) (Reprinted from Walton and Beahm.[74] Copyright 2002, with the permission of Lippincott Williams & Wilkins.)

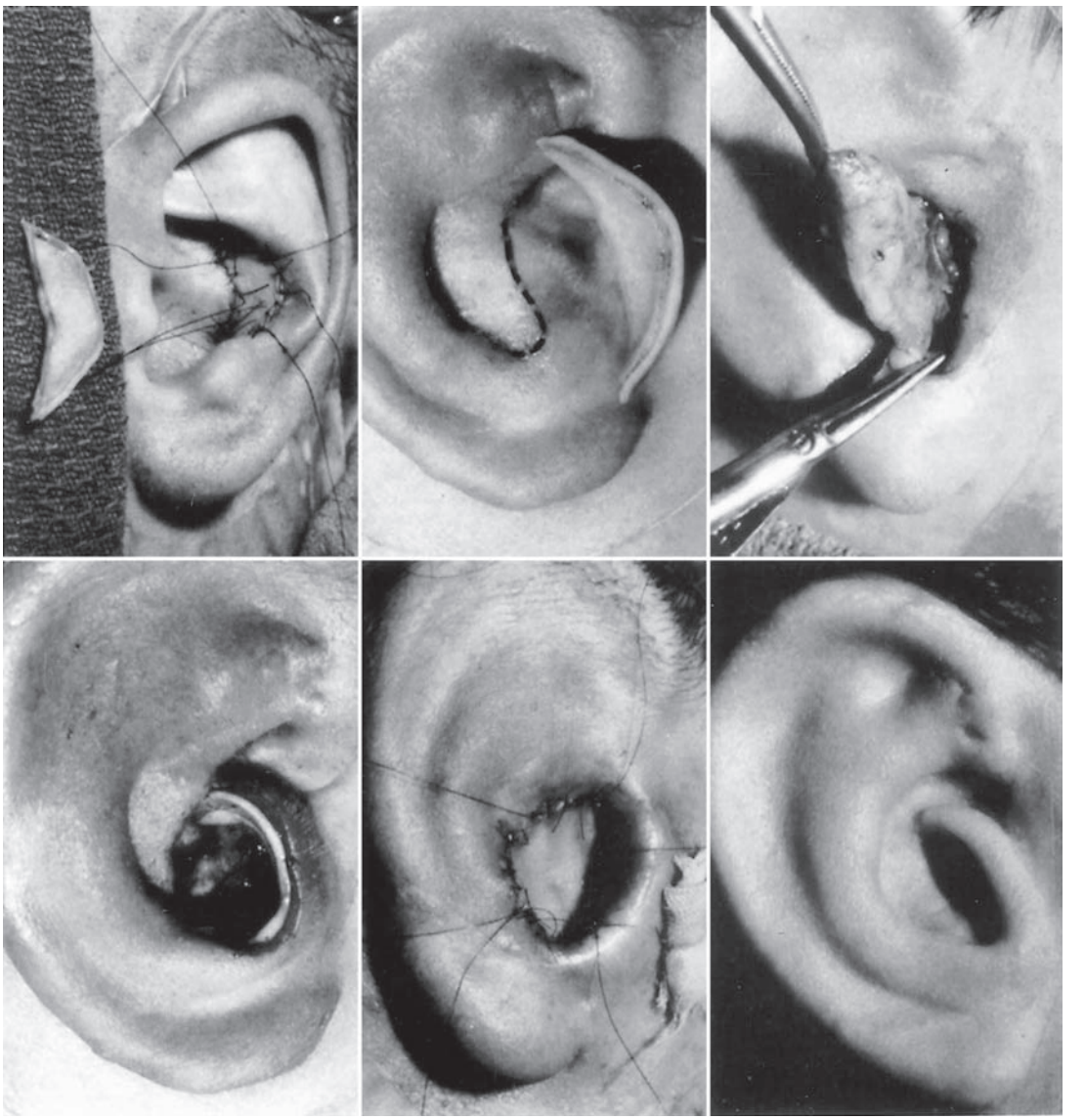

Figure 25.9. Brent technique. Tragal reconstruction, concha excavation, and canal mimicry in the unilateral microtia patient. (*Above, left*) Chondrocutaneous graft harvested from the contralateral ear. To avoid setting this particular ear too close to the head by the usual direct closure of the defect, the concha is repaired with a small skin graft harvested just anterior to the hairline. (*Above, center* and *below, left*) A composite conchal cartilage/skin graft from the opposite ear is applied beneath the "tragal flap," developed by a J-shaped incision placed at the proposed location of the tragal margin and intertragic notch. (*Above, right*) Excision of excess soft tissues accentuates the conchal depth. (*Below, center*) A composite graft is pulled under the tragal flap with a bolster suture; the conchal floor is resurfaced with a full-thickness skin graft harvested from the posterior contralateral ear lobule. (*Below, right*) Result 2 years postoperatively. The shadow cast beneath the constructed tragus mimics the external auditory meatus. (Courtesy of Dr. Burt Brent.) (Reprinted from Walton and Beahm.[71] Copyright 2002, with the permission of Lippincott Williams & Wilkins.)

to the composite skin cartilage graft has been adopted from Nagata and others and involves incorporating a small cartilaginous extension to the framework to create a tragus element. The definition achieved with this technique is less than that achieved with the composite skin/cartilage

graft described above, although the skin cartilage grafts may contract, diminishing the retrotragal hollow and occasionally everting the tragus.

A major criticism of Brent's technique is the number of operative stages, though in some cases the second and third stages may be combined if the local vascular anatomy allows for safe manipulation of the tissues. Some have also criticized Brent's final results for having lack of definition in the conchal bowl, the antitragus, and the intertragal notch. This concern is underscored by the need to perform an additional procedure for conchal excavation. Nevertheless, the Brent technique has proved to be a very reliable and effective technique for ear reconstruction that has proven durable over time (Figure 25.10).

The Nagata Technique

First introduced in 1993, the Nagata technique has enjoyed wide success as an alternative to the Brent technique.[71] Its major advantage lies in its two-staged approach, which provides for reconstruction of the incisura intertragica and incorporates a tragal element in the primary cartilage framework.

First Stage

The first stage of the Nagata technique involves fabrication and insertion of a cartilage framework (including a tragal element) and transposition of the lobule. This roughly corresponds to the first three stages of the Brent technique. Nagata uses the ipsilateral 6th–9th costal cartilages in fabricating the framework, which contrasts Brent's use of the contralateral 6th–8th costal cartilages. Nagata attempts to preserve the entire perichondrium except at the junction of the 6th and 7th costal cartilages. The framework is constructed in three distinct levels or "floors" that represent specific elevations of the ear. These include the cymba and the cavum conchae: first floor; the crus helicis, fossa triangularis, and scapha: second floor; and the helix, antihelix, tragus, and antitragus: the third floor. The base or first floor of the framework is constructed from the 6th and 7th costal cartilages. The helix and the crus helices are carved from the 8th costal cartilage, and the 9th costal cartilage is used to construct the antihelix with its superior and inferior cruces. The cartilage elements are joined using fine-gauge wire sutures (Figures 25.11 and 25.12).

Preparation of the recipient site commences with careful drawing of the skin flaps (Figure 25.13). The skin of the concha is derived from the posterior surface of the remnant lobule, which provides ample soft tissue coverage of the cartilage framework. A "W" incision is drawn with the tip of its mid point located at the apex of the incisura intertragica. The posterior skin flaps are elevated in the subcutaneous, subdermal plane.

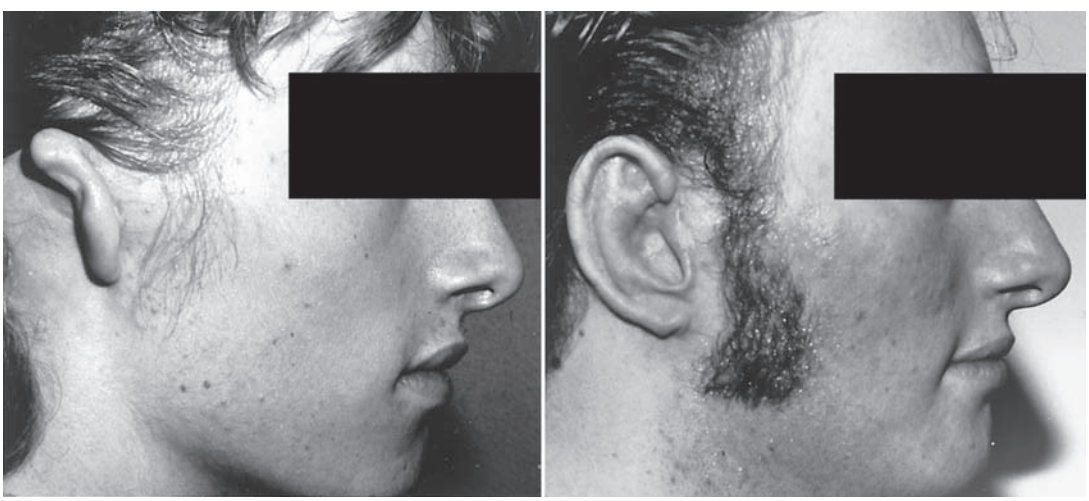

Figure 25.10. Brent technique, long-term follow-up. () A 13-year-old male patient is shown preoperatively. () The same patient 10 years after reconstruction. (Courtesy Dr. Burt Brent.) (Reprinted from Walton and Beahm.[71] Copyright 2002, with the permission of Lippincott Williams & Wilkins.)

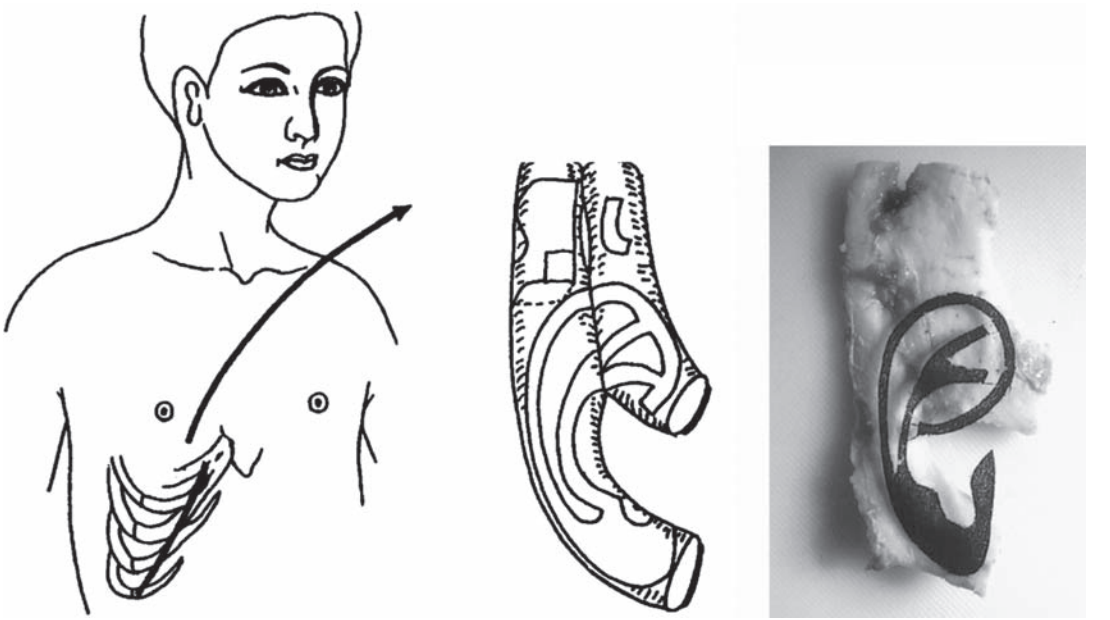

Figure 25.11. Nagata technique. Harvesting of the costal cartilages. (*Left*) The sixth through ninth costal cartilages are harvested from the same side as the reconstruction, leaving the majority (75%) of the perichondrium intact. (Recently, Nagata started leaving all of the perichondrium intact.) The harvested cartilages are reversed to take advantage of their configurations. (*Center*) A three-dimensional frame unit is oriented to the harvested cartilages. (*Right*) Clinical example. (Reprinted with permission from Nagata, S. Microtia: Auricular reconstruction.[80]

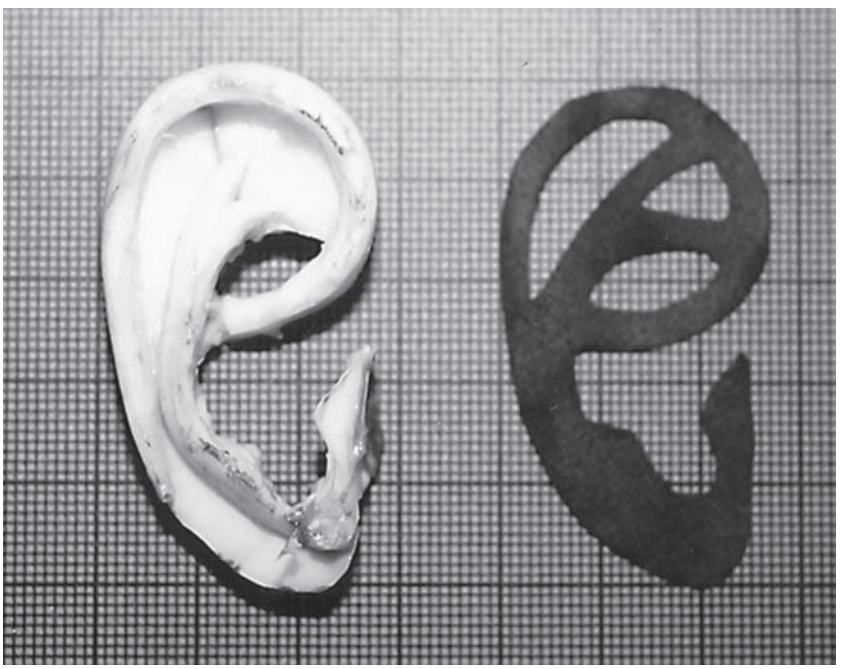

Figure 25.12. Nagata technique. Carved framework. The base frame is constructed from the sixth and seventh costal cartilages; the helix and crus helicis are constructed from the eighth costal cartilage; and the ninth costal cartilage is used to construct the superior crus, inferior crus, and antihelix. The remaining structures are carved from residual cartilage pieces. The cartilage construct is assembled using fine-gauge wire sutures. (Courtesy Dr. S. Nagata.) (Reprinted from Walton and Beahm.[71] Copyright 2002, with the permission of Lippincott Williams & Wilkins.)

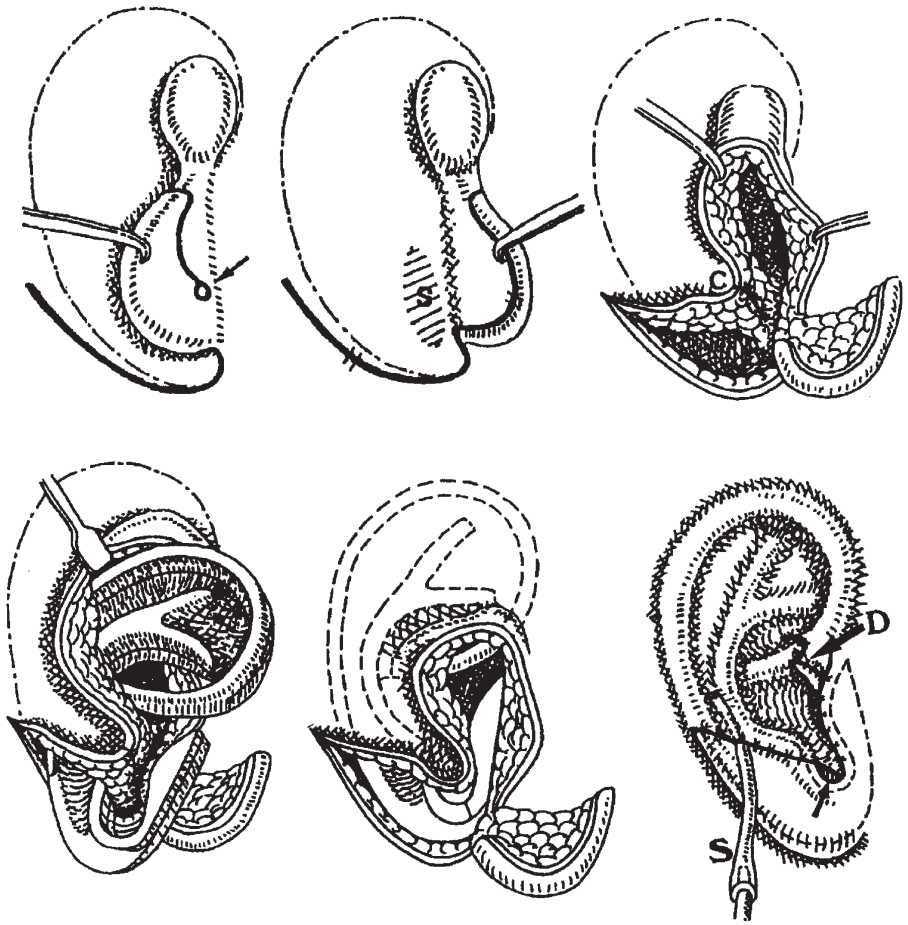

Figure 25.13. Nagata technique. Incision planning, creation of skin flaps, placement of framework. () Incision line for the anterior surface of the auricle. Note that the terminus of the anterior incision is circular (). (,) Incision line for the posterior surface of the auricle; note the W-shaped flap. The shaded area (S) depicts the area of subcutaneous tissue that will be left intact so as to optimize blood supply to the flaps. () The skin flaps are undermined, and the wings of the W-shaped flap have been approximated to form the cup of the intertragic notch (C). (, and) Insertion and positioning of framework. () Transposition of lobule and closure of incisions. Excessive skin shown at (D) is inverted to construct the pseudoacoustic meatus. The skin flaps are then approximated to the underlying framework with cotton cylinder compresses secured by bolster sutures. (Courtesy Dr. S. Nagata.) (Reprinted from Walton and Beahm.[71] Copyright 2002, with the permission of Lippincott Williams & Wilkins.)

At the level of the future intertragic notch, a button of subcutaneous tissue is kept intact to augment the blood supply to the undermined skin flaps. All cartilage remnants are removed. The cup of the intertragic notch is formed by approximation of the wings of the W-shaped flap. The anterior lobular incision forms the edge of the neotragus. At the terminus of this incision, a small 2-mm circle of full-thickness skin is excised. This will coapt with the cup of the joined "W" flaps to form the rim of the incisura intertragica. The anterior skin flap is elevated to receive the tragal element of the framework. After the skin flaps have been sufficiently undermined, the framework is inserted and positioned anatomically. The skin of the anterior surface of the lobule remnant is then transposed over the tail of the framework to create the new lobule. Bolsters of dental cotton are then used to secure the skin flaps to the cartilage framework. These are affixed with 4-0 monofilament mattress sutures. The bolsters are kept in place for 2 weeks (Figure 25.14).

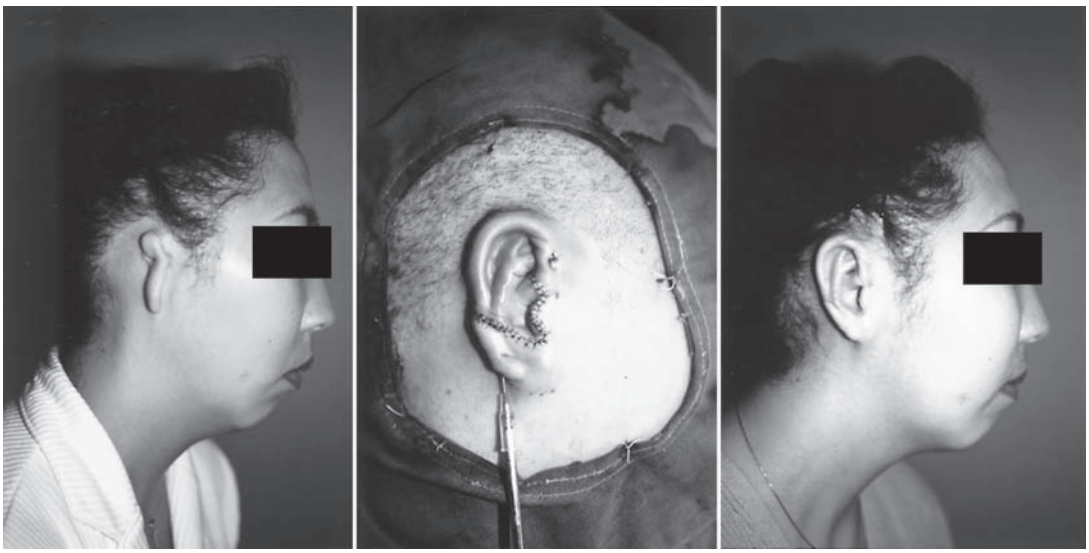

Figure 25.14. Nagata technique, first stage. Clinical case. (*Left*) A 29-year-old woman with lobule-type microtia. (*Center*) Immediate result after placement of framework and skin closure. (*Right*) Six months after the first-stage operation. (Courtesy Dr. S. Nagata.) (Reprinted from Walton and Beahm.[71] Copyright 2002, with the permission of Lippincott Williams & Wilkins.)

Second Stage

The second stage of the Nagata technique involves elevation of the construct (Figure 25.15). This differs from the Brent technique in that the posterior surface of the elevated auricle is covered with a temporoparietal flap and a split skin graft harvested free hand from the occipital/parietal scalp. After elevation of the framework, a crescent-shaped wedge of autologous rib cartilage is placed beneath the framework to maintain the elevated position of the ear. The retroauricular skin is advanced toward the ear as far as possible to minimize visibility of the graft scar. The temporoparietal fascial flap is elevated through a zigzag temporal scalp incision and then tunneled subcutaneously to the posterior ear site, where it is used to cover the posterior surface of the ear, the cartilage graft, and any exposed mastoid surface. After insetting the fascial flap, the wound is closed with a split graft from the occipital/parietal scalp secured with a tie-over bolster dressing.

Criticisms that have surfaced from others who have used Nagata's technique are primarily technical in nature. Of significant concern is the relatively high incidence of compromise of the perilobular skin flaps in which the reported incidence of flap necrosis approaches 14%.[25] This is felt to relate to impaired dermal perfusion in the skin flaps, which is, in part, due to the excessive skin tension resulting from the high relief of the framework. Additionally, the incision required for transposition of the lobule may also impose some restrictions on flap blood supply. Although Nagata feels that the retained subcutaneous pedicle at the midpoint of the posterior flap improves flap circulation, others doubt the reliability of this adjunct for preventing flap necrosis. Additionally, harvest of the posterior skin of the lobule remnant to increase the size of the covering flap is felt by some to destroy the natural appearance of the lobule.

The Nagata technique has been criticized for its piecemeal construction, which may be at risk for delayed resorption and deformity. This complication, however, has not been borne out by long-term follow-up of over 600 patients.[71]

The Brent and Nagata techniques both use autologous rib cartilage for creation of the framework. Both techniques may result in permanent deformity of the anterior chest wall. Because Nagata technique uses more cartilage, the potential for greater deformity exists. Although Nagata attempts to preserve most of the perichondrium during rib graft harvest, the benefit in the older patient remains questionable.

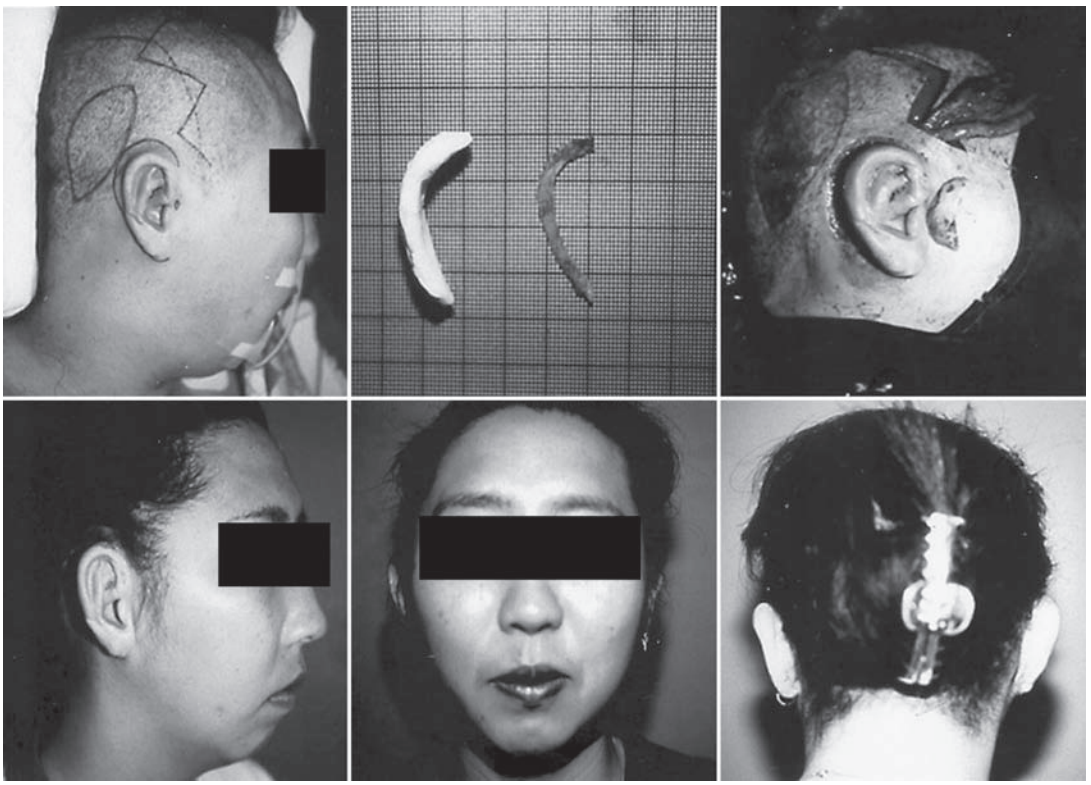

Figure 25.15. Nagata technique, second stage. Clinical case. () The design for incision lines and harvesting of the ultradelicate split-thickness scalp graft and temporoparietal fascia. () A crescent-shaped costal cartilage block is fabricated from the harvested fifth costal cartilage. () The constructed cartilage is undermined and elevated. The cartilage graft is wedged in the postauricular sulcus to create the posterior conchal wall. A temporal parietal fascial flap is then transposed to provide soft tissue coverage over the postauricular surface. The wound is closed with an ultradelicate split-thickness scalp skin graft. () Lateral view shown 2 years after the second-stage operation. () Frontal view shown 2 years after the second-stage operation. () Posterior view shown 2 years after the second-stage operation. (Courtesy Dr. S. Nagata.) (Reprinted from Walton and Beahm.[71] Copyright 2002, with the permission of Lippincott Williams & Wilkins.)

Nagata's use of a superficial temporal fascia flap for coverage of the posterior ear is felt to be excessive by many and increases the risk for hair loss and scarring in the temporal scalp. Use of the superficial temporal fascia may also compromise the ability to use local tissues to salvage an ear reconstruction. Although the deep temporal fascia may also be used in these cases, the size and draping characteristics of this layer are less optimal than those of the superficial temporal fascia.

With either technique, consistently excellent results can be achieved only through practice and experience. Both the Brent and Nagata techniques will produce superb results in experienced hands. The pitfalls of either technique can be minimized by strict attention to detail and a rigid commitment to excellence. As emphasized by Nagata, "the failure to achieve the desired endpoint is the result of compromise of the surgical technique or the omission of certain procedures during the reconstruction."[71]

Alloplastic Materials for Framework Fabrication

Over the years, numerous alloplastic materials have been used to create a framework for use in ear reconstruction. Success has been quite variable and likely relates to the surface biocompatibility of the alloplast material, its pliability, durability,

and propensity for tissue adherence. Medical-grade silicone rubber, commonly used throughout the 1960s and 1970s for ear and nasal reconstruction, exhibited a high extrusion rate likely due to its lack of porosity and tissue adherence.[20,21,47,48] Unlike silicone rubber, porous polyethylene promotes tissue ingrowth and adherence and has proved quite advantageous as a scaffold for ear reconstruction.[52,55,75] Reinish has shown the utility of porous polyethylene in ear reconstruction using a commercially available, standard-sized implant.[52] Complication rates are reportedly acceptable but appear to relate to surgical experience. The major advantage of an "off-the-shelf" scaffold is the lack of chest wall donor-site morbidity and deformity and decreased operative times. The quality or refinement of ear reconstruction using these standardized implants, however, is clearly inferior compared with that of the multistage, autologous cartilage-based ear reconstructions in the hands of masters.

Complications in Ear Reconstruction

As with any complex reconstructive effort, complications in ear reconstruction can and do occur. These can be defined as early and late complications in the ear reconstruction and at the chest wall donor site.

Ear Construct Complications

Infection is an early complication of ear reconstruction and usually follows compromise in surgical sterile technique. Infection may also occur beneath compromised skin flaps. In early infections where the skin envelope has not adhered to the framework, there is great risk for contamination and loss of the entire cartilage framework. Fortunately, this is a very rare, but devastating, complication in ear reconstruction. Late infections are less problematic, as the skin envelope is adherent to the cartilage framework and the infection usually involves only a small, isolated portion of the framework. In these cases, wound debridement and closure with local skin or fascial flaps will resolve the problem with minimal loss of the framework.

Partial necrosis of the skin envelope is an infrequent complication in ear reconstruction. Small isolated patches of necroses less than 1 cm diameter can usually be managed with conservative treatment with topical antimicrobials allowing the wound to heal by secondary intent. Larger areas of necrosis require formal operative debridement and closure with local skin or fascial flaps. The superficial temporal fascia flap has proven utility in these situations. Effective use of the flap requires very careful planning to ensure adequate flap size and inset without tension or kinking. A split scalp graft is used to provide epithelial coverage of the exposed fascia.

Infections and partial skin flap cover loss, once resolved, often lead to residual ear deformities that will require surgical intervention to correct. Use of fascial flaps generally results in loss of definition of the ear reconstruction. These deformities may require subsequent debulking and repeat skin grafting to achieve improved definition.

Cartilage resorption is a relatively common complication in ear reconstruction. The causes of resorption are multifactorial and include infection, scarred or poorly vascularized recipient bed, tight skin envelope, and wire or polymer sutures that are tied too tightly. With respect to framework construction, wire sutures, especially when twisted to secure adjacent elements of the construct, are notorious for inducing resorption of the cartilage directly beneath the suture. The tension caused by tightly twisted or knotted sutures is thought to restrict the passive flow of nutrients through the graft, leading to necrosis. When assembling the framework, the sutures should be tied with little or no tension to avoid this complication. Late complications of local cartilage resorption are notching and distortion of the framework and spitting of the sutures through the overlying skin envelope. Local areas of resorption can be managed by removal of the offending suture and re-augmenting the helical rim or antihelix with fresh autologous cartilage grafts. In cases where there has been general resorption of the framework, more extensive restoration of the framework may be required. If the skin envelope is too tight or otherwise inadequate, a superficial temporal fascia flap may be required to supplement coverage.

Other relatively common complications observed in ear reconstruction include malposition of the framework, a construct that is larger than the opposite normal ear, lack of definition in the reconstructed ear, and inadequate of projection. These complications generally relate to

inadequate preoperative planning and surgical execution. There are relatively few operative procedures in which outcome is so closely related to the precision of the surgical technique as in ear reconstruction. Moreover, this directly relates to experience.

There are a plethora of techniques available for determining the correct positioning of the ear on the head. Tolleth has studied the variations in ear position on the head and proposes that, ideally, the ear top should align with the brow and the lobular tip with the columella.[67] Many surgeons use a grid aligned to the facial/skeletal midline. In cases of hemifacial microsomia, however, there is asymmetry between the normal and microtic sides, making the grid technique and other midline-based techniques somewhat ineffective. In these cases, the surgeon must locate the ear reconstruction at a site that will give the optimal aesthetic appearance, understanding that the measured ear position will be discrepant from the normal side. Positioning of the ear template should be performed with the patient in the upright position and viewed from the front, back, and both sides to ensure correctness. It is also important to discuss this issue with the parents and child preoperatively to avoid any misunderstandings later. Recently, Walsh has reported on the successful use of a device for creating and positioning an autologous cartilage framework based on modeling the opposite, normal side.[70]

The size of the reconstructed ear tends to be larger than the normal ear. The framework has been shown to increase in size through adolescence, and this may account for some of the observed discrepancies between reconstructed and normal ear sizes.[12] In many cases, however, the large size of the reconstructed ear is the fault of creating a framework that is excessively large. Frequently, the framework will be patterned from the normal ear, not accounting for the skin envelope that will eventually drape the construct. To avoid this complication, it is important to make the framework 2–3 mm smaller than the pattern of the normal ear.

Adequacy of projection of the reconstructed ear has not received proper attention in most texts, but it is this asset that truly delineates normal from abnormal on first or casual appearances regardless of the quality of the three-dimensional reconstruction. An ear reconstruction that is of correct size, is positioned anatomically, and has symmetrical projection with the normal ear will be accepted as "normal" in most social interactions regardless of the quality of its three-dimensional rendering. To achieve symmetry in ear projection, it is perhaps easiest to match the normal ear projection to that of the reconstructed ear. If the reconstructed ear has very little or no projection, then a secondary projection of the reconstructed ear should be performed. In these cases, simple placement of a cartilage of alloplast wedge behind the framework is inadequate and results in very little improvement in projection. The tethering forces behind the reconstructed ear (scar, contracted skin graft) must be totally released, and then the framework should be elevated to the desired projection and supported in this position with an appropriately sized and shaped wedge. The exposed framework is then covered with a local or superficial temporal parietal fascia flap and a skin graft.

Chest Wall Donor-Site Complications

Early complications at the donor site include pneumothorax, hematoma, and infection. Pneumothorax is a relatively common event that follows costal cartilage harvest. In most cases, a small rent in the pleura is identified and, after inflating the lungs with positive pressure ventilation, is repaired over a small red rubber catheter that is removed following closure of the subcutaneous tissues. Rarely is the lung parenchyma injured necessitating the placement of a chest tube. Postoperative upright chest x-rays are routinely obtained to assess for pneumothorax.

Hematoma and infection are very uncommon following costal cartilage harvest. Nevertheless, it is important to adhere to strict sterile technique and meticulous hemostasis during cartilage graft harvest.

The most common late complication following costal cartilage harvest is donor-site chest wall deformity. In patients younger than 10 years of age, chest wall deformities are seen in nearly 64% of patients.[46] In older patients, the rate of chest wall deformity is 20%. This has prompted some surgeons to advocate delaying the ear reconstruction until adolescence, a practice common in Asia. Preservation of the posterior perichondrium, as proposed by Nagata, allows for regeneration of the costal cartilage in younger patients and may be effective in stifling the development of donor-site chest wall deformity.[71]

Combined Approaches for Hearing Restoration and Reconstruction of the External Ear Deformity in Microtia

Binaural hearing affords improved sound localization, speech perception, and learning. Studies have demonstrated that children with unilateral hearing loss from any cause are at risk for delayed language development, attention deficits, and poor school performance. Traditionally, it has been held that middle ear reconstruction was not indicated in a unilateral microtia with normal hearing on the contralateral ear. This approach is based on the clinical observation that those patients who have microtia often fail to achieve true binaural hearing following correction of their middle ear deformity, because they continue to rely most heavily on the normal hearing ear. Moreover, experimental observations indicate that the neural structures in the auditory system that are critical for binaural processing develop only if binaural hearing is present and undisturbed early in life.[36,40] Accordingly, children with unilateral microtia have primarily had their otologic treatment focused on preservation of the normal hearing ear with amplification of the affected one. Recently, modifications of this approach have come to light. The plasticity of the developing auditory system appears to be greater than originally suggested, as a number of patients may exhibit binaural processing, albeit some what suboptimal, even after long-term sensory deprivation.[61,69,76]

Middle ear surgery by experienced surgeons results in hearing improvement in approximately 70% of all cases. Although encouraging at first glance, there are inherent risks to the procedure, including injury to the facial nerve as well as a decrease in sensorineural hearing levels.[18,22,34,77] These risks suggest that a risk/benefit analysis be undertaken before each proposed procedure. Middle ear surgery should proceed only when a final air-bone gap of 30 dB or better may be expected.[34]

Many surgeons hold that the modest gain in hearing obtained with surgical intervention in the middle ear does not warrant the risks of the procedure. This thinking is strengthened by the observation that hearing occasionally degrades postoperatively as a result of mechanical conditions, such as stenosis of the new external auditory or chronic infection. Additionally, approximately a third of the patients will require an additional surgical revision procedure. These objections are countered by data that show hearing improvement of 30 dB or greater in nearly two-thirds of patients operated. Although the magnitude of facial nerve injury can be staggering, the risk of facial nerve injury in an experienced surgeon's hands approximates that of cholesteatoma surgery, and the risk of sensorineural hearing loss is comparable to that reported for stapedectomy. This would therefore suggest a favorable risk benefit ratio for middle ear exploration in selected patients.[4,18,22,32]

The current approach to middle ear surgery involves careful evaluation of the status of the ossicles, mastoid, and facial nerves, using and applying specific selection criteria to establish those patients who might optimally benefit from a surgical intervention.[4,32,36] A rating system developed by Aguilar and Jahrsdoerfer assigns a cumulative point scale given for the anatomic variables in the middle ear that can be ascertained from a precisely executed CT scan. One point is given for the presence of each of the following: an open oval window, an adequate middle ear space, a normal facial nerve course, a malleus-incus complex, adequate mastoid pneumatization, an incus-stapes connection, good external ear appearance, and canal stenosis with a malleus bar. Two points are given for the presence of the stapes. A score of 8 or more in total suggests that the patient will be a good candidate for surgery. Cumulative scores of 5 or less would contraindicate surgery as would a predominately sensorineural-type hearing loss. Complete lack of pneumatization of the mastoid or obstruction of the mandibular condyle or glenoid fossa would mitigate against surgery. In general, the less severe the clinical deformity of the external ear in microtia, the better the development of the middle ear.[31]

In bilateral microtia, early and conscientious use of bone conductive hearing aids is imperative for the patient's social hearing and speech development. Traditionally, it is has been held that if adequate auditory acuity is not achieved with the use of these aids by approximately 1 year of age, middle ear exploration should be

undertaken on the most favorable side of the hearing deformity. Unfortunately, not all patients are considered favorable candidates. If otologic surgery is deemed appropriate, the anticipated success rate approximates only 50% in bilateral cases.[18,34,63] Due to this relatively poor success rate, hearing deficits in bilateral microtia children are usually not treated surgically or are deferred until the patient can participate in the decision to undergo surgery later in life. The majority of hearing deficits in children with bilateral microtia are managed primarily with hearing aids.

There have been a number of advances in conductive hearing aids, but a major limitation of these has been their fixation to the mastoid bone. Bone aids have been traditionally applied to intact skin with adhesives, headbands, or eyeglasses, and these limitations can be particularly problematic in young children. Recently, bone-anchored hearing aids have proved to be quite promising.[2,28,29,38,50,53] The implant is usually composed of ceramic and titanium or gold, and is compatible with magnetic resonance imaging (MRI). The bone-anchored hearing aid has demonstrated marked improvement over the last 20 years and demonstrates a reliably more favorable response over conventional aids in terms of hearing threshold levels as well as in aiding ears that have been previously refractory. This efficacy is likely due to the improved osseointegrated anchoring of the device to the mastoid bone itself. The success of these implants has obviated the need for middle ear exploration in a number of cases. In general, these implants are well tolerated in children and have minimal adverse effects, such as soft tissue reaction and minor infections in 30% of patients.[53] The aids have a retention rate of over 95% on long-term follow-up.

When considering restoration of the external ear deformity combined with middle ear surgery for hearing, localization for placement of the external ear canal as well as a conductive hearing aid is very important. This planning must take place with full collaboration and consensus between the otologist and plastic surgeon to ensure both good coaptation and hearing and to avoid the surgical incision site used for the external ear. In middle ear reconstruction, the canal position, the vascular axis of the flaps, and the location of the incisions that will be used in the external ear reconstruction must be carefully considered. It may require at least an additional stage in the auricular reconstruction and necessitate flexibility on the part of both the otologist and the external ear surgeon. Integrated protocols with atresia repair and auricular reconstruction in cohesive stages have also been proposed in a three- to five-stage procedure.[5,58] Brent prefers to delay the middle ear surgery until after the auricular reconstruction is completed, and this has become the standard approach in the majority of patients.[10,11,13,14]

The combined techniques for microtia repair and canal atresia have been updated by Siegert et al.[58,59] Following placement of the costal cartilage framework, transposition of the lobule, and framework elevation, the ear canal and tympanic membrane are constructed from prelaminated composites of fibrovascular stroma and cartilage using silicone molds stored at the rib cartilage harvest site. A series of microtia reconstructions using this combined approach have yielded good results with the majority (76%) having an air-bone gap of 30 dB or smaller, which is consistent with or better than other reports. Two-stage atresia repair and microtia reconstruction have been described by Cho and Lee.[19] Additionally, successful cochlear implantation was recently reported in a child with microtia, aural atresia, and profound, bilateral sensorineural hearing loss.[35]

Ear Prosthetics

The field of anaplastology has evolved in the recent years to become an important adjunct to reconstructive surgery. In failed prior autologous ear reconstructions, reconstructive sites compromised by prior radiation, or reconstructive needs in elderly patients or those patients with high operative risks, ear prosthetics offer a reasonable solution for restoration of the missing anatomy.[62,74] Surprisingly life-like fabrications of the missing ear can be made from latex or silicone rubber. The prosthesis can be attached by glue or anchored to osseointegrated implants in the temporal bone. The latter method is much preferred and carries a high patient satisfaction rate. The osseointegrated implants are well tolerated and elicit minimal skin reaction. When exposed to ultraviolet light, all prosthetics tend to fade over time, necessitating their replacement.

Future Considerations

Ear reconstruction has evolved as a technical feat of tissue manipulation, and rendering this is based on a keen understanding of wound repair and is wholly related to the artistry and experience of the operating surgeon. In the best-case example, the reconstructed ear approximates the fine details of a normal ear but does not duplicate them. The reconstructed ear is relatively rigid and has a variable degree of insensibility. The reconstructed ear also changes over time with distortion or resorption of the cartilage scaffold. Further refinements in ear reconstruction will likely not come from improved surgical techniques but rather from successful application of tissue engineering solutions in reconstructive surgery. Of this, the evolution of scaffold technology is of paramount importance.[72,73] The ideal scaffold for ear reconstruction must have a high degree of biocompatibility and elicit little cellular or soft tissue reaction. It should be thin, lightweight, flexible, have a shape memory, and should promote tissue healing/re-epithelialization if it becomes exposed. The surface of the ideal scaffold should promote cellular and soft tissue adherence. Intuitively, it would seem that autologous cartilage is an ideal scaffold material, as it has all of the desired physical and biomechanical attributes except for genetically defined shape. Vacanti and others have shown that transplanted chondrocytes, under special experimental conditions, can be induced to produce de novo cartilage.[33,49,54,56,60,69] When placed in molds, the neocartilage can be configured into complex three-dimensional forms that resemble ears. These early experiments used fetal cartilage to induce chondroneogenesis, thereby making applications in older patients problematic. Recently, Mesa and others have demonstrated the feasibility of inducing adult chondrocytes to produce neocartilage just as do chondrocytes from young donors.[39] The question remains, however, if these three-dimensional constructs will retain their size and configuration over time. To date, the ideal scaffold material for ear reconstruction remains to be discovered.

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Aesthetic Surgery of the Aging Face and Neck

James E. Zins and Colin M. Morrison

Summary

Although procedures for the correction of facial aging in the 1980s–1990s emphasized sub-SMAS approaches and operations of increasing complexity, more recently, minimally invasive techniques are gaining increasing popularity. This chapter reviews the concepts behind these approaches, describes procedures to correct areas recalcitrant to facelift correction, and offers minimally invasive options or lesser procedures that can be used to correct various aspects of facial aging when more involved procedures are not a viable option.

Introduction

Statistical analysis documents dramatic aging of the United States' population. At present, more than 10% of the people in the United States are older than 65 years. Life expectancy has similarly increased. The average male reaching age 65 years is now expected to live to 81 years, whereas the average female of 65 years will statistically live to be 84 years of age.[45]

Given the above data and recent emphasis on quality-of-life issues, it is not surprising that cosmetic surgery has seen a similar increase in popularity.

Other trends deserve mention. Although cosmetic surgery in the 1980s was marked by facelift procedures of increasing complexity,[8,25,37,38,42] the 1990s and early twenty-first century have been highlighted by emphasis on minimal downtime and minimally invasive techniques.[43] This includes operative techniques, light-based modalities, and injectables. According to recent statistics, 8 of 10 procedures performed by plastic surgeons and dermatologists whose practices are 50% or more cosmetic-related are nonoperative injectable ones.[2] Recent American Society of Plastic Surgeon statistics[3] estimates that more than 3,000,000 individuals were injected with botulinum toxin in 2004 and over 500,000 individuals received fillers.

According to the American Society of Plastic Surgeons' 1998 statistics, there has been a 113% increase in plastic surgery procedures in patients older than 65 years between 1996 and 1998.[4] This trend has almost certainly accelerated into 2007.

This chapter attempts to emphasize the current concepts in facial aging as they apply to surgical procedures practiced today, analyze why they work, and point out areas where answers are less clear and where opportunity for improvements exist.

M.Z. Siemionow and M. Eisenmann-Klein (eds.), *Plastic and Reconstructive Surgery*, Springer Specialist Surgery Series, © Springer-Verlag London Limited 2010

Facelift

The modern era of facelift surgery began in the mid 1970s with Tord Skoog.[40] Skoog realized that tension could be taken off the skin closure of the skin-only facelift, laxity in the superficial fascia and fat addressed, and a longer-lasting facelift obtained by a facelift performed deep to the platysma and superficial fascia. The Skoog facelift was a totally subplatysmal and sub-SMAS (the SMAS had yet to be described) dissection. In retrospect, the drawbacks of this operation were (1) the platysma tends to be less lax in the neck than neck skin per se and (2) Skoog did not appreciate the importance of release of the yet to be described zygomatic and upper masseteric ligaments[19] to mid-face rejuvenation. Therefore, although this procedure produced excellent results, lack of mid-face correction and early recurrence of neck laxity were limiting factors.

Hamra and Lemon[35] reported their favorable results in over 500 Skoog facelifts in 1984. Hamra[25,26] later modified the Skoog facelift to correct for the above, first in his "Deep-Plane Facelift" and later with his composite rhytidectomy. Realizing the lack of mid-face correction, Hamra's dissection after a short subcutaneous cheek dissection was deep to the SMAS but then became superficial at the level of the zygomatic major muscle. This avoided facial nerve injury where the nerves are mostly at risk while allowing medial dissection and ligament release. In the neck, Hamra's dissection was subcutaneous rather than subplatysmal, again addressing the weakness in Skoog's operation of early recurrence in neck skin laxity.

In the mid 1970s, on Tessier's suggestion, students of Tessier investigated and described the SMAS, its anatomy, and its boundaries. Although some of the Mitz and Peyronie description were later in dispute,[21,31] their findings became one of the bases for modern facelift surgery. That is, that the superficial fascia of the face is contiguous with the platysma inferiorly and the superficial temporal fascia superiorly and that the superficial fascia is an investing layer of the muscles of facial expression. The Mitz and Peyronie findings led to the standard SMAS dissection of the 1970s, which ended at the anterior border of the parotid gland. This facelift technique predated Furnas'[-19] description of the retaining ligaments of the face by over 10 years. Furnas described the retaining ligaments of the face (the zygomatic, masseteric, and mandibular), and he was one of the first to realize the importance of ligament release to mid-face correction. A number of authors have since incorporated this basic concept in their modern facelift variant. These authors all practice release of the zygomatic and upper masseteric ligaments to improve the mid face.[13,25,37]

Mendelson's description is particularly helpful in understanding the three-dimensional anatomy of the face as it relates to facelift surgery.[36] Between the superficial and deep fascia of the face is a cleavage plane. Through this space pass the branches of the facial nerve and the ligaments. Anterior to the parotid gland, the facial nerve branches pass from deep to the parotid-masseteric fascia into this sub-SMAS space to innervate the muscles of facial expression predominantly on their deep surface. (Three exceptions: BLM buccinator, levator labii superioris, and mentalis.) The nerves pass deep to superficial in a predictable manner. That is, they pass deep to superficial *distal* to the retaining ligaments. Therefore, until the ligaments are reached, the nerve branches are deep and relatively safe from injury. Virtually all modern sub-SMAS procedures follow a similar and consistent dissection pattern. That is, the sub-SMAS plane is continued until the zygomatic major is encountered. The dissection becomes superficial or subcutaneous once the zygomatic major is visualized, avoiding the facial nerve branches, which are at that point in the sub-SMAS cleavage plane innervating the muscles of facial expression on their deep surface. Further, medial dissection ensures adequate release of the zygomatic and upper masseteric ligaments. Modern sub-SMAS procedures then vary, not in release, but in means and location of fixation and whether the operation is in a single sub-SMAS plane[25,26] or a separate subcutaneous and sub-SMAS plane.[13,36–38,42]

Facelift procedures do not, however, have to be sub-SMAS to be effective. Until recently, a basic tenet was that ligament release at any level, subperiosteal, sub-SMAS or subcutaneous, allows for adequate mid-face correction. Even this can be questioned given the proven effectiveness of lateral SMASectomy and MACs lift.[7,43,44] A reasonable explanation for the efficacy of the lateral SMASectomy is that there is enough ligamentous laxity that release is perhaps unnecessary.

Patient Evaluation

A firm understanding of both hard and soft tissue cephalometrics in the frontal and profile views is essential to good surgical planning. Although numerous methods have been described, the following has been found to be most useful in our unit, allowing both rapid analysis in the office setting and detailed preoperative photographic analysis. For frontal evaluation, a series of horizontal lines are drawn on the photograph or measurements are made during patient office examination. These horizontal lines are (1) at the apex of the eyebrow, (2) through the medial canthus, (3) through the alar base, (4) through the oral commissure, and (5) through soft tissue menton. The vertical distance from the medial canthus to the apex of the brow (A) is equal to the vertical distance of the upper lip (C) and vertical distance from the alar base to the medial canthus is equal to the alar base to soft tissue menton (C, D). Further, upper lip (C) is equal to one-half of the vertical length of the chin (D) (Figure 26.1).

Profile analysis can be similarly performed. Frankfort's horizontal is estimated by constructing a horizontal line through the tip of the extraoral auditory canal and parallel to the floor (Figure 26.2). A vertical line through the glabella is then constructed perpendicular to Frankfort's horizontal. The chin should fall just posterior to this vertical line construct.

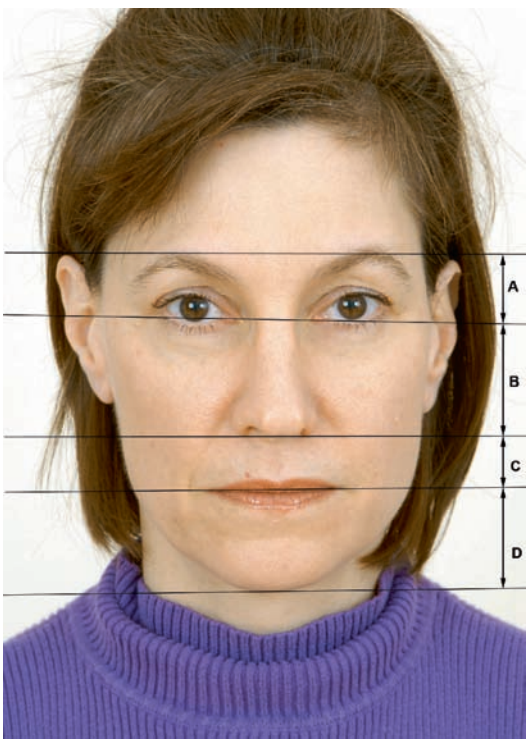

Figure 26.1. Frontal soft tissue cephalometric analysis. The face is readily divided into four areas by the following horizontal lines drawn through the (1) apex of the brow, (2) medial canthal ligaments, (3) alar base, (4) oral commissure, and (5) soft tissue menton. This creates vertical distances A, B, C, and D. Distance A is roughly equal to distance C, whereas distance B is roughly equal to distance D. The upper lip distance (C) is approximately one-half the vertical length of the chin (D).

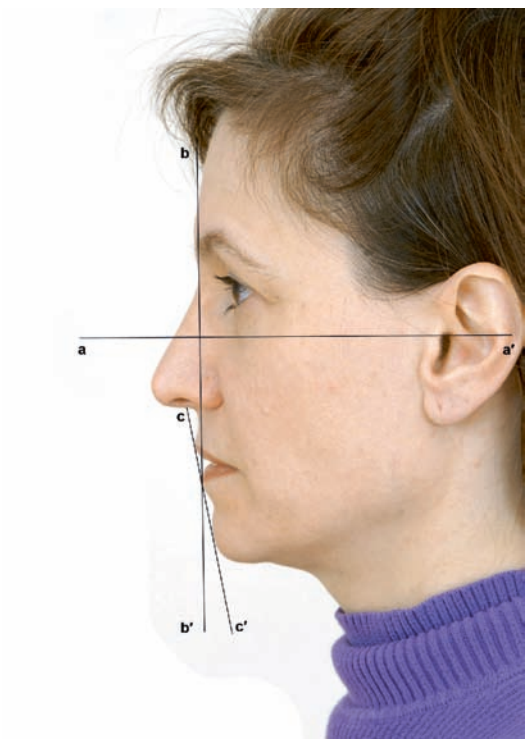

Figure 26.2. Lateral soft tissue cephalometric analysis allows for rapid assessment of chin position. Reidel's line, a tangent connecting the anterior-most projection of the upper and lower lips, determines ideal chin position. If the chin falls posterior to this idealized line, the patient demonstrates sagittal microgenia. If the chin falls anterior to this line, the patient demonstrates sagittal macrogenia.

Reidel's line is also quite valuable and corrects for head tilt. This line is a tangent connecting the upper and lower lips' most anterior projection. The ideal chin should fall on Reidel's line (Figure 26.2). If the chin is posterior, this suggests sagittal microgenia and the need for chin advancement. If anterior to this line, this suggests sagittal macrogenia. The decided improvement in profile and front view can be appreciated when isolated horizontal advancement genioplasty is performed (Figure 26.3). Restoring lower vertical facial height can also have a dramatic affect on facial appearance (Figure 26.4). Chin reduction should be approached with caution as this will deflate the soft tissue envelope and have an adverse affect on aging.

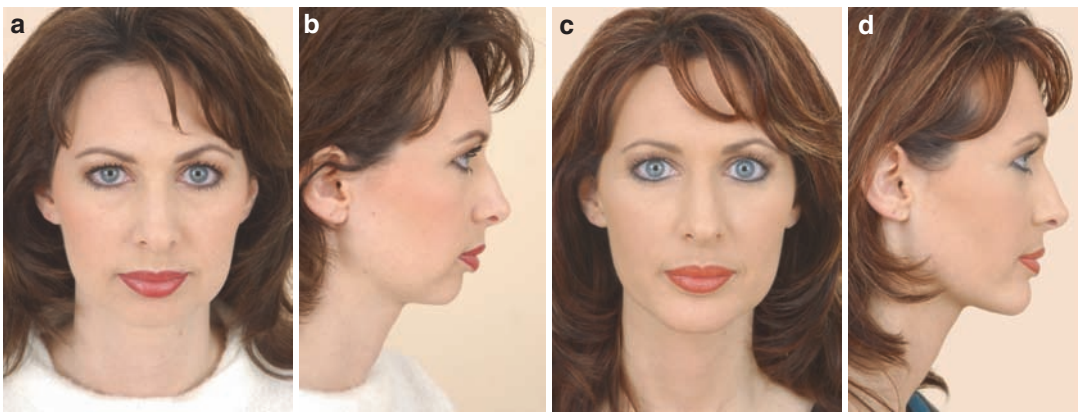

Figure 26.3. (**a**) Preoperative frontal and profile view of this 30-year-old female with sagittal and (**b**) vertical microgenia. Six-month postoperative (**c**) frontal and (**d**) profile views following correction with a horizontal advancement genioplasty.

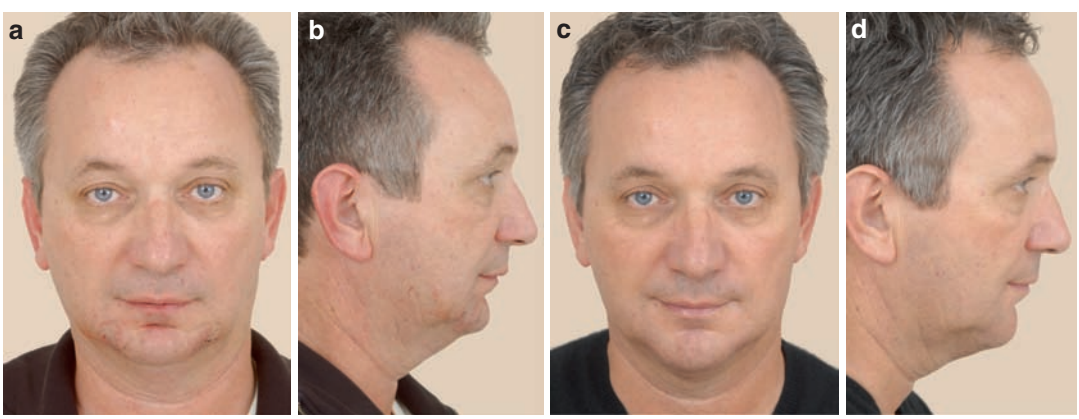

Figure 26.4. (**a**) A 55-year-old male who presented with a decrease in his lower vertical facial height due to vertical microgenia. Postoperative (**b**) frontal and (**c**) profile views following vertical lengthening genioplasty and interposition implant of tricalcium phosphate.

The Neck

Patient Evaluation

Neck laxity is one of the earliest signs of facial aging and can be treated either as an isolated entity or at the time of face/necklift. Once corrected by skin and platysmaplasty techniques, its results are long lasting.

There are easy necks to correct and there are necks that are more difficult. Most of these problems can be predicted preoperatively. From the skeletal point of view, good chin position as predicted by soft tissue cephalometric analysis is critical to the postoperative result. If sagittal and/or vertical microgenia are present, correction of these entities should be discussed as their correction will dramatically enhance the result. Gonial angle configuration is also an important preoperative criterion. A prominent gonial angle will enhance the surgical result, whereas an obtuse angle is not ideal. Defatting just posterior to the angle and inferiorly will unmask the angle hidden by soft tissue laxity and subcutaneous fat (Figure 26.5).

A low hyoid bone is a well-described limiting factor to neck correction. Although it should be appreciated preoperatively, it is not readily corrected.

In the patient with an obtuse cervicomental angle skin excess, platysma laxity and fat above and/or below the platysma may all play contributing roles. Although fat superficial to the platysma is readily apparent, subplatysmal fat is more difficult to assess preoperatively and usually requires intraoperative identification.

A ptotic submandibular gland should be noted in the preoperative examination and pointed out to the patient. Jowl fat can sometimes be confused with a ptotic gland but should be relatively easily differentiated from it. The gland lies in the mid mandible, whereas jowl fat is found more medially.

Ideal neck correction includes (1) restoration of ideal skeletal form with a horizontal genioplasty or anatomic chin implant, (2) platysma tightening where it is most lax, that is, medially, (3) defatting above and below the platysma, and (4) plication over the submandibular gland if after platysmaplasty the gland remains prominent. Although some authors advocate submandibular gland removal,[16] this is not practiced in our unit and should be approached with great caution, as complications can be significant.

The importance of skin excision deserves special attention and discussion. Consistent with the trend toward less surgery, the senior author (JEZ) as well as others[16,33,47] have described

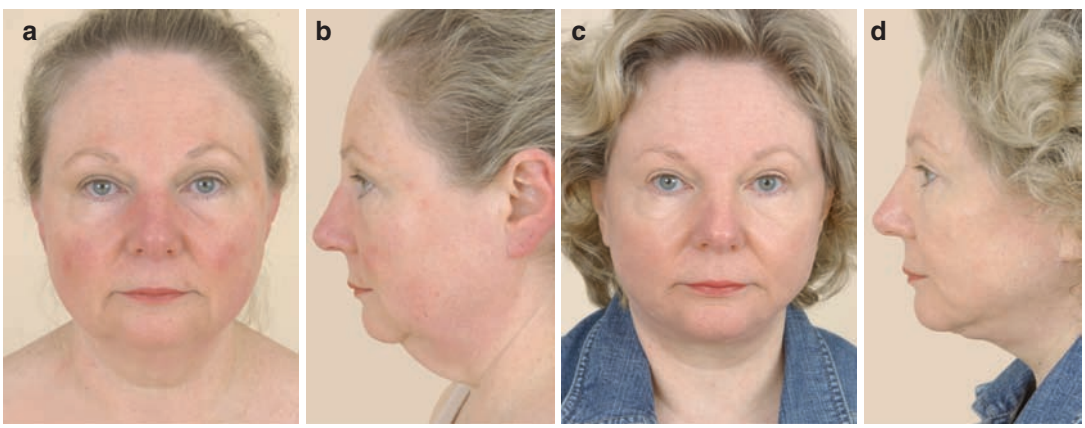

Figure 26.5. A 55-year-old female demonstrating facial aging, an obtuse cervicomental angle, and a loss of jaw line definition preoperative (**a**) frontal and (**b**) profile views. One year following facelift with extended SMAS and aggressive defatting of the submental, submandibular and gonial angle areas, (**c**) frontal and (**d**) profile views.

approaches to neck correction while avoiding a preauricular incision. Knize's[33] approach is through a submental incision only, while the senior author (JEZ) uses a submental and postauricular sulcus incision.[47] Feldman[16] described his submental and postauricular sulcus approach in great detail in his recent textbook.

Conceptually, all of these techniques are extensions of lessons learned from liposuction. Once the skin is separated from the platysma muscle, it has a unique and inherent ability to contract. With proper patient selection, no skin needs to be removed from the neck in neck rejuvenation. As stated by Feldman,[16] there is no "lifting" in this necklift approach. How extensive the skin release needs to be is dictated by the degree of skin laxity. A good rule of thumb is that skin undermining in the neck should be as extensive as it would be if a traditional neck/facelift were being performed. However, a critical difference is that no skin is removed.

After skin undermining has been accomplished, platysma alteration is addressed. Once defatting above the platysma has been performed, the platysma is opened in the midline. Subplatysmal fat is assessed and removed flush with the digastric muscles superiorly and down to thyroid cartilage inferiorly. Rarely is digastric shaving or removal performed. A platysmaplasty of choice is then completed either pants-over-vest or corset variety.[17] Suction drainage is always performed with the suction drain exiting posteriorly. Duration of drainage is dictated both by the amount of drainage and extent of undermining and defatting but is essential if seromas are to be prevented. A chin strap is then used continuously for 5 days and for 2 weeks at night only.

Patient Selection

The ideal patient for a necklift without a preauricular incision is one who is most interested in profile change only (Figure 26.6). There will clearly be no improvement in the mid-face, although the jowl area up to the level of the mandible will be improved. Early on in our experience, this procedure was limited to those with mild and moderate skin laxity. However, there is a distinct subgroup of patients in the elderly population who refuse facelift surgery but are willing to undergo a lesser procedure (see Table 26.1). These patients must, however, clearly

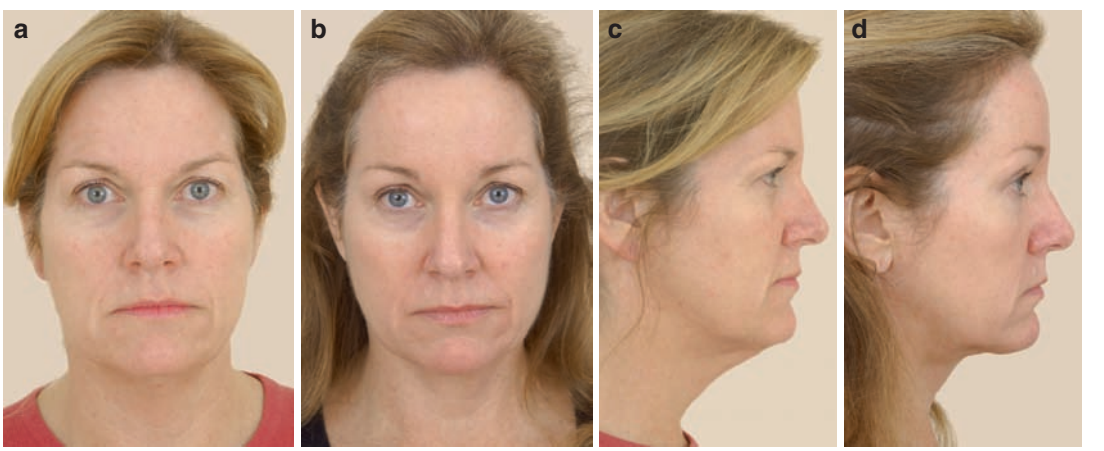

Figure 26.6. (**a**) Frontal and (**b**) profile views of 50-year-old female with obtuse cervicomental angle and neck laxity. (**c** and **d**) Nine months postneck correction with no preauricular incision.

Table 26.1. For Those Who Desire Change in Profile

Age ≥ *40 years*	Liposuction only or liposuction and platysmaplasty if subplatysmal fat suspected
Age 40–70 years	Necklift with no preauricular incision
Age 70 years and greater	Necklift with no preauricular incision or direct neck skin excision and Z-plasty

understand the limitations of the necklift without a preauricular incision. In this population (usually age 70 years and more), the operation will fall short of a traditional face/necklift (Figure 26.7). However, if properly counseled, this is a very appreciative group and can readily be taken on. Therefore, a reasonable paradigm is for those patients desiring predominantly profile changes: (1) liposuction alone in the young group or liposuction plus platysmaplasty if subplatysmal fat is expected, (2) necklift with no preauricular incision in the middle aged, and (3) either necklift with no preauricular incision or necklift with direct excision and Z-plasty in the elderly (Table 26.1, Figure 26.8).

Complications encountered are few and are similar to those seen with either traditional liposuction or open neck/facelift. Hematomas are very rare. Seromas are generally prevented by suction drains, which should always be used to obliterate dead space and eliminate seroma fluid. Drains should be maintained until drainage is minimal. One should not hesitate to maintain the drains for several days, especially in big necks or in necks where significant defatting is performed.

Temporary neuropraxia of the marginal mandibular nerve is relatively common and routinely resolves in 2 weeks. Patients should be warned of this possibility preoperatively.

Figure 26.7. (**a**) Preoperative frontal and (**b**) profile photograph of 73-year-old female with neck laxity and obtuse cervicomental angle. (**c**) Frontal and (**d**) profile views following necklift with submental incision and posterior sulcus incision. No preauricular incision was used.

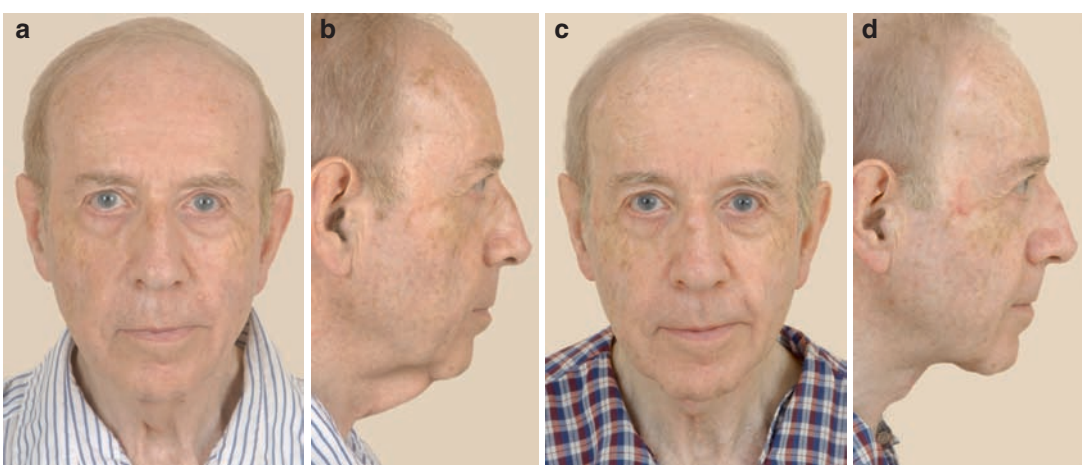

Figure 26.8. A 74-year-old male with significant neck laxity and skin excess in the submental area. (**a**) Frontal and (**b**) profile views. (**c**) Frontal and (**d**) profile views one year following direct neck excision and Z-plasty.

Late complications include visible platysmal banding and contour irregularities. Contour irregularities are minimized by conservative defatting and are most likely to occur in the fat neck. Those patients should be addressed only after significant experience has been gained with the technique. Both result from overly aggressive defatting of the neck and are difficult to treat once they occur.

Direct Necklift

Although frowned upon by many, the direct neck lift by submental vertical skin excision and Z-plasty is a reasonable alternative in the elderly. This includes men with a significant amount of skin excess and laxity who clearly will not have enough skin elasticity to contract adequately postoperatively. These are generally men aged 70 years or older. Women in their 80s with significant skin excess are also readily treated, thus avoiding the morbidity of a traditional facelift.

Patients who fall into this group are the elderly who are unwilling or not healthy enough to undergo a facelift. The procedure has the added benefit that it will often give a better neck contour than a standard facelift because skin is being removed where it is most lax, that is, medially. In the standard facelift approach, these elderly individuals usually have early recurrence or incomplete correction, because lateral pull is at some distance from the area of skin laxity. However, the patients must clearly understand the length and character of the scar before proceeding. Photographs are particularly helpful in this regard.

Although numerous techniques have been described in the literature,[1,9,14,24] we prefer the Gradinger technique.[22]

With the patient in the sitting position preoperatively, a tape measure is run ear to ear to determine the ideal location of the new cervicomental angle and the central limb of the Z-plasty. This is marked by a long horizontal line. The skin is then grasped in the midline, and by pinch, the vertical ellipse is marked (Figure 26.9). Care should be taken not to overestimate skin excision, as this will have a decidedly adverse affect on healing and may result in hypertrophic scarring.

At surgery, the markings are rechecked and the patient injected with 0.5% lidocaine with epinephrine. The vertical ellipse is excised and skin undermined for 4–5 cm superficial to the platysma and then on its deep surface. Subplatysmal defatting is performed as previously described for necklift with no preauricular incision. The platysma is then temporarily closed at the level of the horizontal line. A Z-plasty with limbs of 2.5 cm and angles of 60° is then designed on the platysma so that the central limb once transposed will be on the horizontal line described earlier. Platysmal closure is then completed.

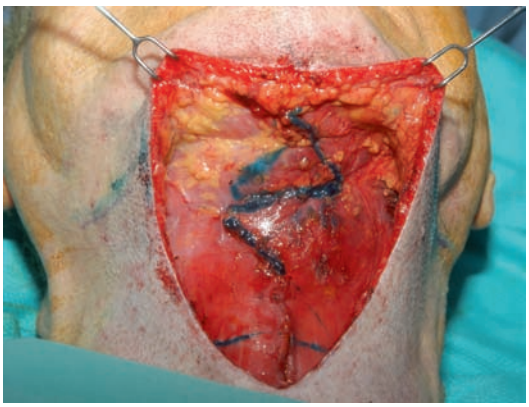

Figure 26.9. Intraoperative view of direct excision of neck skin and Z-plasty. Z-plasty has been completed on muscle, skin will be closed, and a Z-plasty performed.

The skin is then temporarily closed at the level of the new cervicomental crease, an inch above and an inch below. A second Z-plasty on the skin can be done lower down. Scars will go through the usual maturation phases, visible at first but by 1 year are quite acceptable (Figure 26.10). A Z-plasty similar to the Z-plasty described for the platysma is then designed and transposed so that the central limb lies in the new cervicomental crease.

The best patients for this technique are nonobese, elderly individuals with significant skin excess. Fat necks can be problematic with less than ideal postoperative contour. Other complications include mild hypertrophic scarring at 3 months, which resolves with time when it occurs.

The perioral area can present a particularly troublesome area of facial aging, and perioral aging can detract significantly from an otherwise excellent result. Problems peculiar to this area do not generally improve with facelifting

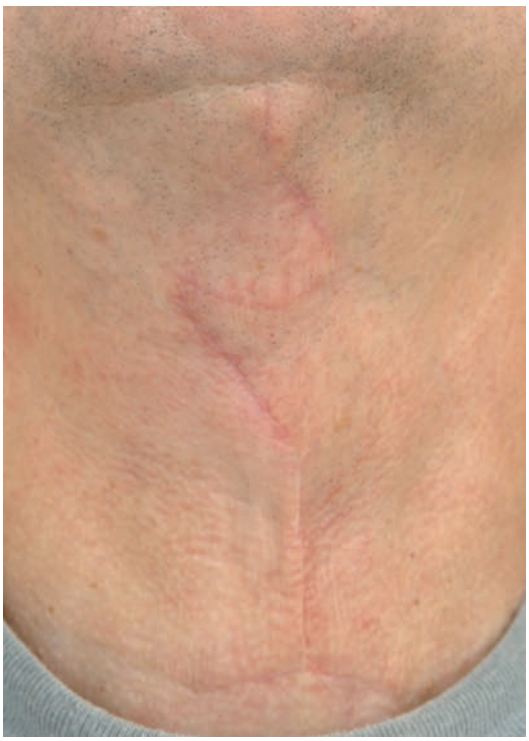

Figure 26.10. Scar from direct neck excision and Z-plasty at 1 year postoperatively.

alone. Causes of facial aging are multifactorial. Bone resorption particularly following tooth loss leads to alveolar ridge flattening and upper lip lengthening. The edentulous patient tends to overclose the mouth, leading to a reduction in lower third vertical facial height.

Inherent changes in the skin including skin laxity and attenuation of the retinaculum cutis lead to lengthening of the upper lip. The combination of loss of subcutaneous fat, thinning of the dermis, and muscle hyperactivity lead to vertical rhytides in the upper and lower lips, especially in thin-skinned, fair-skinned (Fitzpatrick I and II) women. Vertical rhytides tend to be significantly less of a problem in darker, thicker-skinned women (Fitzpatrick III and above) and are never a problem in the male.

The down-turned corners of the mouth again tend to be more prevalent in the thinner-skinned and fairer-skinned women and are generally not effectively addressed by facelift surgery alone. The multifactorial causes and the variety of findings, therefore, require multiple ancillary procedures to effect correction.

Lips

Correction of the aging lips can be accomplished by a variety of surgical or nonsurgical means. Surgical correction of the long upper lip can be performed by crescentric skin excision at the base of the nose[6,15,18,23] or by an advancement of the vermilion-cutaneous junction. In both cases, such patients should exhibit no or minimal, preoperative, upper incisor show. Correction should be performed so that no more than 3–4 mm of upper incisor show is created. This is difficult to judge intraoperatively. Therefore, upper incisor show should be measured preoperatively and 1:1 correction assumed, that is, 1 mm of excision of skin for each millimeter of correction desired (Figure 26.11).

Skin excision extends down to the orbicularis muscle, minimal skin undermining performed and a two-layer closure completed. Generally, 4 mm vertical excision should be the maximum attempted. Sigal[39] provides four lip contours for which crescentric excisions should be avoided in order to prevent untoward results.

Corner-of-the-Lip Lift

The downturned corners of the mouth, although showing significant early satisfactory correction following facelift surgery, also generally exhibit early recurrence postoperatively. This problem, interestingly unlike many perioral aging problems, occurs throughout the Fitzpatrick classifications. Generally, this problem requires a direct approach with local procedures to adequately correct the problem. The corner-of-the-lip lift and its variations have been described in detail by Austin and others.[5] The technique we prefer entails a triangular excision with its base at the commissure and a 12 mm medial limb extension from the commissure along the vermilion, a second limb extending laterally from the commissure toward the top of the ear and, a curvilinear line connecting the two lines. The vertical height of the triangle is 7 or 9 mm depending on the degree of correction needed (Figures 26.12 and 26.13).

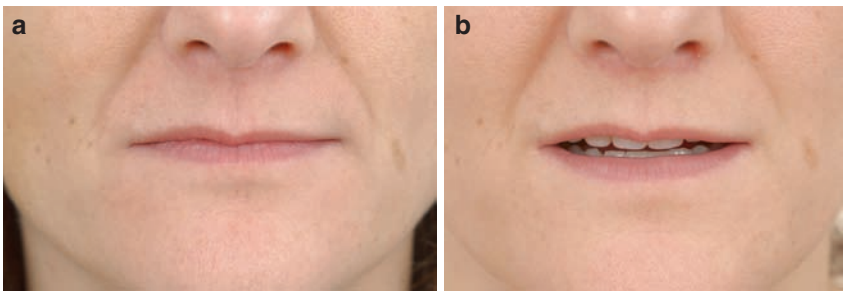

Figure 26.11. A 39-year-old female with vertical maxillary deficiency and a lack of upper incisor show. (**a**) Preoperative frontal view. (**b**) Postoperative view of the same patient following elliptical excision of skin at the alar base with a vertical component of the ellipse measuring 4 mm. Patient now demonstrates 3 mm of upper incisor show and normal lip length.

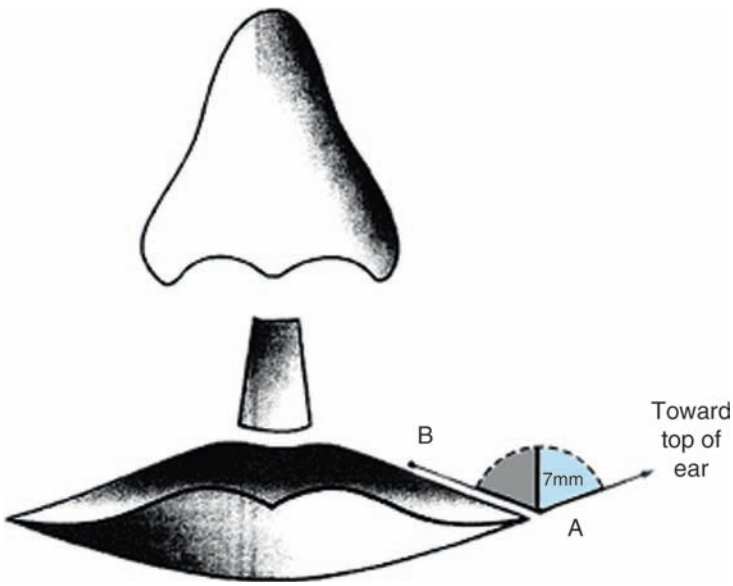

Figure 26.12. Corner lip lift. A triangular skin excision is performed just lateral to the commissure. The base of the triangle is superior. The height of the excision is 7 mm for moderate elevation and 9 mm for maximum elevation.

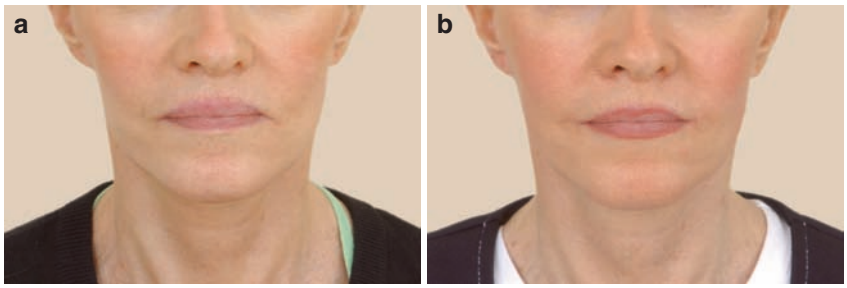

Figure 26.13. (**a**) Preoperative 56-year-old-female who had undergone a previous lip lift with direct excision above the vermilion. She now presents with down-turned corners of the mouth and lip deformity. (**b**) Postoperative photo following corner-of-the-lip lift with correction of downturned corners of the mouth.

Fat Injection

The concept of perceived or real deflation of the skin envelope associated with facial aging has gained a good deal of attention in recent years.[10–12,34] Although the efficacy and long-term maintenance of fat transfers have been claimed by a number of authors, strict outcomes demonstrating long-term volume maintenance are lacking.[32] Despite this, many plastic surgeons have embraced fat transfer both as an isolated procedure or as an adjunct to facelift surgery as a mechanism for volume restoration. Fat transfer as an adjunct to facelift surgery is standard in our unit and is merely considered part of the facelift procedure. As many other plastic surgeons, we have not verified volume maintenance. The technique for fat transfer as espoused by Coleman[10–12] has become the standard procedure. Fat is harvested from the abdomen or thigh using a Coleman aspirating cannula and 10-cc syringe. Centrifugation is performed for 2 min at 3,000 RPM, and fat is loaded into 1-cc syringes after oil (supernatant) is removed. Fat is then injected using multiple passes, multiple levels, and microvolumes.

In reviewing the literature, several conclusions can be drawn with regard to fat transfer: (1) fat appears to demonstrate greater volume maintenance in areas where motion is minimized. Volume maintenance appears to be better in the infraorbital and nasolabial fold areas than in the lips, and (2) volume maintenance varies significantly not only from location to location but also from patient to patient, that is, it is unpredictable.

Clearly, methods to enhance volume maintenance and improve the consistency of fat transfers are needed. Whether this will occur by the identification of appropriate stem cells or by the addition of specific growth factors is unclear at present.

Finally, the list of synthetic fillers continues to expand and will continue to do so in the future. Rather than adjuncts to facelift surgery, fillers tend to be most often used as an office procedure to soften nasolabial folds (hyaluronic acids such as Juvederm, Restylane, Radiesse), correct marionette lines, or fill the nasojugal groove. Sculptra (L-poly lactic acid), initially approved for facial atrophy following HAART therapy for HIV, is finding increasing "off-label" use as a volume enhancer similar to fat transfers in facial aging.

Skin Resurfacing

Skin rejuvenation with minimal to no downtime is the goal of all of the newer lasers, fractionated lasers, and light-based and radiofrequency devices currently or soon to be on the market. Some attempt to do this using a cooling tip to spare the epidermis and affect only the dermis (Syneron Aurora/Elos System), whereas others use a technique that spares areas of the epidermis while heating others in a defined pattern (fractionated erbium or fractionated CO_2).

Finally, radiofrequency alone (Thermage) or in combination with intense pulse light is being used (Syneron Elos). Each has its advocates. At present, rosacea, redness, and dyschromasia appear to be well treated, whereas deep rhytides appear to be more recalcitrant to treatment.

Although deep rhytides in the perioral area, the cheeks, and eyelid areas respond consistently to more aggressive peeling techniques such as CO_2 lasering, phenol–croton oil peeling, and dermabrasion, these treatments have fallen into disfavor because of their relatively prolonged downtime and risk of hypopigmentation. However, for eradicating deeper rhytides, these treatments are unsurpassed. In addition, when deeper peeling is performed as an adjunct to a facelift procedure, downtime in most cases is not prolonged beyond that of the facelift itself. In our unit we have extensive experience with both CO_2 lasering and phenol–croton oil peeling techniques. Recent writings have dispelled certain precepts regarding phenol–croton oil peeling and have reduced phenol and croton-oil concentrations routinely used.[20,27–30,41,46] If these recommendations are followed, results with phenol–croton oil in particular are unsurpassed (Figure 26.14).

Although a thorough review of this subject is beyond the scope of this chapter, several important points deserve emphasis: (1) although phenol–croton oil peeling can be done at the time of facelift surgery, if areas are not undermined (perioral area, eyelids), areas of undermining during the facelift are never peeled simultaneously, (2) increasing concentrations of phenol and croton oil increase the depth of injury, and (3) areas of thinner dermis are peeled with lesser concentrations of phenol and croton oil.

Not only will phenol–croton oil peeling of the perioral area eradicate deep rhytides at the time

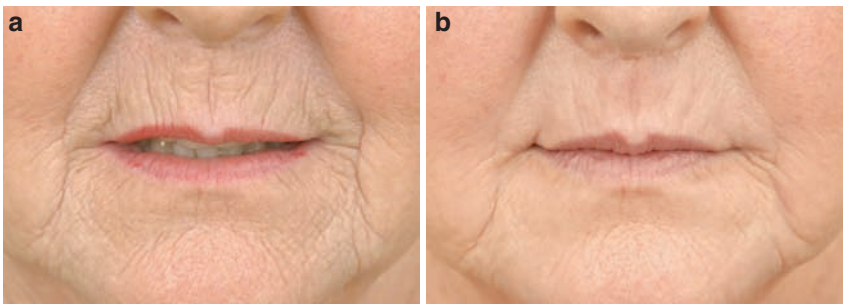

Figure 26.14. (**a**) Preoperative 63-year-old female with deep perioral rhytides. (**b**) One year postoperative photograph following phenol–croton oil peel using 33% phenol, 1.1% croton oil, and shortening of the upper lip by skin excision.

Figure 26.15. (**a**) Preoperative view of 63-year-old female with facial aging and perioral rhytides. (**b**) One year follow-up following facelift with extended SMAS and perioral phenol–croton oil peel demonstrating the synergistic affect of facelift combined with perioral phenol–croton oil peel for improvement in the lower face.

of facelift surgery, but the combination of the two procedures has an, as of yet, unappreciated dramatic tightening affect on the nasolabial fold region (Figure 26.15).

In addition, dilute concentrations of phenol and croton oil have a decidedly beneficial effect on the lower lids (Figure 26.16). Phenol–croton oil peeling of the lower lids can be performed using a variety of regimens. The peel can be done secondary to enhance the overall results from lower lid blepharoplasty. Specifically, the peel will significantly improve fine lines and wrinkles. Alternatively, the phenol–croton oil peel can be performed at the same time as either transconjunctival or pinch blepharoplasty.[20,46]

In conclusion, we have attempted to provide a conceptual approach to facelift surgery, emphasizing the precepts on which modern facelift surgery is based. An understanding of the three-dimensional anatomy of the face is critical to safe facelift surgery and has been emphasized.

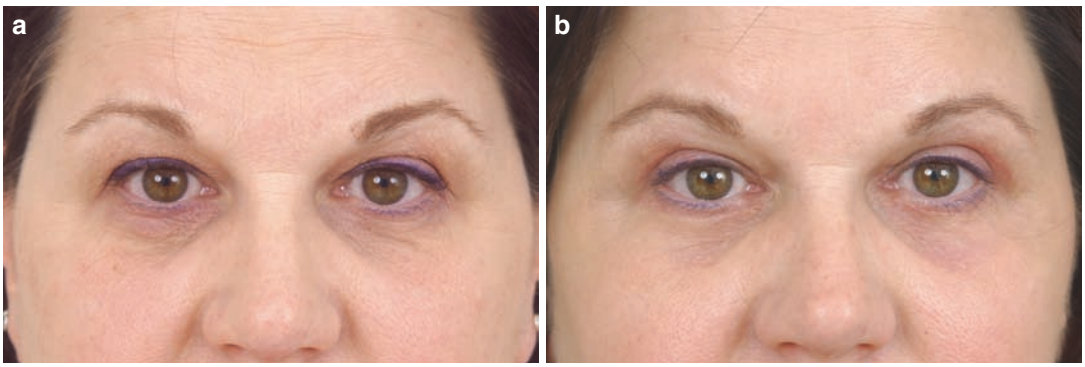

Figure 26.16. (**a**) Preoperative view of 51-year-old female with lower lid rhytides and hyperpigmentation. (**b**) Six-month postoperative view of patient following pinch blepharoplasty and phenol–croton oil peel of the lower lids using 27% phenol, 0.01% croton oil.

Ancillary procedures that can be used to address areas recalcitrant to the facelift procedure alone have also been outlined. Finally, several alternatives or lesser procedures that can be used in appropriate patients in lieu of a facelift with gratifying results and less morbidity have been described.

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27

Treatment of Headaches with Plastic Surgery

Devra Becker and Bahman Guyuron

Summary

Headaches affect millions of people a year and are associated with a great deal of morbidity. Though the causes of headaches are many, a subset of headaches refractory to traditional pharmacologic treatment – namely migraine and cluster headaches – can be treated surgically. All patients should undergo a thorough history and physical examination and evaluation by a headache specialist. Patients who are diagnosed with migraine headaches have potential trigger sites identified by consideration of the constellation of symptoms, intranasal examination, CT scan, and by injection with 12.5 U of botulinum toxin A for each muscle involved. The use of botulinum toxin A as definitive treatment for headaches remains controversial; we use it as a diagnostic aid. Glabellar trigger sites are treated with corrugator supercilii resection, temporal trigger sites are treated with resection of the zygomaticotemporal branch of the trigeminal nerve (ZMTBTN), occipital trigger sites are treated with surgical release of the greater occipital nerve, and patients with intranasal triggers are treated with septoplasty and inferior turbinate resection. Patients with cluster headaches are treated with resection of the ZMTBTN. Surgery does have a role in the treatment of refractory migraine and cluster headaches.

Abbreviations

GMG	Glabellar muscle group
MH	Migraine headaches
ZMTBTN	Zygomaticotemporal branch of the trigeminal nerve

General Epidemiology and Classification of Headaches

Headaches affect millions of people a year, and some have estimated the overall prevalence of headaches to be 46%.[28] It is a common symptom seen in neurological practices, and indeed entire journals and textbooks are devoted to the diagnosis and management of headaches. The treatment of headaches with plastic surgery has been a topic of considerable interest and has been the subject of prior publications.[35] Descriptions of headache date back to several millennia BCE, and detailed incantations about headaches, including proposed treatments, have been described in Babylonian literature in Ur III incantations[30]. Because headaches affect so many people and can be caused by many different factors, it is appropriate to begin with a review of the classification of headaches and specific descriptions of migraine headache (MH) and cluster headache, which are the only headaches for which we perform surgical intervention at this time. We describe the current understanding of pathophysiology that underlies the basis

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for surgical intervention, the treatment algorithm, and the surgical techniques appropriate in MH and cluster headaches.

Classification

In most general terms, headaches can be classified as either primary or secondary. Primary headaches are those that are not associated with another, distinct disease process, whereas secondary headaches are so associated. Headaches are classified by the International Headache Society in the International Classification of Headache Disorders.[15] Though there are no fewer than 45 WHO ICD-10 codes for primary headaches, it is helpful to think of them in four general categories: migraine, cluster/trigeminal autonomic cephalalgia, tension type, and other. A surgeon treating patients with complaints of headache should have a general understanding of the presentation and treatment of the three most common types (migraine, cluster, and tension headaches) in order to distinguish them and identify appropriate surgical candidates. All patients presenting to a plastic surgeon for surgical treatment of headaches, however, should be evaluated by a neurologist or headache specialist.

Migraine

Migraine headaches are a debilitating condition that affects tens of millions of Americans and costs over $7,000/year per patient of medical treatment, as reported in a 2005 study.[9] It causes worldwide loss of productivity – commonly cited MH statistics are that the lifetime prevalence is between 11% and 32% in several different countries[1,9,11,14,21,26], and it is in the World Health Organization's top 20 diseases causing disability.[16,17,34]

The International Headache Society created and updates diagnostic criteria for MH. The diagnostic criteria of MH include "A. At least five attacks meeting B-D. B. Headaches lasting 4–72 hours (untreated or unsuccessfully treated). C. headaches have at least two of the following characteristics/ unilateral location, pulsating quality, moderate or severe pain intensity, aggravation by or causing avoidance of routine physical activity (for example walking or climbing stairs). D. During headache at least one of the following: nausea and/or vomiting, photophobia and phonophobia. E. Not attributable to any other disorder."[15] In addition to these criteria, MH can be associated with auras (although aura is not necessary for the diagnosis). Auras develop over 5–20 min, last less than an hour, and are followed by a migraine. Frontotemporal location, unilateral symptoms, and pain associated with nausea and photophobia are typical of both MH with aura and MH without aura.

Medical management of MH has relied on behavioral changes, such as avoidance of caffeine, alcohol, or tobacco, as well as prophylactic (beta blockers, tricyclic antidepressives, and valproic acid), abortive (triptans), and analgesic (benzodiazepines, opioids, barbiturates, and OTC analgesics) pharmacologic intervention.[19]

Tension Headaches

Tension headaches are the most common form of headaches and have a wide variation in reported prevalence (30–78%).[15] They can be episodic or chronic and are characterized by a duration of hours to days, mild to moderate intensity, and bilaterality. They are not affected by activity and are not associated with nausea. These distinguishing features are important; tension headaches can easily be confused with MH without aura,[15] but treatment is manifestly different.

Nonpharmacologic treatment of tension headaches includes relaxation and physical therapy. Acute treatment consists of non-narcotic analgesia, and chronic treatment consists of tricyclic antidepressants.[20]

Cluster Headaches

Cluster headaches affect the orbital, supraorbital, or temporal regions exclusively unilaterally and are associated with severe pain, unilateral autonomic disturbances, such as conjunctival injection, lacrimation, nasal congestion, rhinorrhea, forehead and facial sweating, miosis, ptosis, and eyelid edema.[15] During an attack, the headaches occur once every other day up to eight times a day. In contrast to MH, patients with cluster headaches are typically restless and agitated.

Nonpharmacologic treatment consists of avoidance of ethanol, histamine, nitroglycerine, or tobacco. Abortive treatment consists of 100% oxygen, triptans, cafergot, and dihydroergotamine. Prophylactic treatment consists of verapamil, lithium, methysergide, ergotamine tartrate, and prednisone taper.[18]

Pathophysiology

Pathophysiology of MH

MH pathogenesis is a complex interaction of a number of processes. Patients who are susceptible to MH have static and dynamic central nervous system changes and peripheral nerve changes (specifically, the trigeminal nerve). *Interictal cortical derangements* are present in the form of cortical neuronal hyperexcitability. *Periaqueductal gray matter*, an antinociceptive modulator, undergoes progressive derangements. This is important because experimental evidence has shown that sensitized nociceptors increase neuronal activity, and this causes increased sensitivity to both painful and nonpainful stimuli.[3] *Cortical spreading depression* is believed to be the mechanism of aura pathogenesis. In addition, cortical spreading depression can cause irritation of the trigeminal nerve nucleus caudus.[32] The trigeminal nerve releases a number of substances, including substance P, calcitonin gene-related peptide, and neurokinin A in response to irritation.[8,23,32] This *trigeminal nerve irritation* is another factor in MH pathogenesis. The substances that are released in the cell bodies of the trigeminal nerve travel and cause localized meningitis and dilation of trigeminal nerve-innervated vessels and dura mater.[6,9,11,25,32] This dilation is thought to cause MH.

Peripheral branches of the trigeminal nerve cross muscle at defined places. Contraction of the muscle in these places causes the nerve to become irritated. For example, the trunk of the supratrochlear nerve and branches of the supraorbital nerve traverse the depressor supercilii and the corrugator supercilii. Branches of the ZMTBTN pierce the temporalis muscle. The greater occipital nerve pierces the semispinalis capitus muscle.[5,24] Because the headaches originate at defined anatomic places, this is known as the *trigger site hypothesis* of migraine pathogenesis.

Pathophysiology of Cluster Headaches

The pathophysiology of cluster headaches is also associated with abnormalities of the trigeminal nerve, although cluster headaches lack a central nervous system component.[31] In addition, parasympathetic outflow through the facial nerve causes autonomic disturbances.

Seasonal variation, as well as the regularity of headaches during an attack, have led some to investigate the role of circadian rhythms in cluster headaches.[22] In fact, melatonin disturbances, specifically a decrease in peak nocturnal melatonin levels, have been found in cluster headache patients.

Surgical Treatment

History of Surgical Treatment

Surgery for MH is not new. In the first half of the twentieth century, Dandy reported removing the inferior cervical and first thoracic sympathetic ganglions for MH.[4] Over the course of that century, others reported surgeries designed to eliminate headache through the resection of nerves.[7] Because these procedures were associated with a high degree of morbidity, they were not widely adopted.

However, these theories of migraine pathogenesis suggest that surgically addressing the nerves peripherally at their trigger points will have beneficial outcomes. This avoids the invasive and relatively extreme procedures of the previous century while still eliminating a source of MH.

Thus, frontal MH can be treated by resection of the muscles of the glabellar muscle group (GMG, the corrugator supercilii, depressor supercilii, and procerus.) Temporal headaches can be treated with avulsion of the ZMTBTN, a nerve that has been reported to be transected during facial rejuvenation.[9]

Identifying Surgical Candidates

Surgical candidates for MH surgery have the following characteristics: they have been evaluated thoroughly for their headaches and diagnosed as MH refractory to medical management or cluster headaches refractory to medical management, other organic causes of headaches have been excluded, and they pose an acceptable surgical risk. Other factors that help identify those likely to benefit for surgery are patients with corrugator hypertrophy and patients who have evidence of septal deviation,

turbinate hypertrophy, or concha bullosa on direct and indirect nasal endoscopic examination and CT scan.

Migraine Headaches

Trigger Sites and Botulinum Toxin A

Frontal triggers, which include the glabellar muscles, cause frontal headaches. Temporal triggers, which include the temporalis muscle cause temporal headaches. Occipital triggers, which include the semispinatus capitus cause occipital headaches. Septonasal triggers, which include intranasal structures, cause paranasal and retrobulbar headaches.[5,12,24]

Botulinum toxin A (Botox, Allergan, Irvine, CA) use as long-term treatment for MH has not reached widespread acceptance and is still considered investigational.[2,27,33] We use botulinum toxin A to confirm suspected trigger sites in conjunction with a headache diary that patients keep both before and after injection. We inject 12.5 U of botulinum toxin A with a long 30-ga needle into the most likely trigger sites. They are injected one month apart, up to three. After injection, patients are instructed to avoid prophylactic migraine medication.

A response to botulinum toxin is operationally defined as a 50% reduction in headache intensity or frequency from baseline for 4 weeks.[9] The algorithm of botulinum toxin injection is shown in Figure 27.1.

Botulinum toxin A is considered relatively safe in this clinical setting. The most common complication of this use is a temporary temporal muscle atrophy.[10]

Endoscopic Approach for Frontal and Temporal Triggers

Patients who have glabellar muscle (including corrugator and depressor supercilii and procerus) trigger sites are considered to have frontal triggers, and patients who have temporal muscle trigger sites are considered to have temporal triggers. The endoscopic approach allows the surgeon to remove the glabellar muscles, which eliminates the point of irritation of the supratrochlear and supraorbital nerves[13] as well as avulses the ZMTBTN as it pierces the deep temporal fascia.

The patient is prepped and draped, and incisions are marked. There are five total incisions: one midline incision, and two on either temple, approximately 7 and 10 cm from midline. The non-hair-bearing frontal and temporal regions are injected with 1% lidocaine with 1:100,000 epinephrine, and the hair-bearing regions are injected with 0.5% lidocaine with 1:200,000 epinephrine. The lateral incisions are made with a scalpel and deepened with a Baby Metzenbaum Scissors to the deep temporal fascia. A periosteal elevator is used to dissect in that plane to the more medial ports, which are then incised over the periosteal elevator. The central incision is carried down to bone. Endoscopic access devices (EAD, Applied Medical Technology, Cleveland, OH) are placed in each port site. The dissection continues with a periosteal elevator in the subperiosteal plane to the supraorbital and lateral orbital rims and the zygomatic and malar arches. The dissection is then continued immediately superficial to the deep temporal fascia until the ZMTBTN is seen approximately 17 mm (right) or 16.8 mm (left) lateral to the lateral canthus and 6.6 mm (right) or 6.4 mm (left) superior to the lateral canthus.[29] The ZMTBTN is more vertically oriented than the zygomaticofacial branch of the trigeminal nerve (Figure 27.2). Once seen and dissected, it is grasped with a long curved hemostat and avulsed. The periosteum and arcus marginalis are released on the lateral orbit with a periosteal elevator. A rent in the fascia is made just above the zygoma to harvest fat for the glabellar area.

Next, attention is turned to the glabellar area. At the level of the supraorbital rim, the periosteal elevator is used to pierce the periosteum and expose the corrugator muscle and supraorbital and supratrochlear nerves. The central area of the periosteum over the corrugator is left intact. Care is taken to preserve the nerves while taking out the corrugator, piecemeal, with grasping forceps. After resection, the harvested fat is placed at the corrugator site to prevent a contour deformity. Fascial sutures of 3-0 polydioxanone are placed for resuspension, and a drain is placed. The incisions are closed with 5-0 polygalactin (Vicryl) and 5-0 plain catgut.[11]

The most common complications of the endoscopic approach are paresthesias or anesthesia of the scalp. These are usually transient. In addition, temporary alopecia can occur around the port sites; we try to minimize this complication

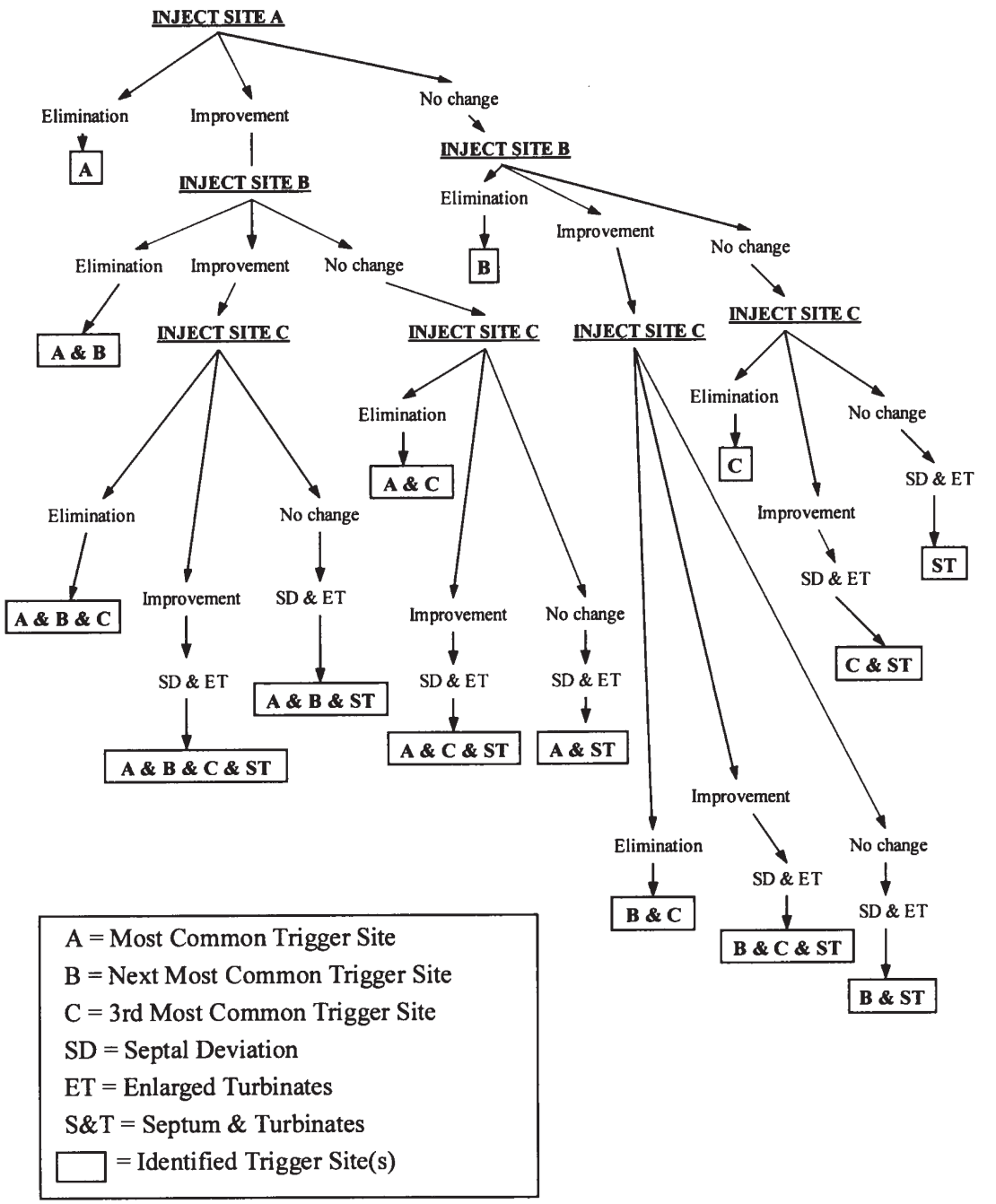

Figure 27.1. Algorithm for approach to botulinum toxin A injection. (Reprinted with permission from Guyuron et al.,[9] January 2005.)

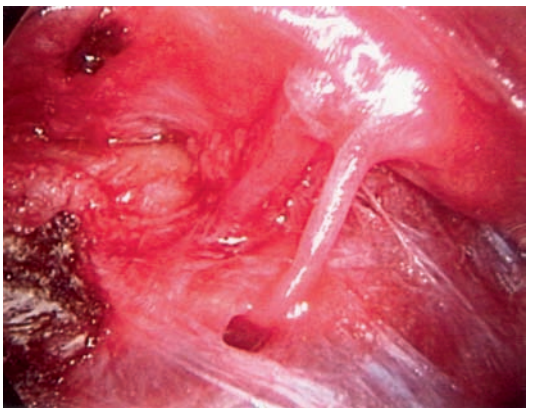

Figure 27.2. Appearance of the ZMTBTN under endoscopic visualization.

by injecting only lidocaine with 1:200,00 epinephrine in the hair-bearing scalp.

Occipital Triggers

The incision is planned in the caudal occipital region, approximately 4 cm in length. After induction of anesthesia, the patient is placed in the prone position, and the incision site is infiltrated with 1% lidocaine with 1:200,000 epinephrine. The incision is made with a 10 blade and carried to the midline raphe using electrocautery. The trapezius fascia is incised to the right of the midline, and the semispinalis muscle fibers are identified. They are distinguished from the trapezius fibers, because they are vertical rather than oblique. If trapezius fibers are in the field, they are retracted. The dissection is subfascial and superficial to muscle until the trunk of the greater occipital nerve is identified approximately 1.5 cm from the midline and 3 cm caudal to the occipital protuberance. The semispinalis muscle is isolated medial to the nerve, and a 1-in. swath is transected. The nerve is then traced to ensure that no fascial bands remain as points of compression. A fascial flap is placed under the nerve, and a drain is placed. The incision is then repaired in layers with 5-0 polygalactin (Vicryl) and 5-0 plain catgut.

Septonasal Triggers

After induction of general anesthesia, the patient is prepped and draped, and the nose is prepared for surgery with cocaine-soaked gauze and two rounds of local anesthesia injection, first with 0.5% lidocaine with 1:200,000 epinephrine and then with 0.5% lidocaine with 1:100,000 epinephrine. An L-shaped incision is made on the left septal mucoperiosteum, and the mucoperiosteum is elevated. Areas of septal deviation and bony spurs are removed. The mucoperiosteal flaps are repaired with 5-0 chromic and running quilting sutures. Doyle splints are placed and fixated with 5-0 prolene sutures.

If the turbinates require resection, the inferior turbinates are resected with turbinate scissors, and the middle turbinates are resected after the overlying mucosa is dissected off the turbinates.

Cluster Headaches

The endoscopic approach as described here to transect the ZMTBTN is our approach for refractory cluster headaches.

Conclusions/Key Points

Patients with refractory MH or cluster headaches may be candidates for surgery. Surgical evaluation consists of assessing response to botulinum toxin A injected into trigger points as well as thorough history and physical examination, including nasal endoscopy and CT scan. Patients with frontal trigger sites responsive to botulinum toxin A can benefit from corrugator supercilii resection. Patients with temporal trigger sites can benefit from avulsion of the ZMTBTN, those with occipital trigger sites can benefit from resection of the semispinalis capitus muscle, and those with intranasal trigger sites can benefit from septoplasty and turbinectomy.

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28

Facial Reanimation

Manfred Frey

Summary

This overview on the most effective reconstructive techniques for reanimation of the unilaterally or bilaterally paralyzed face includes all the important techniques of neuromuscular reconstruction as well as of supplementing static procedures, which contribute significantly to the efficiency and quality of the functional overall result. Attention is paid to the best indications at the best time since onset of the facial palsy, dependent on the age of the patient, the cause of the lesion, and the compliance of the patient for a long-lasting and complex rehabilitation program. Immediate neuromuscular reconstruction of mimic function is favorable by nerve suture or nerve grafting of the facial nerve, or by using the contralateral healthy facial nerve via cross-face nerve grafting as long as the time since onset of the irreversible palsy is short enough to get the paralyzed mimic muscles still reinnervated. For the most frequent indication, the unilateral irreversible and complete palsy, a three-stage concept is described including cross-face nerve grafting, free functional gracilis muscle transplantation, and several supplementary procedures. In patients with limited life expectancy, transposition of the masseteric muscles is favored. Bilateral facial palsy is treated by bilateral free gracilis muscle transplantation with the masseteric nerve branches for motor reinnervation. Functional upgrading in incomplete lesions is achieved by cross-face nerve grafting with distal end-to-side neurorrhaphy or by functional muscle transplantation with ipsilateral facial nerve supply.

Introduction

The functional outcome of reconstructive procedures for reanimation of the paralyzed face has been improved significantly by the introduction of three newer concepts during the last 30 years: First, the replacement of not only the nerve but also of the mimic muscles by muscle transposition[2] or free functional muscle transplantation;[12] second, the use of the contralateral healthy nerve for reinnervation on the paralyzed side;[1] and third, the most recent option to obtain reinnervation by getting regenerating nerve fibers out or putting them into a healthy or partially functioning facial nerve branch without any functional loss for the donor nerve branches.[10]

Besides these changes in operative concepts and the development of new operative possibilities, objective evaluation of the results achieved has been improving a lot during the last years.[18] Comparing the results of different operative techniques for the same indication among own patients together with comparing the results with those of other centers for the same technique or for other techniques for the same indication has been leading to an algorithm for treatment of

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the patient with facial paralysis in its specific clinical manifestation. As a result, it is not one operative procedure that is applied in a patient but usually a combination of procedures applied in the same operation or subsequently.[4] In the following review on facial reanimation, I am trying to line out our actual algorithm for treatment of unilateral or bilateral, of complete or incomplete, of recently acquired or longstanding facial paralysis. This algorithm is reflected in the structure of this chapter with its different sections.

Diagnostics, Indications, and Planning

One of the most important presuppositions of staring surgical reanimation of the paralyzed face is the proof of the *irreversibility of the paralysis*. That is easy in cases in which there is a clear cause of the deficit with no chance of spontaneous recovery. Planning of reconstruction of the facial nerve and its branches should be done as soon as possible to offer reinnervation by the regenerating facial nerve fibers to the paralyzed mimic muscles before irreversible muscle atrophy takes place. The functional recovery achieved will depend directly on the delay of reconstruction. Therefore, nerve reconstruction alone makes sense, only if the facial muscles have not been paralyzed for longer than 6 months, and cross-face nerve grafting is used for not longer than 12 months and reinnervation is achieved by an ipsilateral nerve source. Irreversibility of a postoperative lesion is best proved by the exact operative notes of the surgeon causing the lesion. Electromyograms from the facial muscles 6 weeks after the lesion and repeating 3 and 6 months after the onset of the paralysis are not only necessary for documentation but are also very helpful to monitor the process of reinnervation. Together with the clinical evaluation, they are the basis to fix the correct time for starting surgical reconstruction. On the one hand, every chance should be given to spontaneous recovery, and on the other hand, nerve reconstruction should be performed as soon as possible to find the facial muscles still suitable for reinnervation with a clinically relevant functional result. In the case of late reconstruction, it is important not to try nerve reconstruction alone and not to lose the motivation of the patient by a predictable unsatisfying result but to plan nerve reconstruction and reconstruction of the muscle target as well from the beginning. Similar aspects are relevant for posttraumatic paralysis. Extratemporal lacerations are best identified clinically before wound closure. Intratemporal lesions of the facial nerve are mostly accompanied by fractures of the skull base. CT scans localize the lesion by localizing the fracture, but a fracture line or dislocated bone fragments should not be an indication for decompression of the facial nerve along its bony channel. All attempts of acute operative decompression of the facial nerve have not shown any benefit for the final outcome and should not be performed anymore. Intracranial lesions by bleedings and hematomas lead necessarily to acute neurosurgical intervention.

In idiopathic lesions, operative efforts should not be started before conservative treatment has been applied for 1 year at minimum, and there is absolutely no functional recovery either clinically or electrophysiologically. Reconstruction should be postponed as long as a dynamic is observed for the functional recovery. If there is stagnation for several months and the mimic function is significantly deficient, additional functional reconstruction has to be considered. In the majority of cases, this decision is appropriate 1.5 years after onset of the paralysis.

Congenital or obstetrical facial palsies need principally operative construction of the nerve and muscle system in the face. Starting with surgery usually at the age of 4 years, there is no question that the palsy is irreversible and muscles are missing too.

Immediate Neuromuscular Reconstruction of Mimic Function

Reinnervation of Paralyzed Mimic Muscles

Nerve Suture and Nerve Grafting of the Facial Nerve

With extratemporal lesions, we usually find open wounds, which need exact clinical examination before anesthesia. By this, the pattern of severed branches becomes evident, and divided branches of the facial nerve can be reconstructed primarily by direct nerve suture or nerve grafting at the

time of wound closure. Early anatomical reconstruction of the peripheral branches gives by far the best result. Secondary reconstruction involves nerve grafting and misses the chance of direct suture of a cleancut facial nerve branch.

Unfortunately, extratemporal facial nerve defects created by tumor surgery are rarely reconstructed at the time of tumor resection. Delayed secondary reconstruction has two significant disadvantages: First, identification of the proximal and distal nerve stumps is much more difficult within the scarred tissues, and second, the delay of reinnervation of the paralyzed mimic muscles reduces the functional result obtainable. Therefore, immediate reconstruction should be considered before tumor surgery. The only argument for postponing the nerve reconstruction is the necessity of adjuvant chemo- or irradiation therapy. In this case, immediate reconstruction should be limited to simpler procedures, which, for example, guarantee the immediate protection of the cornea. If healing of the tumor disease is finally achieved using irradiation therapy, reanimation of the paralyzed side might involve the contralateral facial nerve by cross-face nerve grafting.

Foreign Nerves for Reinnervation

Foreign motor nerves, such as the hypoglossal, the masseteric, or the accessory nerves, appear attractive, if a recent facial nerve lesion cannot be reconstructed with the ipsilateral facial nerve. The advantage of a strong motor reinnervation is compensated by far by the disadvantage of the missing facial nerve control of the recovered movements of the mimic muscles. Personally, I prefer reinnervation by cross-face nerve grafting straightforward in this situation. The still very popular use of connecting the hypoglossal nerve with the facial nerve more than 1 year after the onset of the facial paralysis should be definitely abandoned, because no clinically relevant result can be expected and the patient will not be motivated for further surgery, when he or she has already been disappointed by an operation and a long waiting period for a functional result, which will realistically never show.

Cross-Face Nerve Grafting

Cross-face nerve grafting is playing an increasingly important role in recently acquired as well as in longstanding irreversible facial paralysis. Whereas in longstanding palsy, cross-face nerve grafting is the first step procedure preparing for the second step procedure of reconstruction of the muscle target, which is usually done by free muscle transplantation, in irreversible lesions not older than 6 months, we try to reinnervate the paralyzed muscles via the cross-face nerve grafts. The distal end of the nerve graft is then sutured to the distal paralyzed facial nerve branches, but the nerve grafts are kept in overlength. Therefore, the change from the concept of reinnervation to the concept of additional muscle transplantation becomes possible in the case of an unsatisfactory result 1 year after cross-face nerve grafting, without any disadvantage for the patient.

Functional Free Muscle Transplantation

Sometimes trauma or tumor surgery destroys mimic muscles locally, and functional deficit is caused by the missing muscle target. This situation is a good indication for free functional muscle transplantation. We prefer the use of the gracilis muscle to reconstruct the smile function or as a territorially differentiated gracilis muscle transplant, if the eye closure function has to be reconstructed as well. In tumor cases, the free tissue transfer should cover not only the soft tissue defect, but the possibility of a functional free myocutaneous flap should be considered preoperatively, if the facial nerve is likely to be involved in the tumor resection.

Reanimation in the Unilateral, Irreversible Facial Palsy

Cross-Face Nerve Grafting and Free Muscle Transplantation

The idea of combining reinnervation by the contralateral healthy facial nerve through a cross-face nerve graft with a new muscle for reanimation in an irreversible, long-standing facial palsy was considered first by Freilinger.[2] Drawbacks of this new technique were the technical difficulties to completely denervate the temporalis muscle before offering the regenerated facial nerve fibers for reinnervation. One year later, Harii and group published first experiences with the combination of cross-face nerve

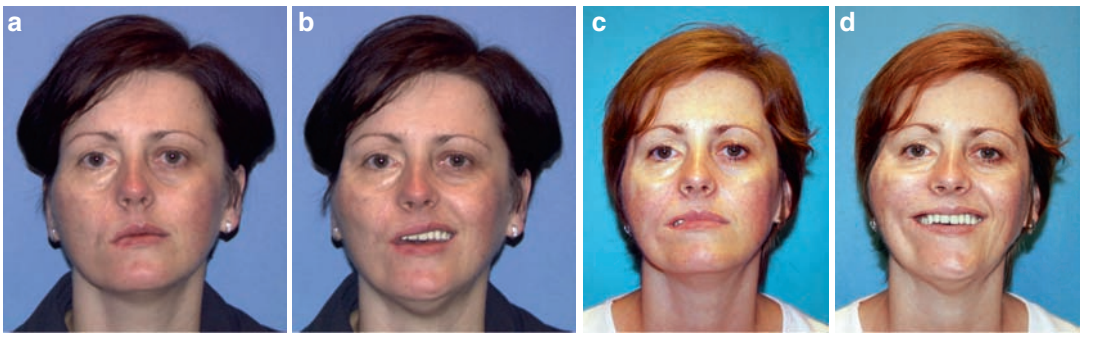

Figure 28.1. This 38-year-old patient suffered from total complete idiopathic facial palsy on her right, at the age of 16 years. The reanimation of the face was carried out by transposing a part of the temporalis muscle to the eye, cross-face nerve grafting, followed by free gracilis muscle transplantation for smile reconstruction and reconstruction of the nasolabial fold. The first two images (**a, b**) show the patient preoperatively in resting position and smiling with showing teeth, respectively (**c, d**) 18 months postoperative.

grafting and free gracilis muscle transplantation with microneurovascular anastomoses.[12] Today, this concept has been accepted as one with the best results of reconstruction of mimic movements with satisfying amplitude and with emotional control (Figure 28.1).

Although other muscles have been proposed for reconstruction of the smile, the gracilis muscle is still the favorite for reconstruction of the mimic function. The advantages of this muscle lie in its anatomy with parallel muscle fibers and a constant neurovascular supply with diameters of 1 mm or more of the vessels, which makes the microsurgical part safe. The muscle fibers themselves show long amplitude of contraction even when reduced in length. If we consider the overall loss of function, this is more important not only for the force but also for the amplitude of the contraction. Especially in the face, the amplitude of the contracting muscle graft is more important than its force. A functional deficit in the donor area has never been reported by the patients. The longitudinal scar on the medial aspect of the medial thigh is well hidden but should be kept as short as possible.

Although a one-stage transfer of a muscle with a long motor nerve branch reaching the healthy facial nerve branches on the contralateral side has several attractive aspects,[13–17] we still prefer the two-stage procedure with the need for cross-face nerve grafting at first.[4] Owing to this, problems in reaching the other side by a too short muscle nerve and to be limited in positioning of the muscle graft are prevented. In addition, the use of the gracilis muscle as a territorially differentiated muscle graft for functional reconstruction around the eye and the mouth at the same time and anastomoses of the vascular pedicle of the muscle graft to the superficial temporal vessels are only possible in the two-stage transfer.[3]

In the unilateral palsy, free functional gracilis muscle transplantation is most often combined with cross-facial nerve grafting. In the majority of cases, reconstruction by free gracilis-muscle transplantation is concentrated on the smile function, whereas closure of the eye is achieved by transposing a part of the temporalis muscle to the upper and lower eyelid. If the latter possibility is no longer available, the gracilis muscle is transplanted by its two functional territories to the eye and the mouth, which are connected to two separate cross-face nerve grafts, one bringing facial nerve fibers from a temporal branch of the healthy side responsible for eye closure and the other from a zygomatic branch responsible for smile function.[9] We are using this technique in children as a principle not to interfere with the growth of the facial skeleton and to take advantage of the better central reorganization in children.

Preferably the sural nerves are used for cross-face nerve grafting. Harvesting is easy over a long distance using a nerve stripper and exposure through three small transverse incisions: One between the lateral malleolus and the Achilles tendon, one in the middle of the calf to separate a branch hindering the stripping process, and a third one on the level of the fibula head. The branches of the healthy facial nerve

are exposed by a facelift incision alone. Blunt dissection creates a subcutaneous tunnel between the two preauricular incisions; only a small incision on the floor of the nose is used in between, to introduce a single or two sural nerve grafts in reverse position. End-to-end epi-perineural nerve sutures are performed under the microscope. The healthy facial nerve branches are selected by intraoperative electrostimulation and divided as peripherally as possible to be most selective with the function to be reconstructed.[8] 10-0 nylon sutures are used for coaptation of the nerve ends. The free ends of the nerve grafts are fixed to the parotid fascia near to the tragus by nonabsorbable sutures. Therefore, the distal ends of the cross-face nerve grafts are easily reexposed and identified, when muscle transplantation with microneurovascular anastomoses is performed 8–10 months later. This interval is necessary to give enough time for regeneration of the facial nerve fibers over the long distance from the healthy to the paralyzed side of the face.

As the donor site, we choose the thigh ipsilateral to the paralyzed side of the face. This way, the neurovascular pedicle will be positioned under the gracilis muscle graft and the muscle hilus will not be exposed on the surface, when a revision procedure on the muscle is necessary later on. During harvesting of the gracilis muscle, drugs for relaxation should not be used. The design of the muscle transplant is transferred to the gracilis muscle after preparing the vessels and nerves in the recipient area. By preferring the superficial temporal vessels to the facial vessels, the skin incisions can be limited. Usually two-thirds of the muscle width is taken for transplantation. Three fingers to the philtrum, to the mediolus, and to the lower lip are prepared in different lengths, with the gracilis muscle still in situ. If eye closure has also to be covered by the function of the muscle transplant, two additional fingers are formed for the upper and the lower eyelid, finally reaching the medial canthal ligament. In this case, the two territories of muscle innervation have to be identified, and the part of the muscle graft for the eye has to be separated from that for the mouth without disturbing the intramuscular distribution of the neurovascular supply. Before cutting the muscle, the resting tension has to be documented.[7] Insertion of the muscle graft is performed in the remnants of the paralytic orbicularis oris muscle. The origin is fixed to the deep fascia around the zygomatic arch. After the neurovascular anastomoses, tissue O2 is monitored (Figure 28.2).

Transposition of Masseteric Muscles

The transposition of the central part of the temporalis muscle for eyelid closure has already been proposed by Gillies in 1934.[11] Although this operation was modified by several authors and finally by us, it is still the favored technique to achieve symmetry of eye fissures and protection of the cornea on the paralyzed side in the adult patient with acquired, complete facial paralysis.[9] For a patient in the age group of younger than 60 years, we combine the transposition of the temporalis muscle to the eye with smile reconstruction with cross-face nerve grafting followed by gracilis muscle transplantation[4] (see "Reinnervation of Paralyzed Mimic Muscles").

In patients older than 60 years or with short life expectancy, temporalis muscle transposition is combined with masseter muscle transposition to achieve some extent of reanimation of the paralyzed face besides a significant improvement of the static symmetry by the suspension effect of the transposed muscles. In these patients, the sagging of the skin on the paralyzed side of the face is met in addition by dermis-suspension-plasties of the brow and of the nasolabial fold, and by a unilateral facelift of the paralyzed side. The de-epithelialized part of skin above the brow is fixed with nonabsorbable sutures to the frontal pericranium. By this, the brow on the paralyzed side is stabilized in a middle position of rest and active elevation on the healthy side. The limitation of the field of vision from superior is removed too. The nasolabial dermis suspension reconstructs not only a nasolabial fold similar to the healthy side but also fixes the ala of the nose laterally and upward against the periosteum of the maxilla. Therefore, some resistance is established against the pull and deforming effect of the healthy side, and even the airway is liberated again on the paralyzed side.

Some operative details are especially important to get good results of muscle transpositions: When transposing the central part of the temporalis muscle, the temporalis fascia in front of the transposed part has to be kept as hypomochlion, and the transposed muscle strips for the upper and the lower eyelids have to be elongated by

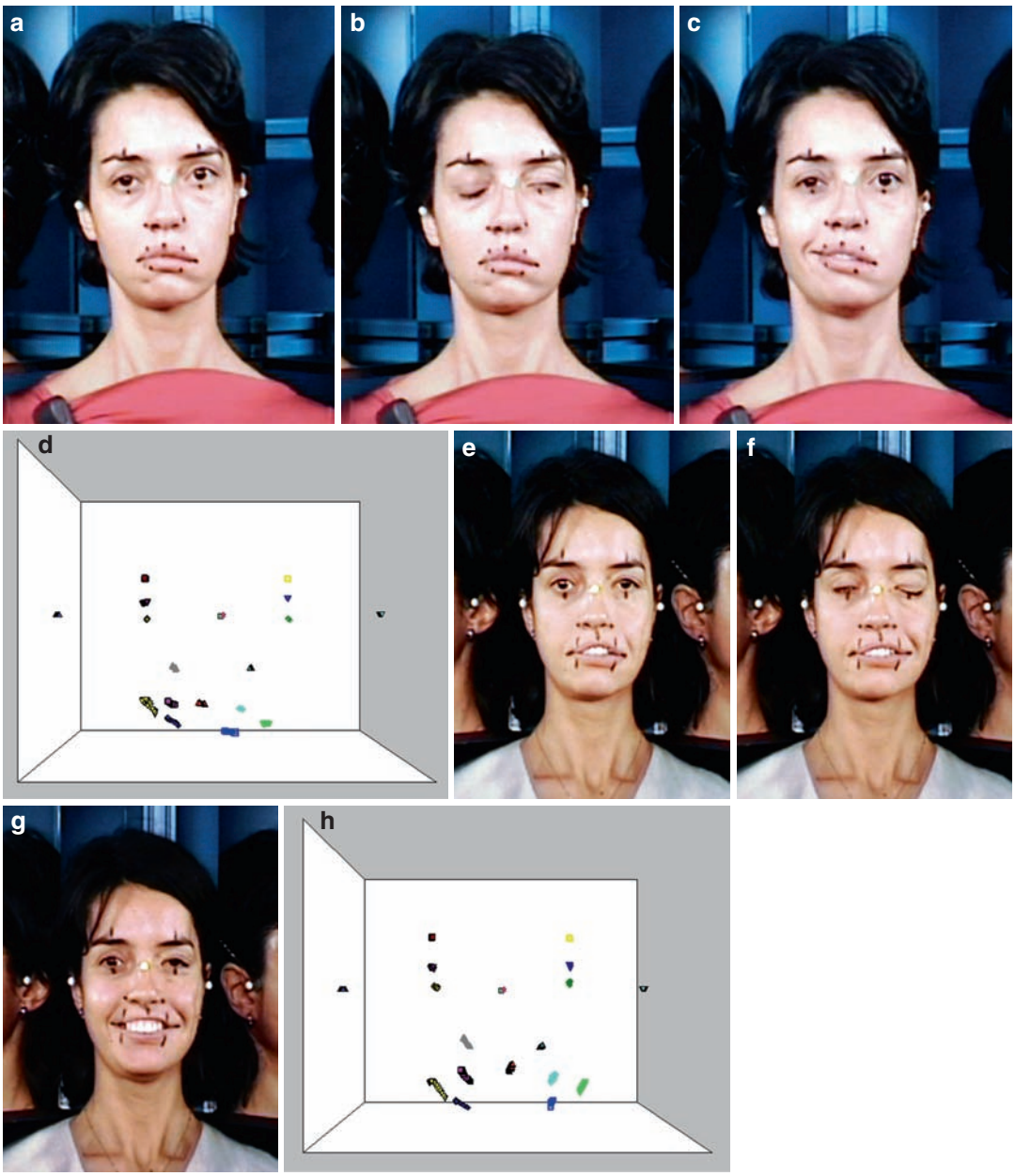

Figure 28.2. A 21-year-old patient, with a total complete idiopathic facial palsy on the left side, since the age of 11 years. The two-stage facial reanimation was carried out using cross-face nerve grafting, followed by free, territorially differentiated gracilis muscle transplantation. The upper row of images shows the preoperative condition at resting position (**a**), closing the eye as in sleep (**b**) and smiling with showing teeth clinically (**c**), as in the three-dimensional (3-D) graph (**d**) of the landmark movements in the 3-D video-analysis system. The lower row of images (**e, f, g, h**) was taken 24 months postoperatively, showing an independent closure of the left eye and a symmetrical emotionally controlled smile.

strips of the temporal fascia to reach the medial canthal ligament. Only in this way, an effective transmission of the muscle contraction becomes possible. Deformity has to be prevented by removal of fat tissue in the area lateral to the orbit to prevent bulging and by filling up the created depression with the posterior part of the temporalis muscle.

The best access to the masseter muscle is the lateral incision along the nasolabial dermis suspension and the preauricular incision for the coincident facelift on the paralyzed side. By that access, the ventral and superficial part of the muscle is separated from its insertion at the mandible, taking care of the course of the parotid duct. Insertion to the remnants of the orbicularis oris muscle is achieved with nonabsorbable sutures to the upper lip as medially as possible, to the mediolus, and to the lateral lower lip under adequate tension. This means some overcorrection. The lateral border of the de-epithelialized skin of the nasolabial dermis suspension is sutured on the surface of the transposed masseter muscle. In the case of severe translocation of the philtrum to the healthy side, circum-oral tendon suspension might be helpful, using a palmaris longus tendon graft.

Procedures Supporting Mimic Function

In reanimation surgery of the paralyzed face, mimic function is not only reconstructed by dynamic procedures but often also by static procedures. The aims of reconstruction are not only emotionally controlled mimic movements but also static and dynamic symmetry as far as possible. Some of these operative procedures have already been mentioned here, because they have become a standardized part of the operative concept of reanimation (see "Reinnervation of Paralyzed Mimic Muscles" and "Functional Free Muscle Transplantation").

Additional procedures after establishment of a functional plateau in the muscle transplant are playing a significant role in the overall result. Therefore, every patient is informed by us that he or she has to expect a finalizing third operation about 1.5 years after muscle transplantation.[4] Dependent on the functional recovery in transposed or transplanted muscles, corrective procedures might improve the result. For example, the amplitude of the contraction of the gracilis muscle transplant is increased by increasing the resting tension of the muscle by shortening the muscle or by elevation of the muscle origin. This often optimizes the resting symmetry of the angles of the mouth. Smaller corrections of the nasolabial fold or of the width of the lips contribute a lot to the mimic function. Around the eye, often tightening of the muscle transposed or transplanted to the upper or lower eyelid increases the efficiency of eyelid closure. If functional recovery in a muscle transplant is turning out to too strong, weakening by excision of functioning muscle might become necessary.

An important factor for the quality of reanimation around the eye is a natural symmetric appearance of the eye fissure when the eyes are open. Increased lateral scleral show and the tendency to ectropium of the lower lid might be corrected by lateral tarsorrhaphy and lateral canthopexy.

Bilateral Facial Palsy

Moebius Syndrome

Bilateral congenital facial palsy is a leading symptom of the Moebius syndrome. Often other cranial nerves such as the abducens nerve, the glossopharyngeal, the hypoglossal, the trigeminal, and the accessory nerves are involved completely or incompletely in this syndrome. Malformations of the heart or of the neuromuscular system of the upper extremity might coincide. Although severe impairments of organ functions are usually well diagnosed by the pediatrician, minor deficits of the cranial nerves additional to the bilateral facial palsy need to be evaluated thoroughly before reconstructive surgery. Before planning to use the masseteric nerve or a part of the hypoglossal nerve for reinnervation, it must be cleared that this source is differentiated and not part of the congenital lesion. In addition, the presence of the muscle planned for transfer has to be proven.

In our hands, bilateral gracilis muscle transplantation to restore the smile has proved to be most effective. On both sides, the same motor nerve source should be used for reinnervation, the masseteric nerve giving the best results. If the mimic movements are controlled by the same nerve on both sides, for example, the trigeminal nerve, and they are therefore synchronous, the missing emotional control by the facial nerve system does not become evident. One of the most significant functional problems in Moebius syndrome is the sagging of the lower lip by gravity. Even if the angles of the mouth are lifted upward by the bilateral functional muscle transplants, the lower lip will not cover the lower teeth, the mouth will stay incontinent for fluids, and the always open mouth will create the impression of cerebral impairment of the patient. Tendon suspension of the lower lip with a palmaris longus tendon graft is a very effective method for secondary correction.

The tendon graft connects both muscle grafts. When they contract at the same time, the lower lip is lifted with the help of the tendon graft, which prevents dilatation of the paralyzed lower lip.

Acquired Bilateral Palsy

Acquired bilateral palsy is very rare and usually caused by trauma or by a complicated operation. We apply the same principles for the concept of treatment as those in the unilateral irreversible facial palsy. The important difference is the absence of a healthy facial nerve, which can be used for reinnervation by cross-face nerve grafts. If there is no proximal segment of the facial nerve accessible, bilateral gracilis muscle transplantation with the masseteric nerve branches for motor reinnervation is the treatment of choice for the younger patient. In a patient with shorter life expectancy, muscle transpositions and static procedures will be preferred to improve static and dynamic symmetry.

Operative Procedures for Functional Upgrading in Incomplete Lesions

Cross-Face Nerve Grafting with Distal End-to-Side Neurorrhaphy

After we published a report on the successful clinical application of the end-to-side neurorrhaphy for sensory and motor nerves[5,6] we designed a new operative technique to improve the motor function in incomplete, but clinically significant, facial paralysis by offering additional regenerating facial nerve axons from the contralateral side through an end-to-side neurorrhaphy to the insufficient facial muscles.[10] The sural nerve graft is interposed between the zygomatic branches for the smile function on both sides. The nerve suture is performed in an end-to-end fashion on the healthy side and in an end-to-side fashion on the side with the partially recovered facial palsy not to destroy the limited function recovered. The nerve graft is used in overlength on the incompletely paralyzed side. By this, the possibility of later additional muscle grafting is still preserved in the case of a clinically disappointing result of the cross-face nerve grafting alone. Therefore, the patient does not need to accept any disadvantage by this new technique, because the conventional way of treatment is still kept open. The technique is showing very promising results, because more than half of our patients did not need the additional muscle transplantation. These results are beyond our own expectations. It makes sense to include a greater number of so far not treated patients with partially recovered facial palsies into this algorithm of reconstruction (Figure 28.3).

Free Functional Muscle Transplantation with Ipsilateral Facial Nerve Supply

An alternative method for functional upgrading of a partially recovered facial palsy is the free functional gracilis muscle transplantation with reinnervation by an ipsilateral zygomatic branch, which shows clear but very limited smile effect on the paralyzed side. The theory behind this concept is the fact that finally well regenerating facial nerve fibers do not contact the paralyzed mimic muscles in time, and the resulting functional recovery is clinically insufficient. Offering a large amount of freshly denervated muscle fibers within a gracilis muscle transplant stimulates the regenerated nerve fibers to sprout, and the recruitment of more muscle fibers by reinnervation leads to a visible contraction and a sufficient smile movement. This technique is, however, limited to extremely selected cases and should be used by very experienced surgeons in this field.

Postoperative Treatment

Physiotherapy

In facial palsy, physiotherapy plays a significant role before designing an operative concept. It is important to treat surgically an irreversible lesion, which would not be improved by time or by physiotherapy. Postoperatively, exercises of the facial movements in front of a mirror not only increase the force and the amplitude of the contraction of the reinnervated muscles but are especially important to learn again the newly acquired movement and to integrate the new movement into the mimics of the whole face. Dosage of the movement is important to fit in symmetry to the healthy side. Programming by learning under

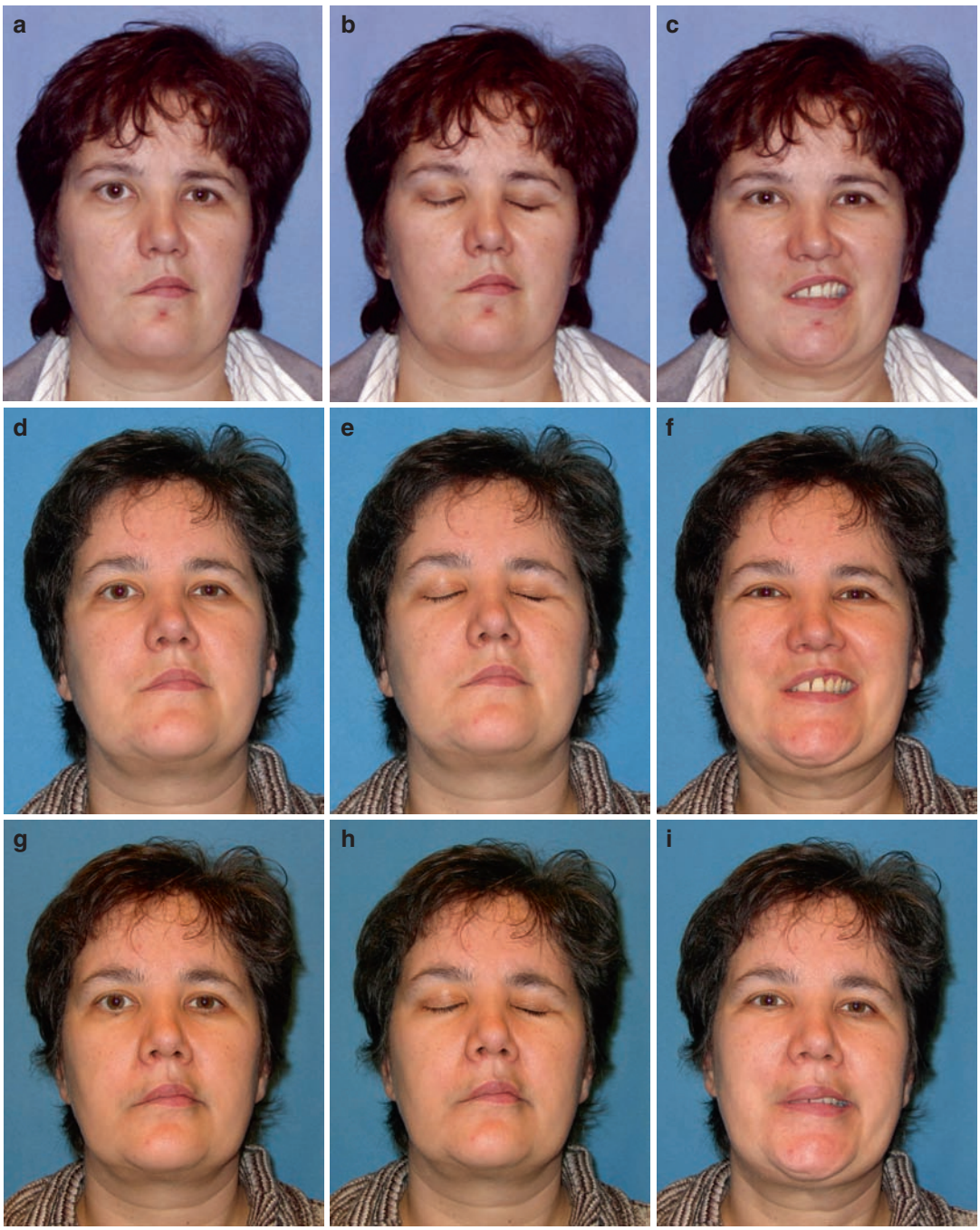

Figure 28.3. Preoperative images (**a, b, c**) of this 41-year-old patient show a total incomplete idiopathic facial palsy on her right face, which set in at the age 13 years. The postoperative images show the patient 24 months after the technique of "cross-face nerve grafting with distal end-to-side neurorrhaphy" at resting position (**d**), closing the eyes as in sleep (**e**) and smiling with showing teeth (**f**). A blockade of the cross-face nerve graft (**g, h, i**) 24 months postoperatively, applying local anesthetic led to the same extent of paralysis as before reanimation surgery.

visual control leads finally to an adequate, emotionally controlled movement. The importance of training the facial movements twice every day increases with the incidence of muscle reinnervation, which means control of muscle contraction by the patient. The duration of the training sequences should be kept to 10–15 min per session not to overload the muscle. Fatigue of the muscles during the training or in the evening is normal and should not lead to increase in the training load. The movement exercises have to be learned and applied by the patients themselves. Supervision by the physiotherapist in increasing intervals is enough, when the training has to be performed for more or less 1 year.

Electrostimulation

Electrostimulation should not be applied, when spontaneous recovery is expected. Spasticity would be the consequence and should be avoided. It should be used to keep a transplanted muscle in good condition and to prevent muscle atrophy as much as possible until reinnervation takes place. We start with electrostimulation 6 weeks after muscle transplantation and decrease it when contractions show evidence of the beginning of reinnervation of the muscle. The accessibility of the muscle contraction for the patient's control increases the meaning and the need for active muscle training. After good muscle control is taken over by the patient, electrostimulation is ceased, which is 6 months after muscle transplantation on the average.

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Part V

Breast

29

Congenital Breast Malformations

Armand Lucas and Serdar Nasir

Summary

Congenital breast deformities pose a challenging dilemma in plastic surgery. The expression of the abnormality may be complete, and anatomic components are often missing, deformed, or weakened. Most breasts are naturally asymmetrical. These asymmetries may include discrepancies in breast size and shape, location of the nipple–areola complex, the inframammary fold, or skeletal abnormalities (see Figure 29.1). Breast abnormalities can cause emotional and psychological trauma, resulting in social maladjustment and associated behavioral problems. The majority of patients are young healthy individuals who seek aesthetic restoration of their deformities.

Preoperative evaluation begins with a thorough medical and surgical history, particularly related to breast disease. Physical examination includes identification of chest wall or musculoskeletal deformities; shape, symmetry, and volume of both breasts; the presence and degree of ptosis; the position of the inframammary folds; and anomalies of the nipple–areola complex. Most of these anomalies require a multistage surgical approach, and the patient must be extensively counseled.

Abbreviations

DIEP	Deep inferior epigastric perforator flap
LDF	Latissimus dorsi muscle myocutaneous flap
SGAP	Superior gluteal artery perforator flap
TRAM	Transverse rectus abdominis myocutaneous flap

Poland's Syndrome

Poland's syndrome was first described by Alfred Poland as a medical student in London in 1841. Although Lallemand and Froriep presented patients with similar anomalies, Alfred Poland's description is by far the most precise and comprehensive.[7] The sine qua non of Poland's syndrome is the absence of a sternocostal portion of the pectoralis major muscle, a hypoplastic or absent breast and/or nipple–areola complex, and may include upper-extremity abnormalities such as hypoplasia of the hand, forearm, and arm. This may also include complete or incomplete syndactyly and short fingers. The chest wall can have abnormalities with depressed ribs and occasional absence of the latissimus dorsi muscle, serratus anterior muscle, and external oblique muscle. Absence of the pectoralis major muscle occurs with an incidence of approximately 1:7,000 to 1:1,00,000 live births. It commonly affects males

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in a 2:1 to 3:1 ratio and has a right-sided predisposition in 60–75% of cases.[8] The etiology of this condition is unknown, and there is no genetic pattern or familial predisposition. The most accepted theory refers to a circulatory defect of the subclavian artery around the 45th gestational day.[9] This has been termed the "subclavian artery supply disruption sequence" and is supported by Merlob et al.'s evaluation of subclavian artery diameter and flow.[18]

Poland's syndrome has a large spectrum of clinical presentations which correlate well with the degree of functional impairment and cosmetic deformity. The surgical correction of Poland's syndrome deformity requires addressing four separate issues: (1) Addressing the chest wall deformity, (2) addressing the breast deformity in females, (3) surgical correction of any upper-extremity deformities, and, lastly, (4) nipple–areola reconstruction.

The ideal reconstruction goals in female Poland's anomaly patients require achieving breast symmetry, correcting the chest wall deformity, recreating the anterior axillary fold, and providing an adequate infraclavicular fullness for aesthetic and psychosocial aspects.[3] In female Poland's syndrome, some clinical situations correcting the breast contour may be enough to camouflage the deformity without the need for extensive muscle flap transposition (i.e., latissimus dorsi flap) (Figure 29.1).

The latissimus dorsi muscle flap and muscul cutaneous components have been used for patients with Poland's syndrome since first introduced for breast reconstruction. Latissimus dorsi transposition reestablishes the anterior axillary fold, filling in the supraclavicular hollows, particularly in males with thin subcutaneous tissue. This replaces the pectoralis major muscle, which is strongly associated with the male gender. In females, it provides excellent coverage for a breast implant or a tissue expander. In patients in their developing adolescent years, an expander implants such as the Becker implant can be used temporarily and later on exchanged for a permanent implant when the patient has completed growth. The timing of this reconstruction, at least with a muscle transposition, should be seriously considered at age 5–6 during the preschool years before serious anxieties and psychological problems develop. To create the anterior axillary fold with a latissimus dorsi flap, some surgeons advocate dividing the latissimus insertion tendon on the humerus and transferring it to a more anterior position to increase its projection across the axilla.[16] In females performing a latissimus dorsi muscle transfer without an expander or a mammary prosthesis may result in an inadequate volume in most cases. If the patient has a larger tissue defect or is older and has significant lower abdominal tissue, a TRAM flap, even a deep inferior epigastric flap (DIEP) flap, or a superficial inferior epigastric artery flap can be transferred as an option. Despite the aesthetically pleasing results that can be achieved with the latissimus dorsi transposition, substantial donor-site morbidity can exist because of the large incision needed to harvest and transfer the

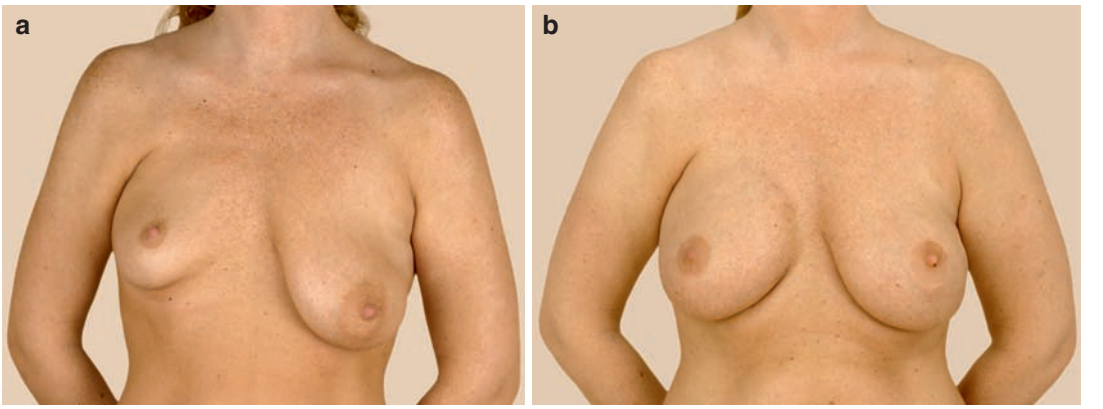

Figure 29.1. (**a**) A 32 year-old female with Poland's syndrome. Right breast corrected with a 350-cc gel implant and a simultaneous left breast mastopexy. No need for latissimus transposition. (**b**) Postoperative result 3 years later.

flaps.[17] Borschel et al described a two-stage minimally invasive technique that requires only a single small incision in the axilla for the muscle dissection and implant placement. In the first stage of this technique, a tissue expander is placed endoscopically to increase the size of the skin envelope. After a period of expansion, at a second stage, the tissue expander is removed endoscopically and a permanent saline implant is placed. The latissimus dorsi muscle can then be transferred. For some patients, the opportunity to reduce the dermasite scar through endoscopic techniques is a more attractive option. In situations where the syndrome includes hypoplasia of the latissimus dorsi muscle, free tissue transfer procedures can provide excellent results. The free superior gluteal myocutaneous flap and free rectus flaps have been reported by various authors.[14,24] However, the use of the TRAM flap particularly if tripedicle may have detrimental effects on the abdominal wall of young patients. Donor-site morbidity can include abdominal hernias, bulging, prolonged pain, and anesthesia of the involved tissues. For those reasons, perforator free flaps have provide an excellent alternative method of autologous reconstruction for this syndrome. The DIEP[13] and the superior gluteal artery flap (SGAP),[9] are currently used for breast reconstruction by Poland's syndrome. The advantage of the DIEP flap is that it lessens the amount of fascial dissection and rectus muscle dissection at the donor site. Thus, it may decrease postoperative pain and lessen recovery, and some studies have shown a slightly lower incidence of hernia formation and bulging compared to the TRAM flap.[10] The Superior gluteal artery prefactor flap (SGAP) may be used if the patient does not have adequate abdominal tissue, as in the case of many younger patients. However, there is a high chance for buttock contour donor-site deformity requiring secondary revisions. Furthermore, perforator flaps need advanced microsurgical skill, and this procedures takes longer operative time.

A separate deformity from Poland's syndrome was described by Spear et al. in 2004, and it was coined the anterior thoracic hypoplasia syndrome. This deformity and Poland's syndrome share similar traits; however, in the anterior thoracic hypoplasia syndrome, the pectoralis major muscle is completely normal unlike the situation in Poland's syndrome where the pectoralis hypoplasia or missing sternocostal head is the defining feature. Poland's syndrome has varying degrees of severity and involvement, and it may or may not have involvement of the upper extremity, but the anterior thoracic hypoplasia syndrome tissue deficiencies are similar with regard to chest and breast size and can occur on the contralateral side. The etiology is unknown. This second syndrome is more easily corrected by the use of breast augmentation to increase breast size and projection of both breasts. Augmentation mammoplasty is the preferred method, because it is simpler compared with alternative flap procedures.

Axillary Breast

The human breast develops from ectoderm during the fifth week of gestation. The earliest identifiable breast tissue is the mammary ridge, which expands bilaterally from the axilla in a caudal direction to the groin. Breast tissue can develop anywhere along this embryologic breast line. One of the most common sites of extra mammary breast tissue is the axilla but it can also be found in unusual locations such as the scapula, thigh, posterior leg, or the labia majora (Figure 29.2). Ectopic breast tissue is at risk for similar benign and malignant problems as normal breasts, which include fibrocystic disease, mastitis, fibroadenomas, atypical hyperplasia, and carcinoma.[6] Marshall in 1994 reported an increased incidence of cancer in aberrant breast tissue. Accessory axillary breast tissues are common embryologic alterations found in about 1–6% of women, and they often manifest bilaterally.[1] Axillary breast tissue presenting as a palpable axillary mass can undergo monthly menstrual changes as well as tenderness, swelling, difficulty with shoulder motion and irritation from clothing. All symptoms may be aggravated, becoming more apparent during puberty and pregnancy. Ectopic cancers located in the axilla seem to present with more extensive disease at an earlier age, suggesting that aberrant tissue may be at increased risk for malignant change. The treatment of axillary breast tissue is generally elliptical excision, although axillary breast tissue has also been reportedly removed with ultrasound-assisted liposuction to avoid a large scar.[5]

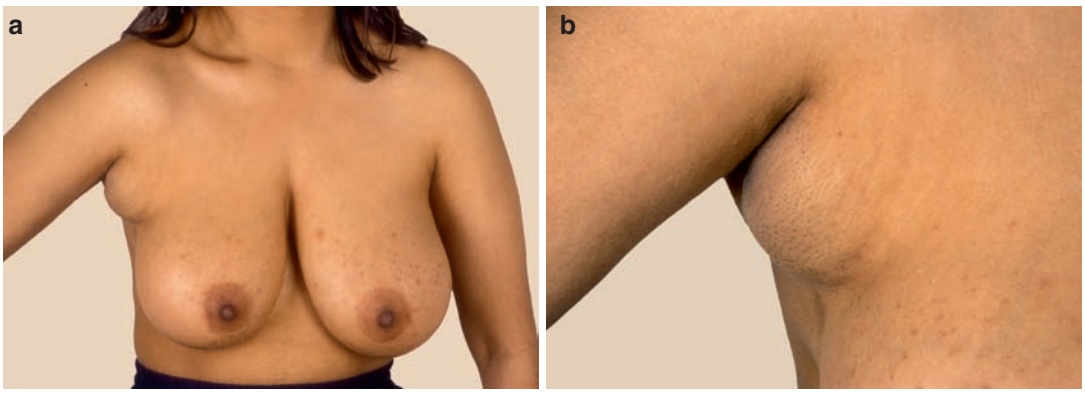

Figure 29.2. (**a**) Axillary breast; (**b**) detail.

Polythelia

Supranumerary nipples or polythelia (Figure 29.3) can be found along the line from the axilla to the groin. This anomaly is the most common anomaly in congenital breast deformities present in about 5% of the general population and can be found in both men and women.[22] A remnant of milk lines after improper regression of the mammary ridges at approximately 3 months of gestation causes accessory nipples.[2] The most common site for an abnormal nipple is inferior to the normal breast but superior to the lower abdominal inguinal regions. In approximately 50% of the patients, it can be bilateral. It has also been described outside the milk line, such as the scapula, posterior thigh, head, and neck.[25] Although polythelia rarely causes more than an aesthetic concern to the patient, sporadic polythelia may be associated with other congenital deformities including kidney urologic abnormalities. This anomaly has also been associated with testicular cancer. It has been reported that male patients with supernumerary nipples have an increased risk of testicular tumors, but the actual relative risk remains uncertain because of the relatively lower incidence of both diseases.[11] These pigmented lesions in the milk line should be excised before puberty, particularly in young girls, because recession may require larger tissue excision if there is glandular growth. There have been a few reported cases of cancerous degeneration of this accessory nipple–areola complex tissue, and that provides an additional justification for excision of these lesions.[22]

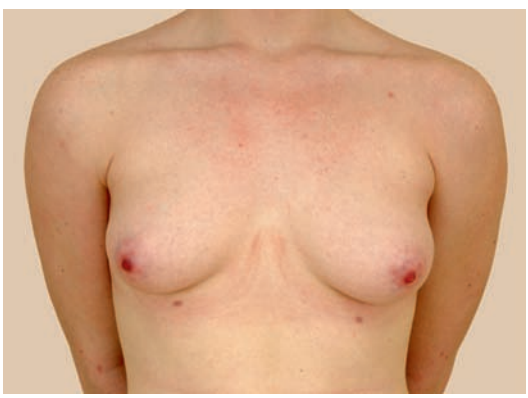

Figure 29.3. Polythelia.

Tuberous Breast

Tuberous breast syndrome is a rare anomaly presenting in adolescent mammary development. It was initially described by Rees and Aston,[20] and it is characterized by a constricted base, hypoplastic breast tissue, a herniated nipple–areola complex, an elevated inframammary fold, and a deficient skin envelope in the lower pole vertically and transversally. Its etiology is unknown. There is no genetic or familial incidence, and a proposed cause of this tuberous deformity is the presence of a ring of fascia that constricts radial growth. It is hypothesized that this constricting ring of superficial fascia limits growth of the lower pole and results in a high inframammary fold. Mammary tissue herniates through a hiatus in the fascia located directly

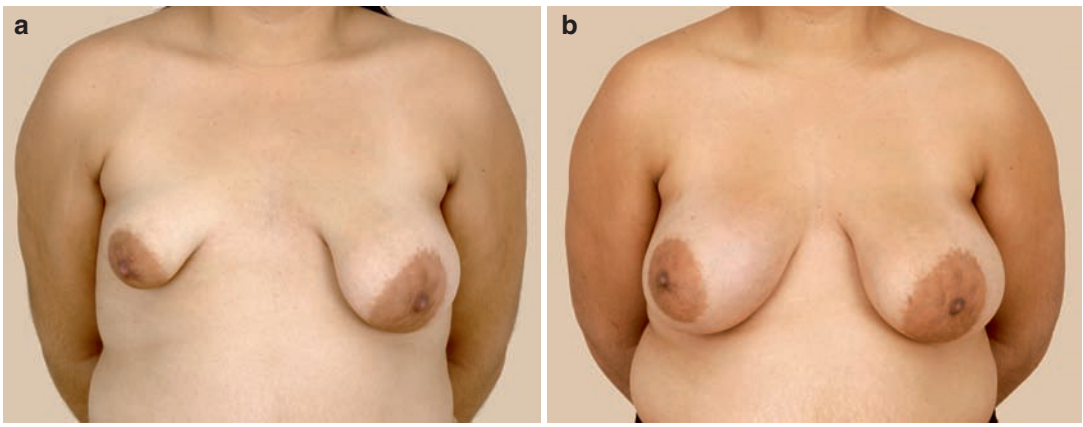

Figure 29.4. (**a**) A 27-year-old female with right tuberous breast deformity, 5 ft 11 in., 220 lb. (**b**). Deformity corrected with 3 months of tissue expansion and later replacement with 600-cc gel prosthesis on right and 500-cc augmentation on left. Postoperative result at 1 year.

deep to the nipple–areolar complex.[26] This breast tissue deficiency results in the tuberous or "potato-like" appearance of the breast that may involve only the lower pole or may involve the breast circumferentially. This deformity becomes evident during adolescence and can generate significant psychological disturbances. Generally, volume deficiency of tuberous breast is at least 27% less than the contralateral breast, and in many cases, it is bilateral to a variable degree. Grolleau et al.[12] reported that in 28% of tuberous breast deformities, the breast was reported to be hypertrophic.

The classifications reported in the literature are based primarily on the shape of the deformity. The von Heimburg classification is divided into four categories based on the degree of hypoplasia of the base of the breast. In this classification, type 1 and type 3 breasts may be considered the same, because the amount of skin envelope is simply dependent on the size of the glandular tissue itself. Grolleau et al.[12] reduced the classification group to three classes based on the degree of hypoplasia at the base of the breast. Type 1 is characterized by hypoplasia of the lower medial quadrant; type 2 breasts are hypoplastic on both lower quadrants, and the areola points downward; and type 3 has hypoplasia in all four quadrants.[12]

There are multiple surgical techniques described for correction of tuberous breast deformity. The main goals are to restore the base dimensions, correct hypoplasia of the lower quadrants, reposition the inframammary fold, correct the nipple–areola complex herniation by reducing the size of the areola, correct ptosis, and provide as close a symmetry as possible with the contralateral breast (Figure 29.4).

The timing of reconstructive surgery for most congenital breast deformities is important to the final outcome. Surgery at a young age can damage the breast bud and cause major growth deformities. In most situations, corrective surgery should not be undertaken until the breast is fully developed by the age of 15 or 16 years.

The tuberous breast with herniation of breast tissue through a constricted fascial ring may be approached through a periareolar incision, allowing for areolar reduction and release of the constricting rim. The inframammary approach is another effective way to release this band and adjust the inferior position of the breast. The chosen technique depends on careful study of the deformities of the breast to achieve proper correction.

The superior pedicle mammoplasty with lateral dermal glandular flap techniques also provides filling of the lower quadrant with breast tissue. The Maillard Z-plasty technique may be used for type 1 and type 2 deformities, and in this technique, the inframammary fold is lowered to a normal position, and the inferior portion of the breast is reshaped by overlapping the elevated inferior skin flaps in the fashion of a Z-plasty.[15] The Ribeiro technique may be used for type 2 and type 3 deformities. In this technique, an inferior flap is made by dividing the lower pole of the breast from the upper pole and the nipple–areolar complex. This flap is then folded over itself to

give the inferior pole projection and as close to normal in appearance as possible.[21] Choupina et al.[4] reported the successful use of this technique in combination with silicone implant placement for type three deformities. In the Puckett technique, the inferior flap is based just deep to the nipple–areola complex; it is elevated, and a submuscular implant is placed, and the breast is unfurled anteriorly over the implant, transferring tissue bulk to the inferior pole.[19]

Tissue expansion has become, in many ways, the mainstay of the treatment of tuberous breast deformities (Figure 29.4a). The constricted base of the breast is approached through the inframammary incision; multiple cruciate incisions are made along the base of the breast to destroy and eliminate the constricted bands, and a tissue expander is placed in the submammary position. The patient then undergoes a period of expansion, which may range from 2 to 4 months depending on the size needed to match the contralateral breast, and then at a second stage, has the tissue expander removed, the pocket capsule adjusted, and a silicone implant placed to match the contralateral side. In many situations, a contralateral augmentation or mastopexy is performed to improve symmetry (see Figure 29.4b).

Congenital Absence of the Breast

Isolated absence of the breast was first described by Froriep in 1839. Unilateral absence can be seen together with Poland's syndrome, bilateral absence is rare, and most cases of amastia are a component of other developmental syndromes such as Ullrich–Turner syndrome or the AREDYLD syndrome.[23] In these situations, breast reconstruction techniques undertaken are similar to those used in breast cancer surgery. That is, the use of the transverse rectus abdominus myocutaneous flap or latissimus dorsi flap plus an implant. Tissue expansion with subsequent placement of a silicone implant is also a viable option.

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30

Breast Reduction and Mastopexy

Marita Eisenmann-Klein

Summary

A vast array of techniques have been developed and abandoned since the first reported breast reduction by Dieffenbach, 160 years ago. The current trend, particularly in Europe, is toward short scar techniques. In this chapter the pertinent aspects of breast anatomy and physiology are delineated as well as the medical indications and psychological aspects inherent in macromastia patients. In addition, necessary preoperative classification and planning are discussed in conjunction with technique selection and execution. This is followed by a detailed description and step-by-step photograph demonstration of our preferred technique, mainly influenced by the Z-plasty technique of Maillard and the short vertical scar technique of Lassus. The complication and revision rates of major studies are compared to our own series of 3,554 breast reductions using this technique. Finally, the medicolegal aspects are discussed focusing on the fact that breast reductions and mastopexies are the most frequent causes of law suits in plastic surgery in the U.S. due to scar problems.

There is no doubt that breast reduction provides enormous benefits to patients, physically als well as psychologically. Even from an economic point of view, insurance companies would benefit from paying for this procedure, since the health condition of patients improves enormously after breast reduction.

Breast reduction and mastopexy are among the most common procedures in plastic surgery. Outcome studies demonstrate that patients' satisfaction postoperatively is very high for this procedure.

However the degree of patient's overall satisfaction is strongly correlated with patient's satisfaction with her scars.

Therefore, there is an urgent need for techniques that are associated with shorter and/or less visible scars.

History

The first breast reduction was reported by Dieffenbach in 1848. His technique did not include the transposition of the nipple–areola complex.[30]

At the end of the nineteenth and at the beginning of the twentieth centuries more techniques were described by Pousson in 1897, Verchère in 1898, and Dartigues in 1924.[30]

The first technique with transposition of the nipple–areola complex was reported by Joseph in 1927.[35]

In 1960, Stroembeck described his technique of an inverted T-incision, which is still in use.

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In the same year, Pitanguy also described a technique with inverted T-incision.[53,54]

Another technique, still in use today, was reported by Mc Kissock in 1972.[49]

Since the horizontal parts of the inverted T-incision caused more problems than the vertical part, some innovative plastic surgeons came up with techniques avoiding the horizontal scar.[50,52,56]

As early as in 1923, Lotsch[44] in Germany presented a technique with a short vertical scar. Hinderer in 1969 described a periareolar dermopexy.[27] In the same year, Benelli presented a personal periareolar mastopexy technique.[3] Lassus published his vertical scar technique in 1970.[41] In 1982, Marchac demonstrated that the T-incision could be done with a much shorter horizontal scar.[47]

Madeleine Lejour gained worldwide popularity in 1990 with her combination of liposuction and vertical scar technique.[42] Innovations also came from Elizabeth Hall-Findlay (short vertical scar),[23] Goes with a "double-skin" technique,[14] and Graf and Biggs, who use a muscle sling as an "inner bra."[21]

Anatomy

Breasts develop from the mammary ridge during the sixth week of fetal development. Between the 15th and the 20th week, primary milk ducts are formed. The milk ducts are surrounded by myoepithelial cells, and finally, the alveoli develop.

Within the gland, there are 15–20 glandular lobes, connective tissue, and ducts.

A normal-sized breast weighs around 200–400 g per side. During lactation, the weight increases considerably.

The breast of an adult woman varies in its position on the chest wall, size, and weight. It extends from the second or third rib to the sixth or seventh rib vertically and from the lateral sternum to the area between the anterior and the mid axillary line in its horizontal diameter.

The inframammary fold is located between the fifth and the eighth rib. There is an extension of the breast tissue into the axillary region, which is called the Tail of Spence.

The distance between the nipple and the sternal notch is quite stable in normally sized and shaped breasts. The sternal notch to nipple distance in a normal young patient should be approximately 20 cm, varying according to the height and the thorax shape of a patient. The position of the breast on the chest wall is subject to major variations. The normal distance between the nipple and the lower fold is in the range of 7–11 cm.

The blood supply is variable. There is a deep and a superficial arterial system. The superficial system supplies the skin and the nipple–areola complex. The deep system provides three major vessels: the internal mammary artery, the lateral thoracic artery, and the intercostal artery system. The internal mammary artery supplies the medial part. The lateral thoracic artery and the intercostal artery perforators supply the lateral breast. The lower breast gets its arterial supply from the anteromedial intercostal perforators. All three systems form a periareolar plexus with multiple anastomoses for the nipple–areola complex (Figure 30.1).

The venous system also combines a deep system and a superficial network. The deep system is located parallel to the arteries, whereas the superficial system provides a network for subdermal drainage.[58]

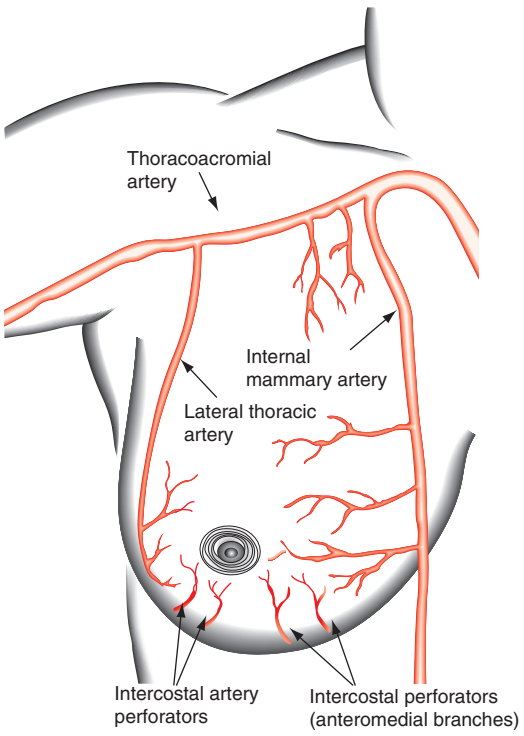

Figure 30.1. Arterial blood supply to the breast.

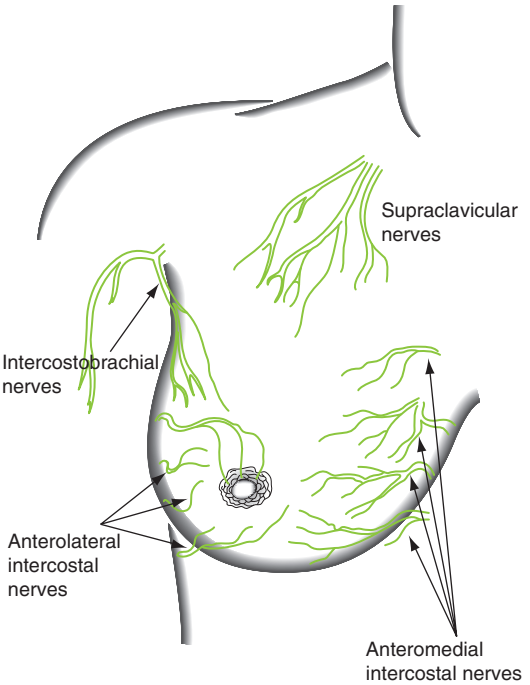

Figure 30.2. Innervation of the breast.

Lymphatic drainage occurs through a superficial system, draining the skin, and a deep system for the gland. The lymph drainage primarily goes to the axilla and to a lesser degree to lymphatics along the internal mammary artery.

The innervation of the two upper quadrants derives from the supraclavicular branch of the cervical plexus. The skin of the lower quadrants is innervated by the intercostal nerves (Figure 30.2).[12]

There is an ongoing discussion about which nerve is mainly responsible for the innervation of the nipple–areola complex: Most authors agree that the lateral cutaneous branch of the fourth intercostal nerve is predominant for the sensation of the nipple.[58]

Physiology and Pathophysiology

Due to maternal estrogen influence, the glands secrete colostrum in the newborn child.

At the beginning of puberty, estrogen production increases and causes breast tissue and fat to grow. There is a major growth of ducts. Estrogen also induces duct maturation. Progesterone promotes growth of the alveoli. Although these two hormones are most important for the development of the breast, other hormones contribute too: insulin, cortisol, prolactin, growth hormone, and thyroxin.[60]

Macromastia seems to be a multifactorial phenomenon with a hereditary component.

The etiology of hypertrophy of breast tissue is still unclear, particularly in juvenile hypertrophy. Hypersensitivity to estrogen seems to play a major role. The hormone levels, however, are almost always normal. There seems to be a hereditary component, and there is a correlation with bodyweight.

Macromastia, starting with puberty, is called juvenile hypertrophy or virginal hypertrophy.

Secondary development of breast hypertrophy is associated with obesity.

The third group of patients, presenting with breast hypertrophy, experience breast involution and replacement of glandular tissue by fat tissue.

Histological findings in specimens of breast reduction patients are not different from the ones found in normal-sized breasts.[37] The increased risk for developing malignant breast disease in patients with macromastia is due to the higher volume. Decrease in volume lowers the risk to a level comparable to women with normal-sized breasts.[32]

Medical Indication and Psychological Aspects

Heavy breasts cause back pain, mostly located in the upper part of the thoracic spine as well as in the lower cervical spine.[43] Myogelosis of the trapezius muscle is common. The typical unphysiological posture of these patients includes a frontal rotation of the shoulders, so that in many cases even the muscles of the anterior neck are shortened. Frequently these patients suffer from severe headache.

The bra strings cut into the skin leaving deep grooves behind. During summer, frequently skin irritations and sometimes skin defects occur in the area of the inframammary fold.

Each symptom by itself, back pain, headache, grooves from bra strings, and skin irritation,

deserves to be regarded as a medical reason for surgical intervention.

Most of the European nations and the United States provide insurance coverage for breast reductions with a reduction of weight of more than 500 g per side.[38,39,63]

This agreement is not accepted anymore in Germany: Insurance companies ask for the body mass index (BMI) and refuse payment if the BMI is above 30, regardless of the amount of excess breast tissue. Back pain and even slipped disc are not considered to be medical indications for breast reduction.

Most women also suffer psychologically. They feel unattractive and self-insecure. Many of them avoid physical contacts. This may also cause professional problems, particularly in professions that require close contacts with clients (e.g., dentists, hairstylists).

The results of a breast reduction are usually good. Pain is remarkably reduced; many patients are free of pain, even in cases with signs of severe degeneration in the cervical and thoracic spine.[5,10,11,19,61,62,64,65]

The psychological benefit is just as obvious in all outcome studies. Patients report remarkable pain reduction and a dramatic improvement of self-esteem after breast reduction.[5,10,11,19,34,61,62,64,65]

What is considered to be a "normal" breast size? Here we are confronted with a transcultural phenomenon: a breast that is considered to be normal sized in Europe might be estimated as too small in North America and as too big in South America. There were remarkable differences in the past, but they seem to disappear gradually with increasing acceptance of a global perspective of physical attractiveness. Physical attractiveness, however, is not the main reason for women to ask for breast reduction.

Table 30.1. Classification of excess volume.

Minor	0–200 cc
Moderate	200–500 cc
Major	500–1.500 cc
Gigantomastia	Over 1.500 cc

Source: Data from Regnault and Daniel.[57]

Classification and Preoperative Planning

Classifications of breast hypertrophy have been presented by multiple authors.

For excess volume, Regnault and Daniel suggest a classification shown in Table 30.1.[57]

The family history and the individual history of breast diseases need to be evaluated carefully.

The medical history is essential to avoid postoperative complications: If the patient is a smoker, the risks for infection, necrosis of the nipple–areola complex, fat necrosis, and compromised wound healing are increased considerably. Breast reduction is an elective procedure. Therefore, it should be done only if the patient contributes to the success by giving up on smoking.[17] We request a waiting period of 6 weeks after the patient quits smoking.

Patients often take low-dose Aspirin for prevention of thromboembolic diseases. The patient needs to be advised not to take any medication that contains acetylsalicylic acid 10 days before surgery. In order to protect the patient during this period, she should be put on low-dose heparin instead.

Patients frequently have asymmetries of the thorax without knowing. It is essential that asymmetry is discussed with the patient before surgery. Photographs are essential to document these findings!

Mammography is suggested before surgery in patients who are older than 35 years. In younger patients, it is impossible to judge the findings in mammography due to the density of the breast tissue. During surgery, one should always look for abnormal tissue.

The preparation for anesthesia is done according to the standards of the American Society of Anesthesiologists. Breast reduction, in most cases, should be performed under general anesthesia.

In Germany, informed consent is obligatory, and it has to be discussed with the patient and signed by the patient at least 24 h before surgery. All potential risks and complications need to be addressed. It is advisable to include the patient's ideas about the perfect size for her. We encourage our patients to show us photographs of their desired breast size and shape.

Since the patient needs to wear a made-to-measure compression bra postoperatively, it is advisable that the patient orders the bra before surgery.

Selection of Technique

Scars resulting from horizontal incisions frequently become hypertrophic in the medial and lateral parts. Every effort should be made to avoid this kind of scars.

The resection of breast tissue is possible in all parts of the gland. Most common is the resection of excess tissue in the caudal and/or central sections of the gland.

In order to perform the transposition of the nipple–areola complex, a glandular pedicle, which provides arterial blood supply, venous drainage, and innervation, is formed. A variety of options exist for the formation of this pedicle: Medial, lateral, superior, inferior, mediolateral, and vertical bipedicle flaps.[28,29,66,67,70]

Periareolar techniques are frequently characterized by a lack of adequate projection of the breast postoperatively. With newer techniques, this problem can be avoided.[1,2,4,22,31]

Inverted T-Incision versus Vertical Scar Technique

A huge variety of techniques have been described, as outlined under history.[3,21,23,27,30,35,41,42,47,49,53]

The discussion about inverted T-incision versus vertical scar incision is ongoing. Vertical scar incisions seem to be more popular in Europe than in other parts of the world.

We do not see limitations for the vertical scar technique as many other authors do. Our longest pedicle was at a 60 cm distance between the sternal notch and the nipple. No free nipple graft was needed (see Figures 30.4 through 30.9).

Common Techniques with Inverted T-Incision

All these techniques have advantages and disadvantages. It is important to select the technique that provides reliable results and the lowest complication rate.

Marking is essential in all techniques: The distance from the sternal notch to the nipple should be marked at 20–23 cm. It is extremely important to place the nipple–areola complex in a position that is not too high. Too low is easier to correct than too high. It is also difficult to correct the position of the nipple–areola complex, if it was placed too far medially.

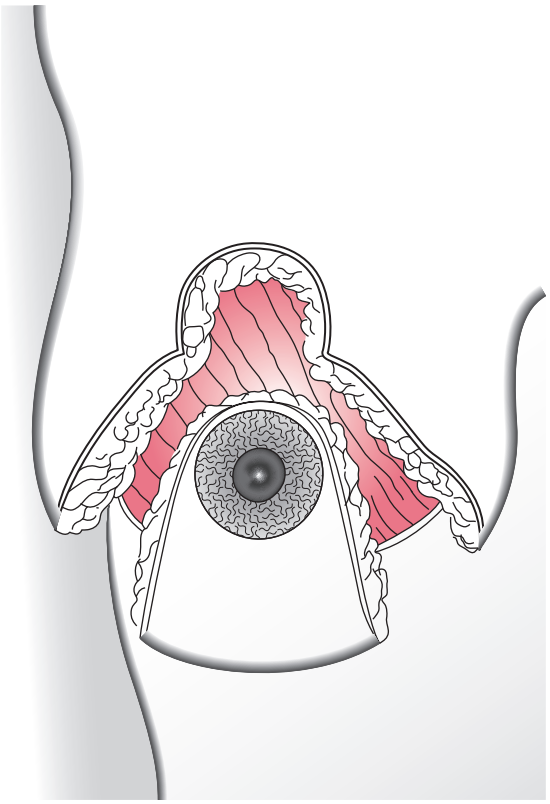

Figure 30.3. Inferior pedicle technique, described by Goldwyn and Courtiss.[8,17]

Today in the United States, the inferior pedicle technique seems to be the most popular procedure in breast reduction. It shows reliable results and a low rate of complications. It is easy to teach, fast to perform, and the complication rate is fairly low.

In this technique, an inferiorly based flap is dissected. It remains attached to the pectoralis muscle. It is essential that enough breast tissue is left behind the nipple–areola complex (at least 3 cm thickness). The resection of the tissue is performed laterally, medially, and superiorly. An inferior pedicle technique was described by Goldwyn and Courtiss[8,17] (Figure 30.3).

Another inferior pedicle technique was first presented by Georgiade. The advantage of this technique is seen in the use of an inferiorly based pedicle in combination with a large pyramid of breast parenchyma. This procedure is considered to be one of the safest techniques for breast reduction.[13]

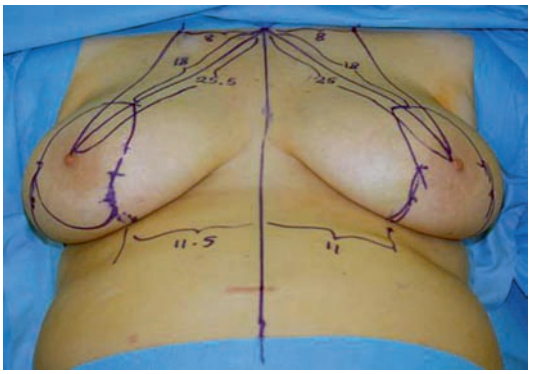

Figure 30.4. Measuring and marking of the breasts in a semisitting position on the operating table.

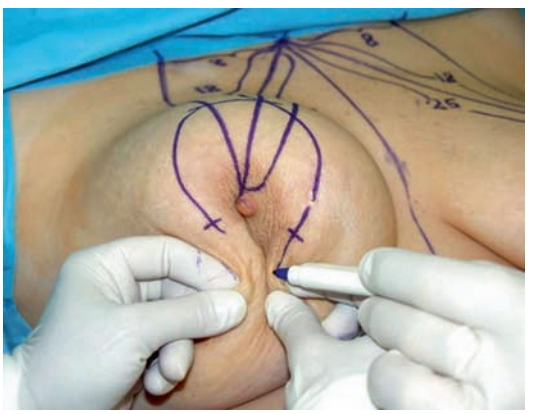

Figure 30.5. Evaluation of the area of skin excess in the lower pole.

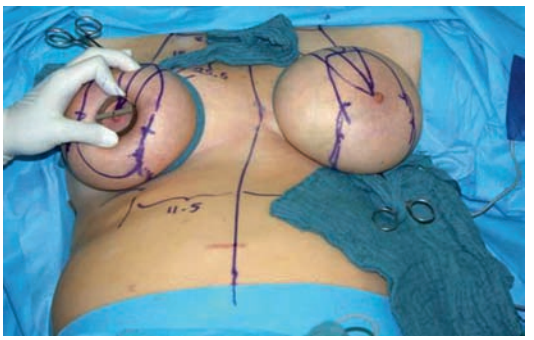

Figure 30.6. Marking the size of the nipple–areola complex, with the towel fixation in place.

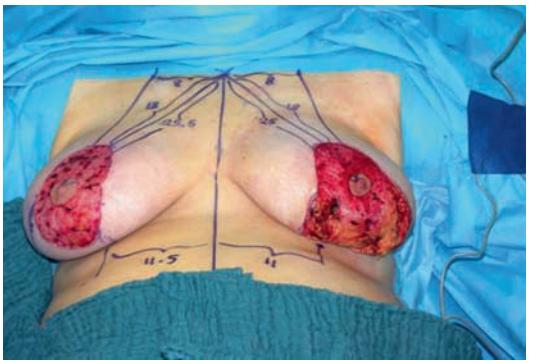

Figure 30.7. Area of de-epithelialization.

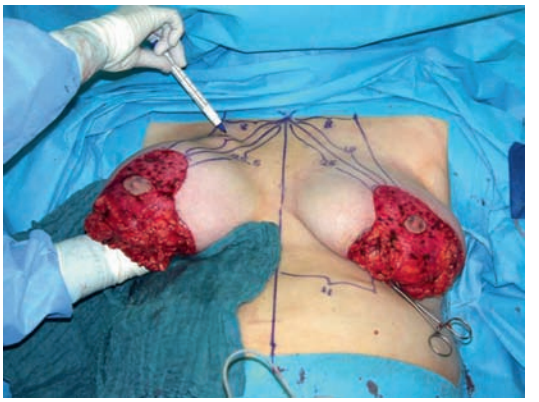

Figure 30.8. Demonstration of the extent of the channel created between the gland and the pectoralis muscle.

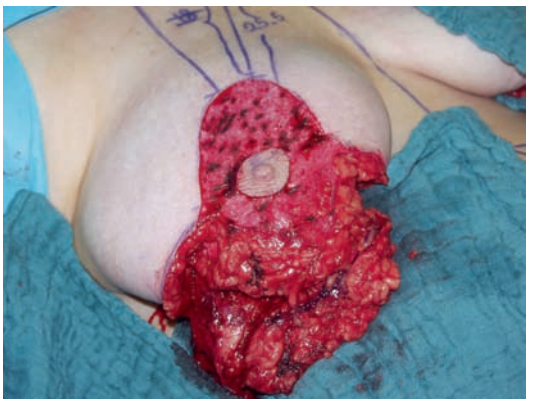

Figure 30.9. Performing the excision of the parenchyma; ink marking of the excision line.

A central pedicle technique was popularized by Hester.[26] It is based on a tangential reduction of the gland in combination with wide undermining of the skin.

Another technique especially suitable for the less-experienced plastic surgeon is the use of the Wise pattern. The keyhole pattern may be combined with a template for the breast surface: the pedicle in this technique can be inferior, central, or superior.[69]

Some authors doubt whether the use of a prefixed pattern and template can give good results.[40]

Preferred Techniques

By gaining more and more experience with a personal modification of the vertical scar technique, the inverted T-incisions became less and less in our service and finally were given up by all team members in 1996.[25] Our technique was influenced by Maillard,[46] who described a Z-plasty technique in 1986, as well as by Lassus.[41]

Between 1988 and 2007, we operated on 3,554 breasts in 2,219 patients in our department. In 2,999 breasts, we used our short scar technique (Table 30.2).

Vertical Scar for All Breast Sizes

The procedure should be performed with the patient in a semi-sitting (beach chair) position with the arms abducted to a 45° angle. The extension in the elbow joint should be 150°. The patient's pelvic region needs to be fixed to the table so that the patient cannot slide down during surgery.

Table 30.2. Total number of breast reductions, patients, and techniques used in our series, 1988–2007.

Total number of breasts	3,554
Total number of patients	2,219
Number of breasts operated with preferred vertical scar technique	2,999
Number of breasts operated with T-incision	320
Number of breasts operated with Z-plasty	170
Number of breasts operated with periareolar technique	52
Number of breasts operated with intra-areolar technique	13

At the beginning of the procedure, the patient receives a single shot of antibiotic, preferably cepholosporins.[55]

The marking is done on the table with sterile markers after skin disinfection and draping.

The line drawn between the sternal notch and the umbilicus marks the midline. Then on both breasts, a line is drawn between the sternal notch and the nipple, and the actual measurements are marked down. The second line is drawn from the mid clavicula (normally in the range of 7–8 cm from the sternal notch) to the nipple. Then the level of the new nipple–areola complex position is marked at 18 cm so that the new nipple position is 20 cm caudolaterally from the sternal notch. A semicircular line is drawn from the line that marks the new cranial rim of the nipple–areola complex medially and laterally along the areola. The drawing looks like the upper part of a figure of 8 (Figure 30.4). To mark the extent of excision in the lower part, a maneuver described by Lejour[42] is performed. Two folds are formed and brought together in the midline (Figure 30.5). If it can be done without remarkable tension, the extent of skin excision should be correct. The folds are marked. They represent the lateral and the medial extent of skin excision. The lower margin of the excision should end at least 3–5 cm above the inferior mammary fold. The marking of the lower pole completes the figure of 8.

In order to facilitate de-epithelialization, a towel is put around the breast and fixed with a towel clip. For smaller breasts, we use the 3.5-cm diameter marking ring for the nipple–areola complex; for larger breasts, 4.0 cm (Figure 30.6). Subsequently, de-epithelialization is performed (Figure 30.7). After de-epithelialization, skin undermining in the medial, caudal, and lateral part is done. In the dorsal part, a channel approximately 6 cm wide is created between the fascia of the pectoralis muscle and the posterior aspect of the gland. The cranial extent of the channel is marked on the skin at the level of the fourth rib (Figure 30.8). This will be the location of the inner fixation stitch later on. Subsequently the extent of glandular excision is marked. The parenchyma (Figure 30.9) is taken from the lateral, medial, lower, and posterior aspect of the gland. After meticulous hemostasis, always with bipolar forceps and irrigation, the fixation of the gland is prepared. Two stitches are used to fix the area behind the nipple–areola complex to the cranial end of the channel (Figure 30.10). We use

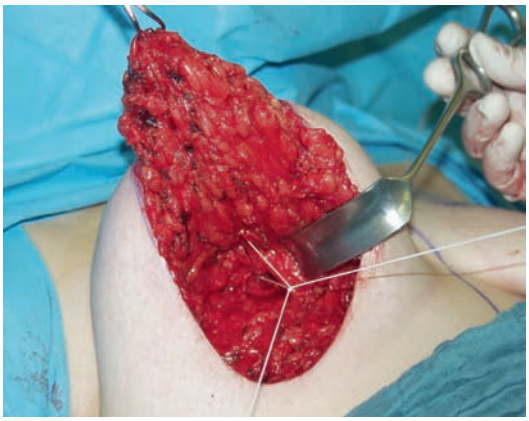

Figure 30.10. Fixation of the posterior aspect of the gland in the retroareolar area to the pectoralis fascia at the level of the third rib.

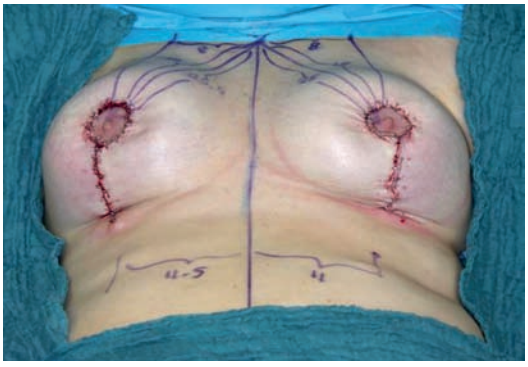

Figure 30.11. Temporary wound closure with staples.

one resorbable and one nonresorbable stitch to fix the gland (2-0 resorbable and 2-0 nonresorbable material). The sutures are tied, and the new position of the gland is evaluated. Then a temporary wound closure is performed with staples (Figure 30.11). This maneuver saves time, and the shape can easily be corrected by fixing the staples tighter or changing the position of the nipple–areola complex. Once the size and symmetry are satisfactory, the staples are removed, one drain is inserted on each side, and the final closure is performed. There is no resorbable material used for wound closure. Two layers of nonresorbable 2-0 suture material are used as an intracutaneous running suture (one deep layer, one superficial layer). On top of these two intracutaneous running sutures, we use single stitches with a 5-0 nonresorbable material. These stitches need to be removed within 7 days.

In order to take away the tension at the nipple–areola complex, a purse-string suture with nonresorbable material is tied around the nipple–areola marker to determine the right diameter (Figure 30.12). After the nipple–areola complex is inserted with intracutaneous running sutures (3-0 nonresorbable material), which need to be interrupted at least three times, and single stitches with 5-0 nonresorbable suture material. The intracutaneous running sutures are closed with an air node to avoid skin necrosis (Figure 30.13). Strips of tape are used for all wounds. Compression dressing and elastic tape are

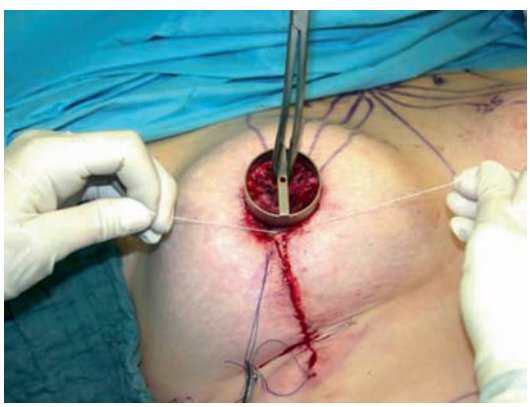

Figure 30.12. Adjusting the diameter of the periareolar skin margins with a purse-string suture with nonresorbable material.

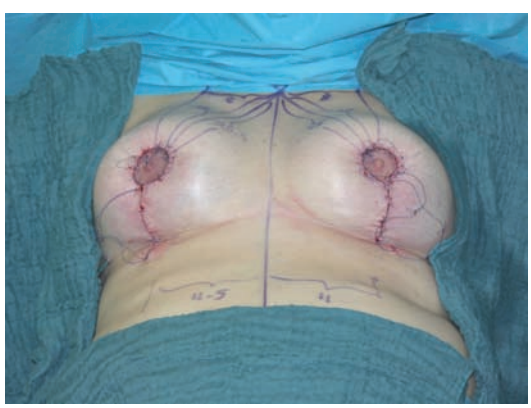

Figure 30.13. Wound closure with non-resorbable material only.

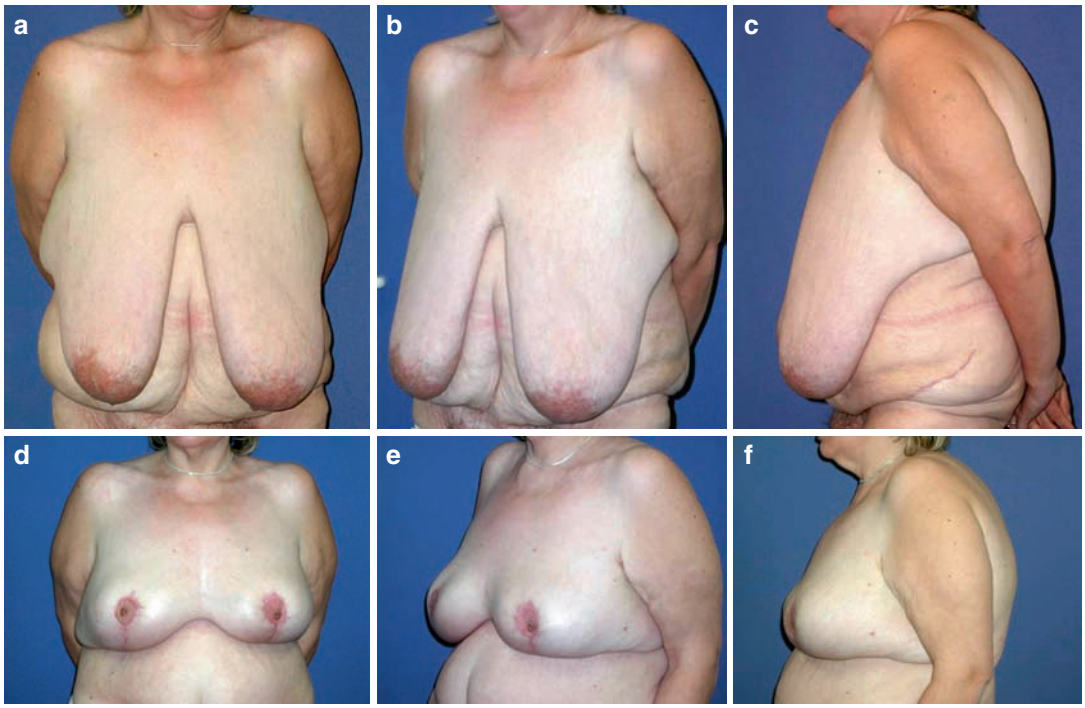

Figure 30.14. Pre- (**A, B, C**) and postoperative (**D, E, F**) images of a patient with sternal notch to nipple distance 60 cm preoperatively and 25 cm postoperatively, using the vertical scar technique described in the section "Vertical Scar for All Breast Sizes."

applied on top. After removing drains (usually after 48 h if less than 20 cc of fluids is evacuated), the patient should start wearing a compression bra for 6 weeks day and night and for another 6 weeks during the day. The bra should create enough compression in the lower pole and avoid sheering forces to the fixation stitches between the gland and the fascia. Postoperatively, the patient is positioned in a 30° elevation of the upper part of the body, and the arms are elevated on pillows. Exercises are not allowed for 6 weeks.

Patients older than 35 years are asked to undergo a mammography 6 months after breast reduction. This is essential to document the amount of scarring for later comparison.

Are there limitations for this technique? The largest volume in our series was 3,250 g resection per side; the longest distance from the sternal notch to the nipple was 60 cm. Pre- and postoperative photographs of this patient are shown in Figure 30.14.

A typical result for an average breast reduction 1 year postoperatively is shown in Figure 30.15.

Preferred Technique for Periareolar Mastopexy

The same procedure as described for reduction can be used for periareolar mastopexy as well. In this case, the skin excision is done with a periareolar excision only (Figure 30.16). After de-epithelialization, the lower pole is opened, and the skin in this area is mobilized. After freeing the gland in the lower pole, the same channel as described in the vertical scar technique is created for the elevation of the gland.

Hemostasis, irrigation, fixation of the gland, and wound closure are done as described in "Vertical Scar for All Breast Sizes."

Intra-areolar Mastopexy

A special technique was presented by Hilton Becker at the IPRAS world congress in Sidney in 2003: reduction of the areola around the nipple. A circular strip of areola skin is excised. Minor reductions can even be performed through this

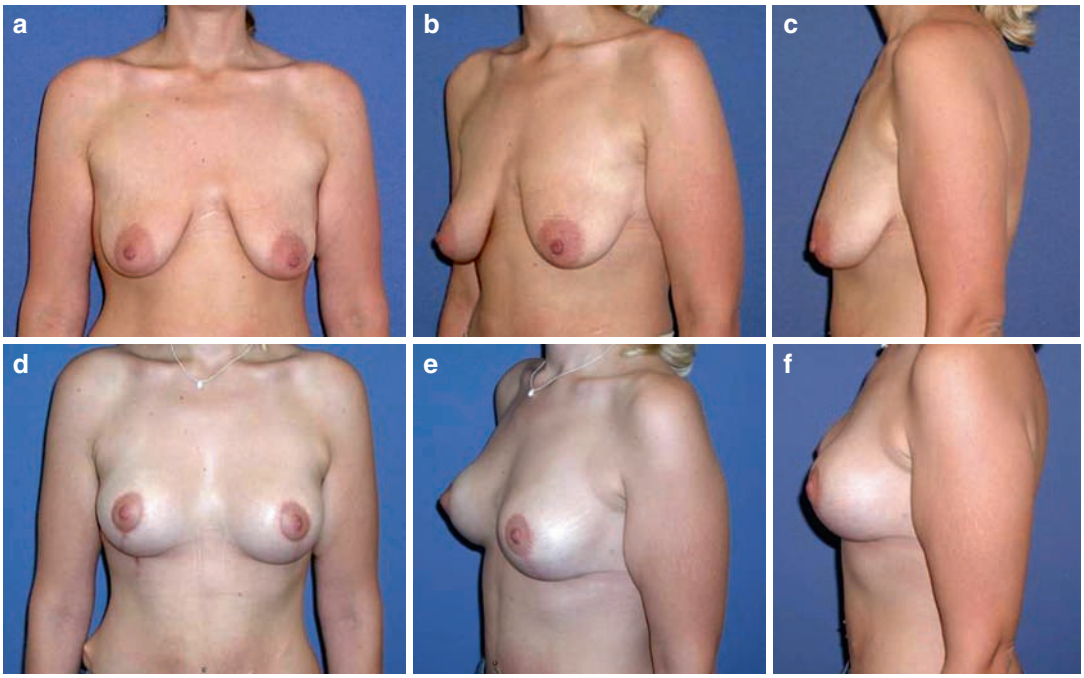

Figure 30.15. A typical result for an average breast reduction 1 year postoperatively.

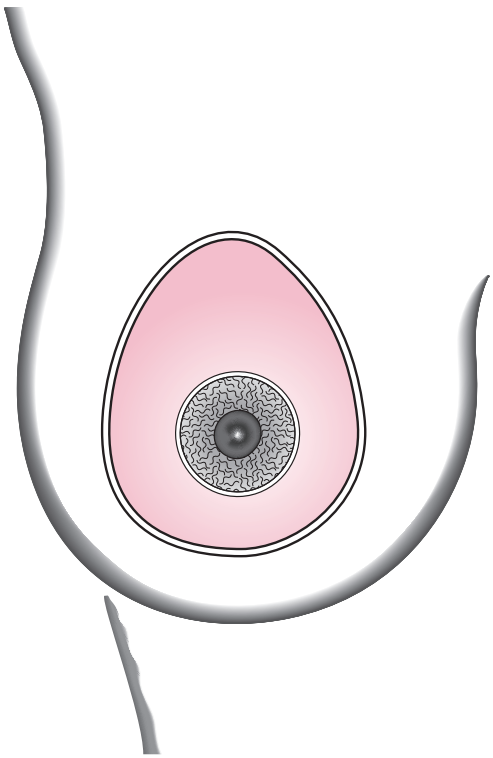

Figure 30.16. Periareolar mastopexy site after de-epithelialization.

excision site (Figure 30.17). This technique can be recommended for minor reductions and/or asymmetries in young patients or for correction of asymmetries of the nipple–areola complex.

Outcomes

Although the complication rate in breast reduction is quite high (between 20% and 32%), with a revision rate of around 10% in most studies, patients' satisfaction is impressively high. All patients report a remarkable improvement of pain and skin problems after surgery.

Patients experience improvement of their psychological condition with better self-esteem and subsequently more energy to start exercising regularly.

These activities are associated with a trend to lose weight and/or the desire for more plastic surgical procedures, e.g. abdominoplasty.[5,10,11,19,34,61,62,64,65]

Complications and Unfavorable Results

Complications and unfavorable results, shown in Table 30.3, are average percentages from multiple studies in which a variety of techniques

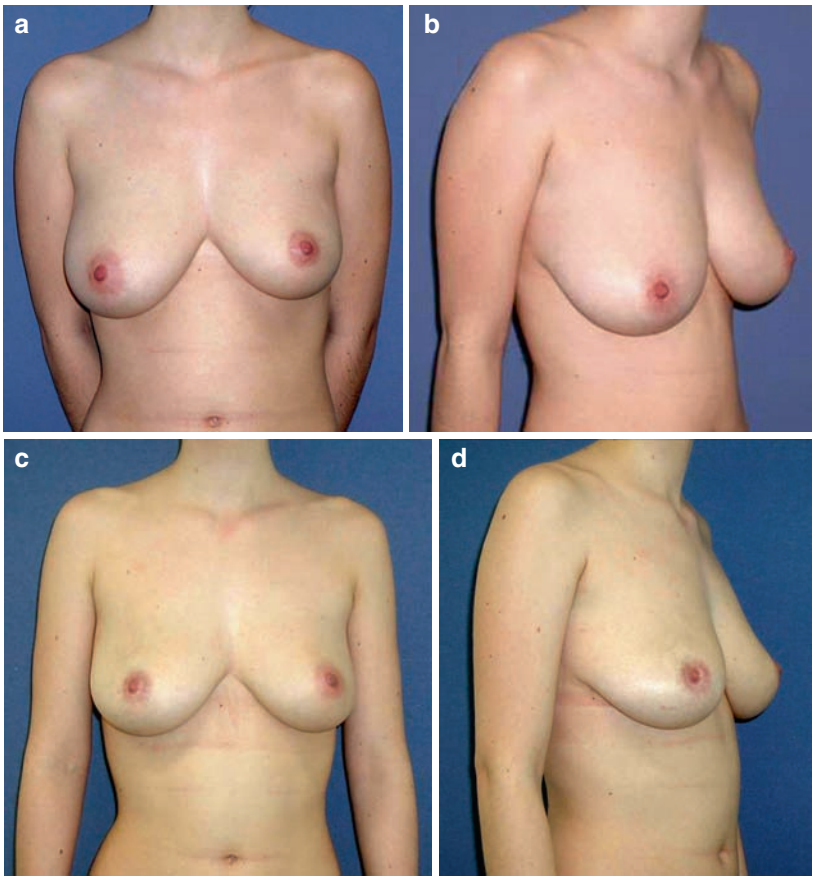

Figure 30.17. Right breast: intra-areolar reduction resulting in a single scar around the nipple.

Table 30.3. Complications and unfavorable results from multiple major studies (in percent).

Partial loss of nipple sensation	80.00
Malposition of the nipple–areola complex	32.00
Asymmetry	23.00
Unfavorable shape	22.00
Nipple areola distortion or inversion	19.00
Wound dehiscence	12.00
Dog ears (estimated)	10.00
Hypertrophic scarring	10.00
Total loss of nipple sensation	8.00
Seroma	4.50
Fat necrosis	1.80
Partial loss of areola/nipple	1.10
Hematoma	0.80
Major blood loss (estimated)	0.50
Total loss of areola/nipple	0.50
Infection	0.40
Deep-vein thrombosis (estimated)	0.04
Average	32.0

were evaluated. It is quite obvious that some techniques have inherent risks, which hardly ever happen in other techniques, but may have less problems with other complications or outcome impairment.[9,16,18,33,36,45,51]

The ability to breast feed varies from technique to technique.[7,24,36,48,68]

Also dependant on the technique are the changes in mammography to be seen postoperatively.[6,59] Some of these changes may be difficult to be distinguished from malignant lesions. Combining mammography with other techniques of examination (ultrasound, MRI) provides a very good safety level for the patient.

Complications, Unfavorable Results, and Outcomes in Our Own Series

In 2002, we had 18 patients with more than 1,000 g reduction weight per side. We were able to examine all of these patients 4 years later.

In this small series of high-volume reductions, we had a complication rate of 27%, which still is within the range of 32% on average, calculated from a number of major studies.

In our whole serious of 3,554 breasts, we had a complication rate of 14%, with a revision rate of 5%.

Most of our complications were minor wound dehiscences. Second were fat necrosis, and third, hypertrophic scarring. The vertical scar hardly ever gets hypertrophic. Normally, it is very inconspicuous although it is not located within the lines of minimal tension and although it is closed under tension. Hypertrophy occurs more often in periareolar scars. Only few of these patients required revision, and in all of our patients, we had only two cases of total areola necrosis due to wound infection (less than 0.1%).

Nipple sensation in our patients was unchanged or improved in 43%, moderately reduced in 50%, and considerably reduced in 7%. We never had a total loss of nipple sensation, except for the two patients with nipple loss.

We had a few reports of uncompromised breast feeding in our patients, although we do not recommend or encourage our patients to breast feed. We tell them that in this technique it would be possible, but they need to know that they are at higher risk for inflammation or formation of cysts!

Two of our patients had a temporary weakness of the deltoid muscle function. Both patients had injury or degenerative disease of the cervical spine prior to breast reduction but they had not mentioned it when their medical history was taken.

Overall, in accordance with other studies, our patients are very content (78%) and content (21%) in our series. The rate of dissatisfaction is below 1%.

We had the chance to take a photograph of a patient 14 years postoperatively: although the patient gained weight, the long-term result of shape was acceptable (Figure 30.18).

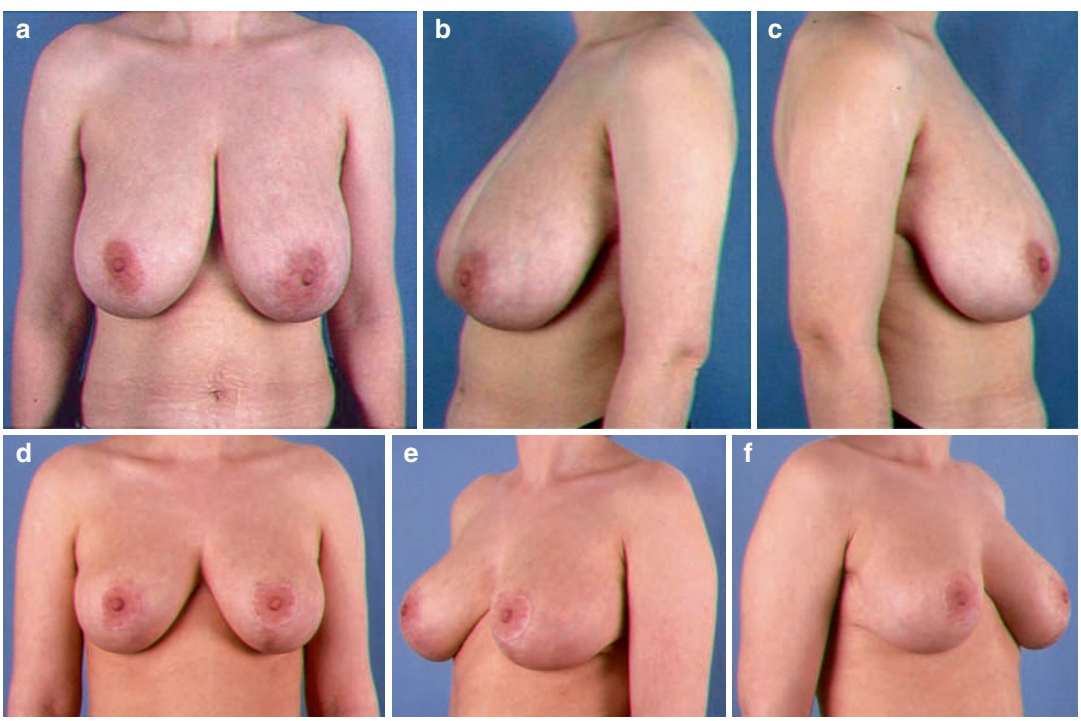

Figure 30.18. Long-term result 14 years after vertical scar breast reduction, described in the section "Vertical Scar for All Breast Sizes."

Medicolegal Aspects

In most countries like the United States and Germany, the rate of lawsuits is increasing. Breast reduction is on top of the statistics in the United States.[20] The majority of breast reduction patients, filing a lawsuit, are dissatisfied with the appearance of the scars.

In our own series, we had one lawsuit: The patient suffered from a weakness of the deltoid muscle function for 4 months, which improved gradually with physiotherapy. The judge decided that the position of the patient during surgery was correct and that the temporary damage to the nerve was an inherent risk.

Outlook

From an economic point of view, it is essential that outcome studies are continued to demonstrate that the morbidity of patients decreases enormously after breast reduction. These patients need less orthopedic treatment and physiotherapy afterward. Mental health is improved.

The conclusion for insurance companies should be that the total amount that they would have to spend for a breast reduction (in Germany currently around €3,000–4,000) is a great investment.[38,39,63] Unfortunately, not all health economists and insurance companies come to this conclusion.

Scars do matter: innovative plastic surgeons continue to look for techniques with less conspicuous scars and less morbidity.

Improving scars would be an enormous benefit for this constantly growing group of patients, mainly due to a growing percentage of overweight individuals in industrialized nations.

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31

Postmastectomy Breast Reconstruction

Robert E. H. Ferguson and David W. Chang

Summary

Among the emotions surrounding the diagnosis of breast cancer is the desire to preserve body form and function. Plastic surgeons have the key role in guiding a patient through the reconstructive options, performing the necessary procedures, and caring for the patient postoperatively. The timing of the breast reconstruction, whether immediate or delayed, should be influenced by potential adjuvant treatments and the patient's psychosocial considerations. A reconstruction may include an implant, autologous tissue, or both. Here, we present an overview of postmastectomy breast reconstruction.

Abbreviations

ALT	Anterolateral thigh (ALT)
DIEA	Deep inferior epigastric artery
DSEA	Deep superior epigastric artery
I-GAP	Inferior gluteal artery perforator (I-GAP) flap
MS-TRAM	Muscle-sparing TRAM
SIEA	Superficial inferior epigastric artery
S-GAP	Superior gluteal artery perforator flap
TRAM	Transverse rectus abdominis musculocutaneous

Introduction

The emotions surrounding a breast cancer diagnosis are complex, but patients generally have two dominant concerns, preservation of life and preservation of body. The plastic surgeon addresses the latter concern by restoring the form of the breasts. Although current methods of reconstruction cannot reestablish the physiologic function of the breasts, breast reconstruction can provide a patient with a sense of restored body image. As one patient confided, "My reconstruction was not part of my cure; it was part of my healing."

The plastic surgeon, the oncologic surgeon, and the patient all play important roles in deciding the type and timing of a postmastectomy reconstruction. The reconstruction options available to the patient are guided by necessary treatments (surgery, adjuvant therapy) and patient characteristics (comorbidities, body habitus, personal preferences). General options for postmastectomy reconstruction consist of autologous tissues, implants, or both.

Timing of Reconstruction

Immediate

Reconstruction of the breast during the same general anesthetic as the mastectomy is known as immediate reconstruction. There are several

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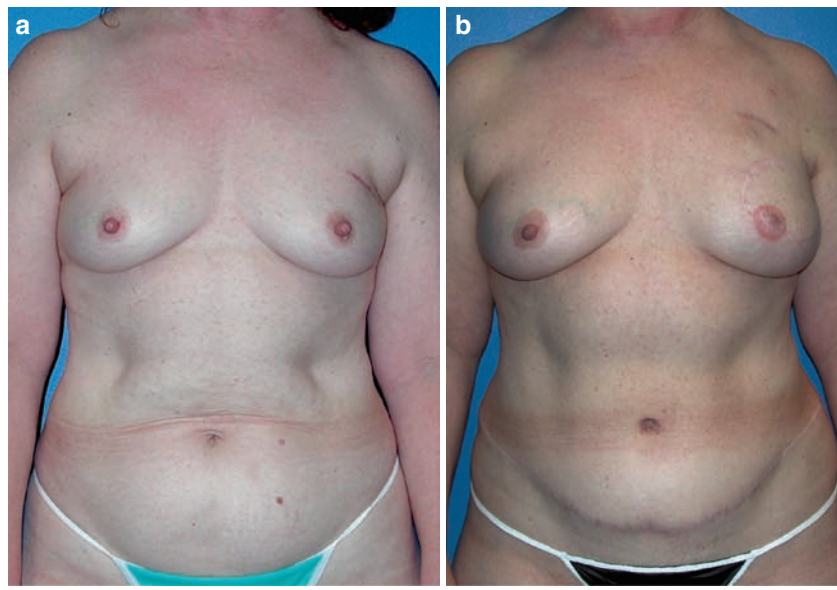

Figure 31.1. (**a**) A patient with a left breast cancer. (**b**) Following mastectomy and immediate reconstruction with a free DIEP flap.

advantages to an immediate reconstruction. For example, if a skin-sparing mastectomy can be performed – in which typically the nipple–areola complex and underlying breast tissue are resected while the breast skin envelope is preserved – the aesthetic outcome of the reconstruction is usually improved, because the form is more likely to be similar to that of the original breast, and the natural skin pigmentations of the patient's chest are maintained (Figure 31.1).

Immediate breast reconstruction may provide a psychological benefit to the patient by decreasing any feelings of physical mutilation and diminished femininity. Studies have demonstrated that women with immediate breast reconstruction may experience less psychosocial morbidity than women who receive mastectomy alone.[7,8,18,21,23,26] The former group may experience less sense of diminished femininity, self-esteem, or sexual attractiveness than the latter group.

Delayed

Breast reconstruction performed at a later date than the mastectomy is known as delayed reconstruction (Figure 31.2). Patients may elect to postpone reconstruction, because they are undecided about undergoing further treatments (such as a prophylactic mastectomy), or because they wish to deal only with the treatment of the cancer before considering reconstructive efforts. Another reason for delay is if the patient needs (or is highly likely to need) adjuvant radiotherapy.

Type of Reconstruction

The general types of breast reconstruction use implants or autologous tissues. Factors affecting decisions about the type of reconstruction may include necessary adjuvant therapies, available tissue donor sites, comorbidities, vocation, and acceptability of donor-site morbidity. Ideally, when consulting a patient about breast reconstruction, the plastic surgeon should know of any neoadjuvant therapy delivered or planned, the surgical oncology plan, and anticipated or planned postoperative adjuvant therapies. At the time of consultation, the plastic surgeon should address the patient's expectations, including those about her ideal breast size, breast shape, and lifestyle. The surgeon should explain the reconstructive techniques available to the patient in terms the patient can understand. The surgeon should also use straightforward terms to explain the risks and benefits of reconstruction as well as alternatives to reconstructive surgery. Each patient places her own subjective value on

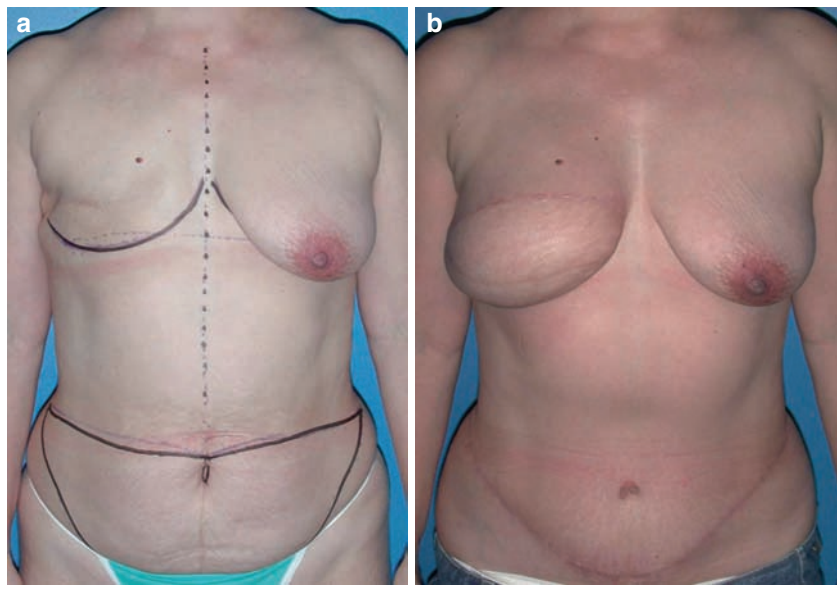

Figure 31.2. (**a**) A patient with acquired absence of right breast following mastectomy is marked for delayed breast reconstruction with a free TRAM flap. (**b**) Following delayed right breast reconstruction with a free MS-TRAM flap.

the presented and perceived risks and benefits of breast reconstruction, and she should feel empowered to make decisions about what is done with her body, provided they are in agreement with sound surgical guidelines.

Tissue Expanders and Implants

Perhaps because of its simplicity, implant-based breast reconstruction is the most common option for postmastectomy breast reconstruction. An implant-based reconstruction may be selected out of necessity or preference; for example, a patient may not have adequate donor sites for autologous tissue reconstruction, or she may not wish to incur donor-site morbidity.

The immediate benefits of an implant-based reconstruction include a shorter operative time, a shorter hospitalization, no donor site, and faster initial recovery, which usually allow the patient to return to work and her normal routines relatively quickly. The disadvantages of an implant-based reconstruction may include less natural ptosis, implant infection or rupture, capsular contracture, and frequent clinic visits during the expansion phase.

Implant-only reconstruction in an irradiated chest is not advisable; the irradiated tissues do not tolerate expansion well, thus increasing the risk of tissue necrosis, implant exposure, capsular contracture, and infection.

As with every method of breast reconstruction, the preoperative consultation should include a discussion of the patient's current or premastectomy breast size and whether she would like to have a breast mound of similar, smaller, or greater volume after the procedure. Measurements of the breast should include, at a minimum, the diameter so that the surgeon can select an expander and implant with an appropriate base width.

The most common method of implant-based breast reconstruction involves a tissue expansion phase followed by exchange of the expander for a permanent implant. Expanders used for breast reconstruction typically have textured surfaces and integrated fill ports. Recently developed expanders are also shaped to allow for preferential expansion of the inferior pole. Expanders vary in width, height, and volume; therefore, a surgeon should select an expander based on the patient's breast measurements and chest wall dimensions.

In an immediate breast reconstruction, the mastectomy incision is used for placement of the tissue expander. In a delayed breast reconstruction,

the mastectomy scar may be used to access the deep tissues to create the expander pocket. Under direct visualization, the surgeon creates a pocket below the pectoralis major muscle and takes care to cauterize or ligate any perforating vessels along the medial and inferomedial borders of the muscle. The inferior and medial aspects of the pectoralis major muscle are detached from the chest wall for optimal placement of the expander whenever possible. The inferior border of this pocket should be just below the level of the inframammary fold. The lateral border of the pocket should correspond to the lateral border of the breast, and the medial extent of the pocket should correspond to the medial border of the breast or the proposed breast mound. If complete muscular coverage is desired, the serratus anterior muscle may be partially elevated from the ribs and sewn to the lateral border of the pectoralis major muscle. The inferior boundary of the pocket may remain below the junction of the rectus fascia and rectus insertion.

Once the expander is placed and the wound is allowed a few days to a week to heal, percutaneous saline expansion is begun. The expander volume suggested by the manufacturer, the initial volume instilled at the time of expander placement, the volume instilled at each subsequent visit, and a running total of the volume should all be accurately and clearly documented. The patient's comfort level and an examination of the patient's tissues help determine the amount of saline added at each expansion session. If the patient expresses discomfort, expansion should stop, and the volume placed should be recorded. Likewise, if the tissues begin to blanch and demonstrate ischemia, or if the incision repair threatens dehiscence, expansion should stop, and the patient should be allowed several days of rest so that the tissue can recover before the next expansion session. The amount of time necessary for expansion depends on the volume goal for the breast mound and the amount of expansion volume tolerated at each session.

When the volume goal is achieved, some surgeons prefer to then overexpand the mound to offset tissue recoil after the expander is removed. Regardless, once the desired mound volume is achieved, it is wise to wait about 3 months before removing the expander and exchanging it for the implant.

Before exchanging the expander for a permanent implant, the type of implant to be used for reconstruction should be discussed with the patient. At the time of this writing, saline and silicone implants are available.

For removal of the expander, previous incisions may be used to access the implant pocket. After the surgeon removes the expander, he or she may perform a capsulotomy if necessary. If the reconstruction is unilateral, any procedures to the contralateral breast needed to achieve symmetry, such as reduction, mastopexy, or augmentation, may be performed during this phase of the reconstruction as well.

Autologous Tissue

The surgeon's overall goals in performing an autologous tissue breast reconstruction are to transfer well-vascularized tissue to the mastectomy site and to create a breast mound that appears as anatomically and aesthetically normal as possible.

Using tissue from her own body to reconstruct a breast can be an attractive option to a patient for a variety of reasons. The structure and consistency of the original breast can be approximated with an autologous reconstruction, particularly in the immediate setting. In addition, a foreign object usually does not need to be implanted in the body – a benefit for women who are not comfortable with the idea of implants. Some patients like to think of autologous breast reconstruction as not only providing a new breast but also eliminating unwanted, redundant tissues, such as tissue from the abdomen; however, the surgeon should review the reality of scars, contour abnormalities, and other potential donor-site morbidities with the patient in such an instance.

Latissimus Dorsi Flaps

The use of the latissimus dorsi musculocutaneous flap was one of the first described methods of breast reconstruction,[13] and this flap remains a versatile option in postmastectomy reconstruction. The latissimus dorsi muscle is a broad, flat, roughly triangular muscle; it extends from the spine of the thoracic vertebrae and sweeps inferiorly and laterally to the posterior iliac crest.[11] The muscle's striations run in a superior and lateral direction and converge onto a narrower, spiraling insertion at the intertubercular groove of the humerus between the pectoralis major and the teres major muscles. The thoracodorsal vessels are the primary sources of perfusion in

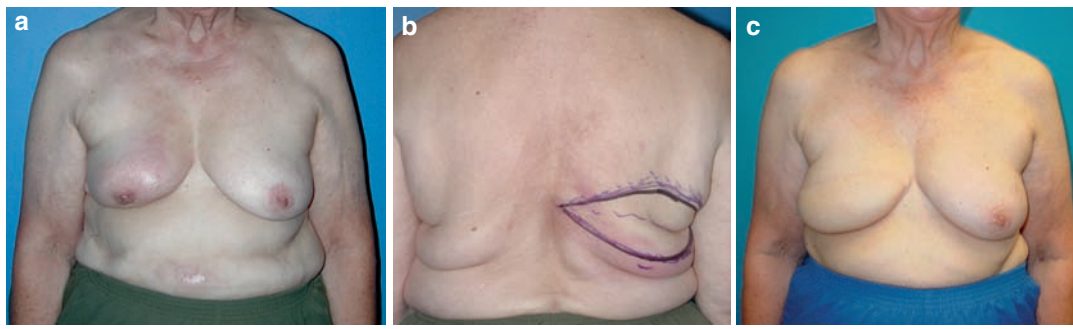

Figure 31.3. (**a**) A patient with a right breast cancer. (**b**) Design of an ELD flap. (**c**) Following immediate reconstruction of right breast with an (ELD) extended latissimus dorsi flap.

the latissimus dorsi muscle; segmental perforating vessels from posterior intercostal and lumbar arteries and veins provide secondary perfusion. The thoracodorsal nerve (C6–8), which courses lateral to the vascular pedicle, innervates the latissimus dorsi muscle. The latissimus dorsi muscle adducts, extends, and medially rotates the humerus.

As a musculocutaneous flap, the latissimus dorsi flap can provide basic coverage for a chest wall reconstruction following a salvage mastectomy or can be the key factor in a more elegant, pleasing breast reconstruction. There are a number of reasons to use the latissimus dorsi muscle for breast reconstruction. The patient may be a poor candidate for reconstruction using abdominal tissue reconstruction because of extensive abdominal scarring, insufficient infraumbilical soft tissue redundancy, obesity, or previous body contouring procedure, or because she desires future pregnancies; she may desire implant-based reconstruction yet has insufficient or irradiated chest tissues, a segmental mastectomy defect, or a salvage mastectomy defect; previous autologous tissue reconstruction may have failed; or she may be unwilling to accept the recovery times or donor-site morbidities associated with other autologous tissue reconstructions.

Because its volume alone is usually insufficient to reconstruct the breast, the latissimus dorsi flap is commonly used in conjunction with an implant. Although the latissimus dorsi muscle is often considered expendable, sacrificing this muscle may produce undesired weakness in patients who rely on upper body strength because of lower extremity disability or in patients with athletic professions or pastimes.

Variants of the latissimus dorsi musculocutaneous flap have been used in an attempt to eliminate the need for an implant and thus allow for a completely autologous tissue breast reconstruction. These flap options include a latissimus dorsi flap with a large, buried, de-epithelialized skin paddle[14]; a fleur-de-lis skin paddle design[19]; and an extended latissimus dorsi musculocutaneous flap[6,12] that may include parascapular, scapular, and lumbar fat and fascia to increase breast mound volume.

At our institution, we reviewed the use of an extended latissimus dorsi musculocutaneous flap for breast reconstruction (Figure 31.3).[6] We found it to be a reliable flap that provides good to excellent results, particularly for reconstruction of small- to medium-sized breasts. Donor-site complications primarily included prolonged drainage and seroma formation and were associated with body mass indexes greater than 30 kg/m^2.

TRAM Flaps

The use of the transverse rectus abdominis musculocutaneous (TRAM) flap in breast reconstruction was first described by Hartrampf[30] in 1982. Since that time, the TRAM flap has undergone multiple modifications and refinements, yet it has remained a reliable option for postmastectomy breast reconstruction. It is an excellent option for an otherwise healthy patient who has the adequate abdominal soft tissue redundancy to achieve the goal volume for the reconstructed breast. TRAM flap-based breast reconstruction has the potential advantage of recreating a breast mound without the need for an implant as well as essentially performing an abdominal lipectomy at the donor site.

The anatomy of the TRAM flap merits review. The rectus abdominis muscles are a pair of muscles that originate from costal margin levels six through eight, traverse the central abdomen longitudinally (with intermittent fascial insertions or inscriptions), and insert at the pubic crest and tubercle.[11] The rectus abdominis muscle has two vascular pedicles: one composed of the deep superior epigastric artery (DSEA) and the other composed of the deep inferior epigastric artery (DIEA).[5] The DSEA and DIEA pedicles arborize as they approach each other under the surface of the rectus abdominis muscle. These two systems connect above the umbilicus through a system of small-caliber vessels that Taylor and Palmer refer to as "choke" vessels.[24]

The DSEA arises from the internal mammary artery at the level of the sixth intercostal space and approaches the rectus abdominis muscle from the muscle's medial undersurface. The DSEA continues along the undersurface of the muscle in a longitudinal direction until it reaches the choke vessels that anastomose with the DIEA. The DSEA generally has two venae comitantes. A small branch of the DSEA courses along the costal margin to join the intercostal artery lateral to the rectus sheath.

The DIEA originates 1 cm above the inguinal ligament from the medial aspect of the external iliac artery directly opposite the deep circumflex iliac artery. The main DIEA pierces the transversalis fascia and enters the rectus sheath just below the arcuate line. The DIEA then ascends obliquely and medially between the rectus abdominis muscle and the posterior wall of the sheath. The DIEA has two venae comitantes that join to form a single vein prior to their junction with the external iliac vein.[5]

These deep arteries supply the TRAM flap's overlying abdominal skin with a system of perforators that traverse the rectus abdominis muscles and communicate with a subcutaneous network.

A TRAM flap incorporates skin from the entire lower abdomen, usually in an elliptical fashion similar to an abdominal lipectomy. Four different skin zones can be included in a TRAM flap.[22] Zone 1 includes the skin overlying the ipsilateral rectus abdominis muscle. Zone 2 comprises the skin across the midline overlying the contralateral rectus abdominis muscle. The skin lateral to the ipsilateral linea semilunaris makes up zone 3, whereas the skin lateral to the contralateral linea semilunaris makes up zone 4. The tissue with the most tenuous blood supply is that of zone 4.

Pedicled TRAM Flap

The conventional pedicled TRAM flap is based on the DSEA and necessitates sacrifice of all or most of the rectus abdominis muscle. Depending on the planned inset configuration, an ipsilateral or contralateral pedicle may be selected; however, the surgeon must ensure that kinking, twisting, or compression does not compromise the blood supply or outflow. Once an opening in the medial aspect of the inframammary fold is established, the skin paddle is positioned into the breast. The surgeon creates the breast mound by tailoring and insetting the flap vertically (typically with zone 3 in the caudal position) or transversely.

Donor sites for conventional TRAM flaps usually leave a fascial defect that necessitates repair with mesh. The repair should provide adequate anterior abdominal wall integrity without displacing the umbilical stalk from its midline position. It may be necessary to plicate the contralateral abdominal fascia to re-center the umbilical stalk and to provide contour symmetry. To avoid compromising skin paddle vasculature, closure of the rectus sheath should end 1–2 cm below the pedicle. To facilitate tension-free closure, it may be necessary to undermine the remaining abdominal skin; however, releasing the tissues past the border of the costal margin is usually unnecessary. Draping of the abdominal skin is simulated to mark the umbilical window. The umbilical window can be created before or after abdominal closure as long as the surgeon's technique allows for nontraumatic retrieval of the umbilical stalk. The superficial fascial system, Scarpa's fascia, is reapproximated before the closure of the dermis and final closure of the skin.

Free TRAM Flap

First described in 1979 the free TRAM flap has several features that make it well suited to breast reconstruction.[9] The DIEA is the dominant vessel of the rectus abdominis muscle and the abdominal wall skin, which accounts for the robust blood supply of the free TRAM flap.[5,24]

As it became clear that the rectus abdominis muscle is necessary to the free TRAM flap only as the structure through which blood vessels pass,

surgeons began to decrease the amount of muscle harvested with the free TRAM flap. This is now known as a muscle-sparing TRAM (MS-TRAM) flap (Figure 31.4). Three types of free MS-TRAM flaps can be performed depending on the location and orientation of the perforators: the medial portion of the rectus abdominis muscle can be preserved and a lateral portion of the muscle harvested with the flap (MS-1M); a lateral portion of the rectus abdominis muscle can be preserved and the medial portion of the muscle harvested with the flap (MS-1L);or a small cuff of muscle around the perforators can be harvested with the flap, leaving most of the rectus abdominis muscle intact (MS-2) (Figure 31.5).[17]

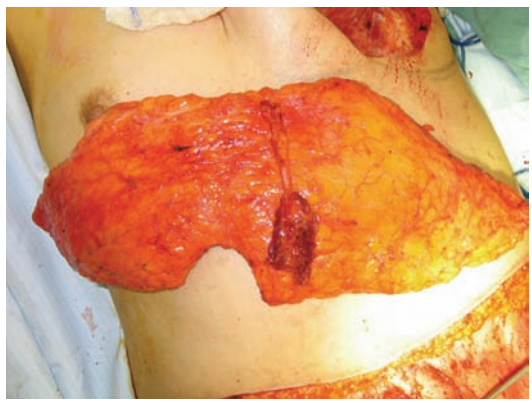

Figure 31.4. A free MS-TRAM flap.

DIEP Flap

If the perforating vessels alone are harvested, thus sparing the entire rectus abdominis muscle, the resulting flap is referred to as a deep inferior epigastric perforator (DIEP) flap (Figure 31.6).

Sparing the entire muscle potentially reduces donor-site morbidity, including abdominal bulge and weakness. However, it is controversial whether DIEP flaps[3,15–17] reduce these complications significantly more than MS-2 free TRAM flaps.[17]

Because a DIEP flap has fewer perforators than a free TRAM flap, there is a concern that the reconstructed breast mound might have an insufficient blood supply and cause fat necrosis in the breast mound.[10] Patient selection as well as intraoperative decision making is crucial to maximize the potential benefit of DIEP flap breast reconstruction.

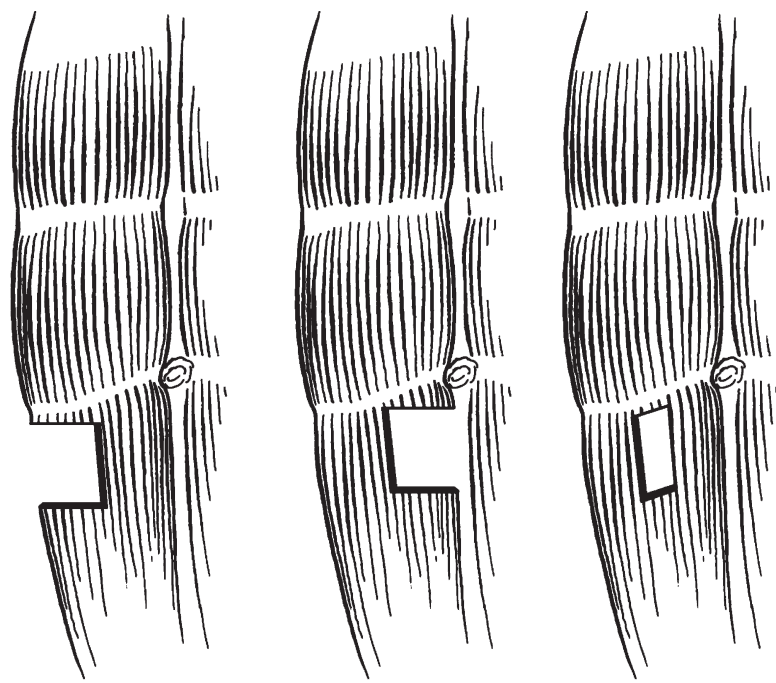

Figure 31.5. Three types of free MS-TRAM flaps can be used depending on the location and orientation of the perforators.

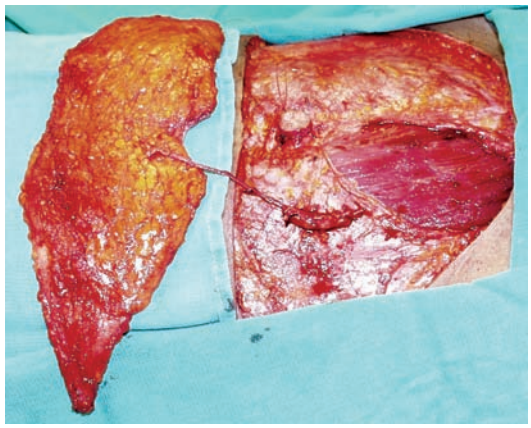

Figure 31.6. A DIEP flap.

The DIEP flap requires advanced reconstructive skills and the ability to adapt the procedure intraoperatively to suit the patient's individual anatomy. The surgeon's goal should be to perform a reconstruction that will leave the patient with the least morbidity possible while at the same time providing her with the greatest chance at a successful reconstruction.

SIEA Flap

The Superficial inferior epigastric artery (SIEA) flap is based on the superficial inferior epigastric artery, which arises from the femoral artery. The main advantage is that the rectus fascia is not violated, thus resulting in minimal donor-site morbidity. However, its use is limited by the variability of the superficial inferior epigastric artery and the reliability of the flap perfusion. Most surgeons limit the use of the SIEA flap to a hemi flap, discarding the tissue across the midline.

Other Flaps

If a patient desires autologous tissue breast reconstruction but is not a candidate for reconstruction with abdominal or back tissues, other donor sites may be available depending on the patient's body habitus and her willingness to accept donor-site morbidities. Options may include the free superior gluteal artery perforator (S-GAP) flap, the free inferior gluteal artery perforator (I-GAP) flap, the free anterolateral thigh (ALT) flap, and the free gracilis musculocutaneous flap.

Gluteal flaps were initially described for breast reconstruction as musculocutaneous flaps. With the development of the perforator concept, the S-GAP flap and then I-GAP flap allowed the surgeon to harvest only skin and fat from the gluteal region, reducing donor-site morbidities such as gait abnormality or femoral instability.[1,4] As with most perforator flaps, sparing the muscle provides the added benefit of a longer effective free flap pedicle. A drawback of all gluteal flaps is that the buttock's adipose tissues differ from those of the abdomen. Buttock tissue tends to be firmer; as a result, using it in breast reconstruction may provide more projection than TRAM or DIEP flaps. The soft tissue volume of the superior gluteal region is usually adequate for the breast tissue requirements for reconstruction. Another criticism of the GAP flap is the donor site. Once the donor site is closed, its scar prominently traverses the buttock, thereby diminishing the buttock's aesthetic appeal.[2] Hollowing of the superior gluteal region and flattening of the inferior region may be exaggerated in unilateral cases and may require symmetry-correcting procedures on the contralateral buttock. The scarring and contour changes at the donor site should be clearly described to the patient preoperatively (Figure 31.7).

Breast reconstruction with an ALT flap or a gracilis musculocutaneous flap is an option for patients who have adequate fat in the thigh for the needed breast volume and who are less-than-ideal candidates for other reconstructive options.[20,25] As in breast reconstruction with abdominal tissues, the location of the donor site is distant from the breast and permits the patient to remain in a supine position throughout the procedure; this facilitates simultaneous harvest and mastectomy.

Nipple–Areola Complex Reconstruction

Some women may choose not to undergo nipple–areola complex reconstruction; however, many women feel that the reconstruction is not truly complete until the nipple-areola complex is reconstructed. The nipple reconstruction is usually delayed until the surgeon and patient are satisfied with the breast mound's position, contour, volume, and relation or symmetry to the contralateral breast or breast mound.

Typically, the new nipple is positioned along the breast median at the level of the inframammary fold or at the point of maximum breast

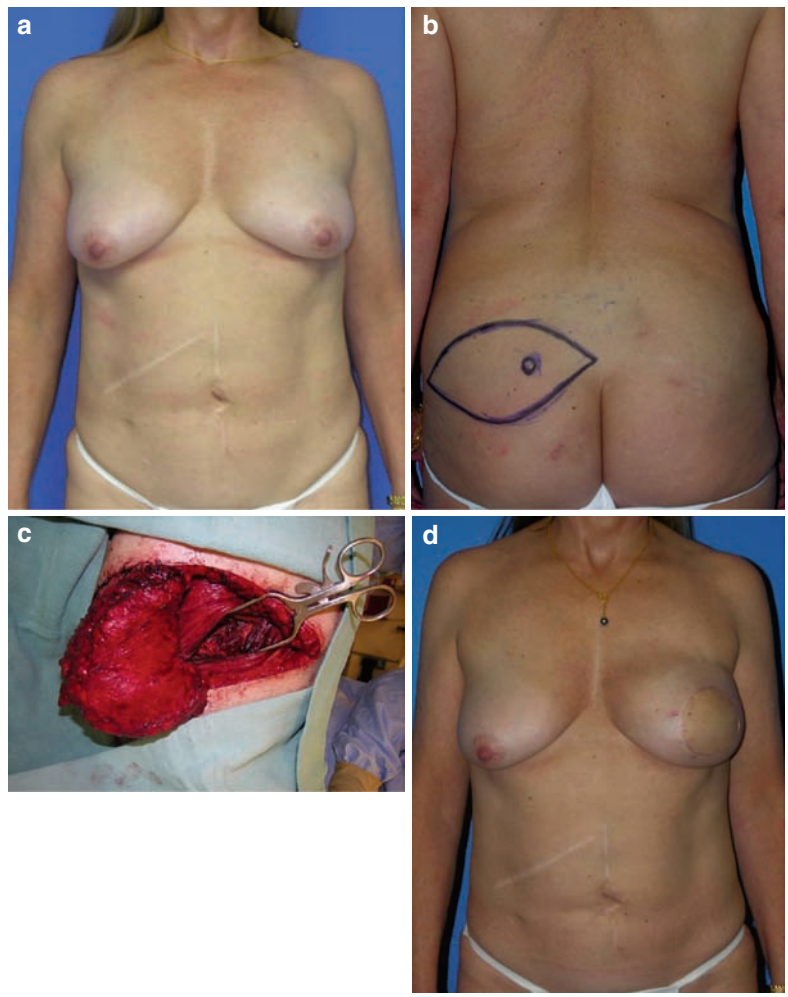

Figure 31.7. (**a**) A patient with left breast cancer and a history of multiple abdominal surgeries. (**b**) Design of an S-GAP flap. (**c**) An S-GAP flap. (**d**) Following immediate reconstruction of left breast with a free S-GAP flap.

mound projection. The patient may participate in choosing the nipple position. Marking this position preoperatively while the patient is in a standing or upright position is recommended.

Different techniques can be employed to reconstruct the nipple. Often, the nipple is reconstructed with skin and subcutaneous tissues on the breast mound. Several designs, including the skate flap, the double-opposing tab flap, and the c-v flap, have been described and are used frequently. In general, a random pattern flap, consisting of skin and subcutaneous tissue, is elevated and wrapped around itself to create a projection similar to a nipple. Because contraction and fat atrophy may reduce the amount of projection over time, it is wise to slightly exaggerate the nipple projection. In unilateral reconstruction, if the contralateral nipple–areola complex is large enough, part of it can be harvested and grafted onto the reconstructed breast. These composite grafts may be harvested from the lower or distal aspect of the contralateral nipple.

An areola can be constructed around the new nipple. The simplest approach is to use the same pigment selected for the reconstructed nipple and tattoo a circle around that nipple that is identical in circumference to the contralateral areola. The areola may also be recreated with a skin grafting technique. After a circle around the reconstructed nipple is de-epithelialized, a full-thickness graft is secured over the vascular bed. This graft may be harvested from a dog-ear

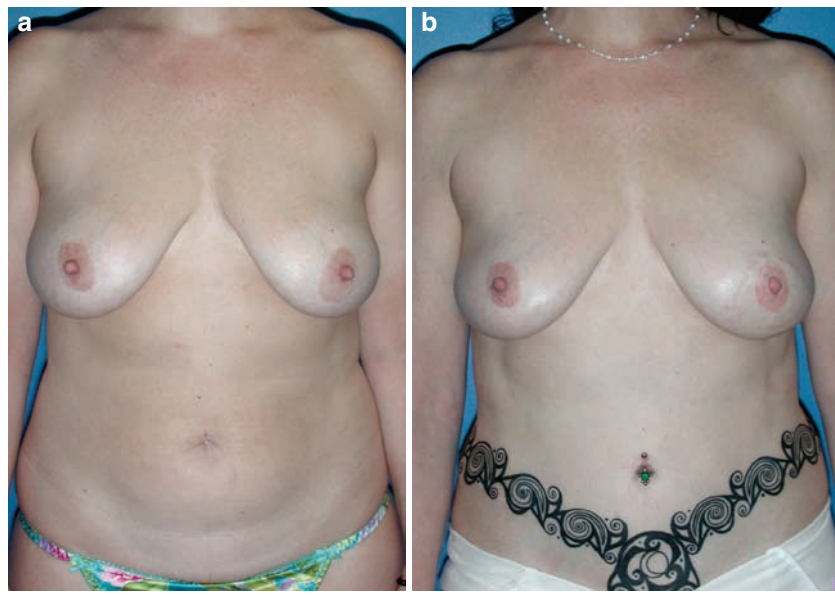

Figure 31.8. (**a**) A patient with left breast cancer. (**b**) Following immediate reconstruction of left breast with a free DIEP flap.

revision at a TRAM or DIEP donor-site scar; alternatively, it may be harvested from the groin, gluteal crease, or upper inner thigh. In Caucasians, a graft from the upper inner thigh often contrasts with the breast mound skin in a way similar to that of the contralateral areola.

Conclusion

In our practice, it is not uncommon for a patient to feel that although treating her breast cancer may have saved her life, reconstructing her breast has restored her life (Figure 31.8). Many options are available for breast reconstruction. When deciding which option to use, the surgeon should consider the patient's disease and treatment as well as her body habitus, comorbidities, lifestyle, and preferences. When the appropriate technique is used, postmastectomy breast reconstruction can be rewarding for both the surgeon and the patient.

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32

Augmentation Mammoplasty

Peter A. Kreymerman and Silvia Cristina Rotemberg

Summary

Breast augmentation increases the size and enhances the shape of a woman's breasts, but more importantly, it will improve her self-esteem. The history of breast augmentation has been made interesting by such sentinel events as the introduction of silicone implants by Cronin and Gerow in 1963, the subsequent placement of a moratorium on silicone gel implants by the FDA in 1992, and the repeal of that moratorium in 2006 after numerous studies demonstrating their safety. The idea that a well-planned operative approach will facilitate good long-term results is paramount. The various types of implants, incisions, and pocket locations are discussed. The indications, advantages, and disadvantages of the various techniques are explained, including endoscopic transaxillary and endoscopic transumbilical augmentation. Clinical examples with patient photographs, understanding of possible complications, and knowing the techniques that are on the horizon are also key parts of the chapter.

Abbreviations

FDA	Food and Drug Administration
MRI	Magnetic resonance imaging
TUBA	Transumbilical breast augmentation

Introduction

The goal of augmentation mammoplasty is to improve a woman's self-esteem by increasing the size and enhancing the shape of her breasts. Without a well-planned operative approach, a long-term result without complications is not a simple task. Breast augmentation has been a popular procedure since the introduction of silicone gel implants in 1963 by Cronin and Gerow. Many different techniques, various types of implants, and a storm of political debate have followed.

In 1992, the FDA placed a moratorium on silicone gel implants used for augmentation. This was done due to fear of product failure, malignancy, and induction of autoimmune diseases. Saline implants continued to be used and were then sanctioned by the FDA in 2000. Subsequently, studies were performed that demonstrated the safety of gel implants, disproved their correlation with autoimmune diseases, and showed that mammography would still be performed accurately. On November 17, 2006, the FDA approved the marketing of silicone gel implants by Allergan Corp. (Irvine, California) and Mentor Corp. (Santa Barbara, California) for breast reconstruction in women of all ages and for breast augmentation in women at least 22 years old. The FDA warns that breast implants are not considered to be a lifetime device. To diagnose rupture, they recommend an MRI at 3 years after the initial implant placement and every 2 years

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thereafter. The FDA also notes that MRI screening may not be covered by medical insurance.

Augmentation techniques vary based on incision type (inframammary fold, periareolar, axillary, or umbilical), location of the implant (subglandular or submuscular), and type of dissection (open or endoscopic). Through the years, many types and generations of implants have been introduced, but implant selection can essentially be reduced to four questions: Size? Silicone or saline? Smooth or textured? Round or anatomic?

Anatomy

The bulk of the breast is composed of subcutaneous fat and parenchymal tissue. The pectoralis major and minor muscles are just posterior/deep to the breast mound (Figure 32.1). The ideal female nipple location is in the midclavicular plane and overlays the fourth intercostal space. The vascular supply to the breast is rich and redundant. The breast receives branches from the thoracoacromial artery superiorly, specific mammary branches from the internal thoracic artery medially, branches from the lateral thoracic artery laterally, and perforators from the intercostal arteries (Figure 32.2).

Cutaneous breast innervation is via the lateral and anterior cutaneous branches of T2 to T7 intercostals. Cutaneous branches pass through the chest wall deep fascia laterally at the midaxillary line and anteriorly just lateral to the sternum. The nipple–areola complex is most consistently supplied by the lateral branch from the T4 intercostal nerve. Independent of the type of incision, the incidence of permanently

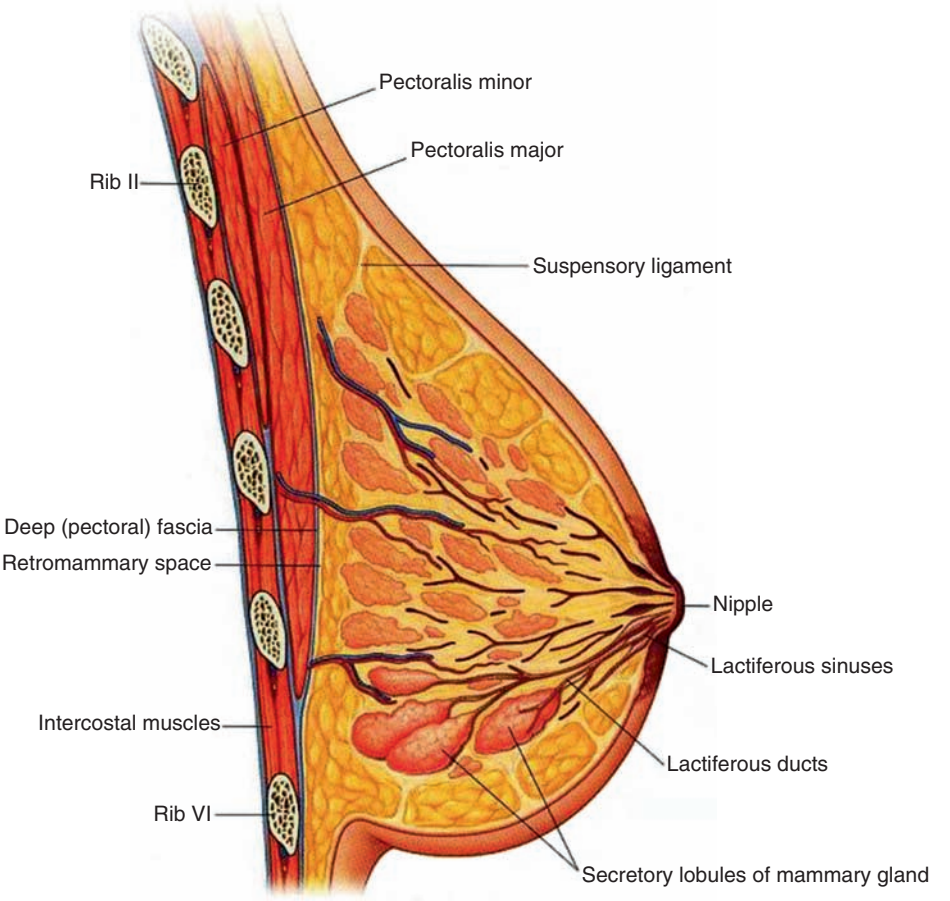


Figure 32.1. Breast and chest wall relationship. (Reprinted with permission from Drake R, et al. *Gray's Atlas of Anatomy*. Copyright Elsevier 2008.)

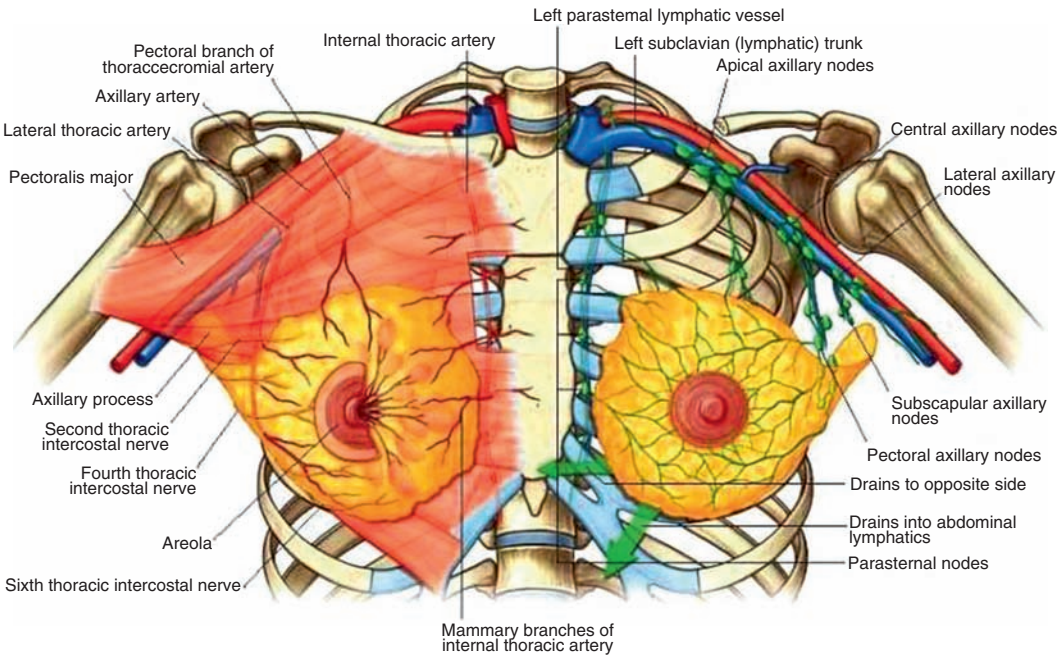


Figure 32.2. Vascular supply, innervation, and lymphatic drainage of the breast. (Reprinted with permission from Drake R, et al. *Gray's Atlas of Anatomy*. Copyright Elsevier 2008.)

decreased sensation after subglandular breast augmentation is near 15%. This may occur from dissection of the supramuscular lateral pocket, whereas submuscular augmentation reportedly preserves greater nipple sensitivity.[12,16,18]

Preoperative Planning

Patient Evaluation

The preoperative evaluation must include assessment of patients' overall health, their expectations, and motivations. Patients seeking augmentation to please their partners should be a red flag to the surgeon. A complete personal and family history for benign and malignant breast disease should be obtained. Mammograms are performed as indicated by the screening guidelines of the American Cancer Society. A pregnancy history should be asked. During physical examination, it is important to point out to the patient her breast asymmetries: nipple and inframammary fold position, amount of ptosis, and the fact that one breast is never the same volume as the other. Chest wall asymmetries and spine deformities can produce an asymmetric result and should therefore be noted preoperatively. Skin quality and soft tissue thickness should be noted. The presence of stretch marks can be accentuated after implant placement, whereas the implant can become easily palpable, or rippling may be a problem in a patient with minimal soft tissue coverage. It is important to document nipple sensation preoperatively. Measurement of the breast base diameter is an important tool to help choose implant size and give the patient a natural looking and shapely breast. The implant diameter should not exceed the base diameter of the breast. Additionally, photographic documentation is vital.

Informed Consent

The procedure should be thoroughly discussed with its risks, alternatives, complications, and need for possible secondary surgery in the future. Two consultation visits are encouraged. On the first visit, the patient is given information to read regarding saline and silicone implants, while on her return visit questions are answered, implant selection is made, and the surgery is scheduled.

Marking

In the preoperative anesthesia unit, with the patient in a sitting or standing position, both inframammary folds and the midline are marked. The inframammary folds must be marked preoperatively, because their exact position becomes distorted once the patient lays supine on the operating table.

Selection of Implant, Incision, and Pocket Location

Implant selection is based on the patient's request of gel or saline (Figure 32.3), desired amount of augmentation, and her native breast measurements. Implant companies make different profile implants (Figure 32.4). The patient's breast base diameter must be able to accommodate the diameter of the implant. When a low-profile implant and high-profile implant with the same diameter are compared, the high-profile implant will contain a larger volume and also provide the patient more projection. Both implant manufactures provide tables with volumes and dimensions of all their implant types (Figure 32.5).

Available incisions include inframammary, periareolar, axillary, and periumbilical (Figure 32.6). Selection of the implant type and size can have a direct bearing on the incision location and size. If the patient selects a large gel implant and she has a relatively small areola, a periareolar incision in this patient may be too small to accommodate the insertion of that implant. For similar reasons, a patient requesting a small axillary incision with endoscopic dissection or a transumbilical augmentation will not be able to have a gel implant. Otherwise, incision selection is based on patient and surgeon preference. It should be noted that periareolar incisions theoretically decrease nipple sensitivity and patients may have more difficulty breast feeding. Additionally, a superior periareolar incision with skin excision can be used to correct a small amount of ptosis.

Submuscular and subglandular pockets each have their distinct advantages (Figure 32.7). In submuscular placement, the pectoralis major muscle covers the implant superiorly, making the implant less visible and decreasing medial-superior rippling. Additionally, due to increase in overlying soft tissue, the incidence of clinically significant capsular contracture is lower. With a submuscular dissection, there is a higher preservation of nipple sensation. The lateral cutaneous branches supplying the nipple course within the pectoral fascia and are undisturbed with this approach.[19] Submuscular relative to subglandular placement is also associated with an improved amount of visualized breast tissue during mammography.[13,20]

Subglandular placement is technically easier to perform and less bleeding is encountered. This approach provides a lift to the breast if mild ptosis is present, gives a more natural looking result, and is associated with less postoperative pain.

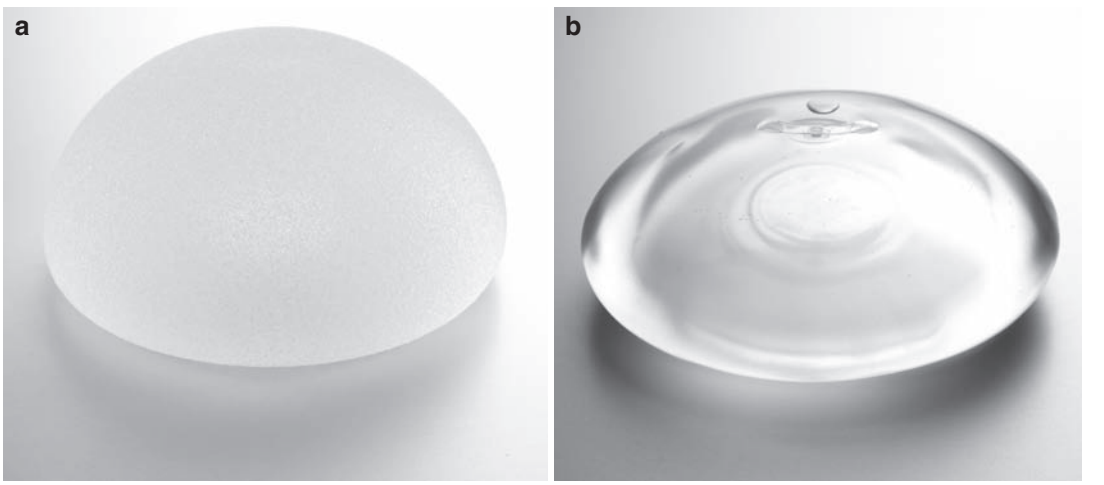


Figure 32.3. Example of breast implant. (**a**) Textured silicone. (**b**) Smooth saline. (Images provided courtesy of Allergan Corp.)

Figure 32.4. Example of different implant projections. (**a**) Silicone. (**b**) Saline. (Images provided courtesy of Mentor Corp.)

Surgical Techniques

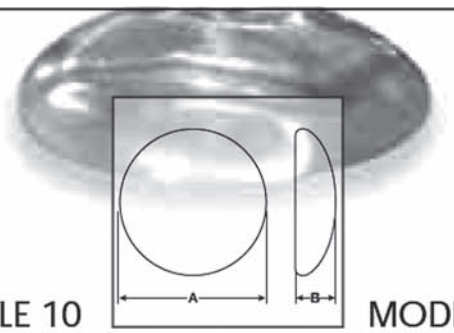
Inframammary Augmentation

Submuscular implant placement via inframammary approach is currently the most common method of breast augmentation. The incision is placed at or slightly below the inframammary fold depending on the planned size of the implants. This will hide the incision within the crease. The incision is marked lower with larger implants because their placement will require lowering of the inframammary fold and the incision will therefore ride up on the breast. The length of the incision also depends on the implant type and size. To accommodate larger gel implants, the incision may need to be made as large as 4.5 cm (Figure 32.8). The incision should begin at the midareola and extend laterally.

With submuscular placement, the lateral edge of the pectoralis major is identified and elevated. Controlled dissection under direct vision with electrocautery is used to elevate the pectoralis major off the chest wall and create the submuscular pocket. A lighted retractor is often helpful. The attachments of the pectoralis minor are left undisturbed. Perforators are identified and coagulated. The inferior third of the medial sternal muscle attachments and all the inferior muscle attachments are transected. The implant will rest in a pocket where approximately the superior two-thirds are covered by muscle and the inferior third only by breast parenchyma (Figure 32.7a). This is partial submuscular placement, sometimes referred to as biplaner, because complete submuscular placement creates a suboptimal shape and is therefore rarely used.

Careful attention should be paid to the boundaries of dissection. Overdissection of the submuscular pocket medially can lead to symmastia. Lateral overdissection can cause the implant to slide toward the axilla. Too much superior dissection can lead to a superiorly displaced implant

a



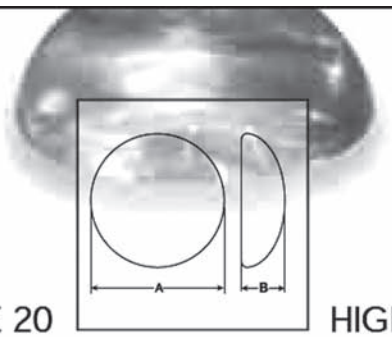
STYLE 10 MODERATE PROFILE

Implant Volume (cc)	Catalog Number	A Diameter (cm)	B Projection (cm)
120 cc	10-120	9.4	2.5
150 cc	10-150	10.1	2.7
180 cc	10-180	10.7	2.9
210 cc	10-210	11.2	3.0
240 cc	10-240	11.7	3.2
270 cc	10-270	12.2	3.3
300 cc	10-300	12.6	3.5
330 cc	10-330	13.0	3.6
360 cc	10-360	13.4	3.7
390 cc	10-390	13.6	3.8
420 cc	10-420	14.0	3.8
450 cc	10-450	14.4	3.9
480 cc	10-480	14.8	3.9
510 cc	10-510	15.1	4.0
550 cc	10-550	15.4	4.0
600 cc	10-600	15.8	4.3
650 cc	10-650	16.0	4.5
700 cc	10-700	16.4	4.6
750 cc	10-750	16.8	4.8
800 cc	10-800	17.2	4.9

Figure 32.5. Example of a volume/diameter/projection table. (**a**): Style 10 – moderate profile. (**b**) Style 20 – high profile. (Images provided courtesy of Allergan Corp.)

and excessive upper pole fullness, whereas overly aggressive inferior dissection can violate the inframammary fold and cause the implant to drop. The pocket is irrigated with saline or antibiotic solution and hemostasis is verified. Triple antibiotic irrigation with bacitracin, cefazolin, and gentamicin has been noted to most effectively decrease bacterial colonization and subsequently

b



STYLE 20 HIGH PROFILE

Implant Volume (cc)	Catalog Number	A Diameter (cm)	B Projection (cm)
120 cc	20-120	9.0	2.7
140 cc	20-140	9.1	3.3
160 cc	20-160	9.4	3.5
180 cc	20-180	9.6	3.8
200 cc	20-200	9.7	4.0
230 cc	20-230	10.0	4.2
260 cc	20-260	10.4	4.3
280 cc	20-280	10.6	4.5
300 cc	20-300	10.9	4.5
325 cc	20-325	11.2	4.6
350 cc	20-350	11.4	4.9
375 cc	20-375	11.7	4.9
400 cc	20-400	11.9	5.0
425 cc	20-425	12.0	5.2
450 cc	20-450	12.4	5.2
475 cc	20-475	12.6	5.5
500 cc	20-500	13.0	5.2
550 cc	20-550	13.5	5.6
600 cc	20-600	13.8	5.7
650 cc	20-650	14.2	5.9
700 cc	20-700	14.5	6.2
750 cc	20-750	15.0	6.0
800 cc	20-800	15.3	6.1

Figure 32.5. (continued)

decrease capsular contracture.[1] In 2000, the FDA listed betadine irrigation as a contraindication in augmentation mammoplasty as its contact with the implant has been implicated in implant wall compromise. The incision is then closed in two or three layers by reapproximating the subcutaneous layer and dermis with absorbable monofilament sutures and the skin with a running subcuticular suture (Figures 32.9 and 32.10).

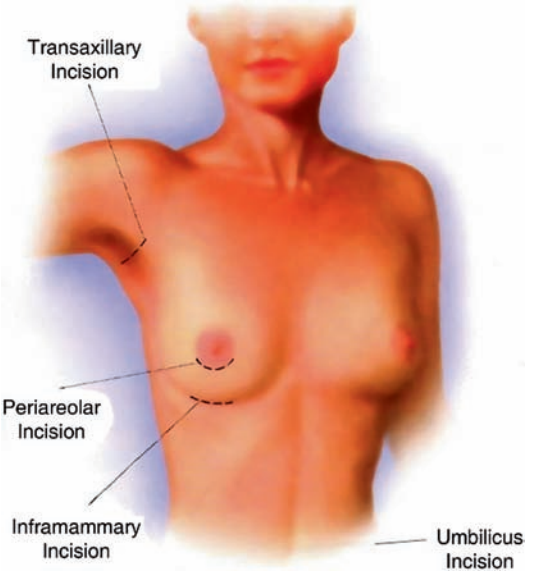


Figure 32.6. Available choices of incisions. (Image provided courtesy of Mentor Corp.)

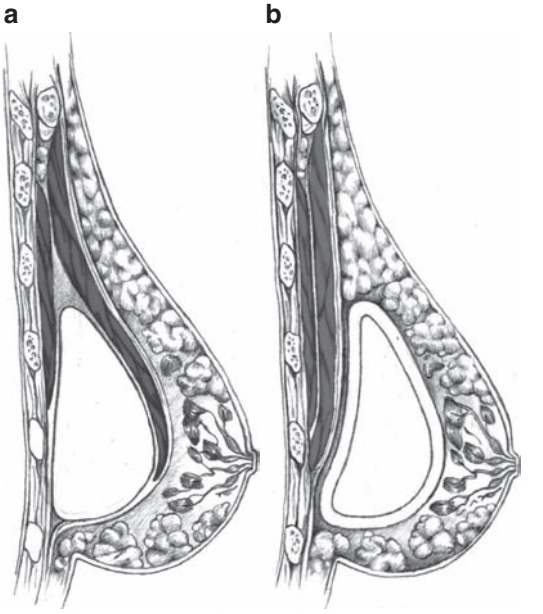


Figure 32.7. Implant pocket location. (**a**) Submuscular. (**b**) Subglandular. (Images provided courtesy of Mentor Corp.)

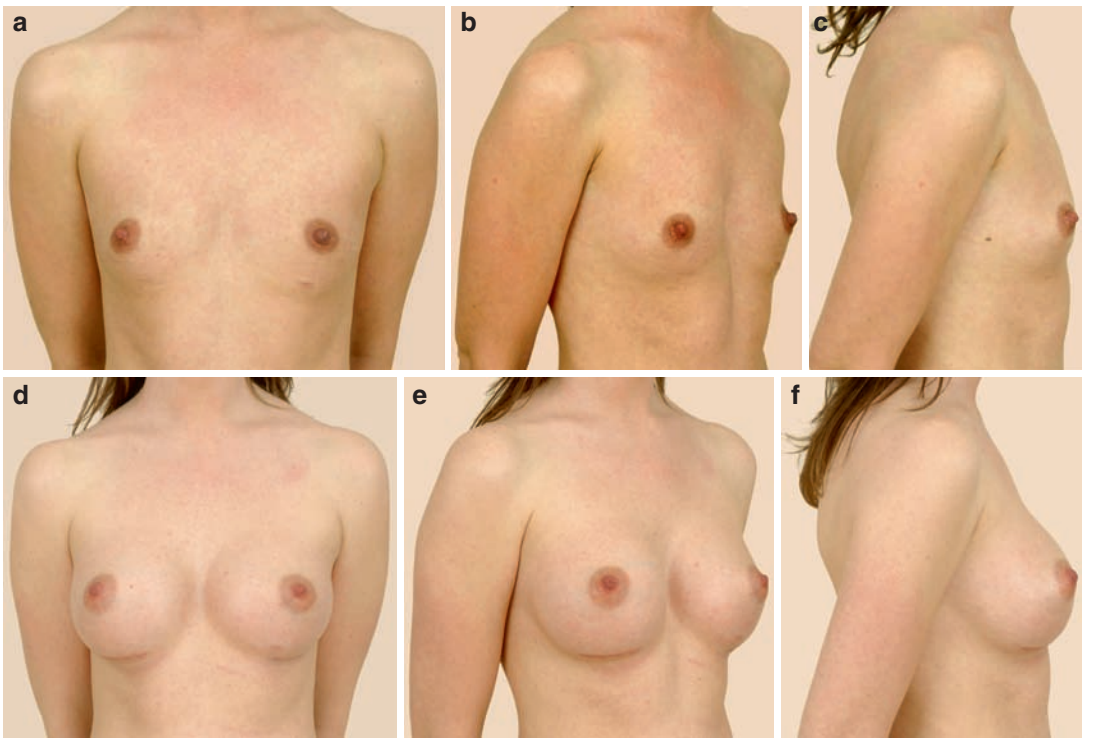


Figure 32.8. A 29-year-old female who underwent placement of bilateral submuscular 295 cc anatomic textured silicone implants to correct severe breast hypoplasia. The 4.5-cm incision was placed in the inframammary fold but migrated superiorly when the fold was lowered to create the implant pocket. (**a**–**c**) Preoperative. (**d**–**f**) Three months postoperative.

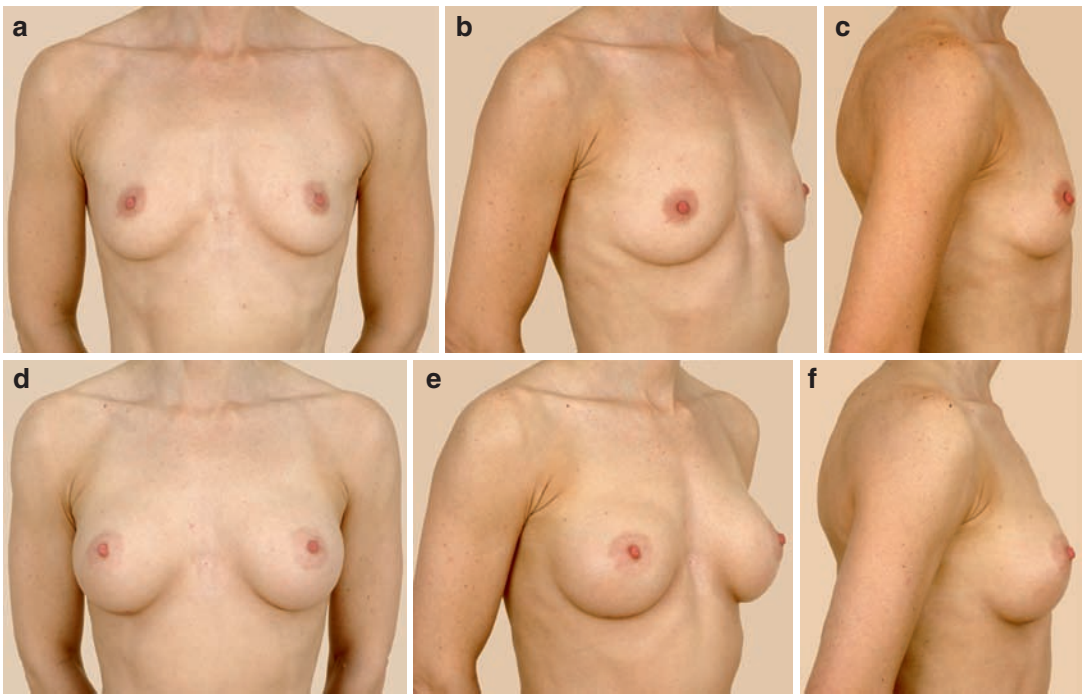


Figure 32.9. A 44-year-old female who underwent placement of bilateral submuscular 230 cc smooth round saline implants via inframammary incision. (**a**–**c**) Preoperative. (**d**–**f**) Two months postoperative.

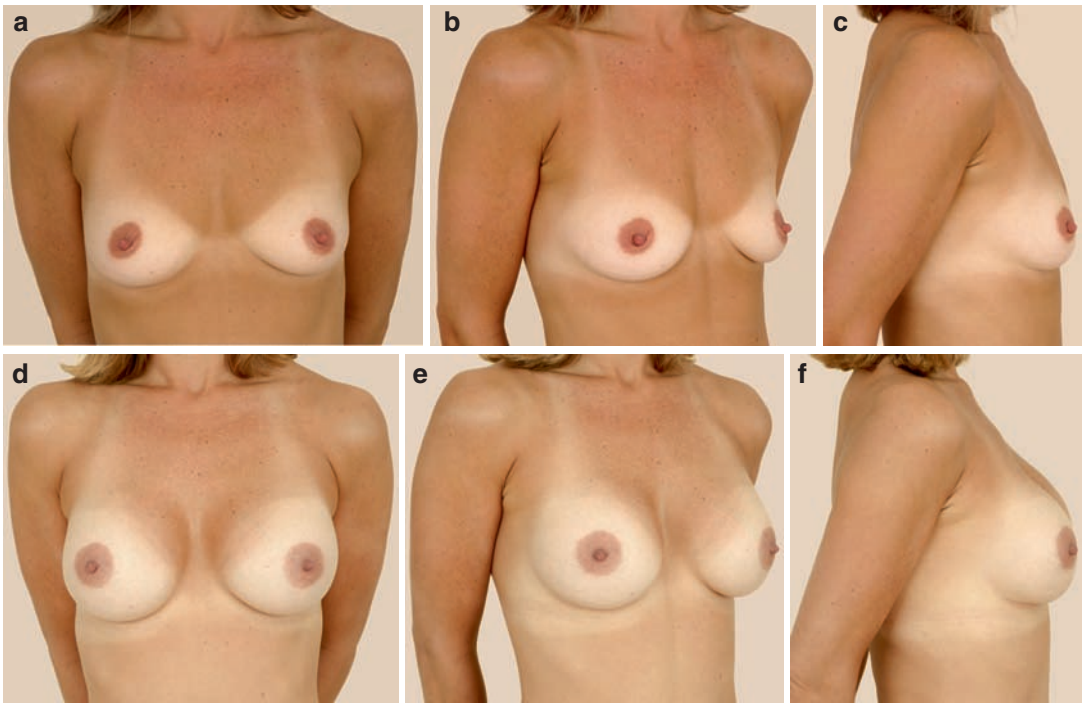


Figure 32.10. A 38-year-old female who underwent placement of bilateral submuscular 330 cc smooth round saline implants via inframammary incision. (**a**–**c**) Preoperative. (**d**–**f**) Nine months postoperative.

With subglandular placement, the implant pocket is created in a plane of dissection just superficial to the pectoralis muscle fascia (Figure 32.7b). This is typically done with electrocautery and the assistance of a lighted retractor. Again, overdissection can lead to symmastia, shift of the implant too lateral, or violation of the inframammary fold. The pocket is irrigated and hemostasis is verified. Closure is again performed in two or three layers. Drains are not necessary. Subglandular implant placement provides more anterior projection and can correct mild ptosis.

Periareolar Augmentation

The periareolar incision should only be used if the patient has adequate areolar circumference to allow dissection and implant placement. An areola diameter of 2.5 cm will give a semicircular incision of about 4 cm in length. It is best used in cases of tuberous breast or when the inframammary fold needs to be lowered. This approach can be used for submuscular or subglandular placement. Typically, the incision is made at the inferior areola–skin interface, but can be made at the superior aspect if a small amount of skin excision to elevate the nipple is desired (Figure 32.11). The dissection is carried down through the breast parenchyma or at the breast parenchyma–subcutaneous interface until the pectoralis fascia is encountered. In submuscular placement, the lateral aspect of the pectoralis muscle is identified, elevated, and the pocket is dissected. For subglandular placement, the pocket is created by dissecting a plane between the gland and the pectoralis fascia. Overdissection can create similar problems as those described in the inframammary approach.

Transaxillary Augmentation

The incision is placed at the hairline of the midaxilla. The length of incision is dependant on whether an open or endoscopic dissection is to be performed. The subcutaneous tissue is spread, and the lateral pectoral fascia is identified. The submuscular plane is entered, and blunt

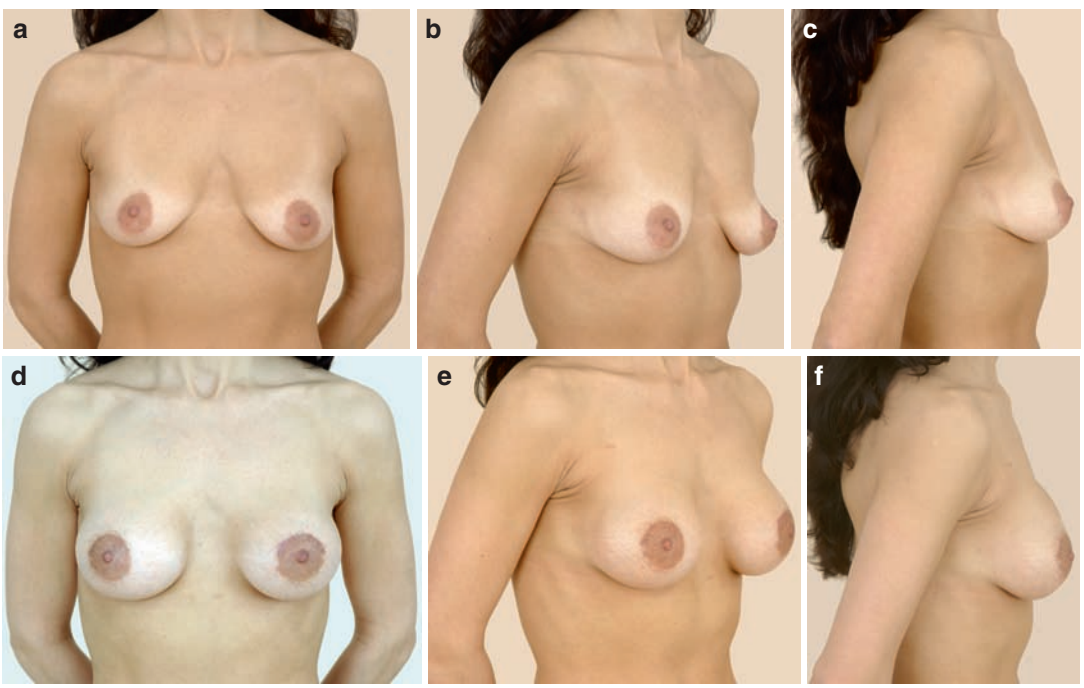


Figure 32.11. A 39-year-old female who underwent placement of bilateral submuscular smooth round saline implants via superior areolar incision. Volume on the right was 240 cc and volume on the left was 220 cc. A crescent of skin was excised on the left to elevate the nipple. (**a**–**c**) Preoperative. (**d**–**f**) Two months postoperative.

dissection of the pocket is performed. In the open method, the dissection and muscle transaction are performed with the assistance of a lighted retractor. The use of an endoscope not only allows the surgeon to make a smaller incision but also facilities a more controlled pocket dissection and transaction of the pectoralis muscle attachments. The open method allows placement of any implant type, whereas the endoscopic approach is typically limited to a saline-filled implant (Figure 32.12). Malposition is more common with the open approach and overdissection can lead to similar problems as previously discussed. Besides the scar position, the main advantages are that breast parenchyma is not transected and there is a decreased incidence of sensation loss with this approach.[15,17]

Transumbilical Augmentation

The transumbilical breast augmentation (TUBA) procedure requires training, endoscopic proficiency, and experience in order to perform the operation safely and well. A detailed description

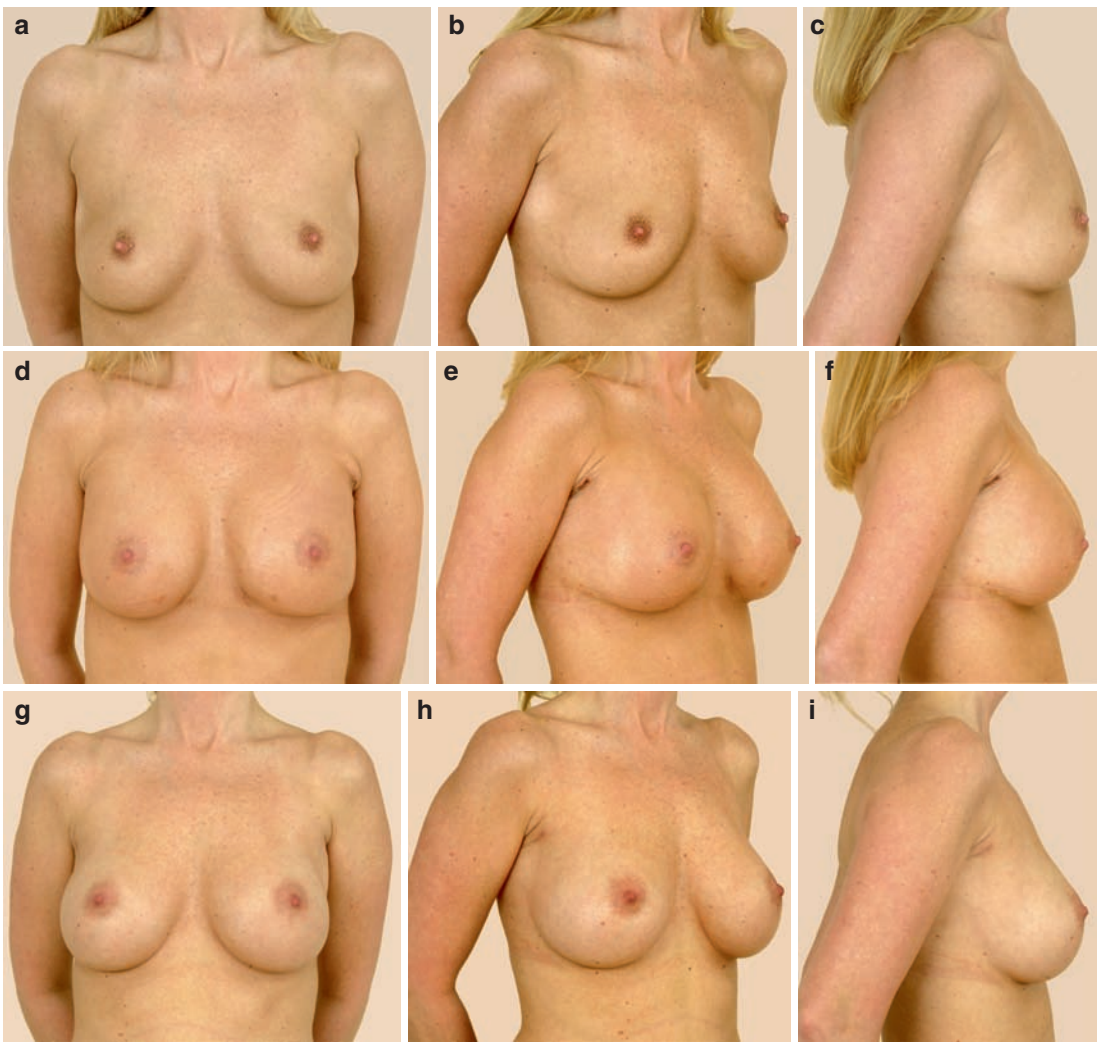


Figure 32.12. A 38-year-old female who underwent endoscopy-assisted placement of bilateral submuscular 360 cc smooth round saline implants via transaxillary incision. The implants appeared high initially but dropped within a year. (**a**–**c**) Preoperative. (**d**–**f**) Six weeks postoperative. (**g**–**i**) One year postoperative.

is beyond the scope of this chapter. Briefly, the incision is placed at the superior aspect of the umbilicus. With the assistance of endoscopy, the inframammary fold is pierced by way of a narrow tunnel. The tunnel is made by dissecting in a plane superficial to the rectus sheath. This allows access to the subglandular or subpectoral space, and a balloon dissector is used to create the pocket. Subpectoral placement is more technically challenging. Saline implants are inflated in the pocket, but adjustment can often be difficult.[7]

Complications

Capsular Contracture

Breast augmentation carries a high revision rate. Capsular contracture is the most common reason these patients seek surgical revision, and rates of significant clinical contracture (Baker grade III/IV) are approximately 20% after 10+ years of implantation.[6] The Modified Baker Classification (Table 32.1) quantifies capsular contracture based on the patient's feel of the breast and its appearance.[21] Malposition, rippling, and poor scarring are other reasons patients seek revision. The incidence of capsular contracture is influenced by pocket location, implant type, and postoperative complications. Submuscular placement allows the implant to be covered by more soft tissue and therefore decreases clinical capsular contracture.[23] Silicone implants have a higher incidence of contracture, but this is modestly improved if textured implants are used. In contrast, saline and textured implants increase the incidence of rippling. Underfilling and subglandular location in a women with minimal native breast tissue are common reasons for noticeable rippling.[8,9] Hematoma (1–3% incidence), seroma, and infection (1% incidence) are uncommon but are associated with higher capsular contracture rates and occasionally necessitate exploration and/or implant removal. Grade III and IV capsular contracture should be managed with capsulectomy and possible repositioning of a subglandular implant into a submuscular pocket.[6,11]

Table 32.1. Modified Baker capsular contracture classification.

Class IA	Breast completely natural
Class IB	Soft but visible implant
Class II	Mild firmness
Class III	Moderate firmness
Class IV	Excessively firm and symptomatic

Malposition

Malposition is the second most common cause of patients seeking revision. Underdissection of the pocket, overdissection of the pocket, and inadequate division of the pectoral attachments during submuscular placement are the typical causes. Transaxillary and TUBA approaches are typically associated with higher rates of malposition due to sometimes blind blunt dissection. Failure to lower the fold with insertion of a larger implant or inadequate release of the muscle will force the implant superiorly and cause a high riding appearance. Management includes capsulotomy and additional dissection of the pocket and/or completing the muscle release. On the contrary, overly aggressive dissection and violation of the inferior fold may cause the implant to drop and produce a "double bubble" appearance (Figure 32.13). This is managed with plication of the inferior aspect of the capsule and recreation of the inframammary fold.

Implant Rupture

Workup of a patient presenting with concern of implant rupture must begin with a thorough history and physical examination. Ruptured saline implants present with deflation, whereas a leaking gel implant can present with pain, a number of different breast irregularities, or can just be silent. Underfilling of saline implants below the manufacture's minimum is associated with more wrinkling and a shorter lifespan.[8,9] Mammography and ultrasound are capable of identifying a silicone rupture, but MRI (Figure 32.14) is the most sensitive and specific study.[2,4] It should be noted that studies have proved that effective mammographic screening can be performed in the augmented breast.[10]

Postoperative Care

Patients typically spend a few hours in the recovery room and then return home. Oral analgesia is usually sufficient for pain control. A soft support

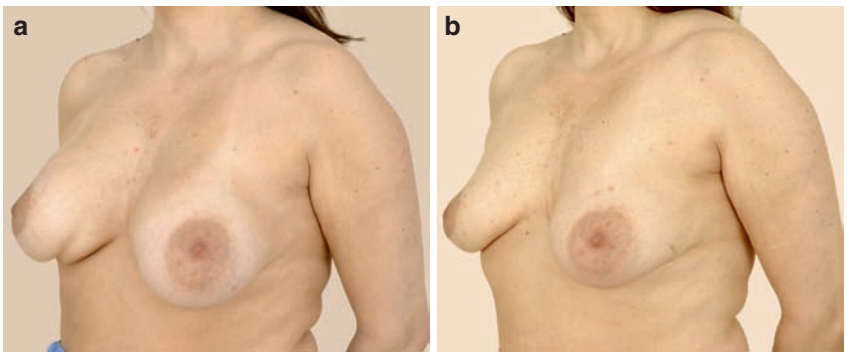


Figure 32.13. Example of a "double bubble." (**a**) The right inframammary fold is violated. (**b**) The right implant has been removed

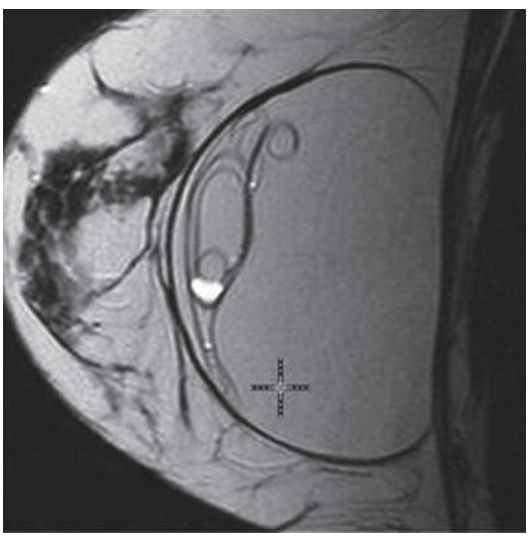


Figure 32.14. Example of subglandular implant with intracapsular and extracapsular rupture as seen on MRI.

bra is used continuously for a week and underwire bras are restricted for a few months. Patients often return to work within a week and can restart exercising.

Emerging Technology

Cohesive Gel Implants

The first anatomic cohesive gel breast implant was manufactured in 1994 by Allergan/Inamed/McGhan Corp. with the brand name "Style 410". They are filled with a highly cohesive silicone gel, resulting in a stable and firm form. There are 12 different implant shapes based on variable height and projection. Height options include low, moderate, and full, whereas projection options include low, moderate, full, and extra full (Figure 32.15).

Cohesive gel implants have been used clinically in Europe and South America for more than 10 years. They are under a clinical trial in the United States and have not yet been approved by the FDA. The trials are open to patients seeking a first-time augmentation, a revision of a breast augmentation, or breast reconstruction following mastectomy. Patients must be committed and willing to participate for a full 10 years of follow-up in order to be considered for the study. Cohesive implants offer plastic surgeons additional options for breast augmentation and may possibly lower complication rates, improve aesthetic outcomes, and give a high satisfaction rate.[3,14]

Autologous Fat Augmentation

Fat grafting to the breast has been championed by Coleman as an alternative or adjunct to breast augmentation and reconstruction. Patients with tuberous breast deformities, micromastia, and Poland's syndrome could also benefit from the procedure. The limiting factor is the safety of breast cancer detection postoperatively. Fat necrosis, calcifications, and even palpable masses can occur. An 8.5% complication rate (including cellulitis and small superficial lumps) with fat injec-tions to the reconstructed breast has been reported. More studies with approved IRB protocols need to be done to further examine the incidence of complications and the efficacy of breast cancer detection.[5,22]

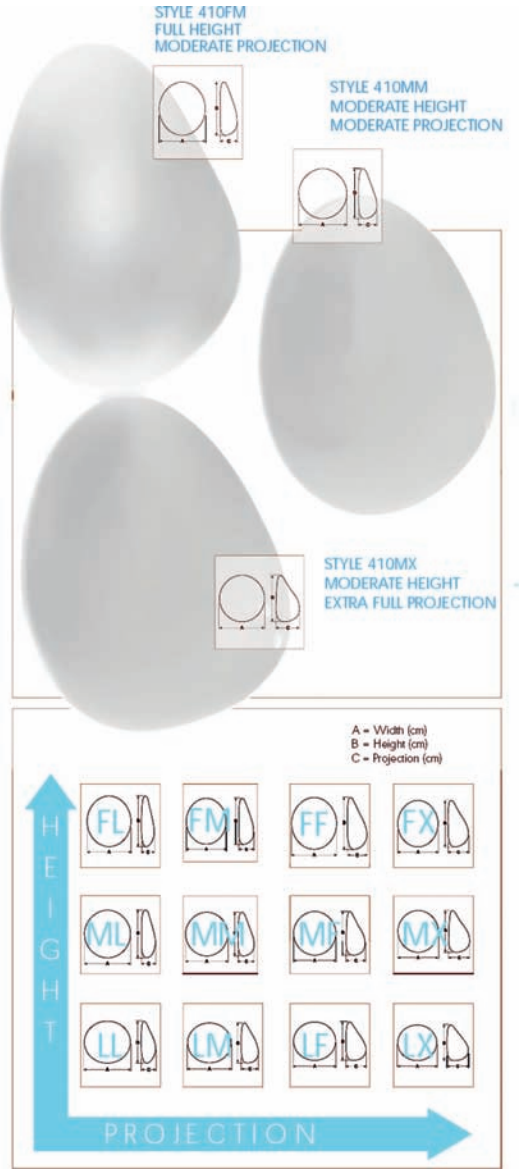


Figure 32.15. The style 410 family of cohesive gel implants. (Image provided courtesy of Allergan Corp.)

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33

Gynecomastia

Raymond Isakov

Summary

Gynecomastia is the benign enlargement of the male breast. There are many potential causes, physiologic, pathologic, and iatrogenic. True gynecomastia is a hypertrophy of breast glandular tissue, and pseudogynecomastia is a more localized adipose within the chest subcutaneous tissues. These entities often coexist. There are several peaks of incidence based on age. Neonatal, adolescent, and senile ages are the peaks and are present for various reasons. Workup is by a good history and physical examination. Further laboratory examinations may be beneficial.

The plastic surgeon often sees a gynecomastia that is either physiologic or idiopathic. Rarely, medical treatment is needed. Surgical treatment is based on patients and their desires and expectations. This may range from liposuction alone to a significant skin reduction.

This chapter reviews the etiology, workup, and options for surgical treatment of gynecomastia.

Abbreviations

SAL Suction-assisted liposuction
UAL Ultrasound-assisted liposuction

Introduction

Gynecomastia is the benign proliferation of breast tissue in boys and men. The incidence of breast proliferation is quite high in the general population. Approximately 50–70% of adolescent boys develop some degree of gynecomastia.[11] Approximately 30% of men have palpable breast tissue.[5]

Gynecomastia is mostly bilateral, but 25–30% may be unilateral. The degree of gynecomastia requiring treatment is individual and is based on the patient and his desires.

Etiology

There are many potential causes for gynecomastia; however, in many cases, an exact etiology is uncertain (see Table 33.1). Hormonal influence on breast tissue stimulates breast growth. Males and females have both androgens and estrogens. The alteration of the balance of the hormones in males likely contributes to the pathophysiology of gynecomastia. What throws this balance off is often unclear, but it may be pathologic, physiologic, or iatrogenic.

Male breast tissue has estrogen and progesterone receptors.[12] These receptors are activated by estrogens and inhibited by androgens. Aromatase converts the androgens to estrogens, which helps to explain the development of breast tissue in obese men and older men who have a higher

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Table 33.1. Etiology of gynecomastia.

Physiologic
Neonatal
Pubertal
Senile
Drug linduced
Table II
Hypogonadism
Trauma, Mmumps, Cchemo
Congenital
Secondary causes such as pituitary failure
Tumors
Testicular, Aadrenal, Bbronchogenic carcinoma
Systemic lillness
Thyrotoxicosis
Renal failure
Hepatic failure
Congenital
Klinefelter's
Testicular feminization
Anorchia
HIV
Alcoholism
Malnutrition

Table 33.2. Common drugs as an etiology for gynecomastia.

ACE inhibitors
Amiodarone
Anabolic steroids
Androgens
Cimetidine
Diazepam
Digoxin
Exogenous estrogens
Gonadotropins
Illicit drugs
Amphetamines
Heroin
Marijuana
Methadone
Isoniazid
Ketoconazole
Metronidazole
Methyldopa
Phenytoin
Potassium sparing diuretics
Protein pump inhibitors
Reserpine
Theophylline
Tricyclic antidepressants

percentage of fat.[3] Systemic illness also contributes to the increase of aromatase, which may explain the incidence in hospitalized patients.[6] Therefore, estrogen excess, androgen deficiency, malfunction of testosterone receptors, or decreased action of testosterone can cause gynecomastia.

Excess estrogens can be from consumption of exogenous sources. The uses of estrogens in prostate cancer or in a transgender population are examples. Increased endogenous estrogen production is also a potential source. This is a very important fact as the age of presentation may often correlate with the common age for presentation of testicular cancers. Adrenal tumors may also be an etiologic factor. Certain drugs (Table 33.2) and alcohol may also contribute to excess estrogens. Cirrhosis may lead to decreased estrogen metabolism, effectively increasing levels and decreasing metabolism. The many causes of excess estrogen may all lead to gynecomastia.

Androgen deficiency also has many etiologies. These can be acquired as in mumps or testicular trauma. Older men have relative deficiencies of testosterone, resulting in an excess in estrogens. This fact also explains the higher incidence of gynecomastia in the later decades of life. Klinefelter's syndrome is the most common chromosomal disorder in which gynecomastia routinely exists. This 47 XXY, and mosaic, 46XY/47XXY, is at significantly higher risk, 20–60 times higher, of developing male breast cancer.[16] A more diligent approach must be taken in this syndrome. Androgen receptor deficiency may also lead to male breast development.

Presentation

Gynecomastia commonly presents in three peaks. The first is in the neonatal period when maternal estrogens are still circulating. This usually disappears within a few weeks after birth.

The presence of pubertal gynecomastia is very common. This second peak usually presents in early to mid puberty and lasts 6–18 months. It is usually a 1.5–3 cm retroareolar mass. At times, it may be tender, but the most common reason for medical presentation is significant emotional distress to the patient due to the appearance. The patients do not take their shirts off for fear of being mocked. This may have significant psychological effects. There is also a component of obesity. Obese boys often have gynecomastia.

Pseudogynecomastia is localized adiposity of the breast without a significant glandular component. In the adolescent population, there is often a true glandular component as well as adiposity of the breast.

The third peak of gynecomastia is in advanced age. Twenty percent of 60+ year old men and 50% of 80+ year old men will have gynecomastia.[7] This population has multifactorial etiology due to advanced age physiologically and with pharmacologic use. These cases also have a component of fatty breast, which varies from patient to patient.

The classification first described by Simon[15] is as follows:

I: Minor breast tissue without skin excess

IIa: Moderate breast tissue without significant skin excess

IIb: Moderate breast tissue with mild to moderate skin excess

III: Significant breast tissue with significant skin excess

The patient will present to the plastic surgeon based on his concerns, not necessarily based on the severity of the gynecomastia. It is not uncommon for a patient to be severely bothered by what others consider a very mild problem. Conversely, some men with severe gynecomastia are not bothered by it.

Diagnosis

The most common presentation in most plastic surgery practices is an aesthetic concern. Most often in adolescent age, boys have severe emotional distress over the appearance of their breasts. Older patients often have had gynecomastia for a significant period of time and are just hoping to look better. History should include time of onset, pain, medications, and drug use. If the gynecomastia occurs prepubertally, a more extensive workup, including ultrasound of the testes, may be indicated.[2] If the presentation is in the early phases of adolescence, no specific diagnostic tests or treatment is needed, as the yield is low. A good physical examination including a testicular/genital examination and abdominal examination to rule out masses and organomegaly should be routinely performed. Noting the overall virilization is important, as any deviation may indicate a need for further workup. For example, a more feminine appearance and Marfanoid habitus should have a karyotype ordered to rule out Klinefelter's syndrome. The breast examination often reveals fatty tissue of varying degree, with dense tissue in the retroareolar position. Skin excess and quality are assessed. Dominant masses need to be addressed with imaging and/or biopsy.

If there is any concern, further evaluation may be appropriate. An endocrine consultation may be helpful. Laboratory tests include kidney and liver function, testosterone and estrogen levels, LH, and FSH. Prolactone and thyroid functions may be helpful. DHEA to check adrenal androgens and tumor markers such as alpha-fetoprotein are checked when appropriate suspicion of malignancy exists.

Treatment Options

The treatment is based on the pathophysiology, physical examination, and patients' desires. If there is a clear inciting agent, (medication, drug, etc.), stopping this may reverse the gynecomastia. Drugs blocking estrogens are a potential medical therapy. Raloxifen and tamoxifen are options.[9] Danazol may also be used.[4,17] It is unusual for a plastic surgeon to dictate medical therapy; however, if the onset is less than 12–18 months, observation is performed. If the onset has been greater than 18 months, fibrous conversion makes it unlikely to recede, regardless of etiology. Therefore, long-standing gynecomastia without pathologic, syndromic, or neoplastic etiology is best treated with surgery.

Many surgical techniques have been described through the decades.[13] The surgical plan is based on skin quality and redundancy. Gynecomastia severity is a spectrum like many issues in medicine. It can range from extremely mild to patients with grade III ptosis of the breast with significant volume and extra skin. When and how to excise skin is a difficult decision, because unlike female breast reduction, scars on a male chest are obvious and unappealing. The trade-off, scar for improved contour needs to be discussed at length with the patients. Photographs should be shown preoperatively.

In mild to moderate gynecomastia, the decision to directly excise breast via a periareolar incision verses liposuction alone needs to be

made. If liposuction alone is to be used, ultrasonic-assisted liposuction (UAL) may break up fibrous tissue and give nice results.[8,14] Suction-assisted liposuction (SAL) with direct excision through a periareolar incision is an excellent method and the author's procedure of choice (Figure 33.1). The operation starts with tumescent or super-wet liposuction SAL or UAL. This removes the fatty component and breaks the inframammary fold. The periareolar incision is then made, and skin flaps are developed leaving 1–2 cm of breast under the areola to avoid a "saucer deformity." The gland is then easily removed off the pectoralis fascia in a bloodless plane. This is sent for pathology evaluation. The clear disadvantages to this are the need for drains, possible seroma, possibly longer operating room time, and the increased risk for hematoma over liposuction alone. The advantages over UAL alone are a more precise excision of the fibrous breast parenchyma, a more aggressive resection, and potentially better symmetry.

In severe gynecomastia with skin excess and, especially, ptosis, it is difficult to get a good result without skin excision. UAL alone does very little to address the skin excess, and revision with later skin excision is almost certain. Proponents of the staged approach with UAL state that the skin contraction will decrease the size of future incisions. However, it is unclear how much smaller the skin excision will be in the second stage when compared with primary skin excision.[10] The amount of skin excision is based on the breast and patients' willingness to accept scars. Different techniques of skin excision are available and vary based on size and skin excess (Figures 33.2 and 33.3a). A Wise pattern looks good on the three-dimensional female breast but is usually unacceptable on a flat male chest. Mastectomy with free nipple graft versus pedicle nipple technique is commonly used (Figure 33.3b). The grafts do not look as good as a vascularized nipple–areolar complex, so this is reserved for very severe skin excess and ptosis. Milder ptosis

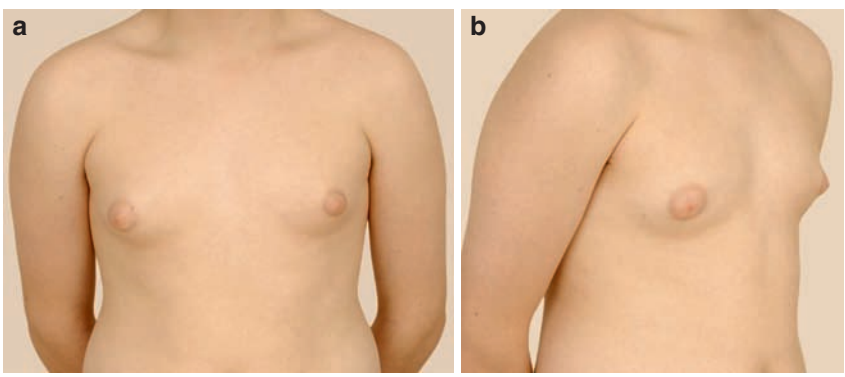

Figure 33.1. (a, b) Mild gynecomastia with no significant skin excess.

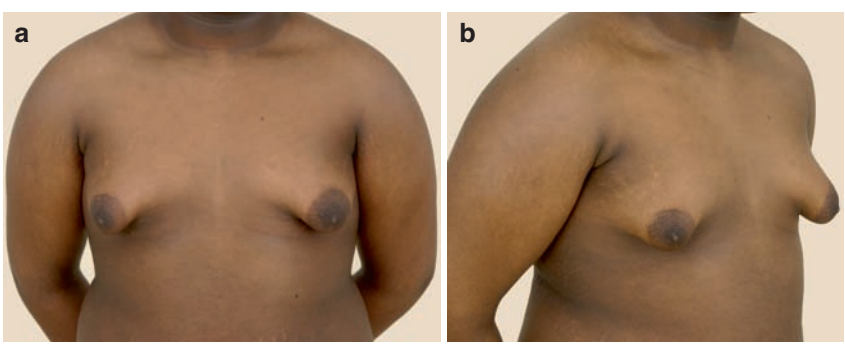

Figure 33.2. (a, b) Moderate gynecomastia with skin excess.

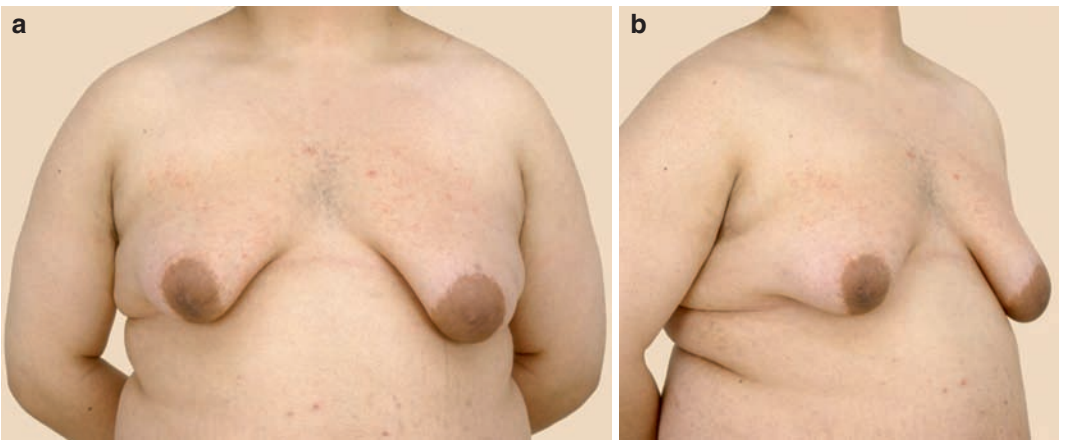

Figure 33.3. (a, b) Severe gynecomastia with skin excess and ptosis.

with significant skin excess is treated well with the nipple on a pedicle. The placement of the nipple has been described.[1] The average areolar diameter is 2.8. The nipple position is easy to estimate at the forth intercostal space with the medial portion at the mid-clavicular line. The mid-humerus has also been used commonly as a reference point.

Postoperative care is usually uneventful. If a direct excision is to be used, hematoma must be looked for. Immediate evacuation of the hematoma once recognized is necessary. Drains are used if any incision is made to surgically excise the tissue. The drains are removed when the output is 30 ml/day or less. This usually occurs in less than 1 week. Compressive dressings are used as well. If liposuction alone is used, the compressive dressings or garments are used for approximately 1 month. If a direct excision is used, the garments are used for at least 2 weeks after the drains come out. Activity is limited to avoid seroma and delayed hematoma. If a seroma occurs, aspiration as needed in the office usually offers definitive treatment. Overall, the surgery is tolerated well by the patients.

Conclusion

Gynecomastia is a common condition affecting boys and men in various phases of life.

This is usually a benign process; however, it often causes emotional distress to the patient.

The etiology is varied and potentially complex. The plastic surgeon sees mostly benign, idiopathic, or physiologic gynecomastia, which rarely requires an extensive workup. The plastic surgeon should be aware of the etiologies and pathophysiology so that any pathology can be diagnosed and addressed appropriately. The potential scarring when skin excision is needed decreases the aesthetic result but is often unavoidable. Trends toward ultrasonic-assisted liposuction as a primary or a staged procedure may decrease the amount of skin excision that is needed. Overall, these patients can receive excellent results, and the surgery can be satisfying to patient and surgeon.

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Part VI

Hand and Upper Extremity

34

Hand Anatomy and Examination

Steven L. Bernard and Benjamin Boudreaux

Summary

The purpose of this chapter is to give a comprehensive review of hand anatomy and then to further apply that anatomy toward a functional examination. With that in mind, this chapter is divided into sections of anatomy, including subsections on arteries, nerves, muscles, nail, and skin as well as bony anatomy. In each of these sections, fine points on the examination of these structures will be included.

Abbreviations

C	Cervical
DIP	Distal interphalangeal
FDP	Flexor digitorum profundus
FDS	Flexor digitorum superficialis
MCP	Metacarpophalangeal
PIP	Proximal interphalangeal
T	Thoracic
TFCC	Triangular fibrocartilagenous complex

Introduction

The human hand is unique in the animal world in its ability to manipulate the surrounding environment. The intricate movements of the hand are made possible by precise balance between our joints, ligaments, tendons, and muscles. A detailed knowledge of anatomy is necessary for our ability to understand the disease processes that affect the hand. Hand injuries account for a significant portion of all emergency room admissions.[2,10] We injure our hands both at play and at work. Inherited diseases and arthritis often affect the function of the hand as well. The hand examination is even more complicated for children and unconscious patients who cannot cooperate with an orderly hand examination. It is necessary to understand how to diagnose an injury without their specific help. As a result of these factors, a detailed understanding of hand anatomy will lead to the best potential outcome and treatment for the disease processes of the hand.

The understanding of the anatomy needs to span the skin surface down into the joints themselves. Anatomic characteristics specific to the hand include a dorsal skin that is thin, pliable, and elastic. It allows for flexion and extension over a great range, with the superficial extensor tendons gliding beneath the subcutaneous tissue. On the palmar surface of the hand, the skin is more irregular and completely hairless. It is fixed to the underlying skeleton through tough septa, which ultimately blend with palmar fascia to provide wearability as well as traction for pinch and grasp. Without this ability, the gliding skin would develop excess roll on attempted grip. It is notable that the difference between the palmar and dorsal aspect of the hand is also manifested in disease states such as edema, which is seen dorsally, but because of the dense underlying connective tissue on the volar side,

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the palm will appear relatively innocuous. This could lead to the erroneous conclusion that a disease process is more dorsal than palmar. When viewing this from the aspect of pathology, this also prevents accumulation of pus leading to palmar infections that extend into the dorsal skin.

The Hand Proper

The subtleties of anatomic differentiation within the skin of the hand deserve further clarification. The skin over the thenar eminence is thinner than that of the skin over the hypothenar eminence, which in turn is thinner than the skin over the dorsal aspect of the hand and heads of the metacarpal. Superficial examination of the hand reveals an immediate differentiation between the dorsal and palmar aspects of the hand. The dorsal skin is essentially a continuation of forearm skin, whereas the palm contains glabellar skin with the whirls that ultimately make up our fingerprints. The palmar skin is densely invested with connections to the palmar fascia, limiting the mobility of the skin over the underlying bones and allowing us to withstand great pressure during grip and function of the hand. The thickened palmar fascia is called the palmar aponeurosis. This is an extension of the palmaris longus tendon, which forms longitudinal bands that extend into the central portion of each finger. Pathologically, these can become thickened into chords in Dupuytren's Contracture. Proximally the aponeurosis blends into the transverse carpal ligament; distally it widens into four slips that blend into the corresponding fibrous digital sheaths and lateral ligaments of the metacarpals incasing the digital nerves and arteries. At their deepest extent, they attach to fibers that invest in the bones of the hand.

Arteries

The vascular supply to the arm is based on the axillary artery and its continuation into the lower arm as the brachial artery (Figure 34.1). The brachial artery courses along the medial intramuscular septum between the biceps and triceps muscles and enters the antecubital fossa medial to the bicipital aponeurosis. The major branches of the brachial artery include the profound brachii, superior and inferior ulnar collateral, and biceps myocutaneous arteries, as well as direct cutaneous perforators. The artery ends by dividing into the radial and ulnar arteries approximately 1 cm distal to the elbow joint.

The radial artery courses laterally over the tendon of the biceps muscle and over the fascia of the flexor digitorum superficialis (FDS) muscles in between the flexor carpi radialis and brachioradialis at the level of the wrist. After coursing under the "anatomic snuffbox," the radial artery courses beneath the extensor pollicis longus onto the dorsum of the hand. The dorsal carpal branches of radial artery unite with a similar contribution from the ulnar artery as well as branches from the interosseous arteries to form the dorsal carpal rete, which supplies the dorsal skin and dorsal metacarpal arteries. The dorsal branch of the radial artery also gives off a large deep branch that travels between the two heads of the first dorsal interosseous muscle and divides into the princeps pollicis and deep palmar arch.[18]

The ulnar artery is the more dominant branch of the brachial artery in most people. After the splitting of the brachial artery, it runs medially beneath the median nerve and the proximal portion of the FDS muscle. It continues between the flexor digitorum profundus (FDP) and the flexor carpi ulnaris muscle until it reaches the level of the wrist. At the level of the wrist, it is just lateral to the flexor carpi ulnaris tendon and travels along with the ulnar nerve through Guyon's canal of the transverse carpal ligament. At the base of the origin of the hypothenar muscles, the vessel divides into a deep and superficial arch. Within the proximal forearm, the ulnar artery branches into the recurrent ulnar artery at the level of the FDS origin. Just distal to this the common interosseous artery arises deep to pronator teres and divides into an anterior and posterior branch. The posterior branch goes through the interosseous membrane and supplies the dorsal extensor musculature.

Within the hand, the superficial palmar arch is the continuation of the ulnar artery and the superficial branch of the radial artery. It crosses the palm above the flexor tendons and just superficial to the digital nerves deep to the palmar fascia.[8,18,27,28] The level of the arch is approximately equal to a line drawn from a fully radially

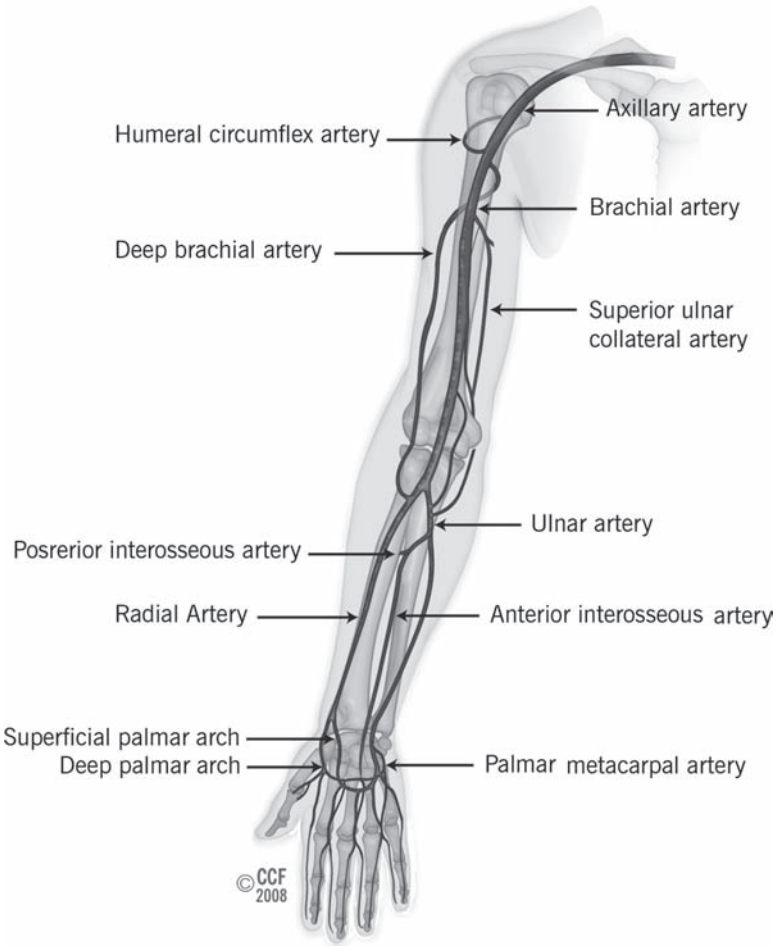

Figure 34.1. Arterial anatomy of the arm.

abducted thumb. The arch terminates into the palmar digital branches, including the common digital arteries that ultimately divide into the proper digital arteries traveling first volar and ultimately dorsal to the digital nerves. There is a great deal of variability[4,20,32] within the vascular system of the hand; however, ultimately consistent anatomy of two palmar and smaller dorsal digital arteries can be found in each finger and the thumb.[3] A deep arch is also formed by a branch of the ulnar artery ultimately connecting with the princeps pollicis from the dorsal branch of the radial artery.

Nerves

Brachial Plexus

The complex origin of the brachial plexus are cervical nerve roots 5 through 8 (C5–8) and the first thoracic root (T1) with occasional contributions from either C4 or T2 (Figure 34.2). Portions of the roots merge to form the upper middle and lower nerve trunks, which travel with the subclavian vessels.[16] At the level of the clavicle, the nerves intertwine to form the posterior, lateral, and medial cords. These structures

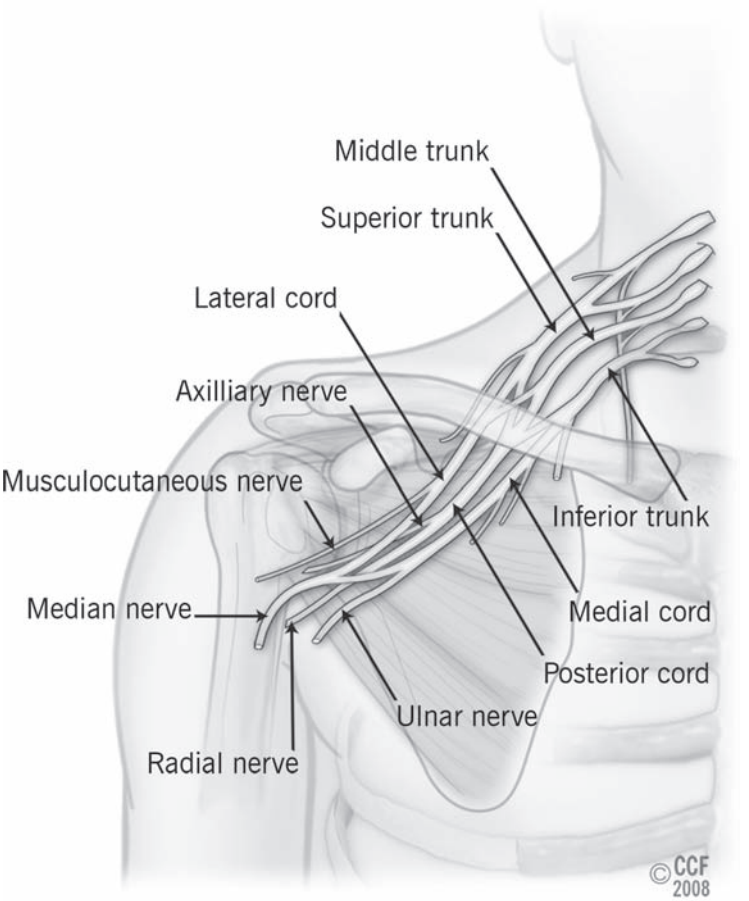

Figure 34.2. Brachial plexus.

intermingle again to divide into the major peripheral nerves of the arm. The posterior cord gives rise to the axillary and radial nerves; the lateral cord gives rise to the musculocutaneous and contributes to a portion of the median nerve. The medial cord gives off the remaining fibers of the median nerve and forms the ulnar nerve as well. As a result of their superficial position, the median, ulnar and musculocutaneous nerves are susceptible to penetrating injury, whereas the axillary and radial nerves are more commonly injured during dislocation of the shoulder. The axillary nerve itself provides motor innervation to the deltoid muscle and sensory innervation to the posterior aspect of the shoulder. The musculocutaneous[22] nerve innervates the coracobrachialis, biceps brachia, and brachialis muscles and supplies sensation to the proximal radial forearm.

Median Nerve

The median nerve is one of three principle motor and sensory nerves of the upper extremity. In its course in the upper arm, it provides no motor or sensory branches. As it enters the forearm between the two heads of the pronator teres, the nerve courses on the deep surface of the FDS muscle and gives off direct motor branches to the pronator teres, flexor carpi radialis, FDS, and palmaris longus muscles. A major branch of the median nerve in the forearm is the anterior interosseous nerve, which runs along the volar surface of the interosseous membrane and

innervates the flexor digitorum profundus to the index and long fingers as well as the flexor pollicis longus and the pronator quadratus muscles. At the level of the wrist, the median nerve travels deep to the palmaris longus tendon traversing the carpal tunnel. In the hand, the nerve provides motor innervation to a portion of the opponens pollicis, abductor pollicis brevis, and the flexor pollicis brevis through its recurrent motor branch to the thenar musculature. From a sensory standpoint, the median nerve gives off branches to the palmar digital nerves of the thumb, index, and the middle and radial palmar digital nerves of the ring finger (Figure 34.3).

As the median nerve terminates into the digital nerves, the nerves themselves lie in the palmar aspect of the hand. Within the finger, the nerve runs with the digital artery and is bordered by Cleland's ligament dorsally and Grayson's ligament volarly.[12] At the level of the distal interphalangeal joint, the digital nerve terminates into three branches: nail bed, tip, and pulp. Two-point discrimination along the axis of each digital nerve should be less than 6 mm in an adult to suggest an intact nerve.

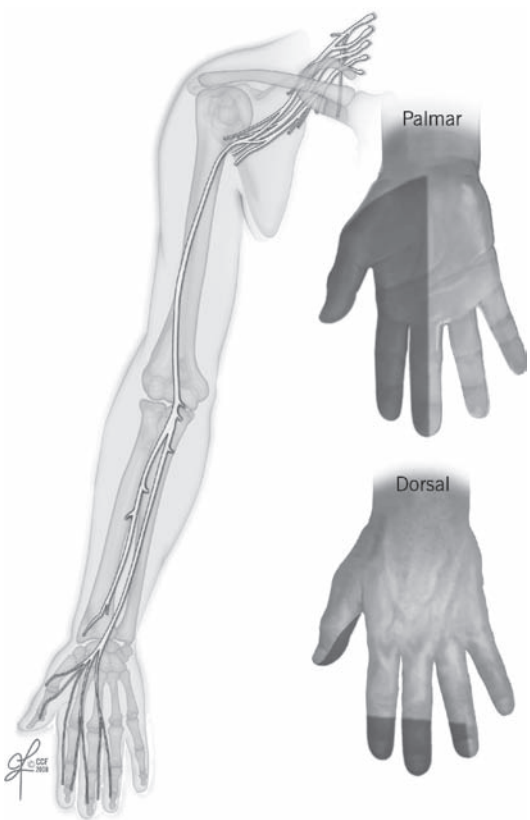

Figure 34.3. Median nerve course and distribution.

Ulnar Nerve

As with the median nerve, the main target of the ulnar nerves is the forearm and hand. It courses along the medial aspect of the arm and gives off the medial antebrachiocutaneous nerve at the mid arm level, supplying sensation to the medial arm and forearm. As the ulnar nerve enters the forearm, it travels between the two heads of the flexor carpi ulnaris muscle and supplies this muscle with innervation. The nerve continues between the plane of the flexor carpi ulnaris and the flexor digitorum profundus muscles. The nerve gives off branches to the portion of the flexor digitorum profundus muscle that supplies the ring and small finger tendons. Just proximal to the wrist, a dorsal sensory branch supplies the ulnar dorsal aspect of the hand. The nerve traverses the transverse carpal ligament through Guyon's canal with the ulnar artery. Beyond Guyon's canal, the nerve divides into a superficial sensory branch, which gives off the palmar digital nerves to the small finger and ulnar side of the ring finger. The other portion of the ulnar nerve just beyond Guyon's canal becomes the deep motor branch supplying the hypothenar musculature, all interosseous muscles, the lumbricals associated with the flexor digitorum profundus to the ring and small fingers, the abductor pollicis, and the deep pad of the flexor pollicis brevis muscle. The proximal ulnar portion on the palmar aspect of the hand is consistently innervated by the ulnar nerve through either a palmar cutaneous branch or the nerve of Henley, which innervates the ulnar artery before giving off branches to the distal forearm and hypothenar eminence (Figure 34.4).

Variability of the Ulnar and Median Nerves

Great variability exists between the ulnar and median nerves from person to person and even within the person from the left to the right upper extremity. Sunderland believes that in only half of the cases the median nerve innervates the flexor digitorum profundus of the index and

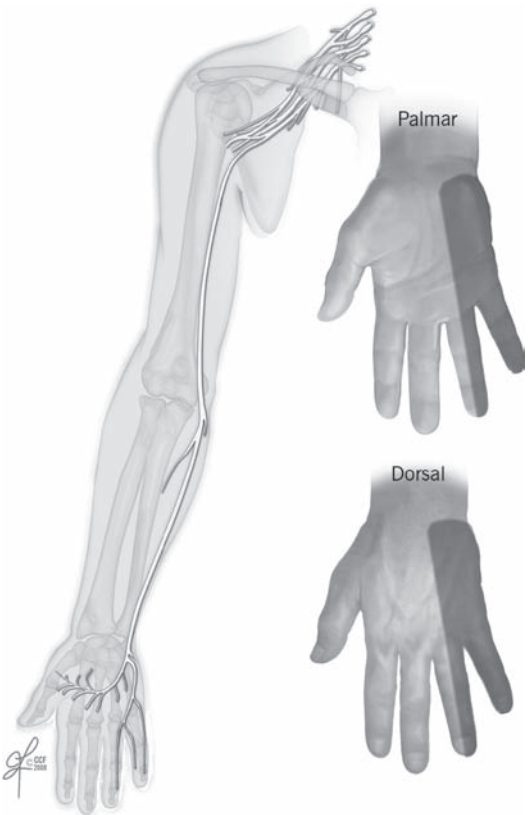

Figure 34.4. Ulnar nerve course and distribution.

middle fingers and the ulnar nerve innervates the ring and little finger profundus. In the remaining cases, there is overlap such that the median nerve encroaches upon the ulnar nerve's territory.[36] This pattern is reversed in the hand, and the ulnar nerve often extends into the territory of the median nerve. Most commonly affected muscles are the thenar eminence, first or dorsal interosseous, and the lumbricals. In addition to the overlap between the two nerves, there are often interconnections between the nerves. The so-called Martin–Gruber anastomoses are interconnections of the median and ulnar nerves in the forearm.

Within the hand, similar connections occur between the median and ulnar nerves, known as Riche–Cannieu[7,31] and may be present in as many as 70% of people.

These interconnections are important when examining for specific nerve injuries, because the function of muscles normally innervated by either the median or the ulnar nerves can remain intact, thereby masking complete injuries to the nerves themselves.

Radial Nerve

Originating from the posterior chord of the brachial plexus, the radial nerve begins medial to the triceps and travels posterior through the muscle behind the shaft of the humerus. The nerve then courses anteromedially between the brachialis and brachioradialis muscles over the lateral epicondyle.[29] In the upper extremity, the radial nerve innervates the teres major, deltoid, triceps, brachioradialis, anconeus, and extensor carpi radialis longus muscles.

Just distal to the elbow, the nerve divides into two major branches. The superficial branch innervates the extensor carpi radialis brevis muscle and provides sensation to the radial aspect of the forearm and dorsal hand. The deep branch of the nerve, the posterior interosseous, runs through the two heads of the supinator muscle beneath the arcade of Frohse and innervates the supinator, extensor digitorum communis, extensor carpi radialis brevis, extensor carpi ulnaris, extensor pollicis longus, abductor pollicis longus, extensor pollicis brevis, extensor digiti minimi, and extensor indicis proprius. Sensation to the fingers extends approximately to the proximal interphalangeal (PIP) joint level on the dorsal aspect of the hand (Figure 34.5).

Muscles and Tendons

The muscles of the upper arm are separated into two functional compartments by the humerus and medial and lateral intermuscular septi. These compartments are labeled anterior and posterior. The muscles of the anterior compartment include the brachialis, and coracobrachialis, which flex the elbow while the other muscle of the upper arm, the biceps, both flexes the elbow and supinates the forearm. The sole muscle within the posterior compartment is the triceps. Several muscles traverse the elbow joint on their way to influencing the flexion of the elbow, the wrist, and, in some cases, the fingers.

Volar Forearm Compartment

The flexor system of the wrist and hand comprises muscles that originate in the proximal

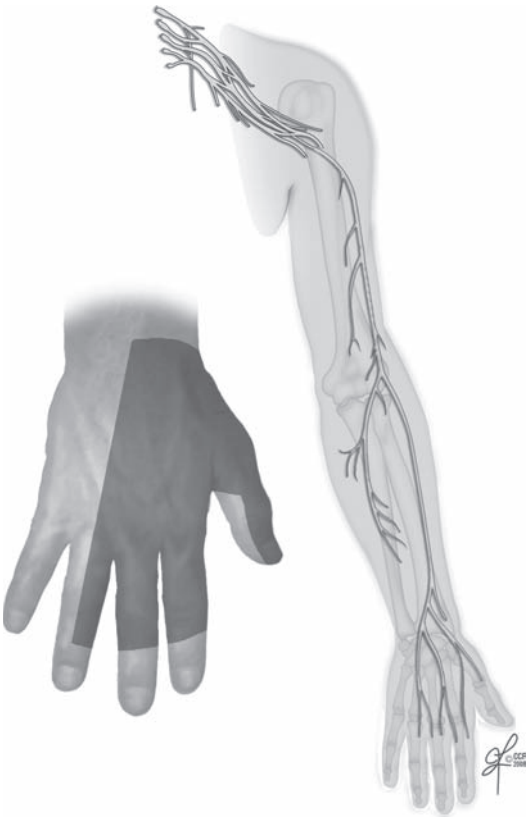

Figure 34.5. Radial nerve course and distribution.

two-thirds of the volar compartment of the forearm, with tendinous extensions that insert into the wrist or digits (Figure 34.6). There are two flexor tendons for each finger and one for the thumb. The FDS inserts into the proximal to mid-portion of the middle phalanx and creates flexion at the PIP joint. The flexor digitorum profundus tendons insert into the distal phalanges and flex the distal interphalangeal (DIP) joints of each finger. The one flexor of the thumb is the flexor pollicis longus that inserts into the distal phalanx of the thumb. This causes flexion of the thumb's only interphalangeal joint.

The cross-sectional anatomy of the forearm is best viewed in layers (Figure 34.7).[9,11,17] The most superficial layer contains the palmaris longus, flexor carpi radialis, flexor carpi ulnaris, and pronator teres. These muscles either insert into the palmar fascia or the metacarpals and are involved in wrist flexion or forearm pronation. The second layer of the forearm contains the FDS muscle. The muscle itself originates from two heads and separates into four tendons. The ring and long finger tendons arise from the superficial portion of the muscle and therefore traverse the wrist in a superficial position. The deeper portion of the FDS muscle belly terminates in the FDS tendon of both the index and small fingers. As the tendons traverse the hand, they flatten and split into radial and ulnar slips, which eventually rotate 180° to encompass the flexor digitorum profundus (FDP) tendons of the individual fingers. They then pass dorsally to the profundus tendon and reunite to insert in the proximal half of the middle phalanx of each finger. The split of the FDS tendon is called the Chiasm of Camper. The next deeper layer of the forearm consists of the flexor digitorum profundus muscle and the flexor pollicis longus. Although we have individual control over the FDS to each finger, in most instances, the flexor digitorum profundus muscle is a conjoined mass that acts as one flexor and lacks individual control to each of the fingers. The tendons insert at the base of the distal phalanx on the palmar aspect of the bone itself. It is of note that when looking at the function of the FDS, and the flexor digitorum profundus tendons[6] Baker, Gaul, Williams and Graves have shown that 34% of small fingers have an insufficient FDS tendon. In addition, it is possible that the FDS tendons to both the ring and small finger act in concert. Both of these cases do not allow for isolated flexion of the PIP joint of the small finger. The deepest layer of the volar forearm contains the pronator quadratus muscle, a flat transverse muscle that assists in pronation of the forearm.

Dorsal Forearm Compartment

Extension of the wrist and fingers can be broken down into an extrinsic and intrinsic portion. In the extrinsic system, the muscle bellies originate within the forearm. Within the intrinsic system, the muscles all originate within the hand (Figure 34.8).

The dorsal forearm is divided into a superficial and deep muscle group. The superficial group consists of the wrist extensors, including the extensor carpi radialis brevis, the extensor carpi radialis longus, extensor carpi ulnaris, the extensor digitorum communis, and the extensor digiti quinti minimi. These muscles arise from the distal forearm and the lateral epicondyle. This group is often subdivided into the muscles of the "mobile wad," including the brachioradialis,

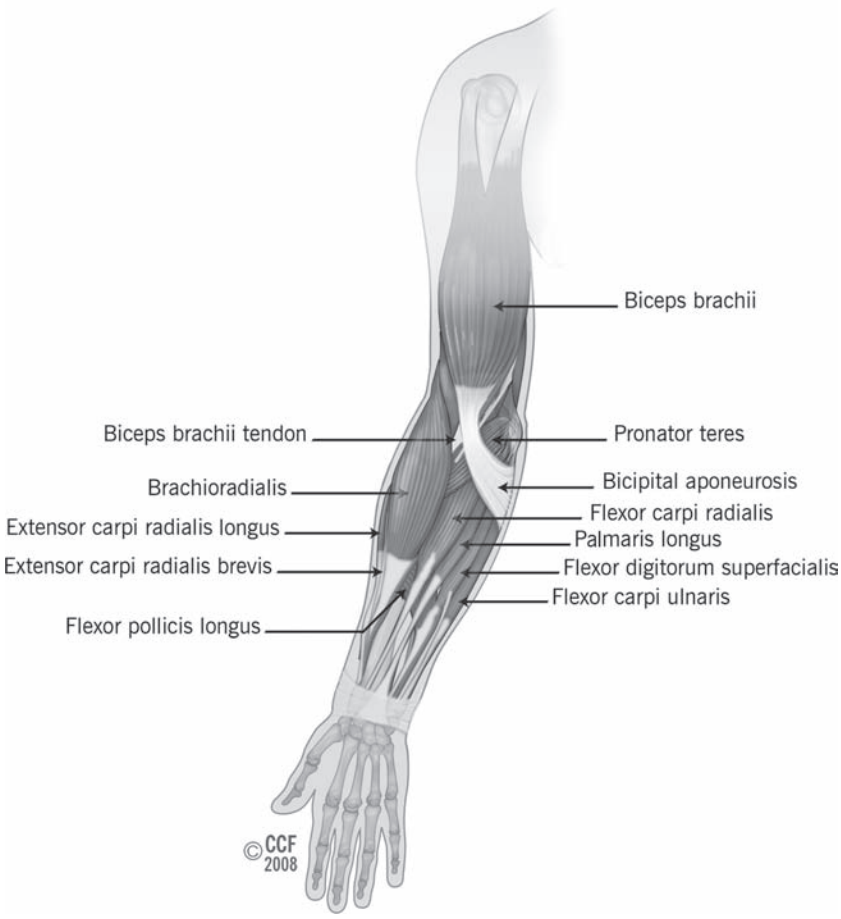

Figure 34.6. Muscles of the volar arm.

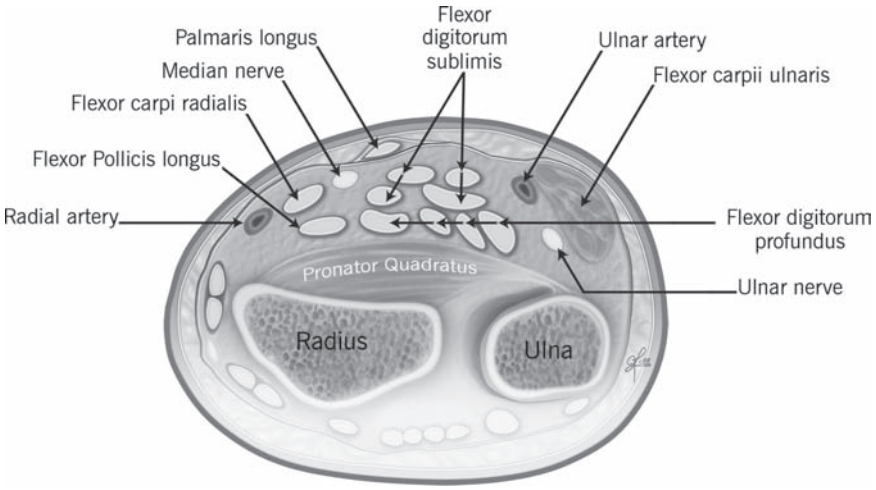

Figure 34.7. Cross-sectional anatomy of the volar forearm just proximal to the carpal tunnel.

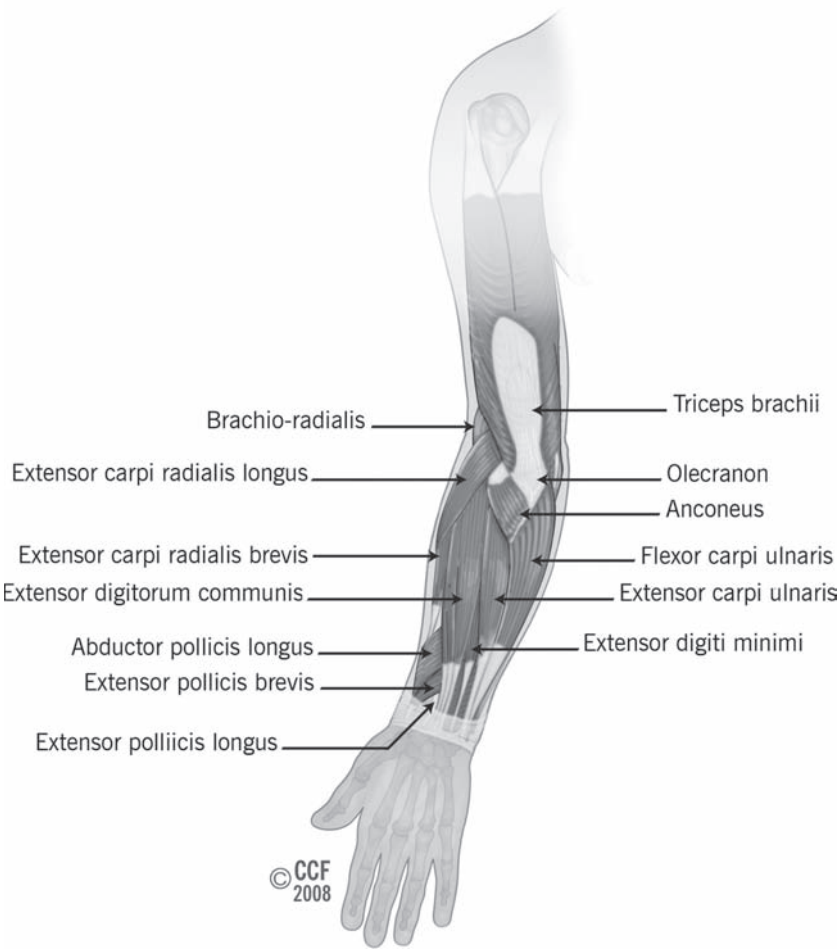

Figure 34.8. Muscles of the dorsal arm.

extensor carpi radialis longus, and extensor carpi radialis brevis.

The deep muscle compartment of the dorsal forearm includes the extensor pollicis longus and brevis, the abductor pollicis longus, the extensor indicis proprius, and the supinator muscles. The supinator arises from the humerus, whereas the remaining muscles originate from the proximal radius, ulna, and interosseous membrane. As these muscle bellies become tendinous and cross the wrist, the deep fascia forms into a strong, thick, retaining structure called the extensor retinaculum (Figure 34.9). Vertical septa originating from the distal radius and ulna create six fibro-osseous compartments. Numbered from radial to ulnar, the first extensor compartment[23] consists of abductor pollicis longus and extensor pollicis brevis tendons; the second compartment, the extensor carpi radialis longus and brevis tendons; the third compartment contains only the extensor pollicis longus, whereas the fourth compartment contains the extensor digitorum communis, as well as the extensor indicis proprius tendon; the fifth compartment contains the extensor digiti quinti minimi tendon; and finally the sixth compartment contains the extensor carpi ulnaris tendon. Just distal to the extensor retinaculum on the radial side of the wrist is the "anatomic snuff box," which is bound by the first and third dorsal compartments. Within the substance of the extensor

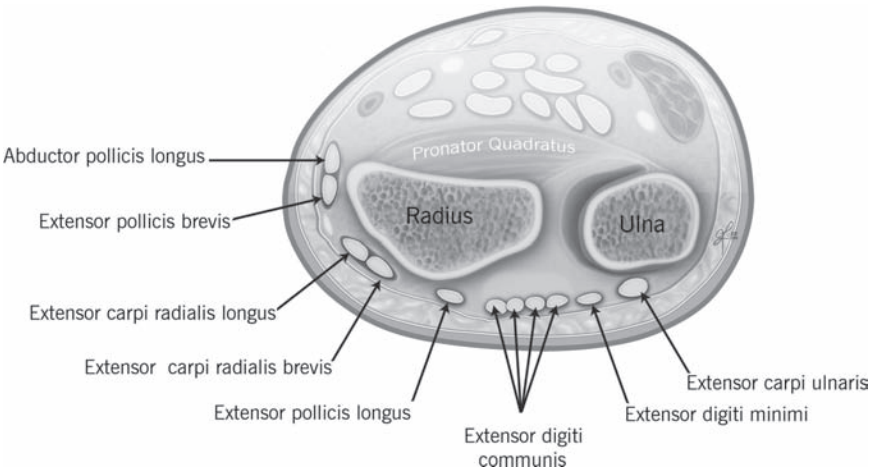

Figure 34.9. Cross-sectional anatomy of the dorsal forearm just proximal to the extensor retinaculum.

retinaculum is *Lister's tubercle*, which protrudes from the dorsal aspect of the distal radius and forms a useful anatomic landmark. The extensor pollicis longus is found immediately ulnar to the tubercle, which acts as a pulley for the tendon, whereas the contents of the second dorsal compartment are immediately radial to the tubercle.

Intrinsic System

The intrinsic muscles within the hand are responsible for the fine coordinated movements of the hand. The muscles themselves are completely contained within the hand distal to the carpus. The thenar eminence has four muscles, including the abductor pollicis brevis, flexor pollicis brevis, opponens pollicis, and adductor pollicis.

The flexor pollicis brevis and opponens pollicis are innervated by contributions from both the median and ulnar nerves. The median-innervated muscles all lie radial to the flexor pollicis longus tendon and include opponens pollicis, abductor pollicis brevis, and the superficial head of the flexor pollicis brevis. The median-innervated muscles serve to position the thumb basal joint. The-ulnar innervated thenar muscles include the adductor pollicis, the first dorsal interosseous, and the deep head of the flexor pollicis brevis. These muscles contribute to key pinch. The abductor pollicis brevis is the only muscle solely innervated by the median nerve. This has clinical significance in that the abductor pollicis brevis can be individually tested to determine if the recurrent branch of the median nerve, which usually arises just distal to the flexor retinaculum, is intact. These muscles lie within a compartment separated by a fascial sheath that extends from the radial margin of the palmar aponeurosis. The final muscle of this group, the adductor pollicis, is a fan-shaped muscle containing both a transverse and oblique head, with its origin from the third metacarpal and its insertion in the base of the proximal phalanx of the thumb. The abductor itself is innervated by the ulnar nerve and has an action similar to the remaining adductors of the fingers.

The hypothenar eminence is completely ulnarly innervated and is made up of the abductor digiti quinti, flexor digiti quinti, and opponens digiti quinti. As with the thumb, a potential space is formed within the hypothenar eminence, which is shut off from the central space by a vertical extension from the palmar aponeurosis. Incisions for draining an abscess in this area are made at the ulnar side of the fifth metacarpal to avoid damage to the deep branch of the ulnar nerve that innervates these muscles.

The remaining intrinsic muscles act to flex the metacarpals and extend the interphalangeal joints (Figure 34.10). These consist of the lumbrical muscles, which are the only muscles in the body to originate from a tendon. They arise from the radial side of the profundus tendon of each finger and insert deep to the transverse metacarpal

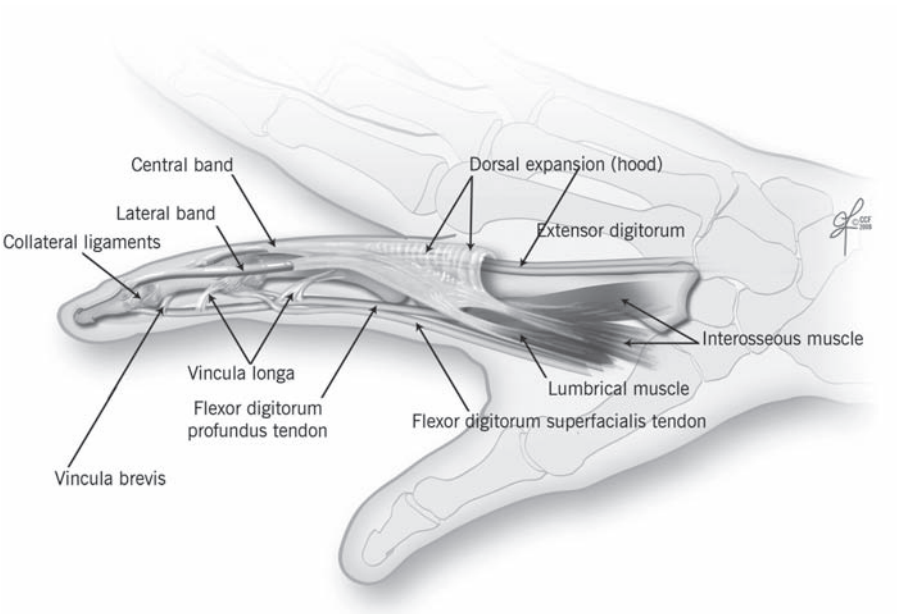

Figure 34.10. Extensor mechanism of the index, including the lumbrical muscle, first dorsal interosseous muscle, and extensor hood. Flexor vincula blood supply represented as well.

ligament joining the radial lateral band of the middle and proximal phalanges.[8] As they start volar to the axis of the metacarpophalangeal (MCP) joint and end up dorsal to the proximal and distal interphalangeal joints, they extend the PIP and DIP joints while flexing the MP joint.[37] When the lumbrical contracts, it pulls the distal profundus tendon toward the lateral band, decreasing the strength of the contraction at the DIP joint while allowing for more effective extension of the DIP joint. This allows for a balanced grip.[5,33]

The remaining muscles, the interossei, can be broken into two groups, the dorsal and volar interossei. Four dorsal interossei act as abductors of the fingers. The anatomic axis for all of the interosseous is the third metacarpal. The small finger is abducted by the abductor digiti quinti previously mentioned and acts similar to the remaining dorsal interossei muscles. The volar interossei are the adductors, with the middle finger acting as the axis again. As such, the middle finger has two dorsal interossei and no volar interosseous. Each of the dorsal interosseous muscles has two heads, with the exception of the third interosseous. The superficial head of the dorsal interosseous muscles terminates in a medial tendon deep onto the lateral tubercle of the base of the proximal phalanx. The deep head of each dorsal interosseous forms a lateral tendon, which ultimately coalesces with the lateral band of the extensor hood of the fingers. This tendon starts palmar to the MCP joint axis and, like the lumbricals, ends up dorsal to the PIP and DIP joints. As with the lumbricals, the interossei flex the MCP joints and extend the PIP and DIP joints.

More distally the oblique fibers or spiral fibers of the lateral bands sweep over the proximal interphalangeal joint joining the central tendon, ultimately terminating in the conjoined lateral bands that unite at the level of the distal third of the middle phalanx to form the terminal tendon inserting on the base of the distal phalanx to extend it.[24]

Unlike the dorsal interossei, the volar interossei have only one muscle head and insert into the ulnar lateral band of the index finger and the radial lateral band of the ring and small fingers. As such, these tendons do not attach onto the proximal phalanx itself. All of the interosseous muscles and the lumbricals to the ring and small fingers are innervated by the ulnar nerve, whereas the lumbricals to the index and middle fingers are innervated by the median nerve.

Bony and Ligamentous Anatomy

Wrist

An understanding of the anatomy of the normal wrist is required before appreciating functional loss from diseases of the wrist. The distal ulna articulates with the radius in the sigmoid notch. This is known as the distal radioulnar joint, and movement through that joint is pronation and supination of the hand. In the normal wrist, the distal portion of the ulna is shorter than the ulnar portion of the distal radius. This allows room for the articular disc known as the triangular fibrocartilagenous complex (TFCC).[1] The TFCC is a fan-shaped structure that acts as a hammock-like sling to the ulnar side of the wrist.

This distal radial ulnar joint supports the eight carpal bones (Figure 34.11). The proximal row includes the scaphoid, lunate, triquetrum, and pisiform bones. The pisiform bone is a load-bearing sesamoid bone of the wrist and is formed within the substance of the flexor carpi ulnaris tendon. Is has a function that is similar to that of the patella and is essentially separate from the remaining bones of the wrist. The distal row is formed by the trapezium trapezoid capitate and hamate. It is of note that the scaphoid itself extends into the distal row as well. The carpal bones have both intrinsic and extrinsic ligaments. The intrinsic ligaments are confined within the carpal bone themselves, whereas the extrinsic ligaments extend from the carpus to the radius and ulna proximally and metacarpals distally. Important ligaments of the extrinsic system include the radial collateral ligament as well as the radioscaphocapitate, radiolunate, and radioscapholunate ligaments. On the ulnar side of the wrist, the important extrinsic ligaments include the ulnar collateral ligament as well as the meniscus, palmar ulnocarpal ligament, and the TFCC itself. The space of Poirier is present due to the absence of a volar lunocapitate ligament. With the exception of this, the palmar ligaments are markedly stronger than the dorsal ligaments. The overall arrangement of the eight bones and ligaments that form the carpus is extremely complex. Motion takes place within the radiocarpal joint and the mid carpal joint, each contributing approximately 50% of the overall motion of the wrist. More distally, the index and long finger metacarpals are firmly attached to the distal row of the carpus and form a fixed unit of the hand. The metacarpals of the ring finger and particularly the small finger have increased motion that allows for cupping of the palm.

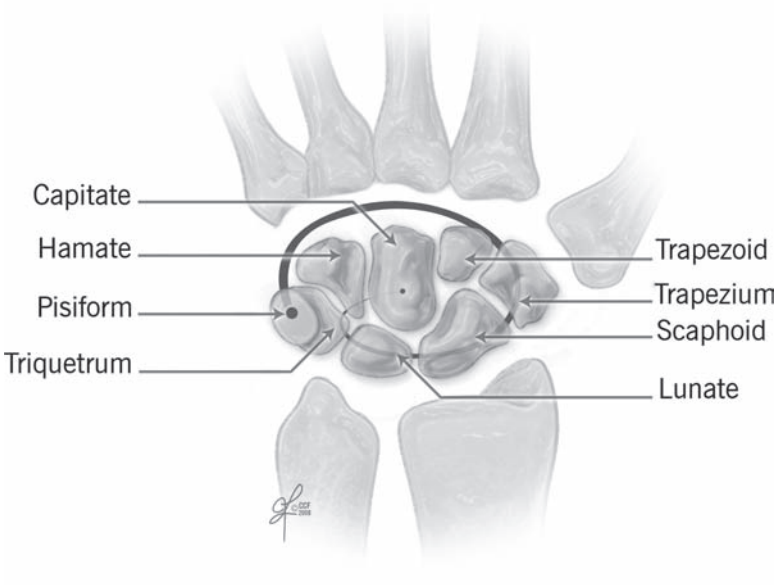

Figure 34.11. Bones of the wrist. Spiral line represents the order of calcification of each bone.

From a pathologic standpoint, it is most helpful to consider the proximal row as one unit that acts as a strong spring in tension. On the thumb side, the scaphoid bone tips palmar to the plane of the wrist, and the tendons pulling of the thumb cause it to act as a cantilever, whose force is held in check by dorsal tension on the triquetral bone. As the lunate is central to both the scaphoid and triquetral bones, it becomes key to understanding pathology of the wrist.[15] When the radial side of the wrist has a disruption either through a fracture of the scaphoid or tear in the scapholunate ligament, the spring is broken, and the scaphoid itself tips down, which in turn releases tension, causing the lunate and the still attached triquetrum to tip dorsally. Likewise, a disruption between the lunate and the triquetrum will cause the lunate, still attached to the scaphoid, to tip palmarly. A more detailed description of this can be obtained in the section on wrist instability. Just distal to the carpal bones are the five metacarpals. Motion between the carpus and metacarpals is limited in the fingers, but within the thumb, the carpal metacarpal joint is a saddle-type joint and allows for much of the complex motion of the thumb, including abduction, adduction, rotation, and opposition.

Bones of the Hand

The hand consists of five metacarpals and 14 phalanges (Figure. 34.12). Between the metacarpals and the phalanges are the MCP joints (Figure. 34.13). The MCP joints are all similar in that they act like a hinge. Because the head of the metacarpal is oblong, as the MCP goes through flexion, the proximal phalanx moves away from

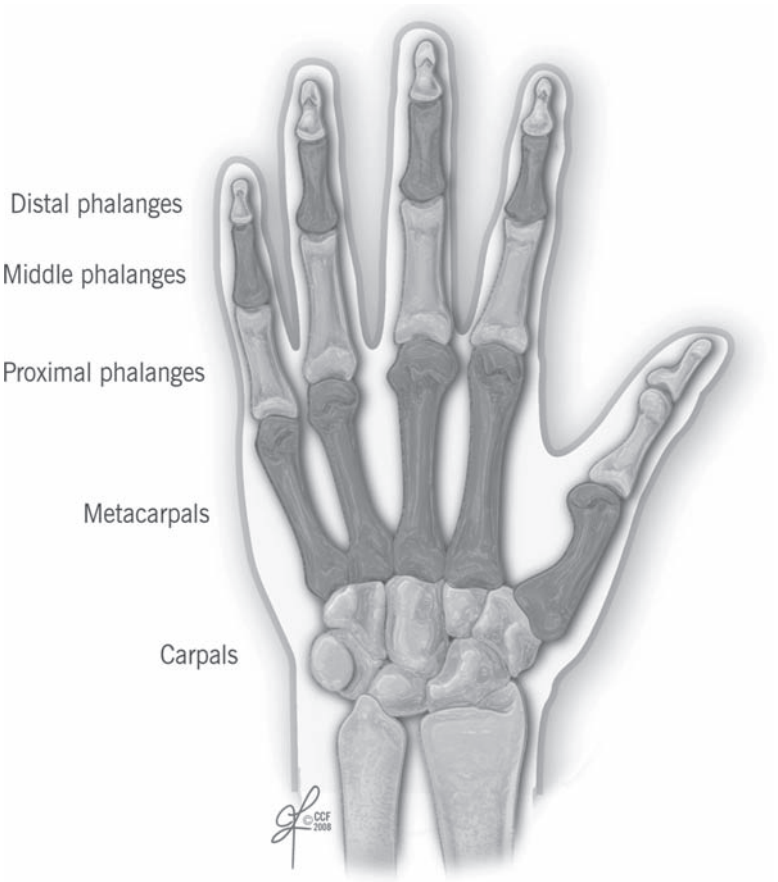

Figure 34.12. Bones of the hand.

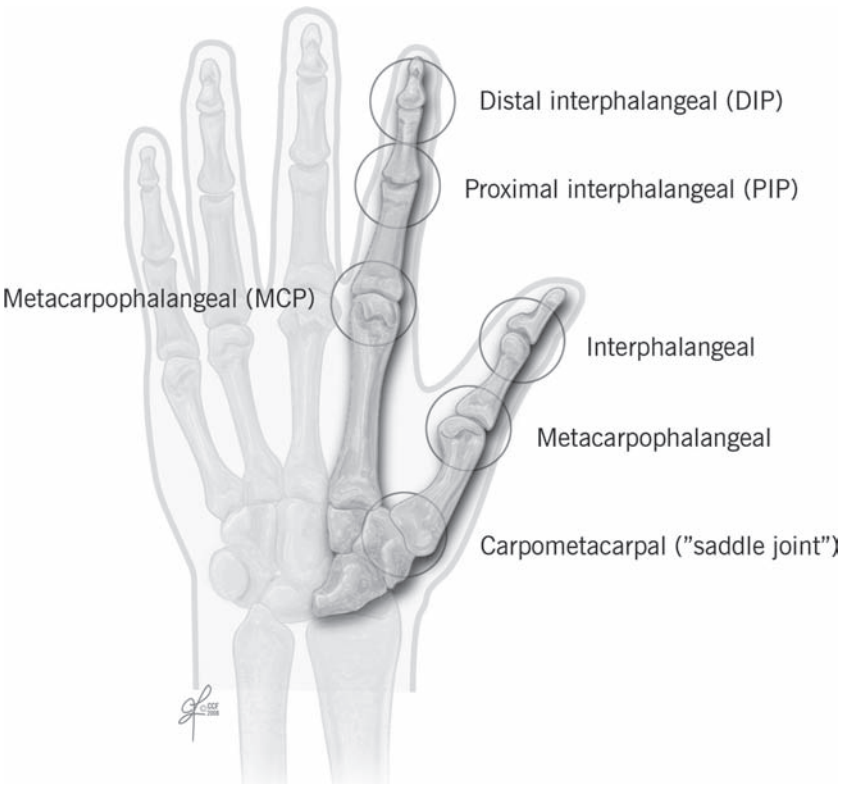

Figure 34.13. Joints of the hand.

the axis of rotation therefore tightening the collateral ligaments. The collateral ligaments of the MCP joint are broken into a proper and accessory ligament.[21] The proper collateral ligaments allow for stability with the MCP joint in extension, whereas the accessory ligaments become progressively tighter as the MCP joint is flexed, and the flare of the head and eccentric axis elongates these fibers, causing greater stability with greater flexion. Deep structures are the joint capsule itself, which is a loose areolar tissue laterally and dorsally and coalesces into the thick volar plate supporting the base of the flexor tendon sheath.

The volar plate of the MCP joint lacks check ligaments as opposed to the PIP and DIP joints that both contain check ligaments lining the lateral aspect of the phalanges proximal to the volar plate. The flexor digitorum profundus and superficialis tendons are contained volarly within the tendon sheaths. These form a retaining system that prevent bow stringing of the flexor tendons as the finger is flexed. Transverse thickenings of the tendon sheaths are called annular pulleys (Figure 34.14). The first pulley (A1)[38] is associated with the volar plate and MCP joint and is deep to the distal palmar crease; the second pulley originates from the proximal phalanx; the third pulley is associated with the volar plate of the PIP joint; the fourth pulley, the middle phalanx, whereas the fifth pulley is just at the level of the volar plate of the DIP joint.[13] Between the annular pulleys are cruciate pulleys C0–C3. These pulleys are made of looser tissue in a *cross* arrangement, allowing for flexion at near the joints, whereas the stiffer annular pulleys still retain the tendons close to the axis of each joint. Functionally, these pulleys improve the efficiency of flexion through decreased tendon excursion.[25,26] The trade-off is less power than if the pulleys were absent.

The anatomy of the pulley system in the thumb differs from that of the fingers in that there are two annular pulleys located over the

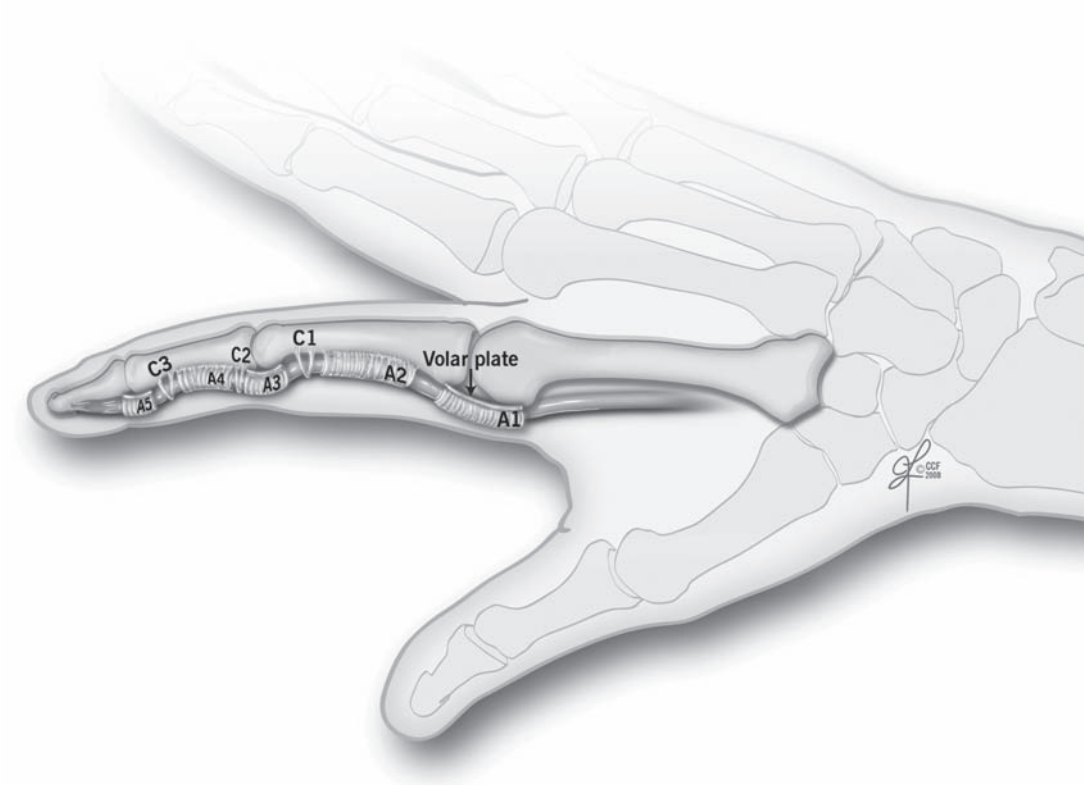

Figure 34.14. Flexor pulley system.

volar plates of the MCP and IP joint, whereas the pulley over the proximal phalanx is oriented in an oblique fashion and is named the oblique pulley.[14] It is an extension of the adductor pollicis aponeurosis and is the main restraint preventing bowstringing of the flexor pollicis longus tendon.

Within the flexor tendon sheath, the tendons obtain a blood supply from the vincula, which are vessels carrying structures that originate on the volar aspect of the bone and insert on the dorsal aspect of each tendon (Figure. 34.10). Each of the FDS tendon and FDP tendon has one short and one long vincula. The vincula brevia is located at the flexor tendon's insertion, whereas the vincula longa is located more proximally. These vascular leashes will often arrest proximal movement of the flexor tendons in trauma cases wherein the tendons have been transected.[34]

Epiphyseal Anatomy

When considering the anatomy of the hand, it is also important to understand the difference between the bony anatomy of children and that of adults. The most obvious difference is noted on radiographs of the epiphysis. When interpreting a radiograph, it is important to note that newborns lack enough calcium to demonstrate a distinct epiphysis or even a bone if young enough. Within the hand, the epiphyses become demonstrable at varying times from approximately 10 months to 3 years, depending on the child and the specific bone (see spiral in Figure. 34.11).[19,35] All epiphyses of the fingers and metacarpals are closed by approximately 14–16 years of age, whereas the epiphyses of the radius and ulna close between 16 and 18 years of age. Boys generally lag behind girls. Within the metacarpals and phalanges, the epiphyses of the

metacarpals are seen distally, whereas in the phalanges they are all proximal. The exception to this is the epiphysis of the metacarpal of the thumb, where it is proximal and is similar in appearance to the three phalanges of the fingers (Figure 34.15).

Finger Tip and Fingernail

The anatomy of the fingertip warrants a separate discussion. The fingertip is defined as everything distal to the insertion of the extensor and flexor tendons of the distal phalanx. Tissues include the skin, nail, subcutaneous fat, and bone (Figure 34.16).[39] The pad of the volar skin is attached to the underlying distal phalanx by vertically oriented tough fibrous septa.

The fingernail is important for stabilization, sensation, and cosmetic appearance. The nail and surrounding skin are called the perionychia. More specifically, the skin folds along the side of the nail are called the paronychia, while the junction between the nail fold proximal to the nail is the eponychium. The hyponychia is the junction of the distal part of the nail and the skin of the finger at the point that the nail leaves the nail bed.

The nail grows approximately 0.1 mm per day. At this rate, it takes approximately 3 months to

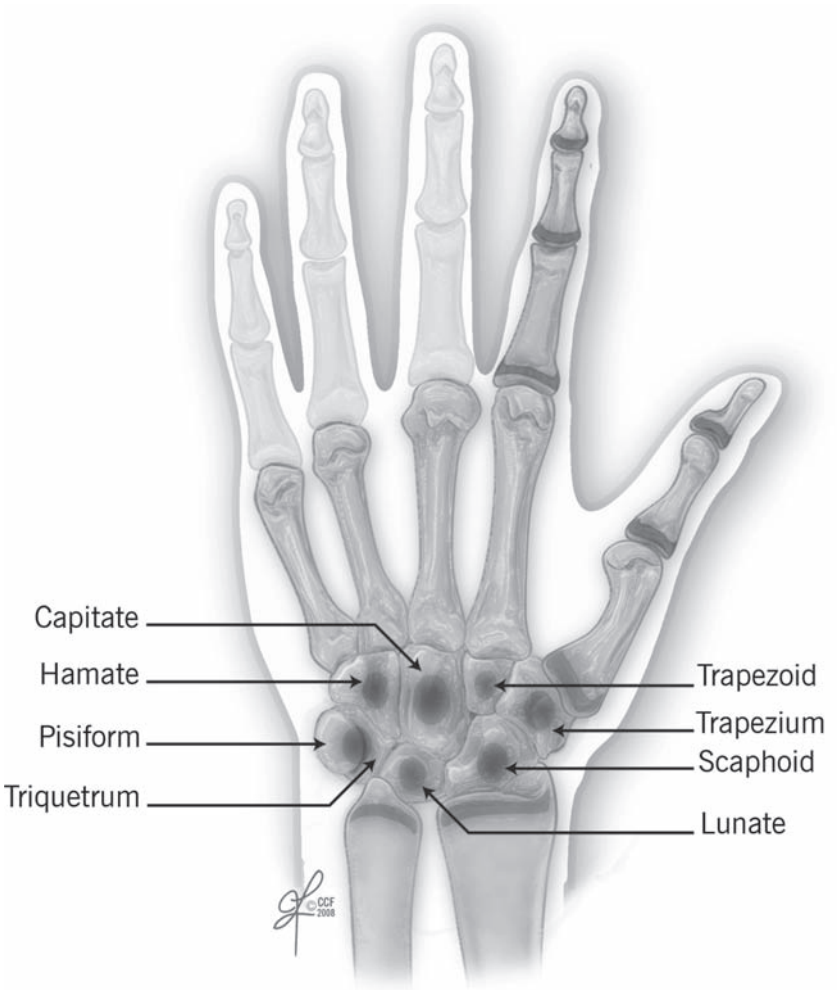

Figure 34.15. Epiphyseal anatomy of the hand.

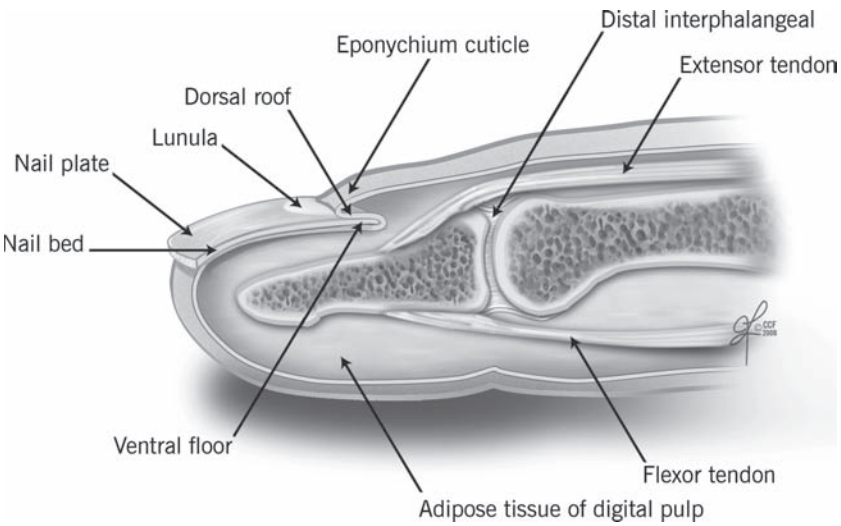

Figure 34.16. Fingertip anatomy.

grow from the eponychium to the fingertip. In addition to this time, there is an approximately 3-week delay before a nail begins to grow after injury. This delay is followed by an increase in nail formation over the next 50 days, causing a bulge. It takes approximately three full nail growths before the nail reaches a final appearance after injury. The dorsal roof and ventral floor of the eponychial fold along with the nail bed all contribute to the growth of the nail. The ventral floor from the proximal end of the nail fold just distal to the insertion of the extensor tendon out to the end of the lunula provides approximately 90% of the growth. The lunula is the moon-shaped whitish opacity at the proximal end of the nail, resulting from nail cell nuclei within the germinal matrix of the nail itself.

Blood supply to the nail is from terminal branches of the volar digital artery. Blood vessels and nerves of the nail coalesce distally to form the glomus, which is a thermal regulatory organ that contributes to the modulation of peripheral circulation. The tip of the distal phalanx reaches to approximately the mid portion of the fingernail bed. The end of the distal phalanx is composed of hard cortical bone and is referred to as the distal tuft. Although this density contributes to the strength of the distal phalanx, it also increases the amount of time required for healing of fractures in the distal phalanx.

Conclusion

In conclusion, before treating diseases of the hand, a deep understanding of the anatomy and physiology is important. Review of this chapter should serve as a basis for further specialized study within each separate anatomic area.

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35

Congenital Deformities and Reconstruction

Paul J. Smith and Gillian D. Smith

Summary

Congenital hand surgery is an interesting but challenging area, where the deformity may not always fit a recognized pattern. Reconstruction may involve the application of innovative thought to basic principles of hand surgery. The challenge is not only to reconstruct to create a functional and aesthetically acceptable hand but also to do so in a way that does not interfere with growth and that will be maintained until skeletal maturity.

This chapter is not an attempt at a comprehensive coverage but reflects the authors' current opinions on areas of congenital hand surgery that plastic surgeons will most frequently encounter.

Abbreviations

EEC	Ectrodactyly-ectodermal dysplasia clefting
MCP	Metacarpophalangeal
PIP	Proximal interphalangeal
TAR	Thrombocytopenia absent radius
VACTERL	Vertebral, anorectal, cardiac, tracheo – esophageal, renal, limb abnormalities

Introduction

The congenital hand abnormality may be readily apparent, but, before embarking on a reconstructive surgical path, the surgeon must first be assured that the child does not have other less apparent congenital anomalies that may require more urgent surgical attention. The child should have been examined by a pediatrician to exclude other anomalies, in particular, cardiac defects. The surgeon must also have assessed the child's functional abilities, assisted by a formal assessment by an occupational therapist. This most likely requires more than one visit, since the cooperation of the child needs to be acquired, and this may vary from moment to moment as well as longitudinally in time as the child develops.

The surgeon must remember that he or she is, in the early stages, treating the child on behalf of the parents whose feelings of guilt and disappointment in having a child who has an abnormality may color their views and decision making. It is important that the surgeon is honest and realistic in what can be achieved for the child. Never will a completely normal hand result from surgical intervention. In the most minor of cases, a scar may be the only price to pay, but, more frequently, the hand that results will not look or function as well as a normal hand.

Later, once the child is able to understand the risks and benefits of surgery, the child may take

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part in decision making. Occasionally, where parental expectations are unrealistic, the parents and child may come into conflict at this point. An older child's refusal to undergo surgery should be explored to understand the child's reasoning, but where sound and not based on resolvable problems such as fear of anesthesia, it should be respected. Here it must be explained to the parents that the child will not come to harm from delaying surgery and that this option will still be available in the future should the child have a change of mind.

Initially, the surgeon seeks to gain the trust of the parents, but as the child matures, the surgeon needs to gain the trust of the child as well. If the child is developing normally, the child's requests should represent a gradually increasing autonomy away from parental reliance as he or she heads toward skeletal maturity.

Classification

The International Federation of Societies for Surgery of the Hand[6] agreed on a classification of congenital hands based on morphology. Approximately 10% of congenital hand anomalies do not fit into this classification, and others fit into more than one category. In the future, it is likely that this classification will be changed with the increase in the understanding of the genetic and developmental basis of limb anomalies.

I. Failure of formation of parts

Transverse – *at any level, no distal elements present*
Longitudinal – *phocomelia*
Radial club hand
Cleft hand
Ulnar club hand

II. Failure of differentiation of parts

Arthrogryposis
Camptodactyly
Syndactyly

III. Duplication

Polydactyly

IV. Overgrowth

Macrodactyly

V. Undergrowth

Thumb hypoplasia

VI. Constriction ring syndrome
VII. Generalized skeletal abnormalities

Syndactyly

Syndactyly, when counted in all its forms, is the commonest congenital hand abnormality. It occurs in 1: 1,000 live births. It may be unilateral or bilateral, complete or incomplete (not extending to the distal phalanx), simple involving skin only, or complex with bony synostosis and may be part of a syndrome, genetically inherited or isolated and sporadic. It may be combined with extra digits either as a complete ray, concealed within a digit, or in its most complex form as a confusing jumble of bones, which spans several rays; so it is uncertain which bones best match with which digits. Acrosyndactyly, where there are distal connections between the digits but proximally the webs are present to some degree, is seen as part of amniotic band syndrome and is considered later.

Syndactyly may affect fingers and/or toes. The commonest form of syndactyly is an incomplete second web syndactyly in the foot, whereas it is the third ray that is most often affected. In the hand the first web is the least commonly affected but requires the most urgent attention to release, as the syndactylized thumb will be unable to function effectively. The thumb should be made independent of the other fingers by the age of 6 months. In addition, both border digits need to be released early to allow adequate growth of the fingers to which they are joined. Failure to do so results in flexion contractures at the proximal interphalangeal (PIP) joints with permanent joint modeling

Before releasing a syndactyly, it is important to examine the passive mobility of the joints and their active motion – a clue to this is given by the presence or absence of joint creases – their absence suggests a failure of active motion due to extrinsic tendon abnormalities. If there are no creases in the digit itself, all motion will be from the intrinsic function of the interossei and lumbricals. The parents should be made aware of this before separation of the digits, as they may wish not to proceed in this instance. However, it is surprising how often the child prefers to have

five fingers even if some of them function poorly rather than an uncorrected syndactyly.

Where there is complete syndactyly, there is a likelihood of abnormalities of the neurovascular supply to that web. This becomes increasingly likely as the syndactyly becomes complex and is expected where there are bizarre osseous anomalies on radiograph. Separation in these situations has a risk of loss of one of the digits as not only are the vessels aberrant but they may be rudimentary or even entirely absent. It may be reasonable to defer separation until the child, and, therefore, all the neurovascular structures are bigger and the procedure may be more reliable. In the Apert's hand, where there is always osseous syndactyly of the third web and often only a single digital artery distally to this web, we operate early to release the thumb, index, and little fingers to produce a functioning hand but defer release of the central digital mass until the child is 5 years or older (Figure 35.1).

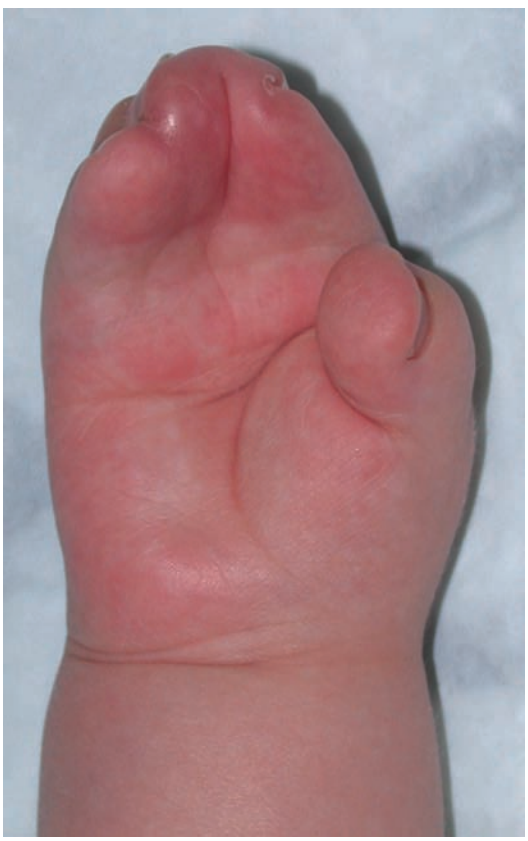


Figure 35.1. Apert's hand with simple and complex syndactyly of the digits.

There are a myriad of techniques described to correct syndactyly, using a volar, dorsal, or combination of flaps of varying sizes and shapes to form a new web. Since in almost all cases there is a major deficiency of skin, most of the techniques involve resurfacing the adjacent sides of the digit with a full-thickness skin graft. For a technique to be effective, it must not result in a straight-line scar across the web space – any that does, is doomed to failure in the long term, with web creep occurring due to scar contracture as the child grows. An appropriately placed and designed commissural flap will avoid this. The timing of surgery is controversial – where the syndactyly is minor; the later the surgical release is performed, the less likely that it will need revision with growth.

Radial Club Hand

This term is used to describe a spectrum disorder of radial longitudinal dysplasia, where the hand is found to be radially deviated, flexed, and pronated on the forearm. It is associated with varying degrees of radial hypoplasia or aplasia affecting the soft tissues, as well as the skeletal elements, on the radial side of the distal forearm and hand. It may be unilateral or bilateral but is rarely symmetrical. The right side is twice as frequently affected as the left. Its estimated incidence[2] is between 1:30,000 and 1:1,00,000 live births.[1] The etiology of this condition remains uncertain, with genetic factors playing a role in syndromic cases but apparently not in isolated ones. Similar defects have been created in chick wings, and it is postulated that they are caused by a deficiency of a morphogen produced by the polarizing zone of the developing limb.

There are a myriad of reported syndromes associated with this condition. Those that require early exclusion include those associated with hematological conditions, cardiac defects, and renal defects. Patients with hematological disorders include those with Fanconi's anemia who may avoid surgery entirely. This autosomal recessive condition produces a pancytopenia, which is fatal in the absence of bone marrow transplant. Siblings may be affected, but, when they are not, they may provide a compatible donor for bone marrow transplantation. Alternatively, those with hematological disorders may come to surgical intervention late. In thrombocytopenia absent

radius (TAR) syndrome, the patients have a platelet count that improves with age until it approaches normal at around 5 years. These patients have thumbs present bilaterally, but these are never normal. They are hypoplastic, supinated, have poor long flexors and extensors, and constricted first webs but are still better developed than those in other conditions.

Cardiac defects, commonly atrial and ventricular septal defects, in association with radial ray deficiencies are seen in Holt–Oram syndrome. This is an autosomal dominant condition in which other family members may be affected to a lesser degree and may be previously undiagnosed.

Patients with VACTERL syndrome form the largest individual syndromic group with associated radial club hand. These patients may have a broad spectrum of severity of other associated features (a minimum of three of the following: vertebral abnormalities, anorectal, cardiac, tracheo – esophageal, renal, limb anomalies, and a single umbilical artery). These patients often have greater degrees of radial deficiency and frequently have stiffness of multiple digits and camptodactyly of multiple digits, most noticeable in the middle and ring fingers.

Bayne and Klug have divided radial club hand into four groups according to the degree of radial hypoplasia: type I – short distal radius, type II – hypoplastic radius type III – partial radial aplasia, and type IV – radial aplasia (Figure 35.2). In our series, type IV is more frequent than the sum of all the others.

The whole length of the limb may be affected, but with the most profound changes in the wrist and hand. In one third of cases, the upper arm is shorter than the contralateral side, with the distal humeral epiphysis involved. There may be bony abnormalities of the elbow joint. Extension of the elbow is usually full, but flexion may be initially limited – provided there is some active motion at the elbow; then this will improve with splintage of the wrist, as originally observed by Lamb. Active pronation and particularly supination are limited, and the patients compensate by using shoulder rotation to turn the hand.

The forearm on the affected side is short, with the ulna often curved and growing only to a maximum of two thirds of the length of the contralateral ulna. Curvature of the ulna may increase with growth. The radius, if present, is always deficient distally and may be replaced by a fibrous condensation into which the combined

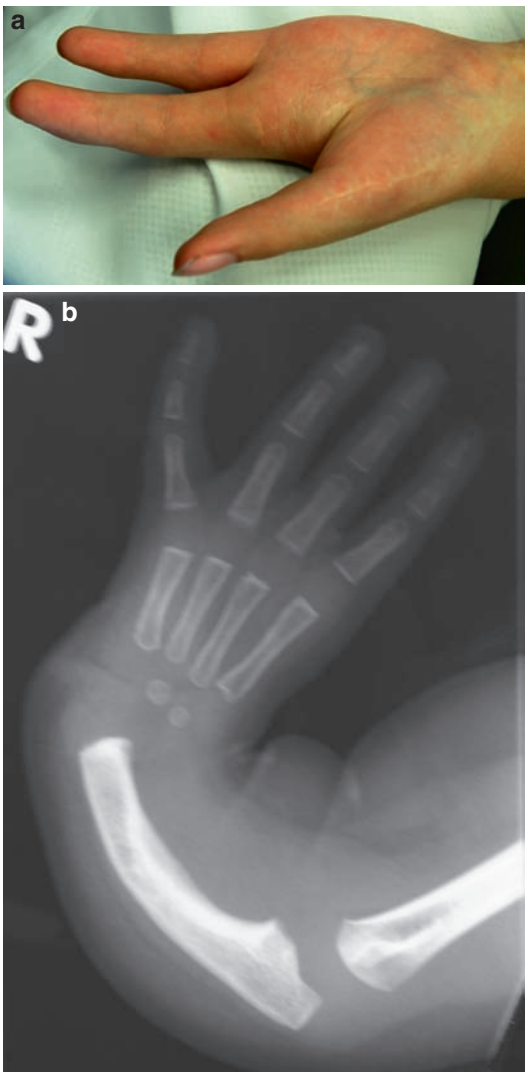


Figure 35.2. Ulnar club hand.

dorsoradial muscle mass inserts, together with, on occasion, the carpus. If the radius is present, then it may also be abnormal proximally. The trapezium and scaphoid are deficient or absent, and increasing carpal coalitions become evident with ossification. The thumb may have varying degrees of hypoplasia or may be absent. The digits may be mobile but typically show the greatest mobility on the ulnar side of the hand and increasing hypoplasia as you travel radially. The metacarpophalangeal (MCP) joints may have a restricted range of motion but normally hyperextend, whereas the proximal interphalangeal

joints may have flexion deformities, especially centrally and particularly in syndromic cases.

Untreated deformity is immediately evident, but a unilateral patient will have good function. A bilateral patient will adapt to performing tasks in an alternative manner, with a dorsal prehensive grip favored between the little and ring fingers where the radial digits exhibit stiffness. Treatment is initially aimed at functional improvement to correct the wrist position while maintaining motion and growth capacity, but with the child maturing, the child's own concerns will often be related more to the cosmetic appearance of the hand.

Treatment begins with physiotherapy by the parents and hand therapist, using stretching of the wrist and digits to attempt to bring them to neutral with splintage, and once this is achieved, to maintain the position. If the wrist is easily placed in neutral passively but tends to lie in radial deviation, then a tendon transfer of the dorsoradial muscle mass into extensor carpi ulnaris may be all that is required to rebalance the wrist.

In types III and IV radial club hand, it is difficult to place the carpus on the ulnar and much may be gained by soft tissue distraction. The distraction process allows gradual stretching of the soft tissues. It simplifies the process of subsequent wrist surgery; since there is no need for complex flaps to approach the wrist and a straight dorsal incision will suffice, the hand can be easily placed on the end of the forearm, leaving the fixator in place for longer than required, for the soft tissues allows remodeling of the ulna to correct ulna bowing and obviate the need for ulna osteotomy. There is a suggestion that it may stimulate ulna growth.

Following soft tissue distraction, the hand needs to be placed on the end of the forearm and the "wrist" rebalanced by tendon transfers. Options for management are radialization, in which the ulna is placed in line with either the second or third metacarpal, or centralization, which involves bony resection with the creation of a carpal slot. The procedure of preference is radialization, since it preserves greater movement at the wrist and avoids carpal resection and associated loss of length. However, some patients have too great a deficiency to be able to perform radialization successfully – we judge this on the quality of the dorsoradial muscle mass suitable for transfer. Of the patients whom we initially considered suitable for radialization, over a third needed reoperation within 3 years because of re-deviation to convert them to centralization. Overall, only half of our radial club hands had maintained their position satisfactorily 3 years later after initial radialization. The construct is inherently unstable, and longer-term results will show whether stability can be maintained with growth.

Many individuals during the last century have attempted to replace the deficient radius with a strut of bone graft to support the hand upon. Both nonvascularized and vascularized fibular head transfers have been tried. In 1998,[7] Villki took an alternative approach with the use of a free vascularized metatarsophalangeal joint transfer to provide a strut on the radial side of the wrist with growth potential, which supports the hand and allows the maintenance of movement at the wrist. This approach has not been adopted elsewhere.

After wrist stabilization, any thumb hypoplasia may need to be addressed.

Later, forearm lengthening may be considered, but the gains achieved here are limited and unpredictable. The degree of forearm lengthening achieved is dependent on the severity of complications that ensue. It is usually these complications, rather than the achievement of a certain amount of lengthening, that herald a halt to further forearm distraction.

Thumb Hypoplasia

Thumb hypoplasia (Figure 35.3a) is a form of radial ray deficiency, but it is classified separately and without consideration of the associated carpal abnormalities. The Blauth classification, since modified by Manske and Buck-Gramcko, is the most frequently used system. It subdivides thumb hypoplasia into five groups with gradually increasing degrees of skeletal abnormality.

Grade I represents a smaller than normal thumb but with all elements present, although some may be hypoplastic.

Grade II thumbs have an adduction contracture of the first web, laxity of the ligamentous structures of the metacarpophalangeal (MCP) joint, and thenar muscle hypoplasia. The senior author (PJS) has previously divided these into two groups.[4] In Grade IIA, ligamentous laxity is confined to the ulnar collateral ligament, whereas

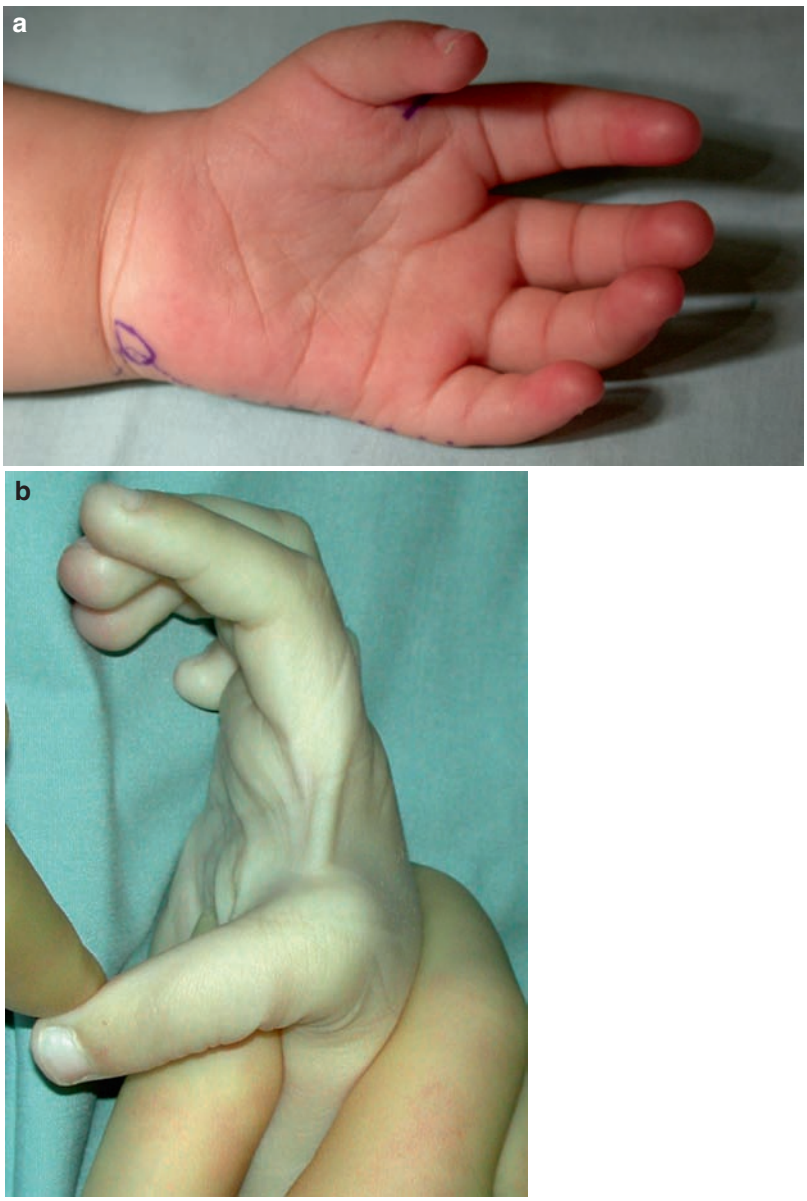


Figure 35.3. (a) Thumb hypoplasia with thenar hypoplasia and anomalous extrinsic tendons. (**b**) Unstable metacarpophalangeal joint in thumb hypoplasia.

in Grade IIB thumbs, there is global instability of the MCP joint.

Grade III thumbs have varying degrees of metacarpal hypoplasia with associated hypoplasia of thenar muscles and extrinsic tendons. Manske divided these into IIIA, where there is radiological evidence of a CMC joint, and IIIB, where there is no CMC joint due to proximal hypoplasia of the CMC joint. Buck-Gramcko added a further category, IIIC thumbs, where only the head of the metacarpal is present. The radiological presence of a first CMC joint does not guarantee its clinical stability, and these thumbs, even following reconstruction, have weak pinch due to first CMC joint instability.

Grade IV thumb has complete metacarpal aplasia and is known as a floating thumb, pouce flottant or Pendaldaumen. Grade V thumb hypoplasia represents complete absence of the thumb (Figure 35.4).

In reality, any grade of thumb hypoplasia can have either or both intrinsic muscle and extrinsic tendon abnormalities, which may affect its functional abilities to differing degrees.

Patients with Grade I thumbs, assuming they exist, do not present to the congenital hand surgeon, since these thumbs function adequately. Grade II thumbs require stabilization of the metacarpophalangeal joint and tendon transfers for opposition plus release of the first web space (Figure 35.3b).

Grade III thumbs are more difficult. Grade IIIA thumbs are considered reconstructable, but, although a CMC joint may be present and appear stable at rest, it may not be stable in pinch, with evidence of posterior subluxation and resultant weak pinch. Usually the MCP joint is also unstable and stabilization of that may cause a greater force transmission through the CMC joint to manifest previously unsuspected instability.

Grade IIIB thumbs and greater are best treated by amputation and pollicization of the index finger. This may be difficult for the parents to understand, and in some cultures, it is unacceptable. Many attempts have been made to reconstruct these highly deficient thumbs, but the results are largely both functionally and aesthetically inferior to those obtained by pollicization.

Pollicization is the pinnacle of congenital hand procedures but is extremely dependent on both the quality of the index finger being used and the surgeon's judgment and abilities. The greatest chance of a favorable result is at the primary procedure. Even so, approximately 45% of our patients have required revision procedures – usually first web release, extensor tenolysis, or opponensplasty. The best results will still produce a thumb that is only one-third to half the strength of the normal thumb.

The stages in performing a pollicization are careful planning of skin incisions, isolation of the arteries to the index finger (frequently there is only one vessel available), teasing back to separate the common digital nerve, isolation of the tendons for transfer, extraperiosteal excision of the metacarpal base and shaft, destruction of the metacarpal epiphysis, repositioning of the thumb in a pronated position with k wiring, and extensor

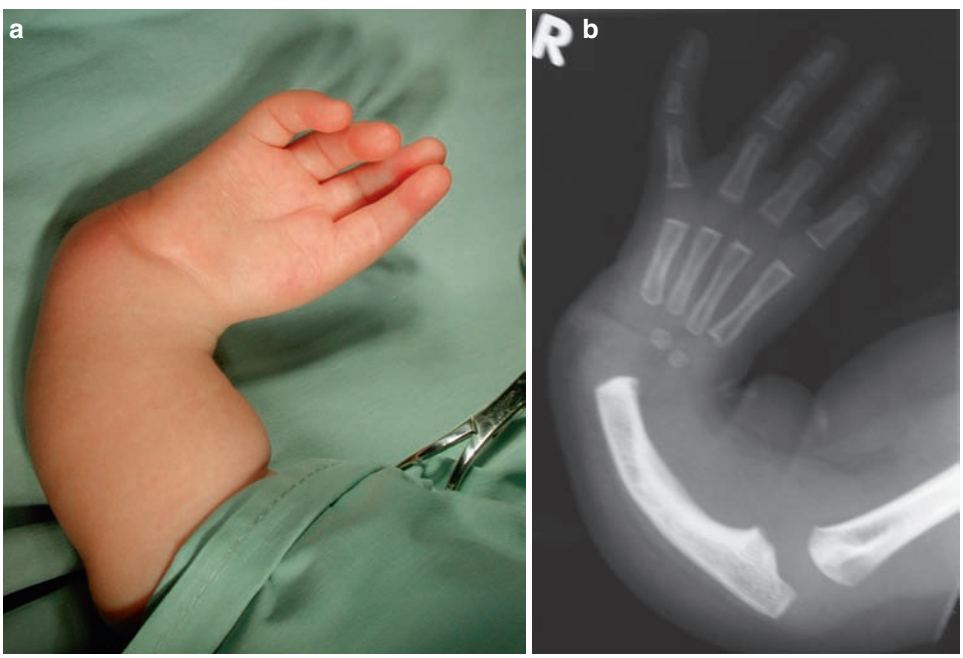


Figure 35.4. (a) Radial club hand with Blauth V thumb hypoplasia. (**b**) Radiograph of type IV radial club hand.

tendon shortening and tendon transfers to maintain that position, followed by skin closure. The flexor tendon is shortened if the child is older than 7 years. The age of the child at pollicization is less important than the quality of the index finger and the surgeon's skill in the result that is obtained.

Ulnar Club Hand

This deformity has an incidence of 1:1,00,000 live births and is frequently bilateral. The deficiency is on the ulnar side of the hand, and other associated anomalies are largely musculoskeletal problems. In contrast to the radial club hand, in the ulnar club hand, the wrist is stable but the elbow may be deficient. There may be absent digits on the ulnar side of the hand, hypoplasia of the thumb and remaining digits, tendon abnormalities, varying degrees of simple or complex syndactyly and, in the most severe cases, a pterygium of the elbow with an associated flexion contracture (Figure 35.2). The degree of abnormality of the soft tissues is not reflected in the degree of hypoplasia of the ulna. The ulna deficiency has been classified by Bayne into the following:

I. Hypoplasia of the ulna distally
II. Partial aplasia of the ulna (proximal third always present)
III. Total aplasia of the ulna
IV. Radiohumeral synostosis

In types III and IV, a fibrous anlage is believed, by some, to produce the problems of bowing of the ulna and subluxation of the radial head seen in this condition. However, excision of this anlage does not appear to alter the likelihood of these deformities occurring, so its significance is disputed. In the mildest cases, the first presentation may be in late childhood or adolescence when growth leads to radial head dislocation, manifesting as a painless lump appearing at the elbow. Excision of this lump produces pain in the DRUJ, so it is best left alone if the patient is approaching the end of skeletal growth.

Duplication

Polydactyly competes with syndactyly as the most frequent congenital hand disorder.

Ulnar Polydactyly

In the little finger, this is usually due to autosomal dominant inheritance, and there is a marked racial preponderance in the black population. It can also be associated with syndromes particularly where the extra digit is more substantial.

Stelling divides ulnar polydactyly into three groups:

- Type I – Soft tissue mass without skeletal structure
- Type II – Digit with all normal components and articulating with a normal or bifid metacarpal or phalanx
- Type III – Complete digit with metacarpal

Type I is seen regularly and can be treated by formal removal in an operating room in the first 8 weeks of life under local anesthetic. Type II may require partial resection of the metacarpal head and reconstruction of the metacarpophalangeal joint. This requires general anesthesia and the child's digits must be large enough to insert a Kirschner wire to stabilize the joint temporarily. Correcting type III requires a ray amputation under general anesthetic and reattachment of the abductor digiti minimi tendon.

Central Polydactyly

Central polydactyly may be seen in association with varying degrees of syndactyly. Polysyndactyly in certain forms is known to be associated with abnormalities in the HOX D19 gene – typically where it produces a polysyndactyly of the fifth ray of the foot together with a suppressed polysyndactyly of the fourth ray of the hand. Gradually a greater understanding of the genetic defects associated with different hand anomalies is emerging.

One form of central polydactyly involves a duplication of the third ray proximally at metacarpal level, which shifts to an apparent duplication of the fourth ray within the digit. This is a genetically inherited condition, and anatomically, both the bony and neurovascular anatomy may be bizarre. Usually these digits have some degree of symphalangism and tendon abnormalities. The results from separation of these are not functionally satisfactory.

Radial Polydactyly

Wassel's classification, although less than ideal since it is based on an immature skeleton, is used to describe thumb duplication.

- Type I – Bifid distal phalanx
- Type II – Duplicated distal phalanx (Figure 35.5a)
- Type III – Bifid proximal phalanx
- Type IV – Duplicated proximal phalanx
- Type V – Bifid metacarpal
- Type VI – Duplicated metacarpal (Figure 35.5b)
- Type VII – Triphalangia, in either or both of the thumbs

Thumb duplication is rarely part of a syndrome and is usually unilateral, with the exception of type VII triphalangeal thumbs. In some cultures, this is considered to confer good fortune on the child, and those affected may not present for treatment until they are older or may not present at all. Each of the two thumbs is smaller and narrower than that on the contralateral side. The two thumbs may be symmetrical or not. In cases wherein there is one obviously deficient thumb, it is easier to decide on which thumb to keep. Where the deficiency is symmetrical, the situation may be more difficult, and, in some cases, one thumb may be deficient proximally and the other distally and a procedure sharing parts of both thumbs may be needed. These rarely prove totally satisfactory but make the best out of the parts available. Most of these duplicate thumbs only possess a single vessel each; particularly where an "on-top-plasty" is performed, there is a risk of vascular compromise.

Even where the decision is clear as to which thumb is to be discarded, the retained thumb will need exploration, as tendon insertions may be asymmetrical and failure to correct this early will produce deviation with growth. The flexor and extensor tendons may have an interconnection (pollex abductus), which limits joint movement and also produces lateral deviation with growth.

In types I and II with similar sized thumbs, the Bilhaut–Cloquet procedure is often recommended, but this gives a residual split nail, which is unsightly and risks epiphyseal damage with later growth disturbance. This procedure is limited in its application, and generally, a modification is performed wherein the entire nail and nail bed are taken from one of the thumbs and the entire nail bed from the other is discarded.

Type IV is seen most frequently, and here there is a bifid metacarpal head. The intrinsic

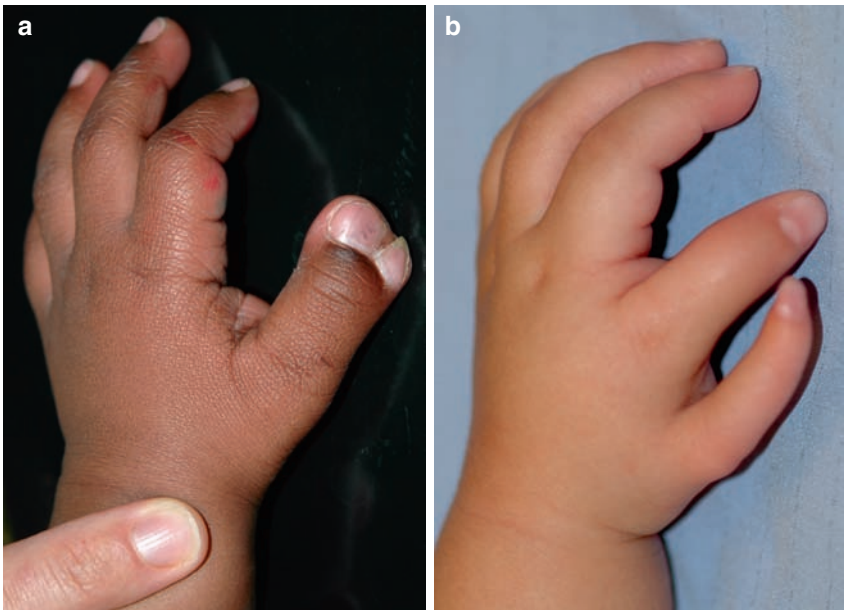


Figure 35.5. (a) Wassell II thumb duplication. (**b**) Wassell VI thumb duplication.

muscles must be detached, anomalous tendinous connections divided, tendon insertions corrected, the metacarpal head shaved to leave a single articular surface, and the collateral ligament reconstructed to the metacarpophalangeal joint with reattachment of the intrinsic muscles and enlargement of the first web space where required.

Type VII thumbs may require an on-top-plasty, wherein the distal part of one thumb is transferred as a vascularized transfer to the proximal part of the thumb with the better basal joint. Alternatively, these thumbs that are long and lie supinated in the wrong plane may require a formal pollicization.

Camptodactyly

Camptodactyly is a flexion deformity of the PIP joint of the digit and can be single or multidigit. The little finger is most frequently affected, and bilateral involvement is more common than unilateral (Figure 35.6). The digits normally have full flexion but are unable to actively extend fully and, in the later stages, unable to be passively extended. Where camptodactyly affects multiple digits on each hand, it is more likely to be associated with a syndrome and is less likely to respond to treatment. Two groups of patients are affected – infants and teenagers. Infants in the first 2 years of life have an equal sex incidence of this condition. In teenagers, it is more likely to be girls who are affected.

The mainstay of treatment is passive stretches and splintage until full extension is obtained and maintained. Radiographs may reveal a joint deformity with flattening of the proximal phalangeal head.

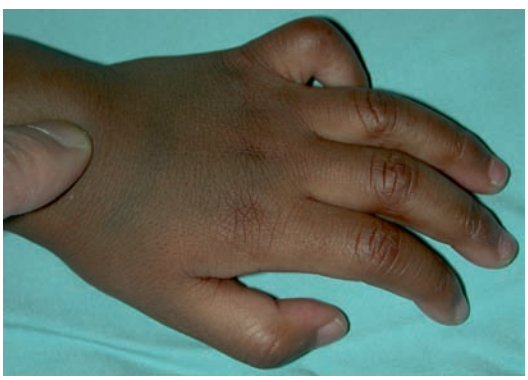


Figure 35.6. Camptodactyly of little finger.

Surgery can be deceptively complex, as all the structures on the volar aspect of the joint may be short. There is deficiency of the skin, abnormalities in the retinaculum cutis, tightness of the flexor digitorum sublimis, and, sometimes, the flexor digitorum profundus, abnormal insertions of the lumbrical, adhesions of the lateral bands to the proximal phalanx and interossei, and attenuation of the central slip. The latter can be demonstrated if despite full passive extension of the PIP joint with flexion of the wrist and MCP joints, the finger fails to actively extend in this position. Shortness of the FDS can be demonstrated by the FDS tenodesis test.

Surgery is thought to be worthwhile only in those with a deformity greater than 60°, but these patients are those either with the most severe disease or are the least compliant, neither of which augurs well for surgical success. Unless all the abnormalities are dealt with during surgery, failure is inevitable. In those where surgery is performed, the approach is volar through a longitudinal incision that can be converted into z-plasties to provide skin lengthening. The shortened retinaculum cutis is divided including the lateral digital sheets and Grayson's ligaments. The lateral bands and interossei may be inserted abnormally, and these attachments to the proximal phalanx are released. The FDS is explored for abnormalities – if it short, its two slips are divided distally and the longer ends reattached so that there is lengthening. If the FDS is absent proximally, then the distal end should be excised. Occasionally, the FDP is short or symmetrically duplicated with no FDS present. Here, lengthening is better performed in the palm or wrist. Lumbrical involvement is frequent, and tendon transfers of either FDS or EIP have been described to augment extension. They will function only if the lateral bands have been adequately mobilized. These muscles are stronger than those they are replacing, which may produce difficulty in obtaining flexion postoperatively. A few patients require a sequential release of the flexor sheath, accessory collateral ligaments, and volar plate, but these deformities tend to recur. No attempt should be made to address any bony deformity – in the infant, a surprising degree of bony remodeling is possible, and in the teenager, osteotomies just add to subsequent stiffness and loss of range within the joint. There is a risk of devascularization

of the digit, since the vessels may not tolerate full extension. Only a minority of patients obtain both full active flexion and full active extension postoperatively, whereas many do with splintage. Surgical intervention should therefore be reserved for those in whom conservative management has failed

Arthrogryposis

The term arthrogryposis refers to a collection of conditions where there are multiple joint contractures. The classical form, arthrogryposis multiplex congenita, consists of joint contractures and muscle wasting from birth, without evidence of any progressive neuromuscular disease. Its etiology is not firmly established although a viral illness affecting the anterior horn cells in the spinal cord is thought probable. The limbs look normal in shape but have few skin creases, tight muscles, and joint capsular contractures in a characteristic form: the shoulders are internally rotated, the elbows extended, forearms pronated, the wrists flexed, and ulnar deviated with the digits flexed and the thumb lying within the palm (Figure 35.7). The lower limbs show talipes equinovarus and dislocated knees and hips. Unfortunately, in the severely affected, the orthopedic surgeon may concentrate on operating to try to allow the child to walk, may achieve this only for a short time with aids, and may neglect the upper limbs. The child who becomes wheelchair dependent will have greater need of effective upper limb function for independence and may have been better served with a more balanced approach to his or her care. Proximal row carpectomy gives best results in these patients when performed before the age of 5 years, but frequently, the child is not referred to the upper limb surgeon until later.

Figure 35.7. Arthrogryposis with flexion deformity of wrist and thumb in palm deformity.

When operating on these children, the principles that must be applied are to avoid loss of the arc of motion of joints, to increase passive range primarily as that may improve function by the use of trick maneuvers, to maintain bimanual function as neither hand will function well independently, to position the limbs in front of the body at the level of the desktop, and to maintain function for assisting lower limb needs for transfers or the use of walking aids.

The shoulder may need repositioning by a humeral osteotomy so that the hands can meet in the midline with the elbows flexed. This is usually performed below the deltoid insertion and positions the arm in 30–40° of internal rotation.

The single most useful function the surgeon can gain is in passive elbow flexion. The elbow needs to remain stable for transfers, but a posterior elbow joint release to regain 90° of passive flexion with a tricepsplasty will improve function by allowing the hand to be brought to the mouth. Attempts at obtaining active flexion can be helpful but may risk producing an elbow contracture and limiting ability to transfer.

In patients older than 5 years, the most useful procedure for the wrist is a wedge resection of the carpus with tendon transfers to augment extension where available. At the same time, a lengthening of the digital flexors may be required.

The thumb exhibits an adduction contracture, laxity of the ulnar collateral ligament, and an absence of abductor pollicis brevis. In the majority of cases, this is best served with first web release with skin augmentation of the web and volar aspect of the thumb and metacarpophalangeal joint epiphysis-sparing fusion. A few patients are suitable for a tendon transfer from the extensor indicis proprius to improve extension instead.

Children with classical arthrogryposis have an intelligence quotient above average compared with their peer group, and, therefore, providing sufficient function to feed themselves, transfer, and operate a wheelchair and a computer keyboard may allow them to lead a relatively normal life for someone with such generalized impairments.

There are a group of arthrogryposes that comprise more distal involvement with contractures and intrinsic muscle dysfunction confined to the

hands – these are a more varied group, but a combination of splintage with stretches and surgery to the thumb where required is often sufficient to maintain reasonable function.

Cleft

The classical cleft hand is an autosomal dominant condition affecting both hands and feet and has an estimated incidence of 1:90,000 births.[1] The typical unilateral cleft of a single hand is usually sporadic. Clefting may be associated with other anomalies such as cleft lip and palate, talipes, ventricular septal defect, Fallot's tetralogy, or be part of ectrodactyly-ectodermal dysplasia clefting (EEC) syndrome. Patients usually have excellent function, but aesthetically the appearance is a disaster. The surgeon must be wary of interfering with function to improve the aesthetic appearance.

The cleft involves absence of one or more central rays and may include metacarpals or proximal phalanges that lie transversely in the web and will cause the cleft to widen with growth. The thumb is usually constrained by a tight web space, syndactyly to the index finger, or even metacarpal synostosis between the first and second metacarpals (Figure 35.8a and b). Syndactyly of the ring–little finger web is commonly associated, and polydactyly may be present.

The aims of surgery are to free the thumb, recreate a first web space by importing tissue from the central cleft, and to close the central cleft by reconstructing the transverse metacarpal ligament across the cleft and reconstructing the central web with a distally based flap. If there is a bone lying transversely across the cleft, this should be removed. As the child grows with an uncorrected cleft deformity, the digits on either side of the cleft rotate gradually toward each other so that they oppose. Correcting this involves realignment of the digits with metacarpal osteotomy so that scissoring does not occur with flexion. If there is a flexion deformity of the PIP joint, this should be explored to resect any abnormal bone, cartilage, or tendons that are contributing with appropriate z-plasties to close.

Several techniques have been described to correct the cleft hand deformity. The most frequently quoted is the Snow–Littler procedure,[5] which involves the transposition of the index finger to the base of the third metacarpal with release of the adductor pollicis and first dorsal interosseous muscles and transposition of a volar skin flap from the cleft into the first web. This is a technically demanding procedure with

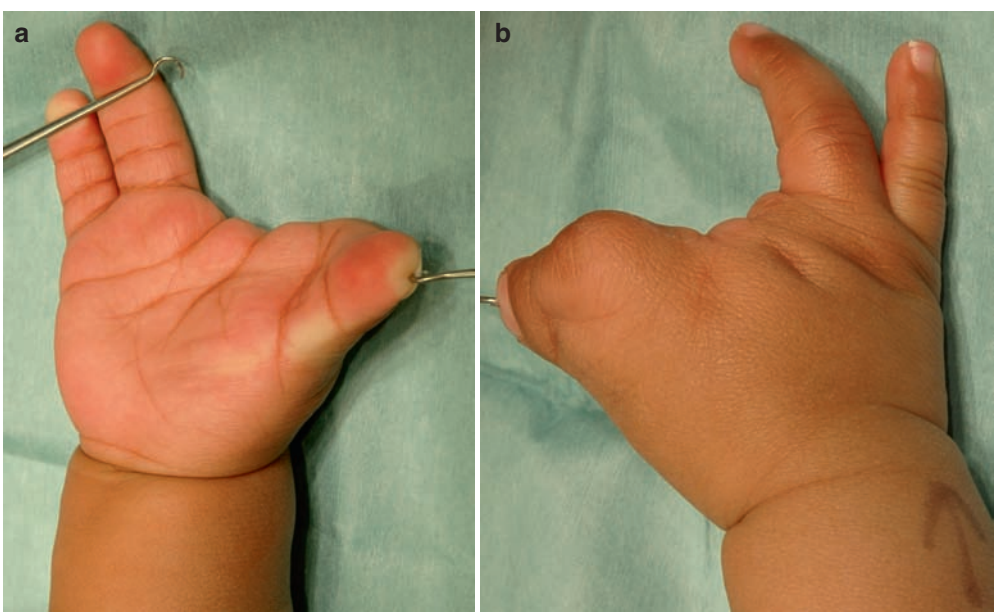


Figure 35.8 (a, b) Cleft hand with first web syndactyly and absent central ray.

a risk of loss of viability of the index finger and the difficulty of mobilizing the palmar skin flap without resulting in distal flap necrosis, which compromises the first web reconstruction. To overcome these problems, other authors, such as Miura and Komada,[3] have devised procedures to mobilize skin flaps largely on the dorsum to reduce these risks. These appear to produce similar results.

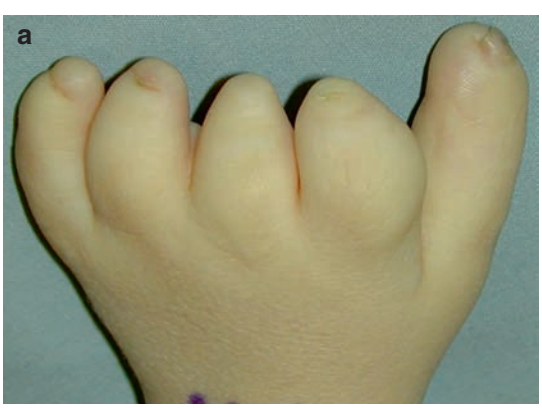
Symbrachydactyly

Symbrachydactyly is unilateral and sporadic and has an estimated incidence of between 1:10,000 and 1:30,000 live births. It may be associated with Poland's syndrome, where it is suspected that an interruption in the vascular supply to the subclavian artery in the early stages of the first trimester results in some underdevelopment of the territories this vessel supplies. The patient presents with loss of the sternal head of pectoralis major, chest wall deformity, nipple asymmetry with later breast asymmetry, abnormalities of the latissimus dorsi and serratus anterior, gradually increasing hypoplasia of the limb as progressing from proximally to distally, and classically a symbrachydactylous hand.

Symbrachydactyly constitutes a spectrum of disorders, with the least severe manifesting as a slightly smaller hand than on the contralateral side with smaller or absent middle phalanges. The most severely affected hand will have small metacarpals but no digits and residual nubbins of soft tissue that constitute all that developed of the distal elements of the digits.

There has been an attempt to group these into four broad subtypes to guide treatment options; short finger type, cleft hand type (U-shaped cleft previously described as an atypical cleft hand), monodactylous type (Figure 35.9a and b) and peromelic type.

The short finger type is functional and rarely needs any treatment apart from syndactyly release. The other types have functional and aesthetic issues, but it is only the former that the surgeon can address. He cannot create a normal hand. The cleft hand type has a thumb and little finger, which are often both mobile but lie in the same plane. Here, the aim of surgery is to adjust the two existing digits to produce



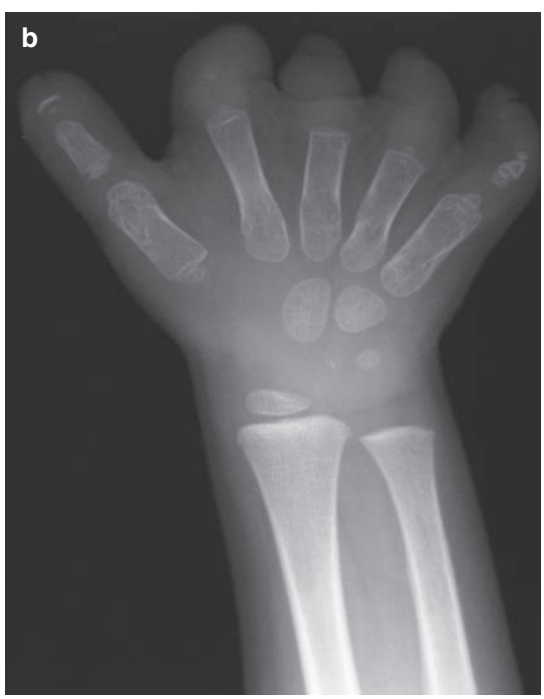


Figure 35.9 (a, b) Symbrachydactyly of monodactylous type.

two digits that oppose with the use of osteotomies and tendon transfers. The monodactylous type has a good thumb but no digits, and options for treatment include functional prostheses, free phalangeal transfers with or without later distraction augmentation manoplasty, and single or double free toe transfer. Free phalangeal transfers are suitable only in patients with empty digital sacs. It is possible to expand these by releasing the constricting fibrous tissue extending to the end of the sac to open the concertina-type constriction and allow placement of a whole proximal phalanx, harvested with its periosteum, onto the intact tendon that extends over the metacarpal head. It is not necessary to harvest the volar plate and collateral ligaments or to divide the extensor and flexor tendons over the metacarpal head. Although this can be carried out at any age, near-normal growth in the phalanx reportedly occurs if the child is less than 15 months of age. However, there are no series large enough to confirm this, and our series suggests it can probably be performed up to the age of 7 years. Some patients will not wish to have any further treatment having obtained a supported lateral pinch. For those who wish for longer digits, distraction augmentation manoplasty can be carried out in the compliant patient from approximately the age of 8 years, which may produce a gain in digital length of up to 3 cm. The syndactyly between the digits then needs division.

The peromelic type has the options of prostheses or double free toe transfer, but the latter is suitable only if it is likely to produce two opposable digits, and if there is an inability to cup the palm preoperatively, this is unlikely.

Trigger Fingers

Congenital trigger digits are believed to have an incidence of 3:1,000 live births. However, several large studies of newborns, where no trigger digits have been identified, have postulated that this is not a congenital condition but is likely to be developmental. Trigger thumbs are 14 times more common than trigger fingers. All trigger digits may present with a nodule in the palm (Notta's nodule) at the base of the A1 pulley, volar to the metacarpophalangeal joint, but although trigger fingers may present with triggering, trigger thumbs, unlike those in adults, most commonly present with a flexion deformity of the digit. This may be misdiagnosed by the unwary as a dislocation, but "reduction" will be followed only by a rapid recurrence of the deformity. The child is not usually in pain unless an attempt is made to straighten the digit.

Trigger thumbs are known to resolve, but the frequency with which this occurs and the time frame in which it is likely to do so are disputed. Estimates vary widely from no spontaneous resolution to 49%. Trigger fingers typically appear earlier than trigger thumbs, are more likely to resolve spontaneously, and, when operated on, tend to do less well.

Initially, stretching exercises are used, and splintage is used where the child will tolerate it. The Japanese have great success with splintage, which is sustained for months or years with improvement rates that are impressive. However, in our own practice, we have found difficulty with compliance at the age group who present with this problem.

Traditional teaching suggests that surgery should be performed in those who have failed to resolve by the age of 3 years, because there is a risk of permanent joint deformity. Some authors defer surgery until later, but there are no documented cases of problems from doing so.

Surgery for trigger thumbs involves an isolated release of the A1 pulley. This has excellent results with few complications. By contrast, correction of trigger fingers usually does not respond to division of the A1 pulley alone, and the best results have been reported when one slip of FDS is sacrificed concomitantly.

Ring Constriction Syndrome

This condition has an incidence of 1:15,000 live births and has associations with talipes equinovarus, cleft lip and palate, and other defects in the child and oligohydramnios in the mother. It has no known hereditary basis. The debate continues as to whether the problem is the result of extrinsic constriction from amniotic bands or an intrinsic defect in the germ cell layer – there is some supporting evidence for both schools of thought.

The defects are often multiple and present with a variety of manifestations (Figure 35.10). Paterson has divided these into four main groups:

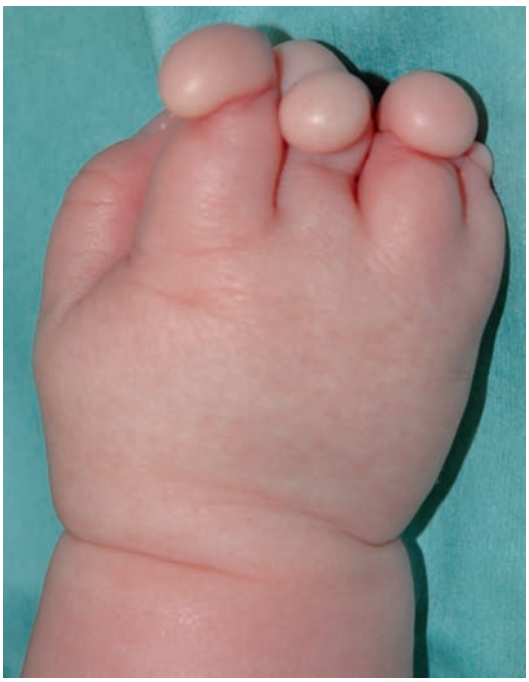


Figure 35.10. Ring constriction syndrome.

1. Simple constriction rings
2. Rings with distal deformity ± lymphedema
3. Rings with associated acrosyndactyly
   a. Type I – joined at the tips
   b. Type II – tips joined + web creep
   c. Type III – complete syndactyly with proximal sinus
4. Amputations

The timing of treatment depends on the presentation. Where there is lymphedema distally, urgent release of the ring may be required within the first few days of life. Early surgery may be required to release acrosyndactyly and allow individual digits to grow independently. Sometimes it is unclear which digit proximally is associated with which distally – here it is useful to consider that the middle finger is usually most volar. It is sensible to avoid complex surgery at that point and simply separate the digits, with grafting where required, as there is a risk of devascularization leading to digital loss in the most severe cases. Deep rings need release to encourage normal growth, while superficial rings may be purely cosmetic in nature. Traditional teaching suggests that circumferential excision is unsafe and multiple z-plasties are required circumferentially. The authors would not concur with this. Circumferential excision is safe except when the ring lies within 1 cm of the end of the digit. Excision is often to the level of the deep fascia and requires broad excision of the shoulders of the ring, not only its deepest levels. One or two z-plasties around a digit are sufficient, and if the ring is not circumferential, none is required. This broader excision of the ring means that only one ring can be excised on a digit at a time if there is to be sufficient remaining tissue for closure. Excess skin excised may be used as skin grafts in syndactyly release, but if the deepest fibrous areas of the ring are used, graft take appears to be impaired.

Patients with multiple amputations may be candidates for distraction augmentation manoplasty or for free toe transfer to recreate better grip and digital length.

Microsurgery

The role of microsurgery in congenital hand deformity is expanding. Free flaps, such as the free groin or lateral arm flaps, used to create an adequate first web, allow the transfer of a substantial quantity of thin vascularized tissue in situations wherein local flaps may be insufficient, such as when separating a complex syndactyly of the first web. Free vascularized muscle transfers allow restoration of flexion or extension after resection or destruction in compartment syndrome of the forearm musculature.

Free vascularized joint transfers have been used with good results in correction of the radial club hand deformity to stabilize the wrist, but they simultaneously provide a good range of motion within it and in Blauth type IIIB thumb hypoplasia to replace the absent first CMC joint.

Free toe transfer is used to reconstruct the thumb and absent digits with good functional results in the appropriate patient. Initially, the aesthetic results of this surgery were poor, but improvements have been made by reducing the pulps of the toes as a secondary procedure. The donor site from harvesting a single second toe from each foot is acceptable, and studies of the foot later have shown no functional problems with this. Harvesting of the great toe but with preservation of the metatarsal head produces a slight decrease in the push off in sprinting.

Harvesting of more than a single toe from each foot leaves an unacceptable aesthetic defect.

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36

Hand Trauma, Dislocations and Fractures, Infections

Frédéric Schuind, Wissam El Kazzi, Jörg Bahm, and Konstantinos Drossos

Summary

The goal in the treatment of a hand trauma or a hand infection is full and rapid restoration of hand function. The main complication is stiffness, which may be of articular origin, or result from ligament shortening, from adhesions within the gliding planes or along the tendons or the consequence of intrinsic tightness or algodystrophy. Understanding the precise cause of stiffness in a particular case allows the instauration of an efficient prevention. Most ligament injuries result from sporting accidents, and the proximal interphalangeal joints of the long fingers and the metacarpophalangeal joint of the thumb are particularly at risk. The principles of treatment of hand fractures are anatomical reduction, followed, when possible, by immediate mobilization and, if necessary, by immobilization of the injured finger in the "protective" position, minimizing that support period as much as possible, and remobilization when the fracture is clinically stable. The indications of replantation include amputation of the thumb, amputation of multiple digits, single-digit amputation distal to the insertion of the flexor digitorum superficialis, amputation in a child, and amputation through the palm, the wrist, or the forearm. Most hand infections result from direct contamination from a penetrating injury or a laceration.

Introduction

The human hand is powerful, stable, yet very mobile. It is exposed to a great variety of injuries. About one third of all accidents involve the hand; one third of all fractures occur in the hand. More time is lost for the industry because of the treatment for the fractures of the hand than for the fractures of the long bones. In developed countries, many hand lacerations result from domestic injuries, while work-related accidents tend to decrease. Crush injuries may result in devastatory lesions. Most hand infections are caused by direct contamination.

Many patients recover good hand function after trauma or infection. However, in a significant proportion of cases, severe stiffness, pain, and joint instability with loss of normal hand/upper-extremity function may result from the severity of the initial lesion and/or from inadequate treatments. Of those complications, stiffness is by far the most common, and understanding the causes of posttraumatic stiffness deserves discussion in the next sections.

Causes of Finger Stiffness

In order to prevent hand stiffness, the surgeon should evaluate immediately, on the day the trauma occurs or on the day the acute infection begins, the chances the patient has of regaining full motion; the surgeon should anticipate the

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risks of stiffness, understanding its origins, and thus apply an efficient strategy of prevention. Stiffness may be of articular origin, result from ligament shortening, from adhesions within the gliding planes or along the tendons, or be the consequence of intrinsic tightness. Algodystrophy is another cause. Occasionally, skin contractures may result in an extension lag.

Articular Origin

Joint stiffness may be the consequence of an insufficiently reduced articular fracture. Even with perfect reduction, proliferation of fibrofatty connective tissue within the joint space, overgrowing the cartilage surfaces like a pannus, may restrict joint motion, and this is especially seen after the joint has been immobilized. Over time, gross adhesions obliterate the synovial folds, which are relatively large in the joints achieving a wide range of motion (metacarpophalangeal and proximal interphalangeal joints). When the stiff joint is later manipulated, the cartilage may be damaged as a result of this additional trauma. Cartilage damage may also result from impaired nutrition, either by the connective pannus or from direct pressure applied on the surfaces of stationary contact.

Ligament Shortening, Adhesions within the Gliding Planes or Along the Tendons

Posttraumatic edema leads to the ingrowth of fibroblasts into the protein-rich tissue fluid that accumulates in the hand, resulting in shortening of relaxed, redundant ligamentous structures and in fibrous gluing within the gliding planes. In this context, the position of immobilization (if immobilization is needed) is critical (see below). With a few exceptions (liver, epithelium, bone), tissues do not heal by themselves, but after injury they are "cemented" together with an interposed material, the scar. All traumatized tissues in a given anatomical area become united by a common cicatrix, and independent function is lost. In the case of an open phalangeal fracture with an associated tendon laceration, or when an open reduction of a phalangeal fracture is performed, imposing tendon exposure, or in the case of a crush injury, there will be a common cicatrix with adhesions between the skin, the fascia, the tendon, and the fracture. The problem of tendon adhesions is particularly critical for flexor tendons, which need marked, differential excursion between themselves and relative to their fibro-osseous sheath. The extensor apparatus constitutes on the dorsal side of the fingers a wide tendinous strip, whose precise tension and excursion determine the finger position in space, under very precise neuromuscular control simultaneously involving the extrinsic extensor and the intrinsic tendons.[26] Minor imbalance may severely impair finger function, as seen for example in the saddle deformity caused by posttraumatic adhesions between the interosseous and lumbrical tendons, at the level of the deep transverse intermetacarpal ligament.[5] In general, tendon adhesions loosen progressively over time if early mobilization is realized. Physical stress exerts in addition a positive influence on collagen remodeling, provoking a polarization of that macromolecule and promoting its strength.[2,7] Excessive physical stresses, however, carry the risk of fracture displacement or of gapping/rupture of the tendon sutures. On the other hand, immobilization has deleterious consequences on the capsuloligamentous structures. Under stress deprivation, matrix disorganization and formation of irregular collagen bundles are observed; in addition, the ligament attachment sites are subject to significant weakening, due to localized osteoporosis.[1,12,16]

Intrinsic Tightness

The intrinsic muscles of the hand – interosseous, thenar, and hypothenar muscles – are contained within inextensible aponeurotic compartments. In the case of a crush injury, the muscles may undergo ischemic retraction, another cause of stiffness. Intrinsic tightness is characterized by limited interphalangeal joint flexion when the metacarpophalangeal joints are held in extension.[23]

Algodystrophy

Algodystrophy, or complex regional pain syndrome (CRPS), as it is now called (the authors feel that algodystrophy remains a better term, as it includes the two cardinal signs of the affection, pain and dystrophy), is a very serious complication whose etiology is still not well understood. The current opinion is that algodystrophy corresponds to an excessive local inflammatory

response to the injury,[27] representing a local form of free radical disease,[28] with secondary sympathetic reactivity. Algodystrophy is a devastating biphasic condition with early exaggerated pain, allodynia, edema, and vasomotor instability, later followed by atrophy, fascial contracture, and joint stiffness.

Prevention of Hand Stiffness

Finger stiffness should be prevented as soon as possible.[17] If an acute compartment syndrome is suspected, fasciotomies should be immediately performed. Edema is part of the inflammatory reaction, a normal response to the injury; it is never totally available. Edema can be minimized by fracture stabilization and by debridement of all necrotic tissues. Primary wound healing, using skin grafts or skin flaps when indicated, is probably essential to reduce the inflammatory reaction. Drainage to limit hematoma formation and antibiotherapy to prevent infection are important as well. If possible, minimally invasive percutaneous procedures should be preferred over more aggressive open techniques. All too frequently, edema is aggravated by inappropriate dressings or casts, acting as venous tourniquets, and/or by postoperative immobility. We recommend avoiding, as much as possible, casts and bulky, circular, compressive dressings. As an example, after stable osteosynthesis of a metacarpal fracture using external minifixation (see below), we use only small dressings around the fixation pins, which are removed after 3–4 days.

The outflow of blood and lymph from the hand results from active motion: because of the existence of venous valves, muscular contractions cause the fluids to be propelled in the proximal direction; in the hand, blood accumulating within the dorsal veins is actively pumped in the proximal direction by tension in the dorsal skin, which occurs during fist-clenching activity. Therefore, there should be no restraint to posttraumatic active mobilization, and early active motion – respecting the patient's pain threshold – should be promoted in all patients. Indeed, our patients are invited to perform the "Möberg's maneuver": every hour, the patient performs ten full fingers flexion-extension movements, with the arm in full abduction. It is psychologically important for the patient to realize that, despite recent surgery, he or she is still able to move his or her fingers without pain. Anti-inflammatory and antalgic drugs can be used to control postoperative pain, but it is important to recognize that, in most cases, the postoperative pain is actually more related to the distension of the soft tissues by postoperative edema, which can be reduced by active motion. Of course, communication with the patient, reassurance, and relief of anxiety are essential. For the rest of the day, the hand is kept elevated using a shoulder sling, two pillows at night, or even a limb suspension device when the patient is supine in bed. The sling should be kept for only a few days, for it can result later in shoulder and elbow stiffness. Shoulder immobilization is also known to impair the venous outflow.[15] Some slings are inadequate as they maintain the hand hanging over the edge of the sling, with palmar flexion of the wrist.

Obviously, allowing early active mobilization is the best method to prevent finger stiffness. An unstable fracture should be converted to a stable situation using adequate operative fixation, to avoid any form of postoperative immobilization. Tendon sutures should be sufficiently resistant to sustain early active motion. Unfortunately, in many posttraumatic situations, some form of immobilization remains mandatory. The involved finger(s) should then be immobilized in the protective position as defined by James,[8,9] the most adequate position to recover joint mobility, actually in the opposite position to that in which the contracture is most likely to develop. The position of protection is with the wrist in slight dorsiflexion, the metacarpophalangeal joints fully flexed, the interphalangeal joints almost completely extended (5–10° flexed), and the thumb held in palmar abduction, keeping open the first web. This protective position is also called the intrinsic-plus position. In this position, irreversible shortening of the ligamentous structures in the presence of edema and inflammation is prevented (Figure 36.1). Unfortunately, posttraumatic edema tends to spontaneously maintain the hand just in the opposite position, with palmar wrist flexion, metacarpophalangeal joint extension, interphalangeal joint flexion, and first web closure. This position is extremely dangerous, even for a few days.

It is unclear if algodystrophy can be prevented, besides controlling as much as possible post-traumatic inflammation and edema. It is possible that free radical scavengers such as vitamin C (ascorbic acid) constitute an effective prophylaxis,[31] but

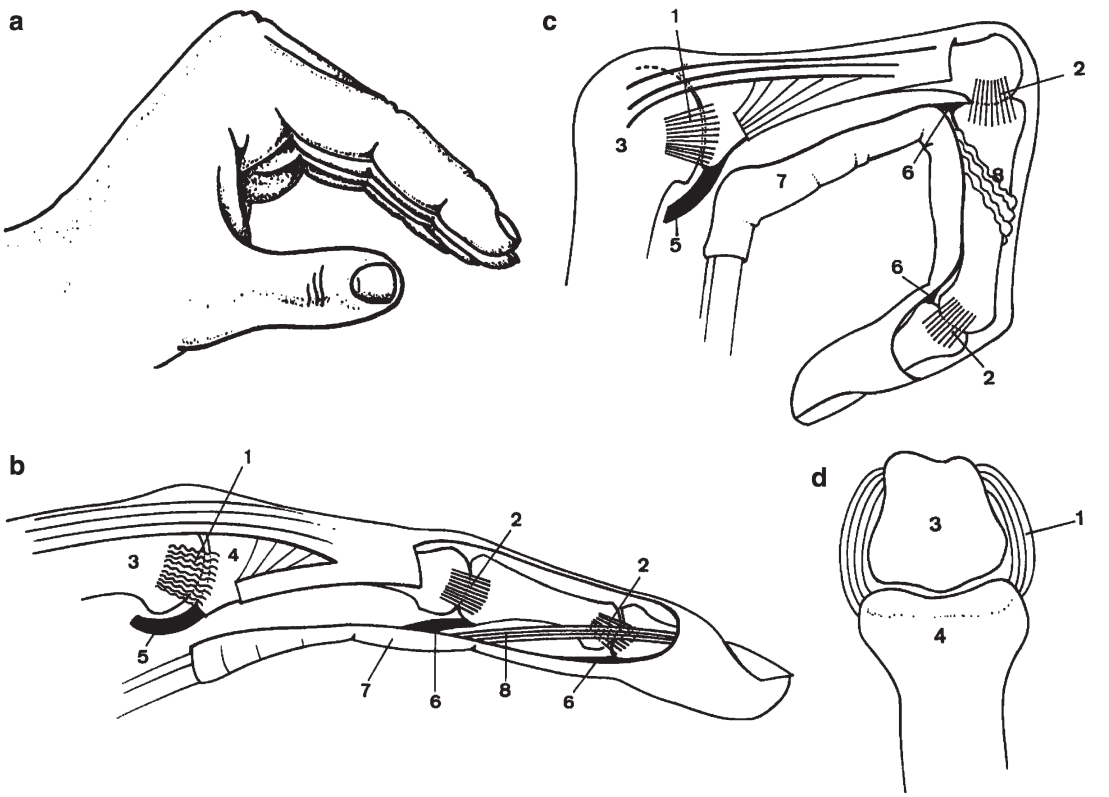

Figure 36.1. The protective position is with proximal interphalangeal joint extension and metacarpophalangeal flexion of the long fingers and with palmar abduction of the thumb, keeping the first web under stretch (**a**). (Reprinted with permission from Schuind and Burny.[18]) With metacarpophalangeal extension, the lateral ligaments (1), which are oblique rather than collateral, are relaxed and redundant: in the presence of edema and inflammation, irreversible shortening of these ligamentous structures may result in metacarpophalangeal ankylosis in extension (**b**). In contrast, flexion of the metacarpophalangeal joint stretches the main lateral ligaments (1) over the enlarged palmar metacarpal head, protecting them from fibrotic shortening. (**c**) The ulnar view of a right index finger. (**d**) The dorsal view of the flexed right middle finger metacarpophalangeal joint; metacarpal head (3), base of proximal phalanx (4). The lateral ligaments (2) of the proximal and distal interphalangeal joints are true collateral ligaments, crossing the joint flexion-extension axis and globally in equal tension in all positions except in extreme flexion (beyond 110°). However, interphalangeal flexion folds the volar plate (6), which is strongly attached at both ends, the flexor tendon sheath (7), the deep fascial complex, and the oblique retinacular ligament of Landsmeer (8); immobilization of the interphalangeal joints in flexion may therefore result in flexion ankylosis. As the metacarpophalangeal volar plate (5) is loosely attached to the metacarpal neck, shortening of that structure will not result in a loss of metacarpophalangeal joint extension (**c**). (**b–d**) – (Reprinted from Schuind.[22])

this remains controversial. In any case, if signs of the development of algodystrophy are observed in the posttraumatic course, immediate medical and physical treatment should be instituted. In addition to psychological treatment, calcitonin, and/or various nerve blocks, we apply the stress-loading program of Watson et al.[29] consisting of active traction and compression exercises.

The hand therapist plays an essential role in the prevention of stiffness. Structuring the exercise programs, controlling edema and scar, applying dynamic thermoplastic splints to improve joint mobilities (prolonged low-energy traction), and organizing occupational therapy are among his/her main tasks.

Joint Injuries

Most ligament injuries result from sporting accidents. The proximal interphalangeal joints of the long fingers and the metacarpophalangeal joint

of the thumb are particularly at risk. Lesions range from simple (partial ligament injury) to severe sprains (total rupture of one or several ligaments), either isolated or resulting from a dislocation, frequently reduced at the site of the trauma. The observation may reveal an obvious dislocation, a subluxation, or more subtle joint malalignment. Gentle examination through the full arc of motion, sometimes under nerve block anesthesia, reveals a possible joint instability. Comparison with the normal, unaffected site is helpful, as some patients are quite lax. Radiographs may be normal or reveal the existence of a small bone fragment related to the avulsion of a ligament insertion. Ultrasound examination is frequently helpful to confirm the ligament rupture, particularly when an injury to the ulnar collateral ligament of the thumb metacarpophalangeal joint is suspected. In case of doubt, an MRI will confirm the capsuloligamentous lesions.

The treatment should promote ligament healing while allowing active motion as soon as possible. Protective splints, avoiding stress to the injured ligament, but maintaining joint motion, are favored, particularly for proximal interphalangeal joint injuries. If immobilization is indispensable, it should be as short as possible, in the already defined protective position. It is followed by progressive remobilization, frequently in "syndactyly" by "buddy strapping." Physical therapy and dynamic splints complete the treatment, if the injured joint keeps some stiffness, which is relatively frequent.

Some joint injuries constitute indications of operative repair.[22] Surgery is of course mandatory in the case of an irreducible dislocation, which is most frequently seen at the metacarpophalangeal joint of the thumb, index, or little finger, by interposition of the palmar plate and because of a buttonhole effect of the metacarpal head through the palmar tendons. An associated palmar wound is frequent. Carpometacarpal dislocations and fracture dislocations are in most cases dorsal, affect the fourth and fifth carpometacarpal joints, and result from high-energy injuries. The diagnosis of carpometacarpal dislocation is frequently missed, because the patient, frequently polytraumatized, suffers from other obvious injuries (including fractures of adjacent metacarpals), because marked swelling quickly hides the deformity, and because the diagnosis is usually not made on conventional X-rays. A CT scan should be obtained. When the diagnosis is made early, closed reduction can easily be accomplished, but it is usually unstable because of the pull of the extensor carpi ulnaris. Percutaneous Kirschner wire fixation is then indicated. In the case of delayed diagnosis, the reduction cannot anymore be obtained by closed means, imposing open reduction by a dorsal approach.

A complete rupture of a collateral ligament with marked instability in the metacarpophalangeal or proximal interphalangeal joint of a long finger may occasionally also justify operative repair, frequently using a minianchor, although conservative management is recommended in most cases. Proximal interphalangeal hyperextension injuries cause rupture of the volar plate and of the accessory collateral ligaments, frequently resulting in a dorsal dislocation. After reduction, most joints are stable, and conservative treatment may be applied. Surgery is, however, indicated when the dorsal dislocation is associated with a large fracture of the base of the middle phalanx, involving 20% or more of the joint surface on a profile view. In this case, the major part of the collateral ligaments remains with the volar plate inserted in the fractured fragment, no longer attached to the middle phalanx. Persistent dorsal instability is usual, and fracture comminution is frequent (Figure 36.2). Open reduction and internal fixation of the fracture are indicated, but technically they are frequently extremely difficult. Some form of dynamic external fixation might be indicated. Alternative techniques include immobilization of the reduced joint in 30° flexion for 3 weeks or resection of the bone fragments with advancement of the volar plate. In all circumstances, stiffness should be expected.

The most frequent indication of operative repair of a hand ligament injury is at the thumb metacarpophalangeal joint, in the case of a complete ulnar collateral ligament rupture, when there is interposition of the adductor pollicis aponeurosis between the extremities of the ligamentous fragments (Stener lesion).[24] In that case, ligament healing does not occur, and the joint becomes less and less stable over time, with progressive distension of the secondary lateral restraints, the thenar muscles, and the aponeuroses. The patient progressively loses possibilities of pinch and develops painful degenerative osteoarthrosis. To prevent this unfavorable evolution, surgical repair of the ligament should be performed within 10–14 days.

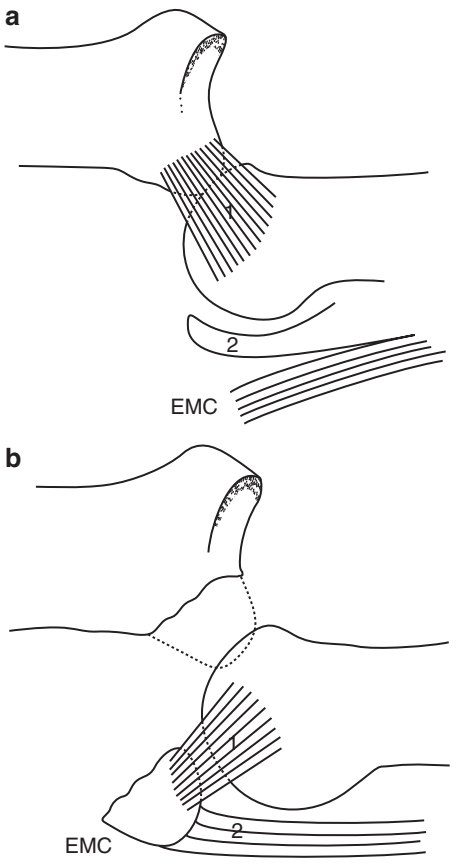

Figure 36.2. Proximal interphalangeal joint hyperextension injury, resulting in volar plate rupture with bony avulsion of its insertion with a small fragment (**a**) or with a large fragment (**b**). The fracture dislocation with a large fragment (**b**) is very unstable because the collateral ligaments (1) and the palmar plate (2) are no longer attached to the middle phalanx; the fracture is frequently comminuted, and the fractured articular surface is in addition frequently impacted by the pressure of the proximal phalanx condyles. (Reprinted from Schuind.[22])

Closed Fractures of the Metacarpals and Phalanges

Diagnosis

The diagnosis of a hand fracture is usually not difficult, except in multi-injured or unconscious patients. In those cases, the fracture of the hand frequently escapes notice and is discovered weeks later. Ironically, these "minor" injuries may represent the principal causes of the final disability.

Radiographs should be obtained in at least two orthogonal planes to show the precise extent of the fracture, ideally face and profile views. However, because of the superposition of the metacarpals and fingers, profile views are frequently not provided, and oblique views are obtained instead (Figure 36.3). However, without profile views, the true extent of displacement of a metacarpal or a proximal phalanx fracture cannot be appreciated,[11] and the existence of small, avulsed bone fragments indicative of significant ligament or tendon injury may be missed. Indeed, a normal X-ray does not exclude an osteoarticular injury. Some undisplaced fractures may not be obvious initially. Frequently missed closed skeletal injuries include carpometacarpal fracture dislocations of the base of the fifth (± fourth) rays (accurate diagnosis can be made by a CT scan – see above) and intra-articular fractures of the metacarpal head, which may cause joint locking (the Brewerton view, with the dorsum of the hand lying flat on the X-ray cassette, may be helpful).[21] If a fracture is suspected but the radiographs are normal, a bone scan should be obtained. If negative, a bone lesion is very unlikely. The surgeon should remember the possibility of multiple injuries, in which the secondary lesion is not obvious and can easily be overlooked: in the case of articular fractures of the proximal and distal interphalangeal joints, one of these injuries may well not be diagnosed.

In pediatric fractures, the possible involvement of the growing physis should be considered. Most fractures are benign Salter and Harris type I and especially type II lesions. Salter and Harris types III, IV, and V – the latter characterized by normal initial X-rays – do occur at the hand and can lead to joint incongruence with decreased range of motion, clinodactyly and/or a foreshortened digit. A pseudo-mallet finger deformity may be seen in a child with a physeal injury at the base of the distal phalanx, occasionally with interposition, between the epiphysis and the metaphysic, of the lacerated nail matrix.

Displacement

Many hand fractures are undisplaced and intrinsically stable, allowing early protected motion (see below). The displacement of the fracture occurs owing to the muscles inserted in the bone fragments. In Bennett's fracture of the base of the thumb metacarpal, involving a triangular

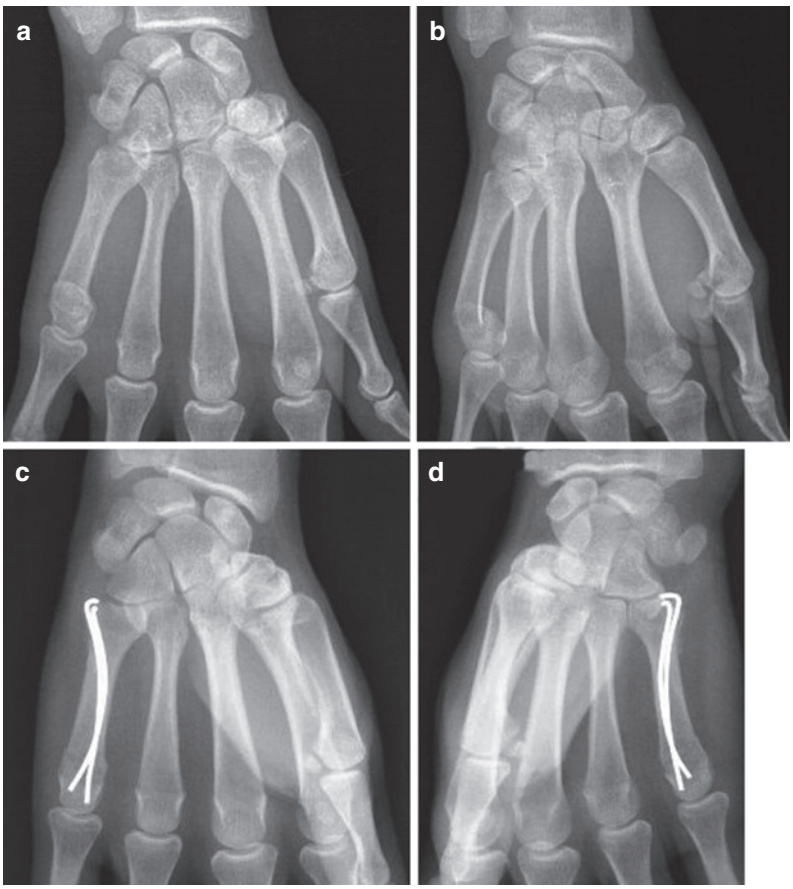

Figure 36.3. (a, b) Closed fracture of the neck of the fifth metacarpal (boxer's fracture). Note that no true profile view has been provided by the radiological department; the anterior displacement of the metacarpal head is more important than that appears on the oblique view. (**c, d**) "Bouquet" osteosynthesis by two endomedullary Kirschner wires.

volar-ulnar fragment held to the trapezium in anatomic position by the preserved anterior oblique trapeziometacarpal ligament, the proximoradial subluxation of the remaining metacarpal base occurs under the muscular preconstraint of the abductor pollicis longus and extensor pollicis brevis. It is characteristic for diaphyseal and neck metacarpal fractures of the long fingers that the displacement consists of a volar tilting of the distal fragment caused by the tension of the interosseous muscles (Figure 36.3). In the proximal phalanx, volar angulation is observed, due to the intrinsic muscles. In the middle phalanx, the displacement depends on the location of the fracture in relation to the insertion of the flexor digitorum superficialis. Whatever the location of the fracture, rotatory displacement is frequently present and should be recognized: in flexion, all digits converge toward the tubercle of the scaphoid.

Principles of Treatment

The goal is full and rapid restoration of hand function. The principles of treatment of hand fractures are: anatomical reduction, followed, when possible, by immediate mobilization, if necessary, by immobilization of the injured finger in the "protective" position, minimizing that support period as much as possible, remobilization when the fracture is clinically stable, and prevention of posttraumatic stiffness.[18]

Anatomical Reduction

It is probably more important than in any other part of the body that the hand fracture is accurately reduced, either by closed manipulation, percutaneously by minidaviers or by manipulating the bone fragments using external minifixation pins or by direct open reduction. Anatomical reduction of intra-articular fractures is mandatory to prevent decreased joint motion amplitudes or even ankylosis, sometimes clinodactyly, and late, frequently painful, osteoarthrosis. Obtaining an anatomical reduction is also important for diaphyseal metacarpal and phalangeal fractures. It is in particular essential to correct any rotational deformity. The deviation error is amplified by digital flexion, with troublesome crossing of the fingers; a 5° rotational error in a finger metacarpal results in almost 1.5 cm crossing when the fingers are fully flexed. Persistence of shortening or palmar or dorsal angulation of the bone fragments must also be avoided, for it will affect the functional results (weakness as a consequence of shortening of the musculotendinous intrinsic units) and may lead to deformities of adjacent joints: for example, an unreduced dorsal angulation after a diaphyseal or neck metacarpal fracture may induce a compensatory metacarpophalangeal joint hypertension, resulting in a secondary pseudoclaw proximal interphalangeal joint deformity, when the digit is fully extended. In the proximal and middle phalanges, an accurate reduction is also needed to preserve the extensor and flexor tendon gliding: the palmar surfaces of the bones form the floor of the flexor tendon sheath. If fractures at this level are not anatomically reduced, the tendons cannot be expected to function normally.

Nonaccurate reductions may be tolerated in certain locations, where the large amplitudes of motion of the neighboring joints may compensate for the consequences of poor reduction. This is the case for the shaft of the first metacarpal bone and for the neck of the fifth metacarpal.

Early Mobilization

As already stated, early mobilization is the best method to prevent late finger stiffness. In clinical series, early mobilization has clearly been demonstrated to provide better long-term results.[3,25,30] Stable, undisplaced fractures should not be immobilized; during the first weeks, some protection may be provided by "buddy taping" to an adjacent digit or by some form of thermoplastic splintage.

Immobilization of the Finger in the "Protective" Position

Ankylosis of the hand joints is seen in two forms: fixation in extension at the metacarpophalangeal joints and fixation in flexion at the proximal interphalangeal joints. The fractured finger should be immobilized in the position of protection, already defined, that is, with the metacarpophalangeal joints fully flexed and the interphalangeal joints almost completely extended (Figure 36.1). Only the joints whose movements will cause motion at the fracture site should be immobilized. This includes the joints immediately above and below the fractured segment.

Osteosynthesis

"Mon but est surtout d'étudier la suture osseuse, ou, pour parler plus exactement, l'ostéosynthèse." That definition of the word osteosynthesis was expressed for the first time by Lambotte[10] in 1907. Burny defines osteosynthesis as a means of fixation of bone fragments with direct anchorage of the implant in the bone.[4] In general, the osteosynthesis of a fracture has two prime objectives: to maintain the precise reduction of the fracture until bone healing is achieved and to facilitate early active motion so as to minimize atrophy, joint stiffness, and tendon adhesions by converting the unstable fracture into a stable system.

Most closed hand fractures are stable, either before or after closed reduction, and do not require operative fixation. Displaced articular fractures and unstable diaphyseal fractures represent the most frequent surgical indications. The predictable consequences of poor reduction and/or of posttraumatic immobilization should always be weighted against the disadvantages and risks of the osteosynthesis. Aggressive attempts at open reduction and internal fixation may cause damage to the delicate soft tissues, tendon adhesions, or even lead to late infection, or the epiphysis may be devascularized, all circumstances leading to poor functional results. Internal fixation may also impose a secondary operation for implant removal.

Apposition Osteosynthesis

These has been in the last decade much improvement in plates and screws: modern implants are of low profile, easy to contour and to cut, and better designed for hand surgery.

Although stainless steel is still largely used, titanium is preferred by some surgeons because of its elastic modulus closer to bone. Cannulated miniscrews are also available for percutaneous bone fixation. Screws are especially used for the osteosynthesis of articular fractures (Figure 36.4) and of simple long oblique or spiral metacarpal and phalangeal fractures. Plates are now less frequently used, because of problems of tendon impingement.

Kirschner Wires

Crossed percutaneous pinning, transverse percutaneous pinning of a fractured metacarpal to an adjacent metacarpal, or transarticular axial Kirschner wire fixation remain popular methods of treatment. The techniques have the advantages of being quick, easy, cheap, and apparently minimally invasive. In fact, it is frequently quite difficult to maintain a good reduction while drilling Kirschner wires. The technique is quite aggressive when the pin obliquely impales a tendinous structure or transfixes a joint. Kirschner wires may also distract the fracture fragments, a cause of delayed- or nonunion. In most of the cases, Kirschner wire fixation does not provide stable fixation and some form of postoperative immobilization is mandatory, and the main goal of the hand osteosynthesis, that is transforming an unstable situation into a stable one, allowing early motion, is not attained. Despite immobilization, secondary displacement may be seen. Pin tract infections may develop. Finally, Kirschner wires may migrate, sometimes imposing major surgery for secondary retrieval (the extremity of the Kirschner wire should always be bent).

Probably the best application of Kirschner wires is the "bouquet" endomedullary fixation of the quite common displaced fracture of the neck of the fifth metacarpal, the so-called "boxer's" fracture.[6] After closed reduction by the Jahss maneuver, a hole is made in the proximal ulnar metaphysis of the bone, allowing the antegrade insertion of prebent smooth Kirschner wires, up to the subchondral bone of the metacarpal head (Figure 36.3). In most cases, early motion can be instaured. The Kirschner wires are removed after bone healing, 4–6 weeks later.

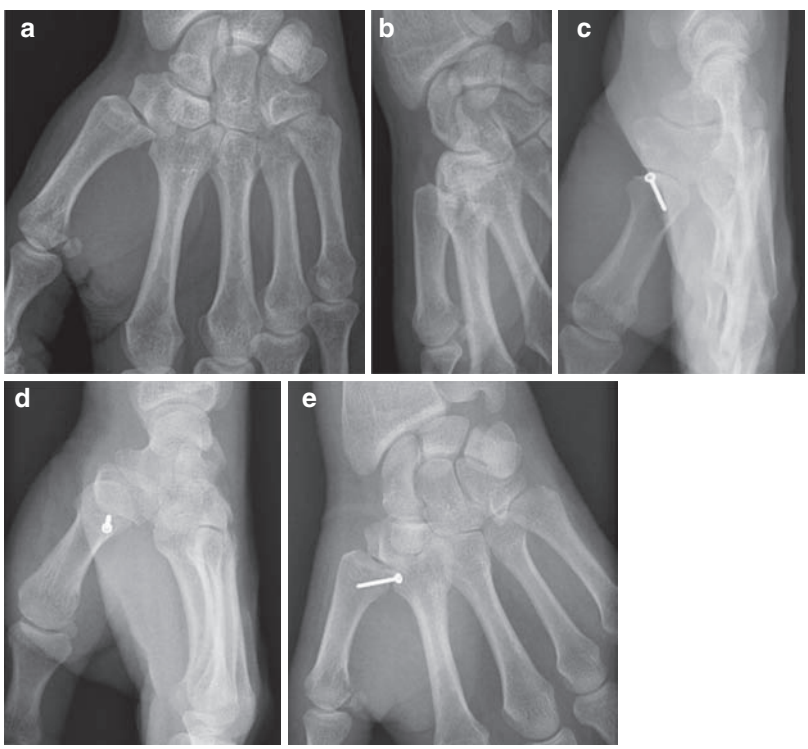

Figure 36.4. (a, b) Bennett's fracture of the base of the first metacarpal. (**c–e**) Open reduction and screw fixation.

Cerclage Wiring

Cerclage wiring, with or without implantation of a single Kirschner wire, has not gained much popularity. If placed dorsally to stabilize a metacarpal fracture, the interosseous cerclage may act as a tension band, provided there is good bone contact and no restriction to early active motion. Extensor tendon synovitis may be seen at the site of the stainless steel wire.

External Minifixation

External minifixation is an excellent technique for the osteosynthesis of selected closed hand fractures, in particular closed unstable diaphyseal metacarpal[19] (Figure 36.5) or phalangeal fractures. In most cases, it is easy to obtain an anatomical closed reduction, by acting on the bone fragments through the implanted minifixation pins fixed in miniclamps. The construct is usually quite stable, allowing early active motion. The external fixator is adjustable to all clinical situations. A second reduction can always easily be carried out, if the initial reduction is not satisfactory. External fixation may also be used to maintain transarticular distraction, in order to reduce comminuted epiphyseal fractures (Figure 36.6), for example Rolando's fractures of the base of the first metacarpal (triangular configuration radius-first and -second metacarpals).[20] Special dynamic transarticular minifixators allow early active mobilization while maintaining distraction and joint alignment (Figure 36.7). There is no secondary operation for hardware retrieval. The drawbacks of external minifixation include pin tract infections (which can be prevented in the vast majority of patients), extensor tendon transfixion (proximal phalanx) limiting early motion by impairment of tendon excursion, and mainly the costs and availability of modern minifixators.

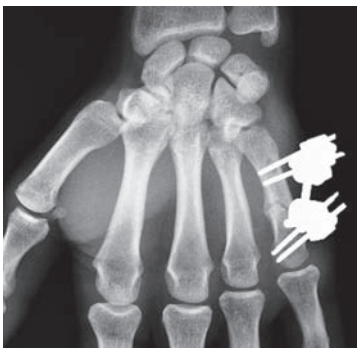

Figure 36.5. Osteosynthesis of a closed diaphyseal fracture of the fifth metacarpal by Jaquet external minifixation. Note periosteal callus formation on these X-rays performed 6 weeks after the initial fracture.

Bone Healing

Contrary to common opinion, bone healing processes in the hand are the same as those in other bones. The healing of fractures through trabecular bone is usually quite quick. Diaphyseal fractures heal with periosteal callus formation (Figure 36.5), unless rigidly fixed by a plate, where slow "primary" bone healing may be observed, with direct "fusion" of the cortices under Haversian remodeling with osteons crossing the fracture line. Nonunions and refractures (after hardware removal) may be seen. Roentgenographic bone healing of hand diaphyseal fractures is quite long, lasting up to several months. Metacarpal fractures heal faster than proximal phalanges, which heal faster than middle phalanges and distal phalanges, and it is probable that open reduction doubles the healing duration. However, it is generally admitted that after 3–6 weeks (metacarpal), 5–7 weeks (proximal phalanx), and 10–14 weeks (middle phalanx), closed fractures become sufficiently stable to allow remobilization, in the case of nonoperative treatment, despite the fact that radiological bone healing is still not obvious.

Complications

Many complications of closed hand fractures are related to inadequate treatment. The causes and prevention of stiffness have already been discussed. Malunion may interfere significantly with hand function and appearance, justifying a corrective osteotomy. Except at the tuft of the distal phalanx, nonunions remain exceptional after closed fractures of metacarpals and phalanges. Nonunions may be related to bone fragment distraction by Kirschner wires, soft tissue interposition, or extensive periosteal devascularization in the case of plate fixation. Posttraumatic epiphyseal avascular necrosis has been reported at the metacarpal and at the phalangeal heads. Growth arrest may be seen in children after physeal fractures. Tendon ruptures may complicate Kirschner wire pinning or screw fixation. Acute carpal tunnel syndrome

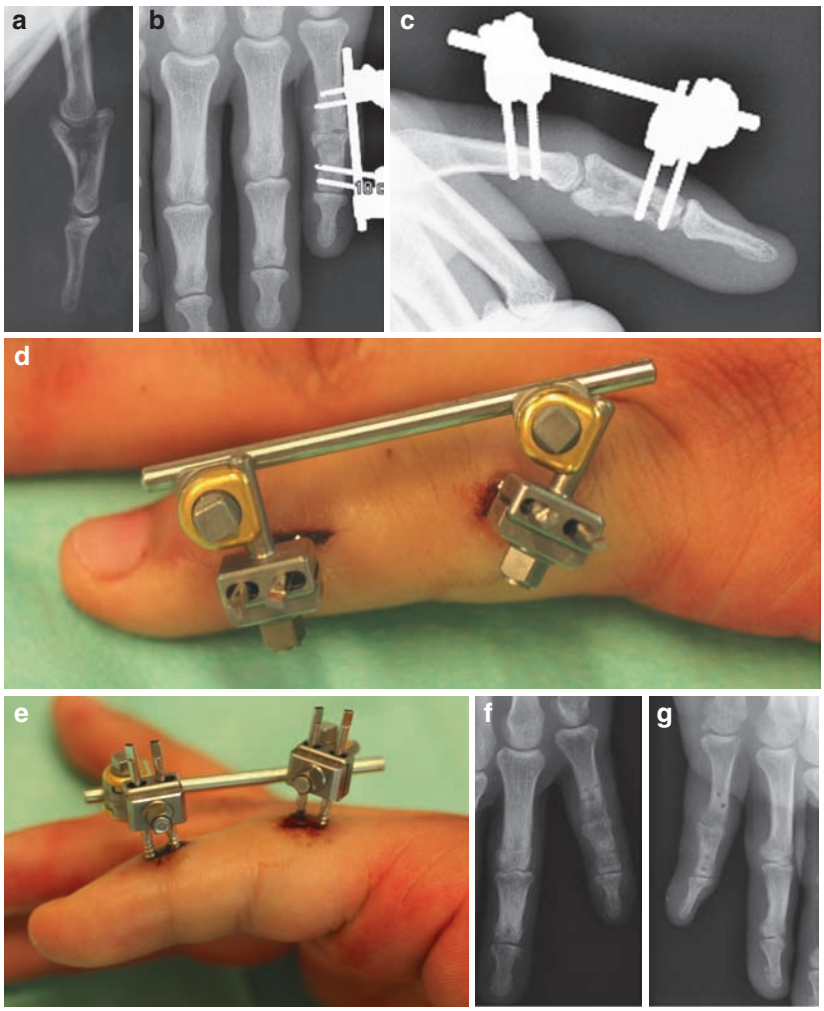

Figure 36.6. (a) Complex closed fracture of the middle phalanx of the little finger, also involving the proximal interphalangeal joint. (**b–e**) Osteosynthesis by distraction external minifixation with a quite acceptable reduction. (**f, g**) Maintenance of the good reduction after retrieval of the external minifixator. The patient has regained good joint motion amplitudes, except for a persistent extension lag at the proximal interphalangeal joint of 30°.

may be observed in the case of a fracture of the base of the first metacarpal, due to a hemorrhage within the carpal tunnel. Plaster cast or traction may cause skin necrosis. Cold sensitivity is common after any hand trauma.

Open Hand Fractures

Most open hand fractures deserve osteosynthesis. In the case of severe contamination or extensive crush, external minifixation is the best choice. The technique is also useful in the case of segmental bone loss. Minigentabeads can then be interposed at the site of the defect, to be replaced, after soft tissue healing, by a cancellous bone autograft. Even more important than in closed injuries, early active motion should be instaured as soon as possible, after an open fracture. This implies solid repair of the associated tendon structures, and in many cases local skin flaps to cover the exposed bone fragments. Modern

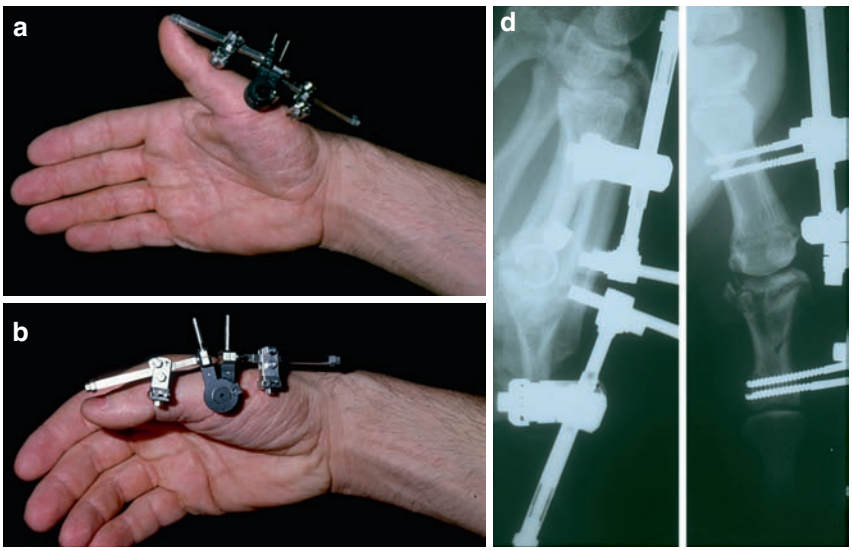

Figure 36.7 (a–d) Comminuted fracture of the proximal phalanx of the thumb involving the metacarpophalangeal joint. Treatment first by static and then by dynamic external minifixation with early active motion.

flexor tendon repair techniques, using at least four suture strands and a circular epitendinous running suture, allow for immediate unresisted active motion. The main limitation remains the necessary 10 days to 2 weeks immobilization, in the case of an associated microsurgical vascular and/or nerve repair.

Complex Open Hand Traumas and Amputations

Complex open hand traumas with contamination and extensive soft tissue and bony injuries pose challenging problems to the hand surgeon (Figure 36.8). As for any hand trauma, a complete history is essential. The pretraumatic state of the patient (hand dominance, profession, leisure activities, general condition, and expectations) should be known. When planning a complex reconstructive procedure, the chronological age of the patient is not so important as the general health and the possible existence of comorbidities. In particular, smoking and the use of vasoconstrictive drugs are generally considered to contraindicate any microvascular reconstruction or at least significantly increase the risks of failure. It is also necessary to appreciate the mechanism and timing of the injury. Indeed, the potential of tissue healing depends on the degree of aggressiveness of the trauma (sharp cut, crush, avulsion, or combination; degree of contamination). It is also mandatory to know the delay of the injury, especially when dealing with devascularizing traumas; in addition to warm ischemia, the wound contamination progresses with the hours lost before debridement.

Although this is not easy, the severely traumatized hand should be examined in the emergency room, in order to determine the need for urgent treatment and to allow adequate organization of the surgical and anesthesiologic teams and preparation of the operating room (microscopic instruments, fixation devices, etc.). Digital photographs should be obtained for discussion with treating colleagues and documentation. After this preliminary examination by the treating hand surgeon, a sterile dressing is applied. There should be no new examination before the operation, for each supplementary opening of the dressing adds a significant risk of additional contamination by hospital-acquired microorganisms, mainly Gram-negative bacteria. Although control of hemorrhage is obviously important, it is quite dangerous to try to clamp or to coagulate vessels in the emergency room, because of the danger of injury to the nearest critical structures,

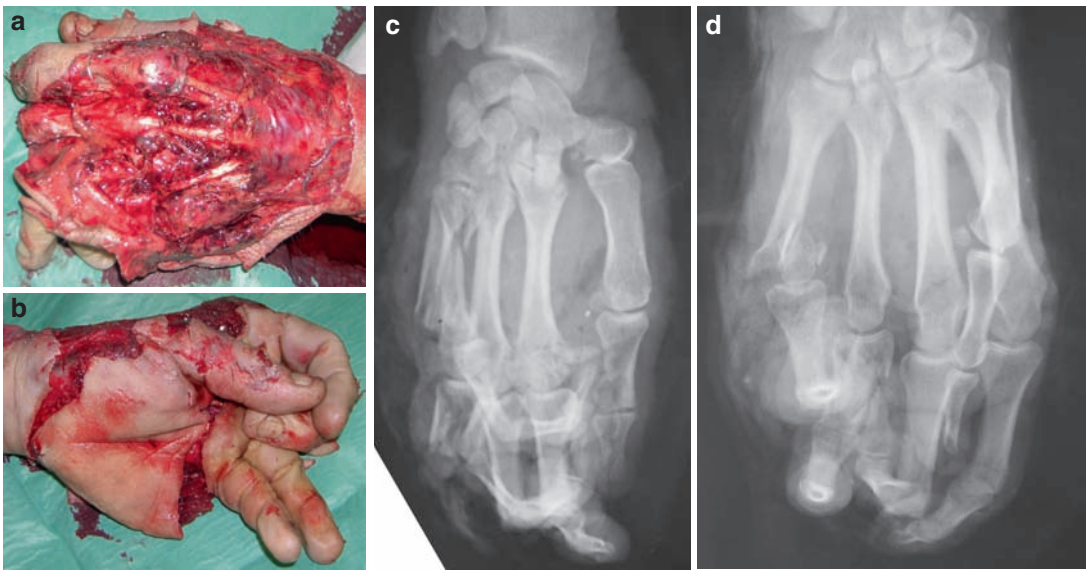

Figure 36.8 (a–d) Severe crush injury to the hand. Documentation by digital pictures and X-rays.

which are frequently nerves; the best method is to apply direct pressure to the wound with sterile material, use semielastic bandages, and/or elevate the traumatized limb. Partially amputated parts are gently manipulated, realigned, and splinted to avoid further damage (kinking of the residual veins). The patient is placed under antibiotics, usually a first-generation cephalosporin, efficient on *Staphylococcus aureus*, the most common infectious microorganism in open hand lesions (originating usually from the skin of the patient himself). An aminoglycoside should be added in case of agricultural or other highly contaminated injuries. X-rays are systematically done; the fractures are frequently markedly comminuted, confirming the intensity of the energy dissipated during the trauma, to the bone and to the adjacent soft-tissues.

In the case of a traumatic amputation, the segment (finger, whole hand) should be conserved in a sterile plastic bag, disposed in a container with ice, avoiding any direct contact with ice (danger of an additional frostbite). At 4 °C, the viability of the amputated digit may be maintained up to 12 h or longer, because there is no muscle tissue. Cooling also delays the bacterial proliferation. All amputated segments should be conserved, even though it seems sure that there will be no indication of replantation, as the segments can also be used as tissue donors for an autograft during the operation (skin, bone, joint, nerve, artery). Radiographs of the amputated segment should be systematically obtained, in addition to those of the traumatized hand.

The patient should be informed before the operation of the possibilities of reconstruction and expected results. In the case of a complete amputation, the patient and his/her family desire in most cases a replantation and expect a miraculous result. Reattachment should never be guaranteed at this stage. The surgeon should explain the chances of success, the duration of surgery and length of hospitalization, the probable necessity of other operations, and the long rehabilitation, in comparison to an amputation. The difference between vascular (viability) and functional (mobility and sensibility) success should be made very clear. The final decision whether to perform the replantation or not is to be taken by the operating hand surgeon, based on the preoperative evaluation, the preoperative assessment of the lesions, and his/her knowledge and experience. There is no justification to perform a digital replantation, if the risks of a poor functional result are high, especially in a manual worker. In general, the indications of replantation are a thumb amputation (all levels), amputation of multiple digits, single digit amputation distal

to the insertion of the flexor digitorum superficialis, amputation in a child, and amputation through the palm, the wrist, and the forearm. The contraindications are severe crush, coexisting serious medical problem (like recent stroke or myocardial infarction, arteriosclerotic vessels, etc.), prolonged warm ischemia, and single-digit amputation in an adult, proximal to the flexor digitorum superficialis tendon insertion (except sometimes in musicians or in females with aesthetical requests).

The surgical procedure is started as soon as possible, especially in the case of warm ischemia (revascularization of partially amputated fingers) with complete wound debridement and identification of vessels, nerves, and tendons. Then bone fixation is performed, sometimes with skeletal shortening. The order of repair is then usually the following: anastomosis of the arteries, flexor tendon repair, nerve repair, extensor tendon repair, venous anastomosis, and skin coverage. Revascularization procedures (partial amputation) may be easier and quicker than replantation (total amputation), offering in general better viability rates.

After microanastomosis, the postoperative care consists in routine precautions for the first 5 days, with salicylic acid (aspirin) 325 mg/day, Dextran 500 ml/24 h, chlorpromazine (anxiolytic), and first-generation Cephalosporin with bed rest in a warm room. Monitoring of the replant is necessary (color, pulp turgescence, capillary refill), initially each hour, and smoking is forbidden for at least 3 weeks. The early complications include arterial insufficiency, venous congestion or thrombosis, and infection, and the rate of early reoperation is significant.

Traumatic Nerve Lesions

The care of an open hand wound is always an exercise of applied anatomy. All structures possibly affected by the traumatizing agent, in particular nerves, should be suspected to be injured. In particular, glass and knife lacerations frequently cause injury with partial or total sections of tendons or neurovascular structures (Figure 36.9). Therefore, detailed knowledge of the three nerves reaching the wrist and hand is mandatory: (1) the superficial branch of the radial nerve, near the first extensor compartment, with terminal branches to the dorso-radial aspect of the midcarpus; (2) the median nerve and its divisions into intermetacarpal and digital collateral nerves, responsible for sensation of the volar and part of the dorsal aspects of the three radial fingers and the radial half of the fourth ray; this mixed nerve at the wrist level carries a single thenar motor branch with variable emergence; (3) the ulnar nerve, with a deep motor branch for intrinsic muscles and a superficial sensory

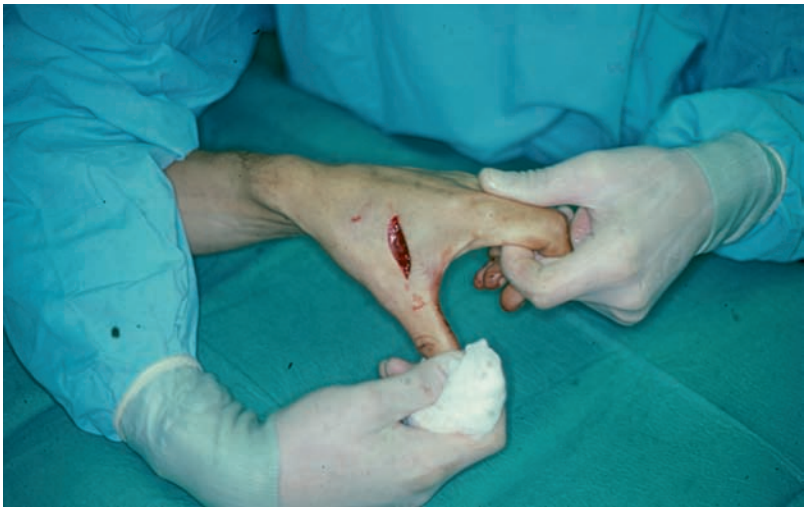

Figure 36.9. Dorsal wound by a knife, resulting in laceration of the palmar flexor pollicis longus tendon and thumb digital nerves.

branch, thereby completing the volar finger sensibility for part of the fourth and the fifth rays. Note that variations do occur, and that there are anastomoses between the median and ulnar nerves at the palm, which may as well be injured.

Pathophysiology

The same rules apply to all peripheral nerves. Age, diabetes mellitus, hypothyroidism, and vascular disease are major negative factors for the quality and speed of reinnervation. Failed motor regeneration results in target muscle atrophy. A long-lasting sensory nerve conduction deficit results in anesthesia, neuropathic pain, and cortical rearrangement. Therefore, precise and early diagnosis of the lesion followed by microsurgical repair is mandatory and best performed by a skilled hand surgeon.

Type of Lesions

Most frequent lesions are clear-cut skin wounds with variable participation of deeper neural, vascular, or tendon structures. An arterial hemorrhage at the volar finger level ascertains a nerve injury, as the collateral nerve runs ventral to the collateral artery. Upon examination, the patient will describe decreased sensibility. Because many nerve lesions are partial, and because of overlapping innervation, total anesthesia is in fact exceptional. Posttraumatic examination is challenging in children, and minor injuries must be examined carefully; if there is any doubt of wound deepness, exploration under anesthesia is mandatory. A very distal repair of the terminal branches of the collateral nerve is technically challenging but worth performing. As stated by Möberg, the fingertip is the "eye of the hand."

Iatrogenic lesions are less rare than one would believe. The superficial radial nerve is at danger whenever de Quervain's tendovaginitis is operated on, especially in the case of reoperation. Carpal tunnel surgery may result in partial nerve tears, especially to the thenar motor branch (the variations of its emergence must be remembered). Excising a deep wrist lipoma might harm the motor branch of the ulnar nerve, which is of difficult direct access for a repair. Sophisticated tendon transfer procedures are frequently indicated in the second stage in such iatrogenic lesions or in cases with persistent palsy despite nerve repair.

Strategy

An isolated nerve repair is almost never a nighttime emergency (except in the case of associated ischemia). Most clean-cut nerve lesions should, however, be repaired within the first few days or within the first week after trauma. In electrical burns or crushed nerves, primary repair often results in anastomosis within the area of damage, followed by poor outcome. In these particular circumstances, one should wait until a proximal Tinel sign shows the level of possible reinnervation, and then cut back the distal stump until healthy fascicular structures appear, before interposing a segmental graft. Neuropathologic examination of the slices before coaptation and/or grafting is very helpful.

Even less important nerves, like terminal dorsal branches of the radial or ulnar nerves, merit a suture to prevent neuroma formation.

Technique

The operation is always conducted under magnification, either by loupes or a microscope, within a bloodless field. Nerve suture tension should be avoided: if a suture with 10/0 nylon ruptures, tension is excessive. Then, further neurolysis of the stumps (to take benefit from redundant nerve length) or an autologous nerve graft are mandatory. Grafts are harvested from the lower leg (sural nerve) or from the volar forearm (sensory branches, running parallel to the superficial veins). The coaptation technique must be very precise and the epiperineural sutures performed without tension, favoring congruent adaptation. Fascicular orientation is mandatory, especially in mixed (median and ulnar) nerves, as motor bundles will regenerate only when put in line with distal motor branches. Alignment may be facilitated using epineural vessel orientation or coloring the stump slices with vital dye, to better identify the organization of the fascicular groups.

Rehabilitation

Any nerve suture can be mobilized after 10 days of tension-free immobilization. In combined tendon-nerve injuries, this delay should be respected before starting active exercises. Motor recovery and return of muscle power take several months and need physiotherapy for both strength

and skill. Sensory reeducation has been neglected for years but is very important, as the reorganization of sensorial areas within the cerebral cortex is nowadays better known and can be visualized, for example, using functional MRI.[13]

Results

Although the repair of tiny nerves at the hand was for long associated with bad prognosis and thus raised limited enthusiasm among surgeons, it is proven today that a perfect microsurgical repair, bringing together healthy stumps (either directly or using a short graft) with good topographic orientation, performed under ideal general and technical conditions, can provide rewarding results. Success may also be observed after secondary suture of a sensory nerve, even after impressive delays of months or years. Skilled physiotherapy and regular controls by the surgeon who performed the repair are mandatory.

Infections

Most hand infections result from direct contamination from a penetrating injury or a laceration or are the consequences of nail deformities or manipulation (nail biting, manicure). Animal or human bites are particularly dangerous. Clenched fist injuries may result in an apparently closed metacarpal fracture, with a small wound caused by the tooth of the opponent, which can be the origin of a destructive septic arthritis (Figure 36.10). It is important to recognize if the patient is immunocompromised (diabetes mellitus, AIDS).

Acute and Chronic Paronychia

Acute paronychia is the most common infection in the hand. It consists of the infection of the lateral nail fold, usually by *S. aureus*. The patient presents erythema, swelling, and tenderness around the nail. After a few days, an abscess develops around and/or below the nail plate. Drainage of the abscess is easily done by opening the thin layer of tissue over the abscess and preserving the nail bed and matrix. More extensive lesions may require removal of a portion of the nail plate. Postoperatively, oral antibiotics are usually administrated for 7–10 days. Chronic paronychia, lasting weeks or months, is usually caused by *Candida albicans*. It most commonly affects middle-aged women, resulting from frequent water immersion with detergents. Treatment is usually conservative with topical antifungal agents.

Felon

Felon is an abscess of the distal pulp of a digit or thumb. Multiple vertical trabeculations divide the pulp into separate septal compartments. Acute cellulitis results in vascular congestion, aggravated by the septal anatomy of the pulp, causing what resembles a compartment syndrome, with early tissue necrosis. Although antibiotics may be sufficient in the early course of the affection, surgical drainage is necessary when palpable fluctuance appears. Several types of incision have been described, aiming to preserve the pulp as much as possible and to efficiently open the septa, in order to drain all abscess cavities. Oral or even intravenous antibiotherapy should be administrated, after bacteriological samples have been taken.

Pyogenic Flexor Tenosynovitis

The flexor tendon sheaths of the fingers begin in the palm, at the level of the metacarpal neck and end just proximal to the distal interphalangeal joint. In the small finger, there is usually a continuity of the sheath with the ulnar bursa, within the carpal tunnel; the thumb sheath may also extend to the carpal tunnel radial bursa, and both radial and ulnar carpal tunnel bursae may be in communication (space of Parona), potentially giving rise to the "horseshoe abscess" after an acute infection of the flexor sheath of the thumb or the little finger. Note that there are many variations in flexor sheath anatomy. Acute infection of a flexor tendon sheath is a very serious affection, deserving urgent treatment, not only because of the risk that the infection spreads to the entire sheath, and possibly to other sheaths as just described, but also because the purulence rapidly destroys the gliding possibilities and causes much adhesion and also because the pressure within the sheath due to the pus impairs the arterial supply to the flexor tendons through the vinculae, resulting in tendon necrosis and subsequent rupture (Figure 36.11).[14] The infection, which results usually from a penetrating injury (bite, puncture wound), progresses rapidly. The

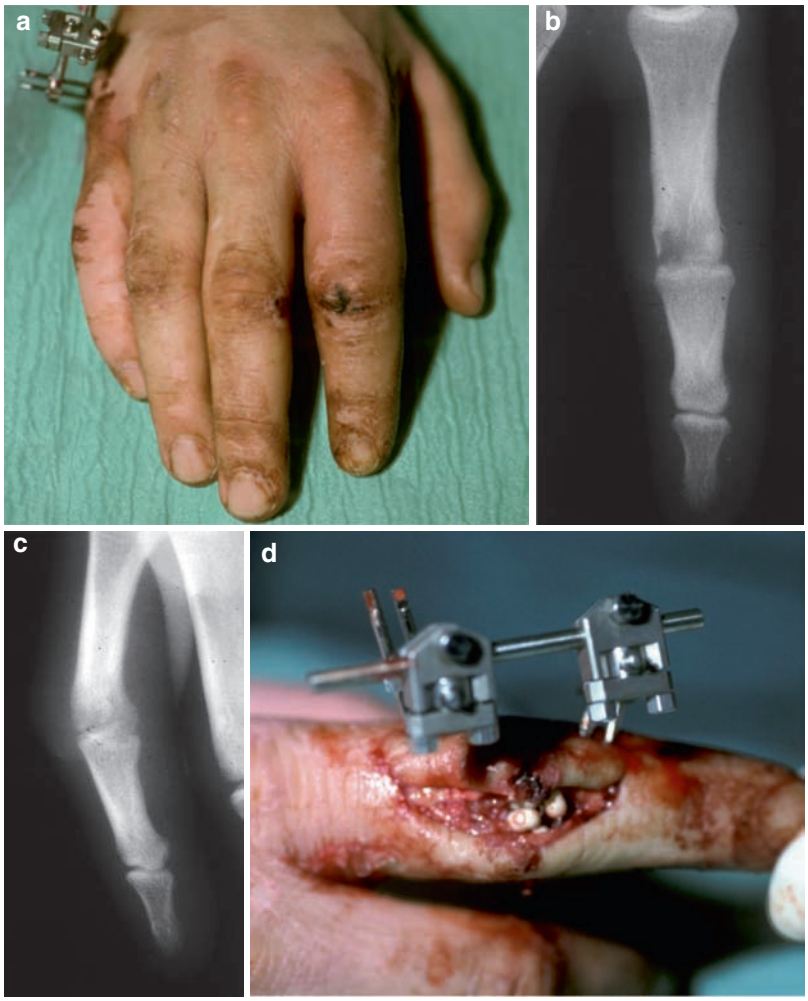

Figure 36.10. (a–c) Closed fracture of the fifth metacarpal, treated by external minifixation. The fracture resulted from a fight. A very small dorsal wound at the level of the proximal interphalangeal joint of the index was neglected. After a few days, the proximal interphalangeal joint of the index was destroyed by septic arthritis. The wound had actually been inflicted by the tooth of the opponent. (**d**) It was necessary to resect the joint, to interpose minigentabeads, and to apply external minifixation. The gentabeads were later removed to be replaced by a cancellous bone autograft, which finally permitted solid arthrodesis.

diagnosis is suspected when the four cardinal signs of Kanavel are present: semiflexed position of the finger, fusiform swelling, tenderness along the flexor tendon sheath, and excruciating pain on passive finger extension. Flexor tenosynovitis may also occur without Kanavel signs, particularly in immunodeficient patients. Urgent drainage is indicated in most cases. In early cases, closed tendon sheath irrigation is the technique of choice, with bacterial samples, followed by broad-spectrum antibiotics: it is better to conclude with a "white" lavage than to miss the diagnosis.

Other

There are many other forms of infection of the hand, either nonspecific (deep-space infection, septic arthritis, osteomyelitis, necrotizing fasciitis) or specific (including viral – herpes – and mycobacterial infections), whose description is beyond the scope of this chapter.

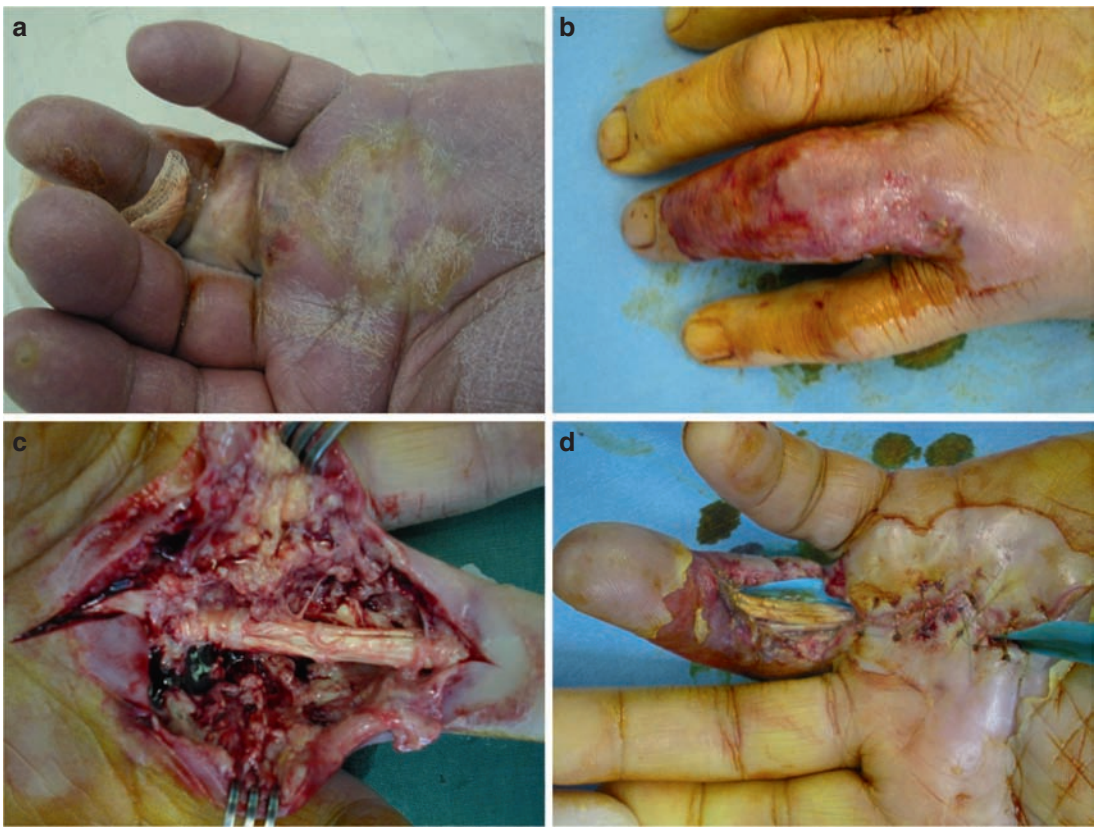

Figure 36.11. (a–d) Necrotic pyogenic tenosynovitis of the flexor tendons. Diagnosis only after several days. (**a**) Palmar view of the affected ring finger: pus outflow. (**b**) Dorsal swelling. (**c**) Urgent exploration after first consultation with the hand surgeon, revealing advanced destruction of the flexor tendons pulley system. (**d**) Picture taken at the end of lavage and debridement. The patient had finally to be amputated at the metacarpophalangeal joint, because of failure of infection control and flexor tendon necrosis.

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37

Peripheral Nerve Injuries

Maria Siemionow and Erhan Sonmez

There are different types of peripheral nerve problems that include nerve trauma, compression, tumors, and so on, and repair of peripheral nerve injuries continues to be a major challenge in reconstructive surgery. Satisfactory functional recovery after a peripheral nerve injury is dependent on accurate regeneration of axons to the original end organs. Reconstruction of peripheral nerve injuries has improved particularly over the last two decades with the experience in traumatic nerve injuries gained particularly during wartimes. In this chapter, we review different types of peripheral nerve injury problems including neuropathies, nerve repair techniques, clinical outcomes, and future strategies.

Abbreviations

CTS	Carpal tunnel syndrome
NSAIDs	Nonsteroidal anti-inflammatory drugs
NGF	Nerve growth factor

Introduction

Peripheral nerve injuries may result in devastating outcome, which is determined by the extent of cellular damage, site of lesion, degree of disruption of connective tissue sheaths that surround the nerve, associated injuries, age, and general health of the patient.[38]

After peripheral nerve injury, a complicated sequence of events commences to begin the reparative process, but this process is unique because it does not involve mitosis and cellular proliferation.[18] Although some of the peripheral nerve injuries recover spontaneously, most of them require surgical intervention. Despite over 150 years of experience in modern surgical management of the peripheral nerves, even meticulous surgical repair cannot guarantee full functional recovery, since the surgeon has no control over the "biological battlefield" occurring inside damaged nerves.[78]

History

The first report of acute nerve injury can be traced to 3,500 years ago. Jacob experienced a sciatic nerve injury with a traumatic hip dislocation during his battle with the angel in the well-known biblical story (Genesis 32:25–33).[24] Hemophilus differentiated peripheral nerves from tendons in 300 BC. He traced nerves to the spinal cord to demonstrate the continuity of the nervous system. [87] The first reference to nerve repair was by Rhazes in AD 900, but peripheral nerve regeneration after nerve repair was demonstrated by Cruikshank in 1795. Nelaton described the secondary nerve repair in 1864, and primary epineural nerve suture concept was introduced by Hueter[25] in 1871. Mikulicz described the sutures that reduced tension on the nerve suture

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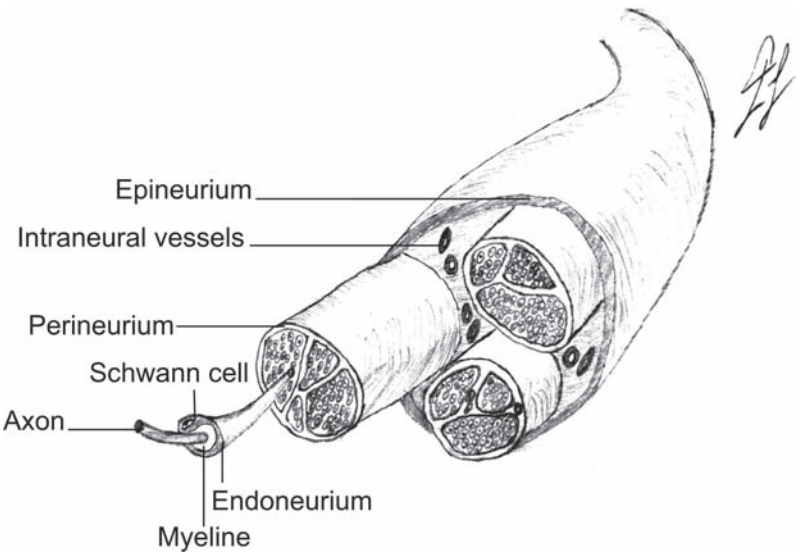

Figure 37.1. A myelinated peripheral nerve branch with associated structures, including epineurium, perineurium, endoneurium, intraneural vessels, Schwann cell, axon, and myelin.

in 1882, and Loebke described bone shortening to decrease the tension on the suture line in 1884. Albert described grafting nerve gaps in 1876. Cajal pioneered the concept of axonal regeneration from neurons and their guidance by chemotropic substances. Plenty of experience in evaluation and treatment of traumatic nerve injuries was gained in wartimes. During World War I, nerve injuries were repaired under tension and risked disruption after repair because of extensive soft tissue injuries and significant infections. After World War II, in 1945 Sir Sunderland demonstrated the detailed internal anatomy of the major peripheral nerves and developed microsurgical techniques to improve nerve repair outcomes.[87]

Peripheral Nerve Anatomy

All members of the mammalian system have the same peripheral nervous system anatomy. In the peripheral nervous system, nerve fibers are ensheathed by Schwann cells, either individually in case of myelinated fibers or in groups in the case of unmyelinated fibers. They form the sheath covering the axons in myelinated peripheral nerves, and they form basal membrane in unmyelinated nerves. The basal lamina layer envelops Schwann cells and supports axonal regeneration. Myelinated and unmyelinated nerve fibers are embedded within a connective tissue compartment called endoneurium. Endoneurium is the layer that covers and nourishes the axons. The endoneurium is encircled by a compact layer called the perineurium, which is a key component in nerve strength. The perineurium partitions nerve fibers into fascicles. These nerve fascicles are embedded within a connective tissue compartment called the internal epineurium. Epineurium is the connective tissue layer of the peripheral nerve, which both encircles and runs between fascicles. Its main function is to nourish and protect the fascicles. The outer layers of the epineurium are condensed to form a sheath-like structure that forms the external epineurium.[58] The perineurium and the nonfenestrated endothelium in the endoneural vessels form the blood nerve barrier, which blocks the infiltration of cells into the endoneural environment of the peripheral nerve (Figure. 37.1).

Nerve Injury Classification

Nerve injuries were classified by Seddon[80] in 1947, and Sunderland[87] further refined this classification in 1951.

Seddon classified nerve injuries into three major groups: neuropraxia, axonotemesis, and neurotemesis. Neuropraxia is characterized by a localized conduction deficiency and local myelin damage, usually secondary to compression.

Axon continuity is preserved and the nerve does not undergo distal degeneration. Axonotemesis is defined as loss of continuity of axons, with preservation of the connective tissue elements of the nerve. Wallerian degeneration is seen distal to the trauma zone. Neurotemesis is the most severe injury, equivalent to physiologic disruption of the entire nerve; it may or may not include actual nerve transactions.[80]

The Sunderland classification is based on the widely variable prognoses of axonotemetic injuries. Axonotemesis grade is divided into three types in this classification depending on the fascicular structure coverage. Neuropraxia is equivalent to Sunderland type 1 injury. In Sunderland type 2 injury, the endoneurium and the epineurium are still intact. Complete functional recovery can be expected, because the regenerating axons from the proximal segment are directed along their original course. The time for recovery is directly related to the level of injury because of the need for axon regeneration distally to the end organ. In a Sunderland type 3 injury, the endoneurium is disrupted, but the perineurium and endoneurium are intact. Endoneurium fibrosis, which hinders axonal regeneration, occurs because of this disruption, and recovery is incomplete unless the intrafascicular fibrosis is excised. If there is severe retrograde injury to cell bodies, which either destroys neurons or slows their recovery with longer delays, end organ changes may develop and the recovery will be incomplete. In a Sunderland type 4 injury, there is full-thickness fibrosis formation, and only the epithelium is intact. Retrograde neuronal damage is intensified. This type of injury requires excision of the damaged segment and surgical repair or reconstruction of the nerve. Sunderland type 5 injury is equivalent to Neurotemesis, and it is characterized by complete nerve disruption. Recovery is impossible without surgical intervention.[87] In 1988, Mackinnon and Dellon presented a "type 6" injury that represented a complex peripheral nerve injury. This grade involved combinations of Sunderland's grades of injuries.[57]

Nerve Degeneration

Waller described the degenerative changes developing in the distal nerve after nerve transaction in 1850, which was later called "Wallerian degeneration." Just after nerve injury, myelin starts to break down. Axonal and myelin debris are removed by the phagocytic action of macrophages and Schwann cells, a process that can take from 1 week to several months. Schwann cells become active within 24 h after injury, exhibiting nuclear and cytoplasmic enlargement as well as an increased mitotic rate. Schwann cells appear to ingest axonal and myelin debris, and then pass this on to macrophages. Same changes also occur in the proximal end of the injury for a varying distance, depending on the severity of the injury. Endoneural sheath of the nerve fibers form the tubes called "Bands of Bungner," which are organized by the proliferating Schwann cells, and they are the potential tubes into which the nerve sprouts will grow.[57]

Nerve Regeneration

Within the first 24 h following nerve injury, while Wallerian degeneration continues distally, axons from the proximal segment start to sprout at the same time. Multiple axons are produced from each axon. These axons are unmyelinated at the beginning even when the axon from which they have originated is myelinated. As the regenerating units mature, they become myelinated fibers, and functional recovery can be seen. If these regenerating fibers cannot reach appropriate distal routes and are lost in the extraperineural environment, then a neuroma will form, which represents a potential loss of functional recovery and may be a source of neurogenic pain. There is a growth cone that consists of filopodia rich in actin in the distal part of regenerating axons. The tips of the filopodia will explore the distal environment, and if they are in contact with the basal lamina of the Schwann cell, they draw the entire growth cone distally. The axonal sprout regenerating rate toward the distal segment ranges between 1 and 4 mm/day.[57,58]

Nerve Repair

Peripheral nerves that are interrupted by trauma or surgical resection of a mass require reapproximation of their ends. Nerve repairs may be primary or secondary depending on the time of repair after injury.

Primary repair is a direct reconnection of the nerve after disruption of its continuity. Historically it was thought that the best approach is to wait 3 weeks before repair to allow for completion of the Wallerian degeneration process, but Mackinnon et al. have shown that immediate repair is associated with better results.[58]

Prerequisites for early repair are a clean wound, good vascular supply, no crush component to the injury, and adequate soft tissue coverage.

Secondary repairs are delayed repairs that may contain different strategies when the prerequisites of primary repair cannot be met. However, within 3 weeks after injury, a nerve may lose as much as 8% of its length.[94]

An end-to-end repair is preferred if the gap is small, and the two ends can be approximated with minimal tension. Best results occur when the nerves are either purely sensory or purely motor and when the intraneural connective tissue component is small, which can vary from 22% to 80%.[93,94] End-to-end nerve repair techniques are "Epineural repair," "Group-fascicular repair," and "Fascicular repair." Sharp lacerations without loss of nerve substance or partial lacerations with proper alignment are good examples of injuries that benefit from epineural repair. In a crushing or delayed repair requiring trimming of nerve ends, group-fascicular repair improves fascicular alignment without an excessive number of sutures. Excessive sutures add to scar tissue production. Fascicular repair is not practiced widely, because it requires numerous sutures, and it is technically difficult.[94] Proponents argue that group-fascicular repair is better, because axonal alignment is more accurate with this technique. However, others have shown that there is no functional difference in outcome between epineural and group-fascicular repair. Furthermore, group-fascicular repair has the potential disadvantage of increased scarring and damage to the blood supply as a result of the additional dissection. Lundborg et al.[55] showed that although this technique ensures correct regeneration of regenerating axons, there is little evidence that it is superior to the less exact but simpler epineural repair.

"Epineural sleeve neurorrhaphy" is another nerve coaptation technique introduced by Siemionow et al.[82] It is a modification of the "epineural cuff" technique described by Snyder et al.[83] before. In epineural sleeve technique, the free edge of the epineurium at the distal stump is rolled back and a 2-mm nerve segment of the distal nerve stump is resected. The epineural sleeve created is then pulled over the proximal nerve end to cover the coaptation site and is anchored to the epineurium 2 mm proximal to the coaptation site with two sutures placed 180° apart. In this study, it was proven that epineural sleeve neurorrhaphy technique revealed faster functional recovery when compared with conventional end-to-end nerve coaptation. This finding was attributed to the fact that in this technique no tension or compression was created at the repair site. Instead, the tension was applied to the epineural sleeve, which is proximal to the coaptation site. In addition, the epineural sleeve provides a biologic chamber at the coaptation site, where axoplasmic fluid is collected, leaving a perfect milieu for regeneration of the nerve. Finally, perfect fascicular matching without bulging was possible using this technique, because the nerve's own epineurium guided the alignment of regenerating fascicles. Faster functional recovery has been shown in the experimental model with this technique, but it has to be verified clinically.[89]

Although the classic technique of neurorrhaphy is devoid of tension, Hentz et al.[40] studied a primate model and showed that direct nerve repair under modest tension actually does better than a tension-free nerve graft over the same regenerating distance.

Neuroma in Continuity

A neuroma in continuity may arise like a bulbous swelling as a sequel to peripheral nerve injury. This lesion may be composed of some functional and nonfunctional fascicular components, which have to be determined by a careful preoperative assessment. Effective treatment of a painful neuroma remains as a challenging problem, because it is possible that the surgeon may sacrifice the functional fascicles while trying to repair the nonfunctional fascicles of the nerve.[63]

A sharp pain associated with a scar and functional loss in the distribution of the involved nerve is typical for neuroma in continuity. The character of pain and its psychological implications are important in the selection of patients for surgery.

In the surgical treatment of the neuroma in continuity, fascicles may be separated by microneurolysis and regenerating axons are directed appropriately to distal ones when the nerve stumps and surgical bed are suitable for this. The functional fascicles can be identified proximal and distal to the injury site via electrodiagnostic testing. These fascicles are preserved, and potentially damaging dissection within the neuroma incontinuity should be avoided.[60]

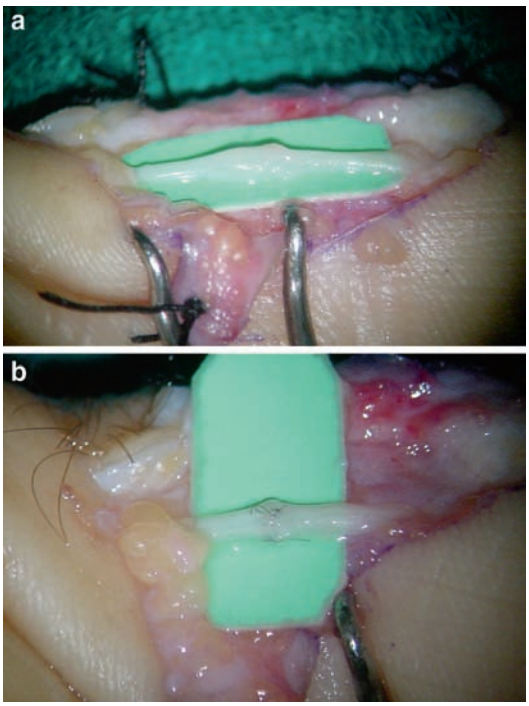

Figure 37.2. (a) A neuroma in the continuity of the digital nerve. (**b**) Epineural end-to-end repair of the digital nerve after excision of the neuroma.

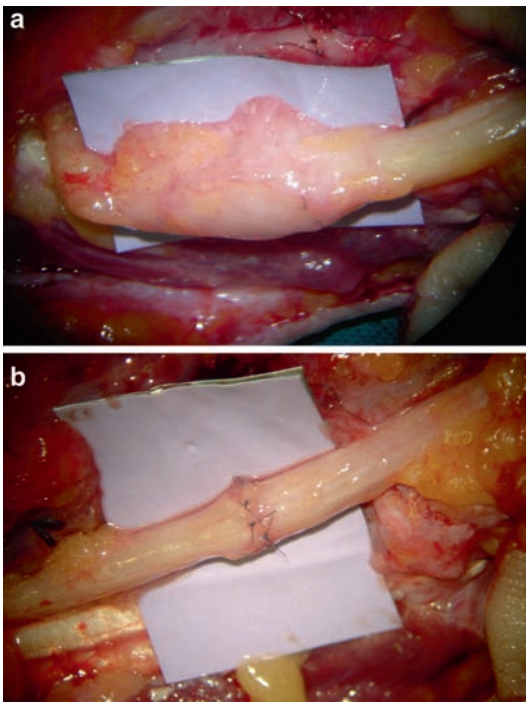

Figure 37.3. (a) A neuroma in the continuity of the ulnar nerve. (**b**) Epineural end-to-end repair of the ulnar nerve after excision of the neuroma

When there is insufficient length for tension-free end-to-end repair, a nerve graft can be used. The second alternative for painful neuroma treatment is resection of neuroma and transposition of the proximal stump into muscle (or bone, e.g., in digits) for noncritical or function distal nerves, in cases of previous treatment failure, or if the wound bed is not suitable for neural repair[72,74,97] (Figures 37.2 and 37.3).

Nerve Sheath Tumors

Neurofibroma (from Schwann cells and fibroblasts), neurilemmoma (from Schwann cells), and neurofibrosarcoma (from Schwann cells and fibroblasts) are the three types of tumors originating from intrinsic cells of the nerve.

Neurofibroma

Neurofibromas are usually seen with neurofibromatosis. They rarely cause some symptoms like pain or paresthesia, and their usual location is along peripheral nerves. They are nonencapsulated fusiform tumors, composed of excess of cells within nerve fascicles.[65,92]

If the tumor size is small without causing major neurological deficit, simple enucleation is recommended. If the tumor size is large, with major neurological symptoms, en bloc resection of the tumor with the peripheral nerve is the treatment of choice.[43,92] (Figure. 37.4).

Neurilemmoma

Neurilemmomas (also called "schwannoma" and "neurinoma") mostly arise from major mixed (motor and sensory) nerves and are solitary unless the patient has neurofibromatosis (von Recklinghausen's disease). They are well-encapsulated tumors, composed of differentiated Schwann cells. Large neurilemmomas may cause some neurological symptoms such as paresthesia and pain, but the small ones are usually asymptomatic. Surgical dissection of the tumor from the nerve is the treatment of choice, and recurrence is rare after excision of neurilemmomas[65,92] (Figure. 37.5).

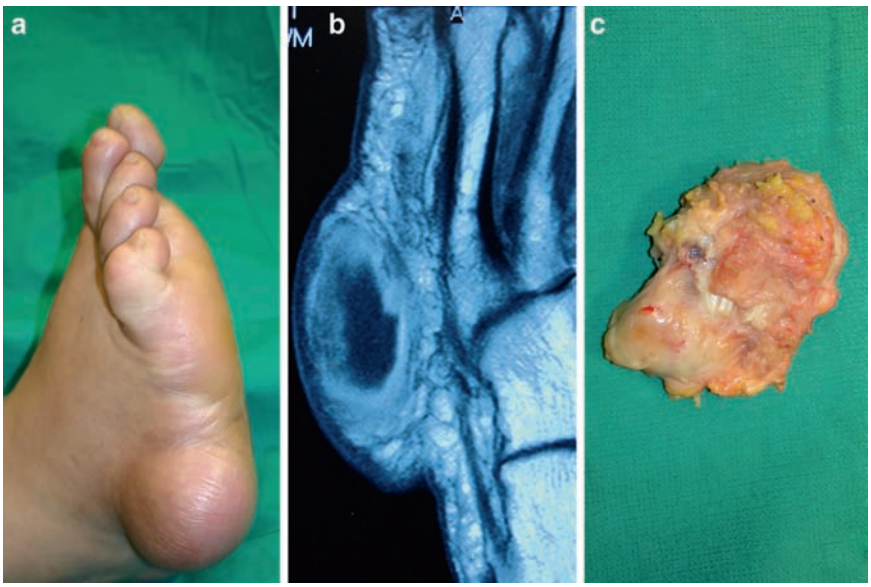

Figure 37.4. (a) A case of neurofibroma of the lateral aspect of the foot. (**b**) Magnetic resonance imaging confirming neurofibroma lesion. (**c**) Neurofibroma measuring 5 cm × 2 cm after excision.

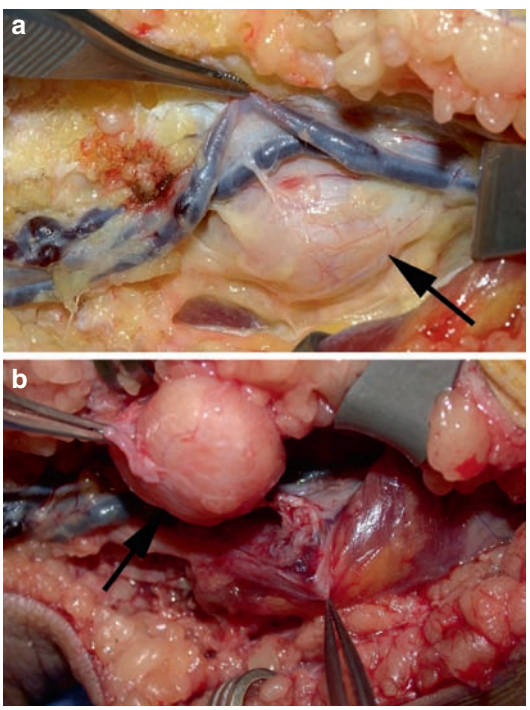

Figure 37.5. (a) Neurilemmoma (*arrow*) of the posterior tibial nerve above the medial malleolus of the foot. (**b**) Posterior tibial nerve after resection of the tumor (*arrow*).

Neurofibrosarcoma

This is a highly malignant tumor, which is found either in a patient with neurofibromatosis or in patients who have undergone radiotherapy. Neurofibrosarcoma may be termed a malignant schwannoma, since the tumors arise from the sheath of Schwann cells covering neural tissues. Radical excision followed by chemotherapy and radiotherapy is the treatment of choice in this aggressive tumor, and patients with neurofibrosarcomas have recurrence rates of more than 10%.[21,31,33,50,62]

Compressive Neuropathies

Although any nerve in the body can be compressed, certain nerves are more vulnerable because of their anatomic locations. The common symptoms include pain, sensorial deficit, and motor loss. Diagnosis is based on physical examination, whereas electrodiagnostic and imaging studies help to confirm the diagnosis and provide information regarding the recovery potential.[85]

Upper-Extremity Compressive Neuropathies

Carpal Tunnel Syndrome

Carpal tunnel syndrome (CTS) is the most frequent nerve compression syndrome. The median nerve is compressed inside the carpal canal by the transverse carpal ligament at the wrist. This compression may be caused by intrinsic (inside the carpal canal) or extrinsic factors (outside the carpal canal). CTS may be related to congenital, traumatic, metabolic/endocrine, inflammatory, infectious, or idiopathic causes. In addition, repetitive use is thought to increase the incidence of CTS.[35]

Physical examination may reveal percussion tenderness of the median nerve at wrist region (Tinnel's sign). Sensory deficits such as decreased sensation to light touch or pain may be present in the radial three digits.[12] Weakness of the abductor pollicis brevis and atrophy of the thenar muscles are common in the late phases of the disease. Many tests have been described for CTS such as Phalen's test, reverse Phalen's test, compression test, tourniquet test, closed fist test, "flick sign," and hand elevation test. Electrodiagnostic tests are helpful in the conformation of the diagnosis and to show the severity of the condition and also in differential diagnosis.

Treatment options for CTS can be categorized as operative and nonoperative. Nonoperative options include activity modifications to avoid exacerbating positions, nonsteroidal anti-inflammatory drugs (NSAIDs), splinting the wrist at neutral position, steroid injections to carpal tunnel, pyridoxine, and diuretics. In cases of nonoperative treatment failure or in advanced cases, operative treatment is indicated.[36] Surgical treatment may be classified as open and closed techniques. In the classic open technique, transverse carpal ligament is released under direct visualization via a mid-palmar incision.[2] In the closed technique (endoscopic), mid-palmar incision is avoided. Earlier recovery and return to work than the open release has been shown in some studies. Neurovascular complication incidence is not increased in the closed technique.[8,95] Incomplete release and longer operation time are among the disadvantages of closed technique, particularly at the beginning of the learning curve[1,16] (Figure. 37.6).

Anterior Interosseous Nerve Syndrome (Kiloh–Nevin Syndrome)

Anterior interosseous nerve is compressed in the forearm region by various anatomic structures. It may be compressed by the deep head of pronator teres and other fibrous bands or vessels in the forearm region.

Patients present with weakness of flexor pollicis longus, flexor digitorum profundi (of second and sometimes third digit), and pronator quadratus muscles of the affected hand. These patients are unable to make the shape of letter "O" between first and second digits. Electrodiagnostic tests are particularly helpful for its differentiation from the "pronator syndrome."

Its treatment is usually surgical release of the structures compressing the median nerve in the forearm region such as lacertus fibrosus, deep head of pronator teres, and other structures such as muscles or vessels overlying the interosseous branch of the median nerve, via an S-shaped incision extending from distal arm to proximal forearm.[29,85]

Pronator Syndrome

The pronator syndrome is described as compression of the sensory branch of the median nerve in the elbow region by the anatomic structures such as pronator teres, the superficial arch of flexor digitorum superficialis, and the bicipital aponeurosis.[6] Patients suffer from pain in the volar proximal portion of the forearm, which may also extend to the distal arm. Electrodiagnostic motor tests are typically normal. Increased pain with resisted pronation of the forearm and elbow extension (compression of pronator teres), pain with resistance to flexion of the middle finger proximal interphalangeal joint (compression of flexor digitorum superficialis arch), and pain with resisted elbow flexion and forearm supination (compression of bicipital aponeurosis) are the provocative maneuvers in this syndrome.[85]

Conservative treatment is the first choice in these patients. Physical therapy, NSAIDs, and local steroid injections are usually effective in most of the patients. In the resistant cases, the median nerve may be decompressed in the forearm region.[69,85]

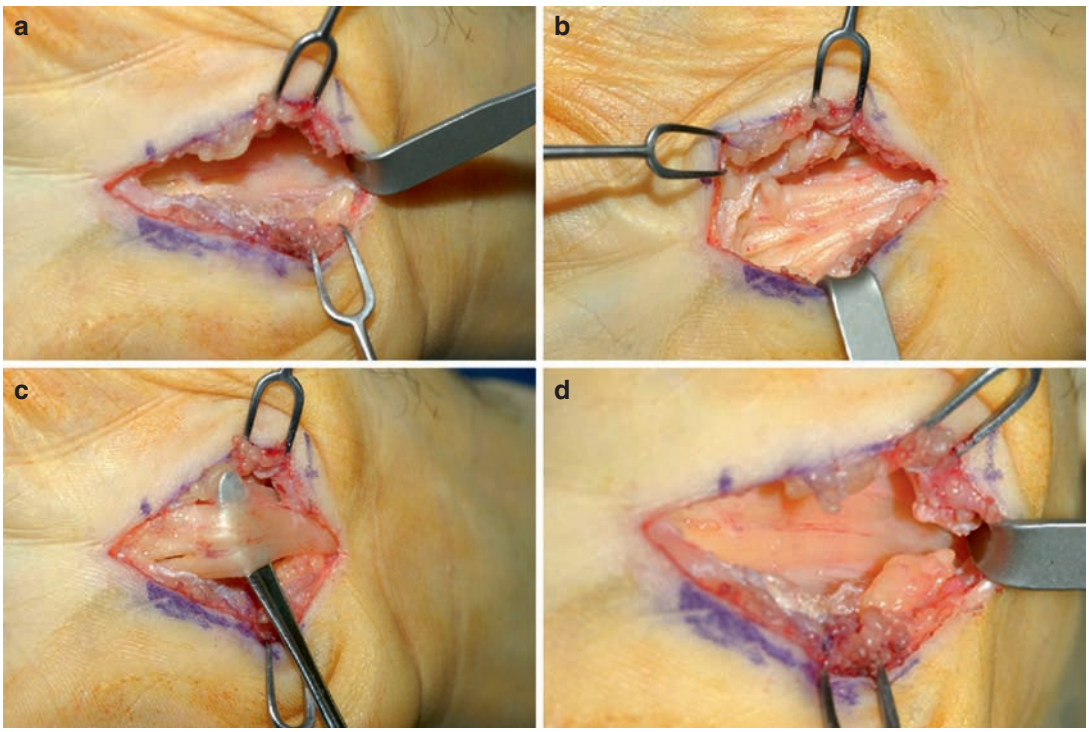

Figure 37.6. Classic open technique of transverse carpal ligament release. (**a**) Mid-palmar incision. (**b**) Exposure of the median nerve after transverse carpal ligament release. (**c**) Presence of engorged vasonervorum after median nerve decompression. (**d**) Median nerve within carpal tunnel following release.

Ulnar Tunnel Syndrome

In this syndrome, ulnar nerve is compressed within Guyon's canal. Trauma, soft tissue masses, and variant muscular and vascular anomalies may cause this compression.[37]

Patients may present with intrinsic muscle weakness of the affected hand, wrist pain, and diminished sensation in the cutaneous dermatome of the ulnar nerve of the affected hand. Motor weakness of the adductor pollicis, hypothenar, and interosseous muscles with positive Wartenberg's sign may be seen. Diminished sensation on the ulnar site of the palm, with normal sensation on the dorsum of the hand, differentiates the distal ulnar nerve compression from a more proximal site than the wrist region. Froment's sign and Phalen test may be positive in this syndrome.[85,98]

Surgical decompression is required in most of the patients, while conservative treatment can be tried in patients with mild symptoms. Ulnar nerve can be decompressed via a curvilinear incision that is centered on the hook of hamate in this region. It is important to decompress both superficial and deep branches of the ulnar nerve.[30]

Cubital Tunnel Syndrome

It is the second most common entrapment syndrome after CTS. In this syndrome, the ulnar nerve is typically compressed beneath the cubital tunnel retinaculum at the elbow. This compression may be spontaneous due to repetitive and sustained elbow flexion, presence of mass, arthritic spur, synovitis, and anatomic variations (e.g., anconeus epitrochlearis).[85]

Medial elbow pain with dorsoulnar-sided palm and digit paresthesias or dysesthesias may be the symptoms of these patients. Intrinsic muscle weakness and hand clumsiness are late symptoms.[19] Tenderness over the elbow at the level of the medial epicondyle may be revealed by examination of the patient. Electrodiagnostic testing reveals motor weakness of the flexor digitorum profundus, flexor carpi ulnaris, and interosseous muscles and slowing at the elbow level.[14]

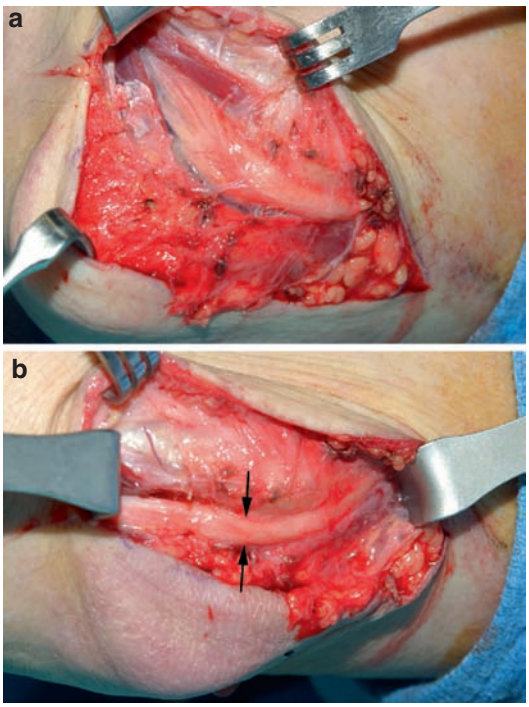

Figure 37.7. Cubital tunnel release. (**a**) Exposure of the ulnar nerve within the ulnar nerve groove. (**b**) Ulnar nerve after decompression showing narrowing at the compression site (*arrows*).

Conservative treatments such as an activity modification, NSAIDs, and nighttime elbow splint are beneficial in some patients.[28] Operative treatment is indicated if the nonoperative treatment fails. Decompression of the ulnar nerve with or without medial epicondylectomy and ulnar nerve transposition are performed as a surgical treatment[75] (Figure. 37.7).

Posterior Interosseous Nerve Compression

Posterior interosseous branch of the radial nerve is compressed in the proximal forearm region in this disease. The most common site of compression is the Arcade of Frosche (proximal edge of supinator muscle), followed by fibrous bands in the vicinity of the radiohumeral joint, vascular malformations (leash of Henry), tendinous edge of the extensor carpi radialis brevis, and substance or distal edge of the supinator muscle.[4]

Forearm pain with extensor weakness is the typical symptom of these patients. Physical examination reveals varying degrees of finger or thumb drop without sensory deficit. Surgery is the treatment of choice via anterior (Henry) or posterior (Thompson) approach.[53]

Radial Tunnel Syndrome

Radial tunnel syndrome is attributed to the cases of proximal forearm pain caused by compression of the posterior interosseous nerve without motor paralysis. Electrodiagnostic studies are usually normal. Local nerve blockade may be useful in differential diagnosis of this syndrome.[32] Long-term conservative treatment with physical therapy is recommended. Surgery is indicated in resistant cases via the Thompson approach.[85]

Posterior Interosseous Nerve Syndrome

"Arcade of Frohse" (proximal edge of the supinator muscle) is the most common site where the posterior interosseous nerve is compressed in this syndrome. These patients present with weakness or paralysis of the extensors of the wrist and digits. Vague forearm pain may accompany the symptoms, without any sensory deficit. Conservative treatment with physical therapy is recommended for 6–12 weeks, and surgery is indicated if no improvement occurs.[53]

Wartenberg Syndrome

Patients with Wartenberg syndrome present with pain over the distal radial forearm along with paresthesia on the dorsal radial hand, which is associated with the compression of the superficial radial nerve at the wrist near the radial styloid. This nerve is susceptible to compression in this region due to extrinsic compression, trauma, surgery, or repetitive movement. Most of the patients are treated nonoperatively, including rest, splinting, and removal of the compression sources. Surgery is indicated in resistant cases[53,84] (Figure. 37.8).

Lower-Extremity Compressive Neuropathies

Lateral Femoral Cutaneous Nerve Compression (Meralgia Paresthetica)

The lateral femoral cutaneous nerve (LFCN) may be compressed by the inguinal ligament. Dysesthesia and hypesthesia of the lateral thigh region are common in these patients. Tinnel's sign may be present on the tract of LFCN under the inguinal ligament.[61,91]

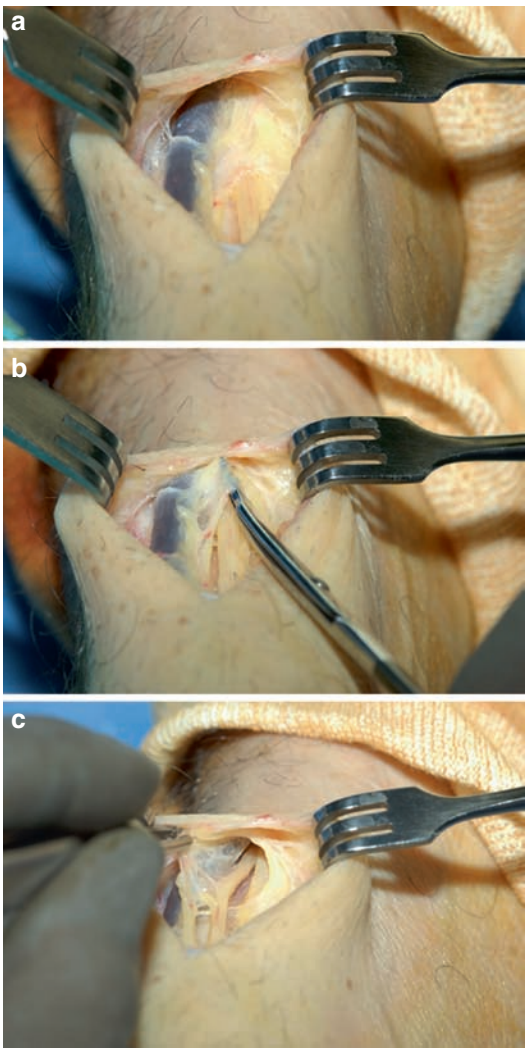

Figure 37.8. Surgical decompression of the superficial radial nerve at the wrist region. (**a**) Exposure of the superficial radial nerve. (**b**) Decompression of the superficial radial nerve. (**c**) Superficial radial nerve after decompression.

Electrodiagnostic tests are not used in this nerve entrapment. Adjustment in the posture may be the first treatment of choice, but if this fails, surgical decompression with transaction of the inguinal ligament is indicated.[46,61]

Common Peroneal Nerve Compression

It is the most common nerve entrapment of the lower extremity. The common peroneal nerve is compressed at the region of the neck of fibula. Patients present with sensorial deficit in the anterolateral aspect of the leg and dorsum of the foot. Foot drop is accompanied in more advanced cases.[42,91]

Although most of the cases were caused by traumas, bony tumors, and ganglia, it has also been reported in athletes due to repetitive trauma or nerve traction injury.[68] Surgical decompression is the treatment of choice in most of the cases via curvilinear incision over the neck of fibula[90] (Figure. 37.9).

Tarsal Tunnel Syndrome

Tarsal tunnel syndrome is the compression of the tibial nerve in the tarsal tunnel in the ankle region. Systemic illnesses such as diabetes mellitus, alcoholism, and hypothyroidism were reported to accompany this syndrome.[10,34]

Sensorial deficit and pain in the plantar region are the symptoms associated with this syndrome. Intrinsic muscle atrophy with clawing of the toes is seen in advanced cases. Electrodiagnostic tests are usually diagnostic. Operative treatment is the treatment of choice via longitudinal incision between the medial malleolus and calcaneus[91] (Figure. 37.10).

Nerve Repair Using Autograft

Although direct nerve repairs demonstrated better results than nerve grafts, nerve grafts revealed superior results when compared with direct repairs performed under undue tension, which produced nerve ischemia.[7,49,56,67] In a study that evaluated nerve tension as a determinant of nerve ischemia in a rat sciatic nerve model, blood flow was inversely proportional to nerve tension. Chronic nerve injuries required nearly three times more of the baseline blood flow when compared with acute nerve injuries. Although an 8% elongation was tolerated for both acute and chronic nerve injuries, a 15% elongation produced a profound decrease in blood flow, which never recovered. When primary repair cannot be performed without undue tension, nerve grafting is required. Acute injuries retract only 4%, making primary end-to-end repairs possible, whereas the 28% retraction in delayed repairs cannot be overcome by nerve elongation, and nerve grafts are usually necessary.[96]

Nerve autografts remain the gold standard for nerve grafting. Three major types are cable, trunk, and vascularized nerve grafts. Cable grafts

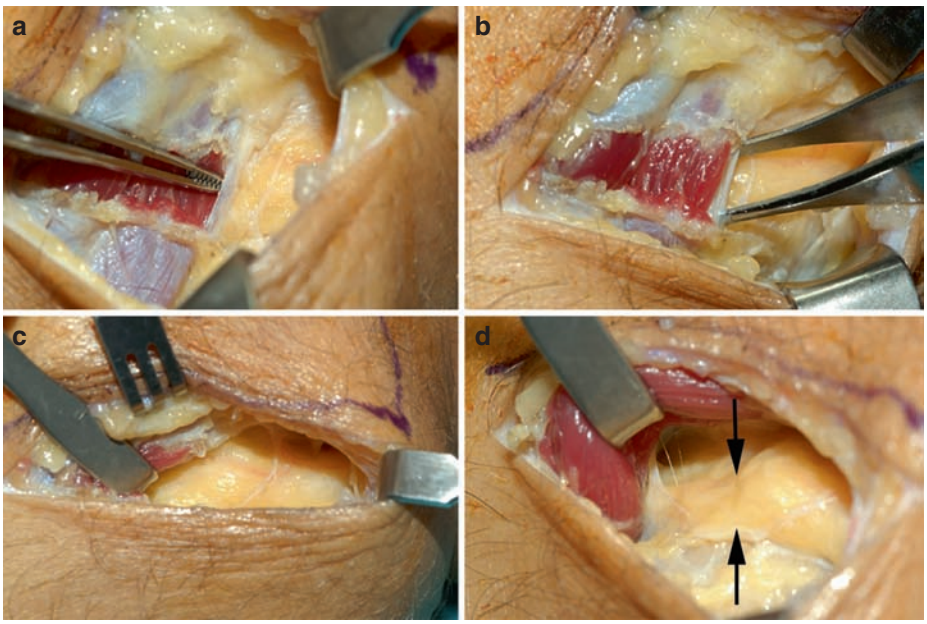

Figure 37.9. Surgical decompression of the common peroneal nerve. (**a**) Forceps are pointing toward the fascia of the peroneus longus muscle 3 cm below the fibular neck. (**b**) Entrance of the common peroneal nerve into peroneus longus muscle after release of the peroneus longus fascia. (**c**) Common peroneal nerve after decompression and resection of the peroneus longus fascia. (**d**) The site of common peroneal nerve compression (*arrows*) on the entrance to the anterior compartment underneath the peroneus longus muscle, which is elevated by the retractor.

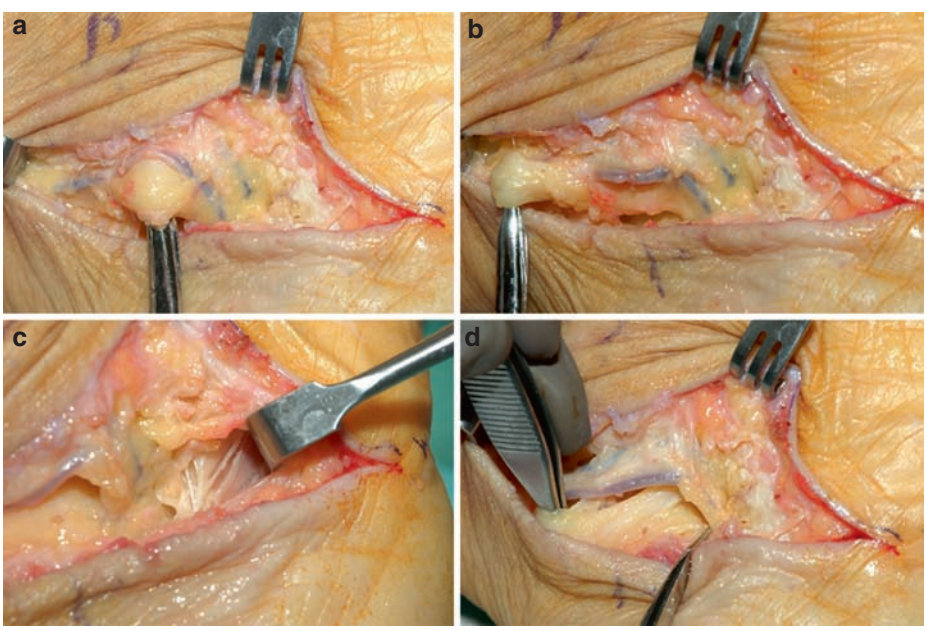

Figure 37.10. Surgical decompression of the posterior tibial nerve in the tarsal tunnel. (**a**) Decompression of the posterior tibial nerve after release of flexor retinaculum. (**b**) Decompressed posterior tibial nerve. (**c**) Decompression of the medial plantar nerve; retractor is holding abductor hallucis muscle. (**d**) Medial plantar nerve entering medial plantar tunnel after decompression.

may be single or multiple small-caliber nerve grafts aligned parallel to span a gap between fascicular groups. The senior author, Siemionow, evaluated the single-fascicle technique for neural deficit repair and proved that a single-fascicle graft resulted in faster functional recovery and better morphologic outcome compared with conventional nerve repair.[89]

Trunk grafts are mixed motor-sensory whole nerve grafts. They have been associated with poor functional results in large part due to the thickness of the graft and the consequent diminished ability to revascularize after implantation.

Vascularized nerve grafts have been used in the past but with conflicting results. They may be considered if a long graft is needed in a poorly vascularized bed. Because of donor-site morbidity, vascularized grafts have been used mainly in brachial plexus injuries.[51]

The most common source of autografts is the sural nerve, which is easily obtainable and relatively dispensable. Other autograft sources include the anterior branch of the medial antebrachial cutaneous nerve and the superficial radial sensory nerve.[87] After end-to-end nerve repair using a nerve autograft, Wallerian degeneration will be seen distal to the coaptation site.

Conduits

Neural conduits are biological (veins) or synthetic structures that guide the regenerating axons to the distal nerve stump. Many researchers have studied different types of conduits that can be used as alternatives to nerve autografts. Both biodegradable and synthetic nonbiodegradable materials, with or without impregnation of growth factors and Schwann cells, have been investigated.

Lotheissen[52] was the first to use gelatin conduits in 1901. In the 1940s, Weiss and Taylor[100] and in the 1960s, Braun[11] and Leeger studied the effects of gelatin on nerve generation. In 1984, Colin and Donoff[22] reported good results by using collagen conduits, which were comparable to nerve autografts. Navaro et al.[73] used lamininfilled collagen tubes in 3–5 mm of nerve defects with successful results. Takashi et al.[88] reported successful results using collagen conduits filled with a collagen matrix in nerve defects of 3 mm. Silicone: poly(dimethylsiloxane),[54] poly(glycolide),[99] poly(3-hydroxybutyrate),[103] poly(L-lactide),[101] Chitosan,[45] D-glucosamine,[102] Polyester urethane,[9] poly 2-hydroxyethylmethacrylateco-methylmethacrylate[27] are the other materials used as conduits so far.

The materials investigated to date are not without disadvantages, such as the induction of scarring, foreign body reactions, conduit collapse, and invasion by scar tissue. Other drawbacks include the technical difficulty of performing the procedure and often the need for a second procedure to remove nonbiodegradable components. None of the conduits has been proven to be as effective as nerve autografts so far.[17,86]

Synthetic nerve conduits can be used for the reconstruction of small-diameter nerves with a defect of not exceeding 3 cm or with large-diameter nerves with a defect of less than 0.5 cm. The use of vein graft as a conduit is recommended only for nerve gaps less than 3 cm in noncritical areas.[70,104]

Nerve Repair Using Allograft

Nerve repair with autograft is inadequate when there is an insufficient amount of autologous nerves available for repair of large defects. This prompted the search for alternative means of reconstruction in extensive nerve injuries. Over the past decades some authors,[5,15,41,44,48,79] particularly Mackinnon et al.,[59] explored the use of host immunosuppression and cadaveric nerve allografts to restore nerve continuity in clinical cases. The cadaveric nerve allograft provides an unlimited graft source without the morbidities associated with autograft reconstruction. Nerve allografts act as viable conduits. Host motor and sensory axons grow to reach host targets via these conduits. Function is provided by the regenerating autologous nerves, and this regeneration is supported by allogenic cells. In order to ensure Schwann cell viability and minimal fibrosis, allograft must be revascularized in an early post-transplant period.[66]

These grafts are rapidly rejected unless appropriate recipient immunosuppression is achieved. The toxicity associated with immunosuppression required to promote graft acceptance must be compared with the relative benefits of reinnervation before nerve allotransplantation can be safely applied in routine clinical practice.[66]

Nerve Graft Alternatives

Another reinnervation technique of a denervated muscle is the "split nerve transfer method." This technique was first described by Conley and Baker[23] in 1979. Although they reported that this technique was not clinically very effective, there are reports in favor of this technique in the literature.[3,26,76]

Neurotization is another technique used for reinnervation of a denervated muscle, described by Gersuny in 1906. In this technique, the motor nerve is directly implanted into the muscle. Zhasng et al.[105] reported the formation of motor end plates by this technique in 1986.

An alternative technique to nerve autograft is "intact nerve bridge" described by McCallister et al.[64] in 2001. In that study, the authors observed the histological evidence that the intact nerve functioned as a bridge for regenerating axons, which traveled in the outer epineurium of the tibial nerve before entering the distal end-to-side coaptated nerve.

Future Developments

Stem cell supportive therapy and cellular therapeutics such as chimeric cell transplantations are among the current strategies used for tolerance and chimerism induction. Both types of cells may be systemically administered via intravenous or intraosseus routes or may be locally administered. Injections may be also delivered into the synthetic, autologous, or allogenic conduits.[20,39,71]

The promising results of the stem cell injections in nerve regeneration made us design a study with subepineural injection of the mesenchymal stem cell. The short- and long-term results of this study are currently being reported.

Schwann cell seeding is another method that may be used for creating an adequate environment for nerve regeneration. They help inflammatory cells eliminate debris and upregulate the synthesis of trophic factors such as nerve growth factor (NGF) and laminin.[20]

Acellular nerve allografts that contain natural extracellular matrix components and structure but not native cells are another valuable alternative to nerve autograft. They are currently used to study the roles of extracellular matrix components and cellular components on nerve regeneration concomitantly.[13,47,77,81] All these new technologies will support future studies on nerve regeneration over long nerve gap defects.

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38

Tendon Repair and Reconstruction

Donald H. Lalonde

Summary

Exciting new developments in flexor tendon repair made possible by the tourniquet-free, sedation-free, pain-free wide-awake approach are covered in detail in this chapter. This approach permits minimal sheath destruction, as flexor tendons are repaired through small transverse sheathotomy incisions. Active intraoperative flexion by the patient allows surgical adjustments to be made before the skin is closed to ensure that there is no gapping of the flexor repair and that the repair glides nicely in the sheath to get an optimal range of motion. Intraoperative patient teaching by the surgeon and the hand therapist present allows the patient to practice the postoperative movement regime in a pain-free comfortable environment. Risks and inconveniences of general anesthesia are avoided in almost all patients. Very functional differential gliding splints allow patients who have undergone dorsal hand extensor tendon repair to return to work 2–3 days after surgery and use the fingers of their operated hand with very little risk of tendon rupture.

Abbreviations

DIP Distal interphalangeal
MP Metacarpal phalangeal
PIP Proximal interphalangeal

Flexor Tendon Injuries

Timing of Flexor Tendon Repair

The repair of flexor tendon injuries does not have to be performed on an emergency basis. The skin can be closed, and the tendon can be repaired in the light of day when the circumstances are ideal. A delay of up to 10 days makes very little difference in the outcome.

It has often been said that injuries that are more than 3 weeks old cannot be repaired because the muscle bellies have been irretrievably shortened and the proximal tendon ends cannot be pulled out to length to permit a repair. However, if the proximal tendon is held out to almost full length by vinculae or by the lumbricals, late repair beyond 3 weeks is possible. Exploration is often required to fully determine the proximal tendon end location and ability to repair a flexor tendon.

In an old tendon injury in which the muscles have retracted the tendon too far proximally and the tendon cannot be pulled out to length, and in which the pulleys have scarred down in the finger, a two-stage tendon reconstruction with a tendon graft can be considered. However, if the superficialis is intact and functioning and only the profundus is lacerated, the cure may be worse than the disease. A superficialis finger functions quite well. A two-stage tendon graft should not be performed unless the patient is extremely well motivated and willing to cooperate fully

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with at least two operations and multiple visits to a hand therapist.

Wide Awake (No Sedation/No Tourniquet) Flexor Tendon Repair vs. Sedation, General Anesthesia, or Regional Block Repair

Conventionally, flexor tendons have been repaired under general anesthesia or with sedation and regional blocks. However, in the last 10 years, the myth that epinephrine (adrenaline) is contraindicated in the finger has clearly become past history.[2,4] Epinephrine hemostasis in the finger and hand has ushered in a new exciting era with wide-awake flexor tendon repair. With the wide-awake approach to hand surgery; the patient receives nothing but lidocaine 1% with 1:0,00,000 epinephrine injected tumescently wherever the surgeon will be cutting in the hand and finger. As finger and hand hemostasis with epinephrine is quite adequate, no tourniquet is required. Because there is no tourniquet required, sedation is not required and the patient is wide awake throughout the procedure. With the exception of young pediatric patients and some severely mangled and amputated hand parts, the author has performed all of his flexor tendon repairs using the wide-awake approach for the last 7 years. Most of his extensor tendons have been repaired with the wide-awake approach for more than 20 years.

Regional blocks such as Bier or axillary blocks paralyze forearm muscles, and therefore the patient cannot actively flex finger tendons during surgery, a distinct disadvantage when testing intraoperative active finger flexion during the flexor tendon repair procedure. Sedation is another distinct disadvantage that impedes intraoperative patient teaching and cooperative active patient movement during the surgery. The sedated patient is often uncooperative and remembers very little about intraoperative teaching.

Pure local anesthesia is injected into the patient's hand and finger in the waiting area before he or she is brought into the operating room. The first injection of 4–8 cc of lidocaine 1% with 1:1,00,000 epinephrine is administered in the volar subcutaneous fat of the most proximal part of the anticipated incision (see Figure 38.1). After 10–15 min, the now numb entire distal hand and fingers where incisions will be made are injected in a tumescent pain-free manner with 2–3 cc per phalanx of 1% lidocaine and 1:1,00,000 epinephrine. Finger injections are all in the subcutaneous fat in the center of the finger to avoid injuring digital nerves with the bevel of the needle (see Figure 38.2). Epinephrine hemostasis is at its best between 30 and 180 min after injection. The hemostatic vasoconstrictive effect of the epinephrine in fingers is completely gone by 6 h postinjection.[3] The duration of local anesthesia after lidocaine with this type of lidocaine with epinephrine is 10 h on average.[5]

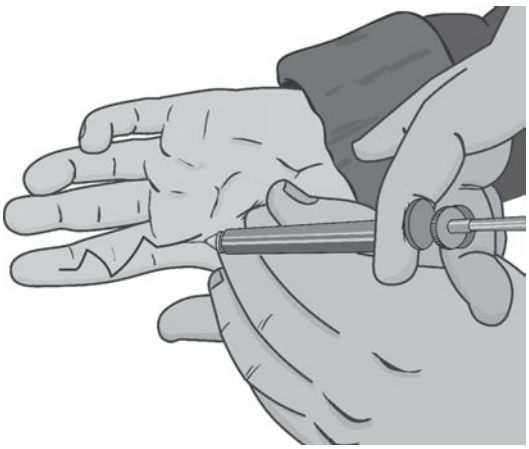

Figure 38.1. The first injection of 4–8 cc of lidocaine 1% with 1:1,00,000 epinephrine in the volar subcutaneous fat of the most proximal part of the anticipated incision. The patient is injected in the waiting area to allow time for the lidocaine and epinephrine to function.

The wide-awake patient will sometimes actively pull on the proximal flexor tendon during the intraoperative attempted delivery of the proximal tendon stump. In this situation, instead of the surgeon pulling against the patient's muscle power, the patient is simply asked to extend the fingers or wrist. With finger extension, finger flexors cannot help but to relax because of a spinal cord reflex. With this maneuver, pulling the proximal tendon stump into the wound is facilitated, and the stump can be secured with a needle, as it is when the patient is asleep.

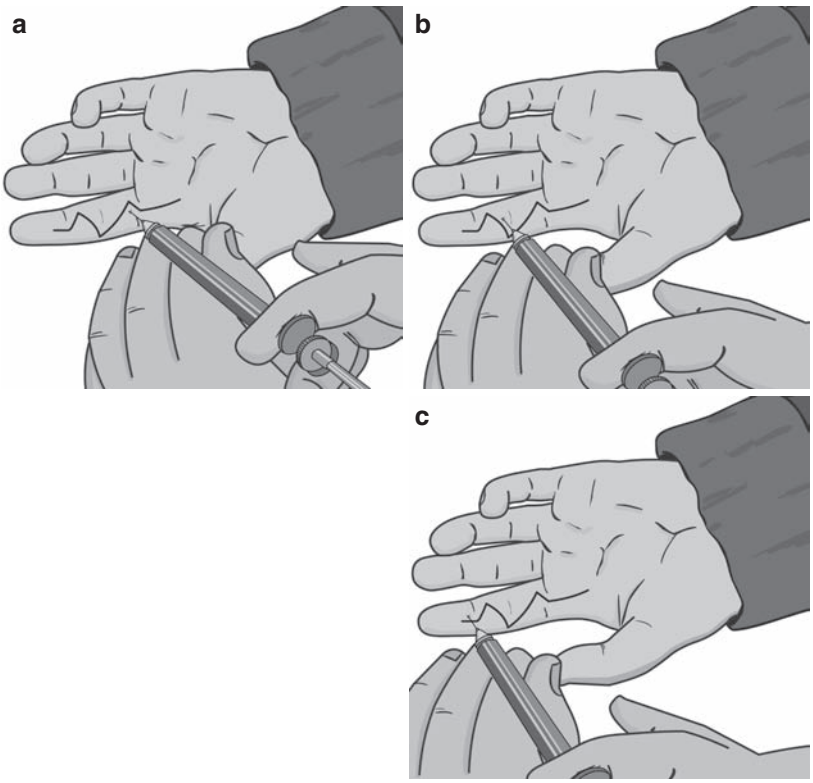

Figure 38.2. Ten to fifteen minutes after the first injection in Figure 38.1, the now numb entire distal hand and fingers where incisions will be made are injected in a tumescent, pain-free manner with 2–3 cc per phalanx of 1% lidocaine and 1:1,00,000 epinephrine. Finger injections – (**a**) proximal phalanx, (**b**) middle phalanx, (**c**) distal phalanx – are all in the subcutaneous fat in the center of the finger to avoid injuring digital nerves with the bevel of the needle.v

Flexor Tendon Injuries in the Finger

Preserving Pulleys and Tendon Sheath

Zone 1 is distal to the distal interphalangeal (DIP) joint. Zone 2 is from the proximal part of the A1 pulley to the DIP joint. In both Zones 1 and 2 injuries, the difficult part is retrieving the proximal tendon ends and performing a repair without injuring the flexor tendon sheath. It is particularly important to preserve the critical A4 (in the middle of the middle phalanx) and the A2 (in the proximal part of the proximal phalanx) pulleys. The more the pulleys and sheath are opened for exposure, the more the postoperative scar generated and the greater the tendency to bowstring the tendon repair. The wide-awake approach permits greater preservation of sheath and pulleys as described in the section Repairing the Tendons through Sheathotomy Incision to Preserve the Sheath and Pulleys.

Retrieving the Tendon Ends in Zones 1 and 2 Injuries

Three classic methods have been described to retrieve the proximal tendon ends.

1. The wrist and metacarpal phalangeal (MP) joints can be flexed and the tendons milked forward.
2. A hemostat can be inserted into the proximal lacerated tendon sheath opening and the tendon blindly grasped for. The problem with this method is that the tendon and the sheath can both be damaged by this rough approach. Postoperative scarring in the sheath and a frayed lacerated tendon end will generate

additional scar tissue, which will result in postoperative adhesions.

3. The proximal tendon end can be retrieved through a second incision in the palm or forearm, and a small rubber catheter can be inserted in the lacerated flexor sheath out to this second incision in the palm or forearm. The proximal tendon can be tied to the catheter and pulled out of the proximal incision in the sheath. Problems with this method are that proximal vincular blood supply attachments and lumbrical attachments to the flexor tendons may be avulsed by the delivery of the proximal tendon ends. In addition, the rubber catheter may not go between the two slips of the superficialis tendon in the proximal sheath where the profundus tendon belongs. Thirdly, the second incision in the palm presents yet another potential area of adhesion.

The author's preferred method of retrieving the proximal tendon ends is to expose the sheath proximally with Brunner incisions (into the hand if necessary) until the point where the vinculae are holding the tendon ends. The proximal tendon end(s) can usually be easily seen bulging in the sheath in the proximal finger. A small transverse sheathotomy incision is made a few millimeters proximal to the bulging tendon ends (see Figure 38.3). The incision that is perpendicular to the long axis of the sheath will expose the tendons and will not generate bowstringing. Two Adson forceps alternatingly grasp the lacerated tendon through the sheathotomy and push the tendon distally (like pushing a rope). The first forceps grasps the tendon proximally in the sheathotomy and pushes it distally. The second forceps then grasps the tendon proximally in the sheathotomy, the first forceps lets go, and the second forceps pushes it another 2–3 mm distally, and so on (see Figure 38.4). The tendon end is therefore delivered through the lacerated sheath without fraying or crushing its lacerated end. The transverse sheathotomy can be easily closed with a 6-0 absorbable suture after the tendon is held in the sheath with a skewering hypodermic needle.

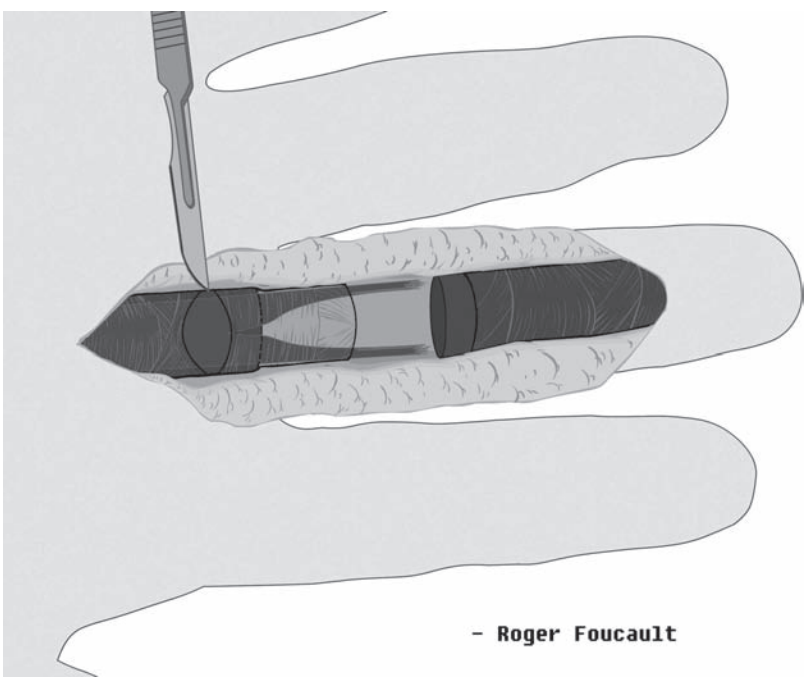

Figure 38.3. The proximal tendon end(s) can usually be easily seen bulging in the sheath in the proximal finger. A small transverse sheathotomy incision is made a few millimeters proximal to the bulging tendon ends. The tendon will be "pushed like a rope" distally though this sheathotomy.

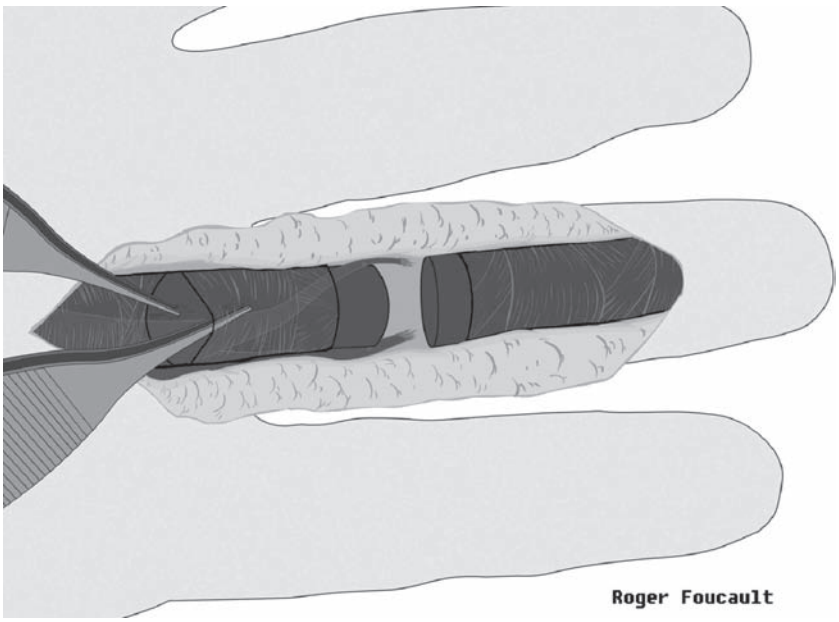

Figure 38.4. Two Adson forceps alternatingly grasp the lacerated tendon through the sheathotomy and push the tendon distally (like pushing a rope). The first forceps grasps the tendon proximally in the sheathotomy and pushes it distally. The second forceps then grasps the tendon proximally in the sheathotomy, the first forceps lets go, and the second forceps pushes it another 2–3 mm distally, and so on, until the untouched tendon end comes out of the lacerated sheath and is secured there with a needle.

Repairing the Tendon Ends in Zones 1 and 2 Injuries

Although many types of grasping suture are used throughout the world, the modified Kessler repair is still the most commonly used flexor tendon grasping suture in North America. Ideally, one would like to get a purchase of at least 5 mm of tendon on either side of the tendon laceration. In Zone 1 injuries, the tendon is anchored to the bone with a pullout suture (though a hole drilled in the bone with a K wire driver) tied on the nail. Alternatively, a suture anchor, a screw, or some other technique of tendon fixation to the bone can be used.

In the last 10 years, it has generally become accepted and preferred to use two core-grasping sutures of 3-0 or 4-0 material instead of a single core suture.[6] Some prefer braided nonabsorbable sutures for scar ingrowth. However, braided sutures tend to get extruded with infection more often than smooth sutures as there are little areas for bacteria to reside in the braided thread. This is why others prefer smooth strong suture. A running epitenon suture of smooth 6-0 suture is added by most surgeons for added strength and for smoothness of intrasheath gliding of the repair.

Repairing the Tendons Through Sheathotomy Incision to Preserve the Sheath and Pulleys

The author prefers using small transverse sheathotomy incisions through which are inserted the needles that suture the tendon (see Figure 38.5). The sheath itself can be used to anatomically reduce the two tendon ends (see Figure 38.6). In this particular situation, an epitenon suture is not possible, but the tendon ends are very nicely approximated.

Before using the wide-awake approach, larger areas of pulley/sheath had to be cut to expose the tendon for suturing. With the wide-awake approach, after a core suture is inserted into the tendon, the patient can actively flex the finger. This maneuver confirms that the intratendinous/intrasheath suture did not inadvertently grasp nontendon structures in spite of the fact that the needle and thread are going though a section of the tendon that is inside intact sheath. This maneuver cannot be performed in asleep

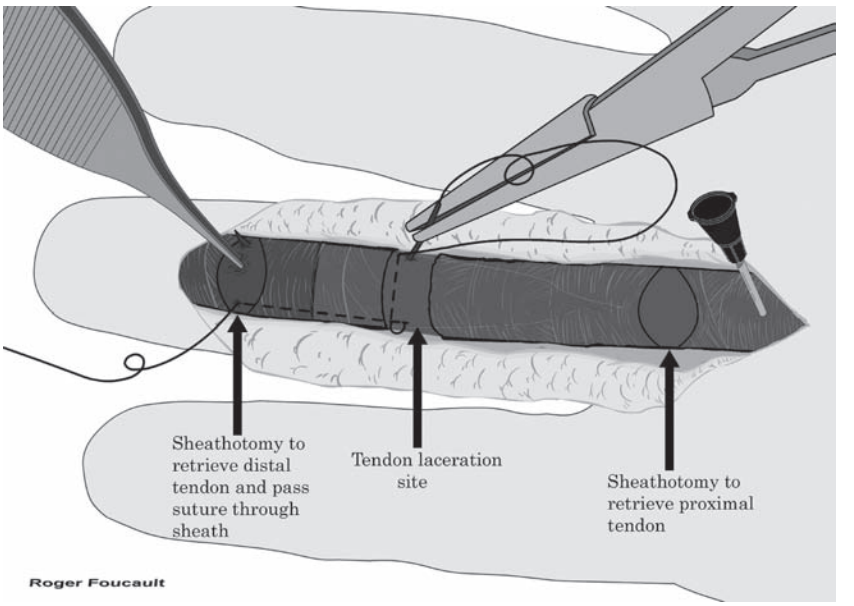

Figure 38.5. The tendon is repaired by inserting the suture through small sheathotomy incisions to preserve as much sheath and pulley as possible to decrease postoperative scarring and bowstringing. The wide awake patient actively flexes the finger during the surgery to be sure that the suture has gone through tendon only.

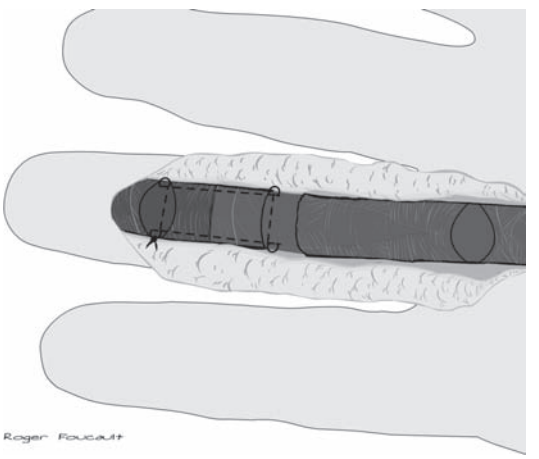

Figure 38.6. The sheath itself can be used to anatomically reduce the two tendon ends as is seen in this illustration. In this particular situation, an epitenon suture is not possible, but the tendon ends are very nicely approximated, as can be seen through the sheath in the patient. After the first core suture is placed, the patient actively flexes the finger to make sure there is no tendon gapping. The second core suture is placed, and the patient flexes the finger again before the skin is closed.

patients, patients with paralyzed muscles, or in those who are uncooperative with sedation. The small transverse sheathotomy incisions that permit needle and thread access can easily be closed with absorbable monofilament 6-0 sutures after the tendon repair. The small repaired sheathotomy incisions are much less likely to generate scarring and bowstringing than long sheath and pulley incisions required to expose both tendon ends that need suturing. This is particularly true when tendons are lacerated in the A2 and A4 pulley areas.

Ensuring There Is No Gap in the Flexor Repair

Intraoperative testing of a flexor tendon repair with active flexion by the patient is a new concept and a very important one. Tendon gaps are felt to be the most common cause of flexor tendon repair rupture, and any gaps in the repair must be rectified. After the first core suture is inserted and tied, the wide-awake patient is asked to flex the finger through a full range of motion. Occasionally, the suture will be seen to bunch up in the tendon and a gap in the repair will be identified. If a gap can be seen with the first core suture and active movement, the gap is rectified with a second core suture, and the first core suture is removed. If active movement reveals a partial gap with the first core suture on one side of the repair, the second core suture is

inserted to reinforce the repair on the gapping side.

With this technique, the surgeon can be confident that postoperative gapping will not occur unless excessive forces are applied to the repair.

Ensuring That the Repaired Flexor Tendon Will Glide Through the Pulleys

After insertion of the two core sutures, the wide-awake patient is again taken through a full range of active movement. Any final adjustment to the repair or to the pulleys can now be completed to optimize the final result. If the repair is too bulky to allow good movement in the sheath, unnecessary pulleys can be divided. Up to 25% of the A2 and A4 pulleys can be divided if necessary. If the size of the repair can be diminished with epitenon sutures, this can be performed as well.

Partial Flexor Tendon Injuries

Injuries of the tendon that are less than 50–60% may not need to be repaired. The wide-awake patient can be asked to actively flex the tendon, which can be seen to glide in the lacerated pulley. If triggering is observed, the pulley can be repaired or the rough edge of the tendon can be trimmed until the tendon glides smoothly without triggering.

Teaching the Wide Awake Patient Throughout the Flexor Tendon Repair Operation

The surgeon gets the entire duration of the surgery for uninterrupted patient education time. The surgeon can teach the unsedated, tourniquet-free patient by showing how the repair can rupture or get stuck in the pulleys if strict adherence to the postoperative therapy regimen is not followed. He or she can talk to the patient about the postoperative regime, about keeping he hand elevated, about return to work, and so on. The patient has a clear visual image of the pulleys and the tendon as he or she has seen them; they are not just a theoretical anatomical structure.

In the author's practice, the hand therapist attends part of most of the flexor tendon repairs. The therapist and the surgeon see and discuss the state of the pulleys, the vincular blood supply, the quality of the repair, and its ability to glide. The surgeon and the therapist decide together what the optimal postoperative regime should be and discuss it with the patient. The patient gets to practice the postoperative hand exercise movements in a totally pain-free state during the surgery before the skin is closed. The glide of the tendons with the different angles of finger and hand position and movement can be examined and adjusted by the surgeon and the therapist.

The surgeon, the therapist, and the patient can see the attainable range of motion result right there during the surgery before the skin is closed. The patient knows what can be achieved when he or she gets through the pain and the therapy if he or she works at it. The patient has seen it with his or her own eyes in a pain-free, drug-free state.

The wide-awake patient gets the chance to establish a good rapport with both the hand therapist and surgeon during the surgery for better team communication and cooperation after the surgery. All three individuals can build a good team rapport during the surgery.

The pain-free patient sits up at the end of the case and helps the therapist apply the splint, with further explanations on splinting given to him or her in a comfortable cooperative state. There is no violent fist clenching behavior, which sometimes occurs on awakening at the end of a general anesthetic. There are also no nausea and vomiting, which can occur in up to one-third of general anesthetic patients, and there is no hospital admission required. Provided it is performed in a sterile environment, the patient's surgery need not be performed in the main operating room.

Postoperative Hand Therapy in Flexor Tendon Injuries in Zone 2

In children and uncooperative patients, these flexor tendon repairs are immobilized for 3 weeks followed by directed hand therapy as much as possible. With cooperative patients, early protected movement is initiated.

If there is no movement after the surgery, these tendons and their repair tend to get stuck in the flexor tendon sheath with scar. If there is too much movement, the repair risks rupturing. The tendon repair is roughly at its weakest point about 10 days after surgery. There is not a lot of point in moving the repair in the first 2 days after surgery, because collagen (scar) formation does not really start in earnest until 3 days after the surgery. In addition, movement in the first 2 days

may generate more blood in the wound, which in turn will generate more scar.

The author's hand therapists apply a static splint after surgery. This keeps the wrist in about 30° of flexion, MP joints at about 90° of flexion, and the IPs fully extended. On day 3 after surgery, active extension and passive flexion are begun in the splint. When the tendons are cleanly cut and vincular blood supply is good with a good 4-strand repair, we sometimes begin active flexion of the IP joints in the splint as well. The decision to start early active movement is often altered by what is seen intraoperatively when the patient takes his or her finger through a full range of motion.

At 3 weeks after the repair, we begin to allow more active flexion of the repair but continue with protective splinting for another 2 weeks. If the active movement is very good at this point, we will become more conservative and protectively splint 2–3 weeks longer. Excellent early active movement will often be followed by rupture.

If the repair is ruptured in the postoperative period, exploration and redo repair are initiated as soon as possible.

Flexor Tendon Injuries in the Hand and Forearm

The wide-awake approach is equally useful for multiple tendon injuries with wrist and distal forearm lacerations. The wide-awake patient can be instructed to flex the index finger, and the proximal index tendon stumps will move more than the others to help in their identification. The same patient education and other advantages of the wide-awake approach apply here.

The forearm, wrist, and hand can be liberally tumesced with lidocaine 1% with epinephrine 1:1,00,000 wherever incisions and exploration will be required. If the area is large and more than 50 cc of local anesthetic fluid will be required, the lidocaine can be diluted to 0.5% with 1:2,00,000 epinephrine, which will allow 100 cc of volume in a 70-kg man or even to 0.75% lidocaine with 1:4,00,000 epinephrine (still very effective), which will allow 200 cc of fluid if a very large area needs infiltration. As long as the local anesthetic is present in the areas to be dissected, epinephrine hemostasis will be effective. We feel that larger volumes of local anesthetic are more important than higher concentrations. As in the hand and fingers, the local anesthetic is injected proximally first and then distally after the proximal anesthesia has taken effect.

Flexor tendon injuries in the hand and forearm do not have the problem of being stuck in the flexor tendon sheath. Early protected movement is therefore less critical in these injuries. The hand is immobilized for 3 weeks and then followed up with hand therapists in hand clinics.

Extensor Tendon Injuries

Mallet Fingers

Fracture-free Closed Mallet Avulsion Injuries

Extensor tendon injuries that create an active extensor loss of the DIP joint result in a mallet finger deformity. It should be stated that mallet fingers do not constitute a major functional loss for many patients. However, function can be improved with treatment in most patients.

Closed injuries without a fracture in which the extensor tendon is simply avulsed from the distal phalanx are best treated with 8 weeks of splinting of the DIP joint in full extension. We tell our patients that they must not even once in the next 8 weeks flex the DIP joint on the pain of starting their 8 weeks of splinting over. Perhaps more importantly, the third month of treatment should include DIP extension splinting every night and during the day when forceful flexion may be expected. The return of flexion of the DIP joint in this month should only be gradual, with careful daily inspection that extension loss is not occurring. If significant extensor loss occurs, a return to full-time extension splinting should be considered.

The author informs his patients that a good result (0 to −10° of extensor lag) can be expected in 90% of patients if they chose to follow the splinting regime over their mallet finger deformity.

Fracture Mallet Injuries

The extensor tendon will frequently avulse a bone fragment on the dorsal intra-articular aspect of the distal phalanx. This is why all mallet fingers should be x-rayed. It is not the percentage of the joint surface that matters in determining the type of treatment of fracture mallets. What is important is whether or not the joint is congruous (joints surfaces parallel) after the mallet splint is applied and the finger re-x-rayed. If the joint surfaces have

become congruous on x-ray with the splint, the finger is treated conservatively with splinting as described in the section Fracture-Free Closed Mallet Avulsion Injuries (see Figure 38.7). These will often give a better result than nonfracture mallet injuries as bone-to-bone healing is often better than tendon-to-bone healing with these injuries. If the joint surfaces are still subluxed (noncongruous) on x-ray after splinting, a transarticular K wire is used in a closed percutaneous fashion, introduced from the finger tip into the middle phalanx to reduce the subluxed distal phalanx onto the middle phalanx (see Figure 38.8). The author prefers the closed approach as the open approach has generated more stiffness and pain in his practice.

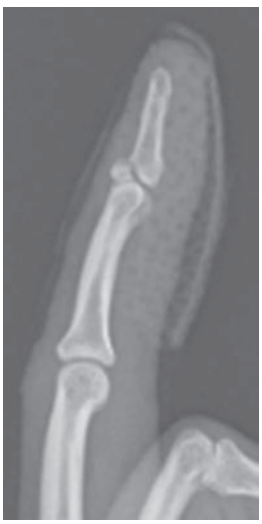

Figure 38.7. In this fracture mallet x-rayed with a mallet finger splint applied, the joint surfaces are seen to be congruent (parallel) in spite of the large fragment size. Only splinting was required in this patient who got an excellent result.

Boutonniere Injuries

Closed Acute Boutonniere Injuries

When the central slip of the extensor tendon avulses off the dorsal proximal lip of the middle phalanx, the lateral bands migrate volar to the axis of the proximal interphalangeal (PIP) joint and become flexors of the PIP joint and hyperextensors of the DIP joint.

If the injury is acute and the PIP joint is easily passively brought into full extension, a boutonniere splint is applied. These splints allow flexion of the MP and DIP joints but keep the PIP joint in extension. We have the patients wear the splint for a full 8 weeks. We tell our patients that they must not even once in the next 8 weeks flex the PIP joint on the pain of starting their 8 weeks of splinting over.

Perhaps more importantly, the third month of treatment should include PIP extension splinting

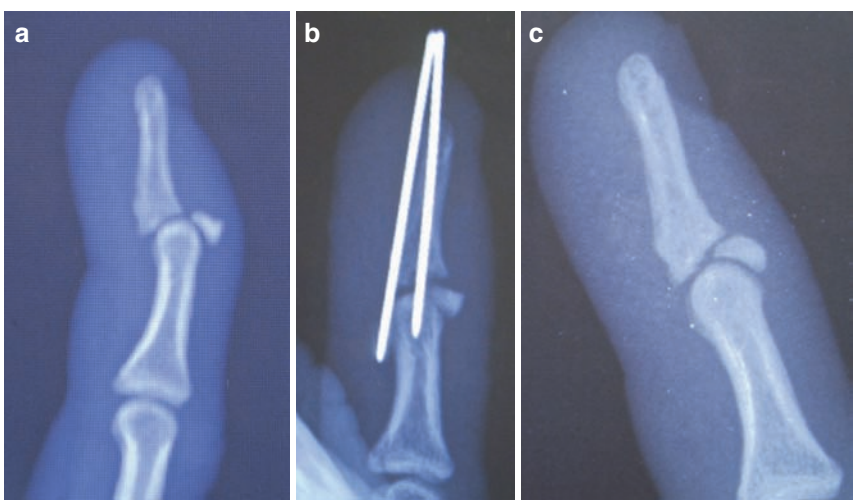

Figure 38.8. (a) Subluxation of the distal phalanx is seen in this 4-week-old injury. (**b**) Using a percutaneous closed approach, the distal phalanx subluxation is reduced. (**c**) The final result at 6 months postoperatively showing restoration of the congruity of the joint, with an excellent range of pain-free motion.

every night as well as during the day when forceful flexion may be expected. The return of flexion of the PIP joint in this month should only be gradual, with careful daily inspection that extension loss is not occurring. If significant extensor loss occurs, a return to full-time PIP extension splinting should be considered.

Closed Chronic Boutonniere Injuries

If the injury is chronic and the PIP joint cannot be passively brought into full extension, and if the PIP joint has a soft end feel, serial casting is begun. The therapist stretches the PIP into as much extension as possible, and then casts it there repeatedly until the PIP is out to full extension. We begin boutonniere splinting only when the DIP is able to fully actively flex. When this milestone is reached, we know that the lateral bands have now relocated themselves dorsal to the axis of the PIP joint, and 8 full weeks of splinting as described in the acute boutonniere section above is begun.

Surgery has been described for closed boutonniere lesions. In the author's practice, surgery has generally not been successful, and splinting has been the mainstay of treatment.

Open Lacerated Extensor Tendons over the Fingers

Sharp, clean lacerations are amenable to mattress sutures in the tendon with skin closure followed by splinting. Even with direct surgical extensor tendon repair, gaps can be present, because the thin extensor sheet on the finger dorsum does not hold a suture well. Fortunately, gaps are better tolerated in the finger extensors as the postoperative splinting allows the tendon to heal in spite of a gap, as occurs with closed mallet deformities. The thin extensor skin does not cover nonabsorbable braided sutures well. This type of suture frequently ends up being extruded with infection. It is for this reason, and the fact that the splinting is as important as the suture, that the author prefers absorbable sutures when the extensor tendon is repaired with buried sutures.

With jagged finger dorsum lacerations, the author often prefers to close the lacerated skin and extensor tendon with large (5–10 mm on each side) composite bites of skin and extensor tendon together with simple 3-0 or 4-0 nylon sutures tied on the outside of the skin (see Figure 38.9).

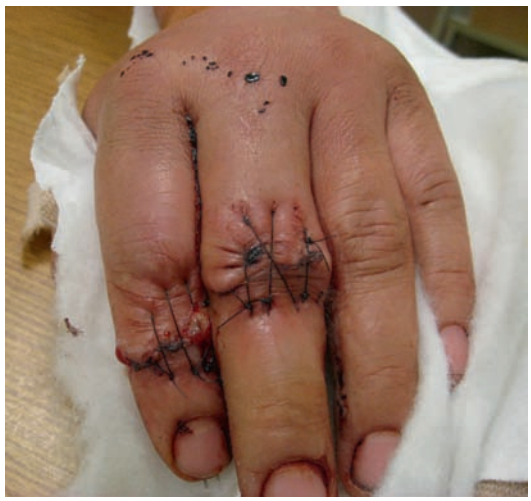

Figure 38.9. This patient suffered a table saw injury, which ripped out skin and at least 2 mm of the central slip and lateral bands of the long finger to create an acute boutonniere and at least 2 mm of the terminal extensor tendon to create an acute mallet. Only external sutures through the tendon and skin were tightened until the boutonniere and mallet fingers were reduced to full extension. The fingers were then wrapped in Coban and splinted with boutonniere and mallet splints.

The skin holds the suture much better than the extensor tendon, and tightening the suture bites of composite skin and tendon until the PIP or DIP joint with the extensor lag is fully extended indicates that the extensor tendon ends inside are closely enough approximated that they will heal with splinting.

After the sutures are in place, the finger is wrapped in Coban tape (stretchy self-adhesive tape), and the PIP or DIP joint that suffered the loss of extension is then splinted as described in the mallet or boutonniere sections above (see Figure 38.10). The large percutaneous/transtendinous nylon sutures can be removed at 2 weeks, but the splinting regime is continued as described above as though these were closed mallet or boutonniere deformities.

Extensor Tendon Injuries on the Dorsum of the Hand

The many intertendinous adhesions such as the juncturae tendinae on the dorsum of the hand prevent these tendons from retracting back up into the wrist or forearm. This is why the lacerated tendon ends are often not very far apart.

The wide-awake approach to surgery as described in the flexor tendon section is preferred. Local anesthesia (1% lidocaine with 1:1,00,000 epinephrine) is tumesced from proximal to distal into the area to be dissected, and local flaps are raised to clearly visualize the lacerated tendons.

If only the long finger extensor is lacerated, a sterile tongue depressor is placed under the long finger proximal phalanx and over the ring and index finger proximal phalanges at the time of the repair. This makes suturing easier, because it takes all of the tension off the repair. The wrist can be extended as well with a towel under the hand to further decrease the tension on the repair.

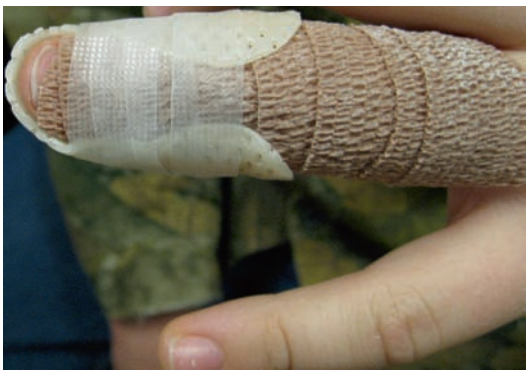

Figure 38.10. Mallet finger splint holding the DIP joint in as extended a position as possible so that the ruptured end of the extensor tendon is as close to the base of the distal phalanx as possible. The finger is wrapped in Coban self-adhesive tape to decrease interphalangeal joint swelling and permit better PIP joint movement.

If only the index extensor is lacerated, the sterile tongue depressor is placed under the proximal phalanges of the index and small fingers and over the proximal phalanges of the long and ring fingers. This takes the tension off the repair of the lacerated tendon, which is then easily repaired with mattress or modified Kessler grasping sutures until there is no gapping with active movement with the tongue depressor in place.

Relative Motion Splinting for Hand Dorsum Extensor Tendon Repairs

This important innovation in postoperative management has permitted patients with extensor tendon repairs to return to work right after surgery with a very functional splint.[1] This splint keeps the MP joint of the lacerated extensor tendon digit extended 30° more than the MP joints of the uninjured fingers (see Figure 38.11). An additional working wrist splint can be added to keep the wrist extended if active movement

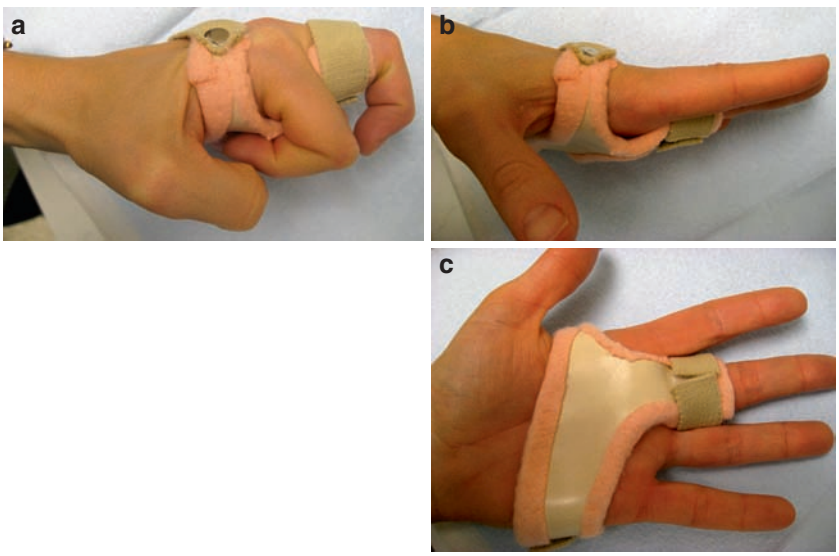

Figure 38.11. The differential gliding splint for hand extensor tendon injuries allows immediate return to work and continued movement of the injured tendon. The risk of rupture of the repaired tendon is very low, because the splint restricts the amount of gliding/excursion of this tendon. (**a**) Fingers fully flexed with long finger held back to keep the long finger repaired extensor tendon relatively tension free. (**b**) All fingers are allowed to extend in this working splint. (**c**) Palmar view of differential gliding splint which allows movement at 2–4 days post extensor tendon repair.

with the sterile tongue depressor at the time of surgery reveals that tendon repair site is under tension with unimpeded wrist flexion.

With the relative motion splint, the repaired extensor tendon will remain tension free and not dehisce in spite of the fact that free active IP flexion and limited active MP flexion are allowed in all four fingers. Surgeons who use this regime for the first time will quickly realize how functional these splints are when their patients come back from working at their manual labor occupations with a dirty splint and a very functional mobile hand. We have been using this splint for 10 years in all of our hand extensor injuries without a single dehiscence. The splint is worn for 4 weeks and then discontinued.

Wrist and Forearm Extensor Tendon Repairs

There are few to no intertendinous adhesions in this region to prevent the proximal extensor tendons from retracting up into the forearm. Large volumes of local anesthetic solution are injected wherever dissection will be required as described in the section on flexor tendon repair in the wrist and forearm.

With the wide-awake approach, proximal tendon identification is facilitated with the patient actively moving the injured proximal tendon stump on request. The same advantages of negating the inconveniences of general anesthesia and the main operating room facility to perform this surgery exist with the extensor tendons as they do with the flexor tendons. Relaxation of proximal extensor tendon stumps occurs reflexly when the patient is asked to actively flex the wrist or fingers as required.

These tendons are repaired in the same way as flexor tendons in the wrist or forearm with two core-grasping sutures followed by a month of tension-free splinting of the affected parts.

The Wide Awake Approach to Tenolysis

We are not aggressive with tenolysis in uncooperative patients, as it is certainly not a substitute for hard work in conjunction with hand therapy. The noncooperative patient is not likely to be any more successful after tenolysis than he or she was before the tenolysis. However, if there has been no change in progress after 3–6 months of good therapy in a cooperative patient, tenolysis is indicated and can be successful.

The wide-awake approach with tumesced lidocaine with epinephrine for hemostasis is preferred. The pain-free, tourniquet-free, unsedated patient aids the surgeon by actively pulling on the long flexor muscles to rupture adhesions at the surgeon's request throughout the procedure. The surgeon and the patient alternate in their cooperative effort to cut and rupture adhesions. The patient is also awake to see the liberated tendon result at the end of the procedure. He or she has seen the gains obtained with his or her own unsedated eyes and knows he or she can keep those gains if he or she works hard at the rehabilitation after the surgery. Patients who are asleep or regionally blocked cannot be as helpful as others, as their muscles may be paralyzed. They may also be uncooperative because of sedation or pain from a tourniquet.

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39

Benign and Malignant Hand Tumors

Leszek Romanowski, Piotr Czarnecki, and Maciej Bręborowicz

Summary

The hand is a region that can be specific for some tumors; on the other hand, some of them can localize there very rarely. It needs special attention because many lesions can be a result of hidden pathology not the neoplasmatic proliferation. Additionally, after treatment, the function of the hand should be preserved at maximum, both with precise and planned operation or appropriate reconstruction when wide resection is a need.

The chapter considers diagnostic and treatment issues and then describes most frequent and relevant tumors that the hand surgeon has to take into account and cope with.

Abbreviations

CT	Computerized tomography
DIP	Distal interphalangeal
GCT	Giant cell tumor
GCTTS	Giant cell tumors of the tendon sheath
LDH	Lactate dehydrogenase
MRI	Magnetic resonance imaging
MP	Metacarpophalangeal
PCBMN	Palmar cutaneous branch of the median nerve
PIP	Proximal interphalangeal
US	Ultrasonography

Introduction

The hand is a frequent localization of specific tumors. These can be classified into benign and malignant, which correspond to general principles. From a practical point of view, both can be divided into soft tissue and bone based on the originating compartment.

The described area can present lesions of a huge variety, from nodules and tumor-like changes to true neoplasms challenging the hand surgeon. Even simple changes can bring a lot of trouble to diagnosis and treatment, including a high risk of recurrence and rare but possible malignant transformation. Malignant tumors are uncommon, but if they occur, the consequences can be serious despite a seemingly small mass.

This chapter starts from the basics of clinical findings, including case history, examination, and imaging. Issues connected with biopsy taking and staging are discussed as an introduction to possible methods of treatment. Two main tumors groups, benign and malignant, are underlined subdividing into soft tissue and bone – most frequent tumors are described precisely and summarizing tables are added at the end of each subdivision.

Clinical Presentation

Symptoms can vary from slight change in shape and cosmetics to severe pain and loss of function depending on tumor type, aggressiveness,

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and time of onset. Patient very often complains of symptoms, which are the effects of the tumor mass growing and pressure on nerves or vessels.

Case history is of the biggest importance: details on precise time of onset, growth dynamics, and symptoms should be gathered. Patient should be asked about possible injury as a trigger of tumor or result (pathological fracture).

Examination should be careful and complex as some changes can be multifocal. The tumor mass is to be looked over for change in color, shape, and size and then palpated to check for local pain, temperature, localization to surrounding tissues (movable to skin and base), and consistency. Hand function must be tested, including range of motion, strength, sense, and blood circulation.

Imaging

In most cases, diagnostics must include imaging, which should be planned and adopted to the possible type of tumor and localization, considering the safety of the patient and the imaging cost (Figure 39.1).

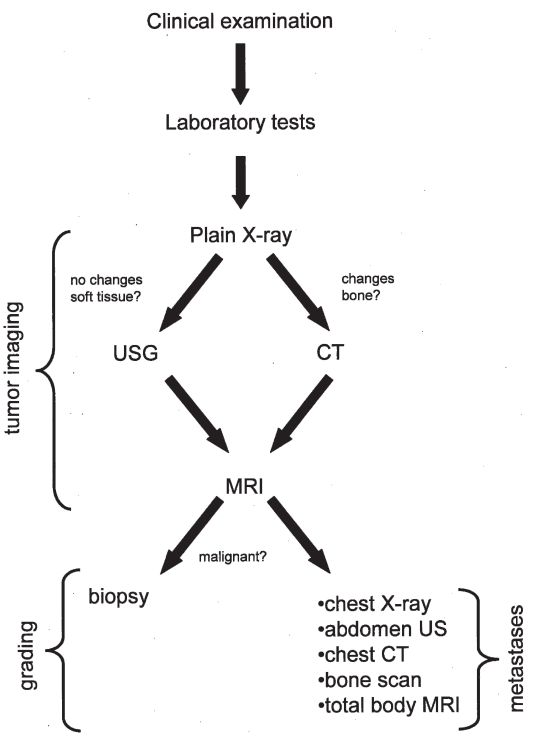

Figure 39.1. Diagnostic chain for hand tumors.

Plain X-Ray

Plain x-ray in appropriate projections is usually the first step in diagnosis of tumors. It should comprise the region within which the lesion is located. It is especially useful with bone evaluation.

While evaluating plain x-rays, one should pay attention to many details. They include location of the lesion within the bone and adjacent soft tissues. In some cases, there is more than one focus of disease. Radiological symptoms may suggest the characteristics of a lesion. Attention should be paid to margins of tumor, how they are defined, and whether there are any changes in cortical bone – such as new bone formation, destruction, and erosion. Soft tissues may present calcifications, and, in some cases, soft tissue invasion might be apparent. Tumor may show osteolytic or osteosclerotic features or both simultaneously. Periosteal reactions might be present in malignant bone lesions. They are described further in the chapter, together with characteristic tumors.[36,46,54]

MRI

Magnetic resonance imaging (MRI) is a method of choice for evaluation of soft tissues.[36,46] However, it is also useful with bone tumor diagnostics. It reveals involvement of bone, together with medullar cavity[42,60] and extent of tumor within bone. Uncalcified cartilage can be visualized.[23,48] The important feature of MRI is imaging the relationship of the lesion to surrounding structures such as neurovascular bundles. Some tumors show enhancement with the use of contrast.[46]

CT

Computerized tomography (CT) is also very useful in the diagnosis of tumors, both in the visualization of primary neoplasm and in the disclosure of theirs metastases. It allows the observation of the extent of the lesion inside and outside the bone.[54] It provides many bony structure details.[36] In addition, information about the relationship to surrounding structures is given, such as muscles, fascial planes, and neurovascular bundles.[54] CT scan is important in the diagnosis of metastases. CT scan of chest, abdomen, and pelvis is performed.[36,46]

Bone Scan

Bone scan is an additional method used in full diagnostics of tumors. With application of radiopharmaceuticals, it shows nonspecific reactive changes within bones. Therefore, it helps in recognition of metastases of the lesions.[36,46,54] Recently, triphase bone scan was performed to distinguish neoplasm lesions from inflammatory changes.

Ultrasound

Ultrasound is less precise than CT and MRI in neoplasm diagnostics; however, it is used to evaluate soft tissue lesions. It helps in the differentiation between solid tumors and fluid lesions.[54] In addition, in some centers, it is used to evaluate metastases within the abdomen. The advantages of this technique are the relatively low price and the high safety for the patient.

Biopsy

The biopsy is the last stage of diagnosis of the tumor. Its technique depends, apart from size and location of lesion, also on the experience of the surgeon and pathologist.[54] It should be performed by the same physician who will perform the operation, in the same center.[46]

There are different biopsy techniques. One of them is needle biopsy. It has no use in most hand tumors because of its limitations,[46,52] although in some patients it is performed. Details will be provided with descriptions of particular lesions.

The best technique is open biopsy. It plays a crucial role in complete tumor diagnostic and therapeutic procedures.[54] This procedure should be planned in detail as definite treatment.[46]

The bloodless field is widely used in hand surgery operations, and it is also accepted during open biopsy and following procedures. A pneumatic tourniquet can be applied, but Esmarck bandage is prohibited because it can cause tumor cell seeding.[54] It may be replaced with elevation of the extremity for a few minutes. During the procedure, evaluation of frozen sections should be obligatory to make sure that a proper sample was taken.[30] There are two possibilities, namely incisional and excisional biopsy.[36,54] The former is mostly appropriate for bone and soft tissue evaluation.

If the tumor is large and there are serious concerns about its malignancy, incisional biopsy is the procedure of choice.[36,54] With the technique of incisional biopsy, the tumor is approached through a longitudinal incision through muscle.[54] Transverse and Brunner's skin incisions are to be avoided.[46] Procedure should be gentle, with avoidance of retraction and wide spreading and all manipulations that increase the risk of surrounding tissue neoplasm cell contamination. It is good if the sample is obtained from the boundary zone including healthy tissue, pseudocapsule, and tumor material. The procedure must be planned in detail, because if the lesion turns out to be malignant, the biopsy tract is contaminated. Therefore, it is to be completely and thoroughly excised en block during definite surgery.[36] Moreover, the biopsy technique cannot compromise future limb-sparing operations, in the end leading to amputation. Hemostasis is very important to avoid hematoma. It can cause the disease to spread to the surrounding tissues and structures beyond the operated site.[54]

In case of little suspicion and a very small lesion, excisional biopsy may be performed. In addition, if the tumor is very small and presumably malignant, in some cases, it can be primarily removed with a wide margin of healthy tissues.[36] The principal rules of the excisional biopsy are the same as those of incisional biopsy. However, there are different indications as mentioned before. If the excised tumor occurs to be malignant and proper wide margins were not primarily obtained, re-excision might be obligatory[36] (Figure 39.2).

Staging

Two systems of tumor classification are mentioned in this chapter. The first was introduced by Enneking, and the second, by the American Joint Committee on Cancer.[25,46]

The former is based on histologic grade, extent of the lesion, and tumor site. The first criterion comprises three possibilities:

- G0 – benign lesion (rare malignant transformation)
- G1 – low grade (rare metastatic spread and local recurrence, usually low mitotic index)
- G2 – high grade (high malignancy with metastases dissemination)[54]

Grading is given by the surgeon and pathologist, based on clinical history, result of examination, imaging characteristics, and histologic appearance.[23]

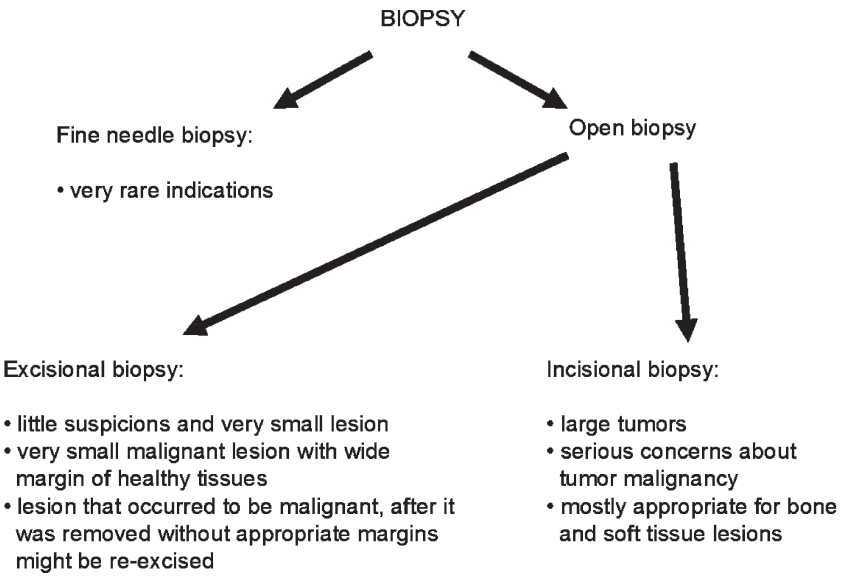

Figure 39.2. Biopsy summary.

Location of tumor can be either intracompartmental (T1) or extracompartmental (T2).[54] Finally, the extent of disease describes lack of metastases (M0) or their presence (M1).[25,46] Enneking divided tumors into three surgical stages (the first two types are neoplasms without metastases):

- Type I – low-grade lesions (G1) located either intracompartmentally (type IA) or extracompartmentally (type IB).
- Type II – tumors of high grade (G2), depending on the relationship to compartments, are divided as in the previous type to type IIA and type IIB
- Type III – any regional or distant disease spread, both G1 and G2, with no consideration of compartment location, belongs to type III.[25,46]

The latter mentioned system of classification is used for evaluation of both bone and soft tissue sarcomas. For bone lesions, criteria include histologic grade, presence of metastases, involvement of lymph nodes, and size of tumor. For soft tissue sarcomas, criteria are the same except for lymph node involvement, which is replaced with tumor depth.[25]

Principles of Treatment

Mostly, treatment is considered to be surgical. In some cases, adjuvant therapy can be beneficial to the patient. The whole treatment process should be based on close cooperation between radiologists, oncologist, and surgeon to provide complex and up-to-date patient support.

Surgery

Because of its complex anatomy and existence of many important structures in a relatively small space, surgical procedures within the hand should be planned well and in detail.[23]

In case of malignant tumors, it is difficult to obtain wide enough healthy tissues margins (2–3 cm).[30] Therefore, in case of fingers, amputation or disarticulation might be considered.[46] If lesions appear more proximally, amputation of hand ray or rays might be the solution. If location is proximal and has no anatomic barriers to the spread of tumor, therapy consists of amputation; an example of such a site is the carpal tunnel.[46] Whenever possible, limb salvage procedures should be considered. In case of superficial lesions, if resection involved a vast superficial region, reconstructive surgery may be needed.[63]

Radiotherapy

Radiotherapy can be used preoperatively and postoperatively. However, not all tumors are radiosensitive. Postoperative radiotherapy is most widely used contemporarily. However, preoperative radiotherapy (neoadjuvant) can decrease size of the tumor, facilitating or enabling further

surgical procedure, but it causes complications in wound healing. Therefore, its use should be limited to patients in whom safe margins during resection cannot be obtained.[36,46]

Chemotherapy

In case of tumors sensitive to chemotherapy, it can be used both preoperatively and postoperatively.[36] It can be used with radiotherapy as neoadjuvant therapy. Reports of different schemes of chemotherapeuticals used in treatment of particular tumors have been published.[36,46]

Follow-Up

After definite treatment, patient should be controlled by physician for 3–5 years. For the first 2–3 years, the patient should be evaluated every 4 months with physical examination, x-ray, chest CT, and MRI of involved region. Later the patient should be followed every half a year for the next 2 years. After that period, yearly control evaluation is suggested, with the use of techniques mentioned before, individually planned for the patient. Depending on the type of lesion, diagnostics might be broadened with additional methods such as CT of the abdomen.[36,46,48]

Benign Tumors

Benign Soft Tissue Tumors

This chapter includes most frequent hand tumors. Some of them are not neoplasms in oncological meaning, but they produce a mass that should be handled with maximal care and interest until confirmation of benign diagnosis.

Ganglion Cyst

It is the most commonly seen tumor of the hand. Made of a capsule derived from joint space or tendon sheath, it includes a jelly-like fluid, which is derived from joint fluid but of slightly different composition. Many authors work on pathogenesis, suggesting carpal instability to be a reason of ganglia appearance with synovium coming out through a weak resisting joint capsule; some of them underline mucoid degeneration.[30] This region is most common for this tumor, especially the dorsal side; some other localizations are also possible: palmar side of a wrist, dorsal side of distal interphalangeal (DIP) joint (mucous cyst), palmar side of metacarpophalangeal (MP) joint, and extensor sheaths.

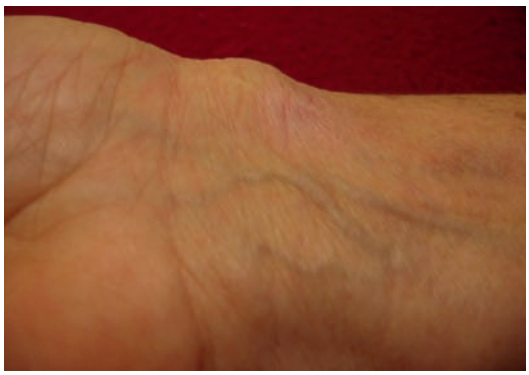

Figure 39.3. Ganglion cyst of the wrist: secondary excision after incomplete resection.

These tumors are mostly typical for females 20–40 years old. Patient presents with a tumor in one of the typical localizations, complaining of cosmetic shape change and pain (this can be a result of main pathology) (Figure 39.3). When ganglion arises in the neighborhood of the peripheral nerve, compression neuropathy can be reported (median or ulnar are mostly involved).[9]

Imaging with x-ray can be done for exclusion of more severe tumors; ultrasound is of the biggest value in diagnostics. Size of the tumor, characteristics, relationship to nerve and vessels, and origin can be described. Some authors suggest that illumination initially differentiates ganglion from solid soft tissue tumors.

Ganglion has a tendency to resolve spontaneously and for frequent recurrence if not treated. When symptoms are presented, a few treatment options should be considered: punction (aspiration with/without steroid injection), percutaneous rupture, or operative resection. The last is the most effective one, but it should be begun with pathogenesis investigation to find the possible reason of ganglion cyst. Resection can be simple, leaving the pedicle open or ligatured. A high rate of recurrence is reported when the resection is incomplete (up to 40%).[47]

Pigmented Villonodular Tenosynovitis (Giant Cell Tumors of the Tendon Sheath)

Pigmented villonodular tenosynovitis is a frequently seen soft tissue tumor in the hand (nodular

type), mostly on the palmar side at the level of the DIP joint, but other locations have also been described (diffuse type).[10] The most widely accepted cause of giant cell tumors of the tendon sheath (GCTTS) is a reactive or regenerative hyperplasia associated with an inflammatory process.[37]

Patient complaints on cosmetics deficit, some problems with sensation, and range-of-motion deficit due to large mass can be also expected.

Local aggressiveness can be excluded by plain x-ray (bony erosion is possible). There was no malignant transformation reported.

Treatment simply includes mass resection with joint synovectomy if this is a point of origin. These should be carefully performed because of the high rate of recurrence (7–29% depending on author).[1,29]

Resected tumor is brown-yellow in color, has a lobular structure, and is sometimes encapsulated, which may influence the treatment result; benign type should be confirmed using microscopy so that giant cells are found[1] (Figure 39.4).

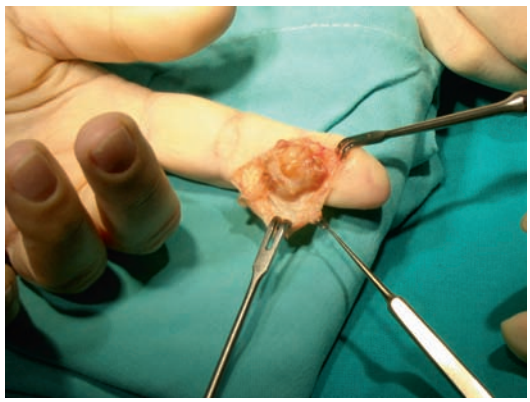

Figure 39.4. Pigmented villonodular tenosynovitis tumor exposed at DIP joint.

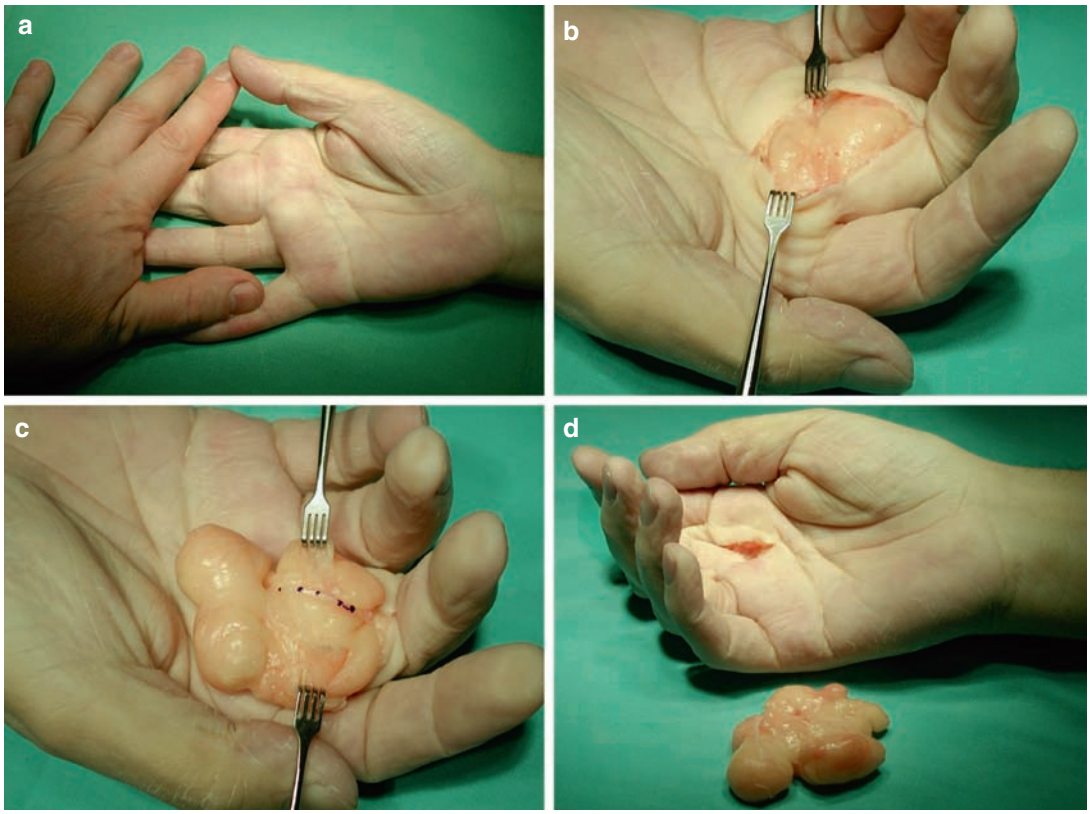

Figure 39.5. Lipoma. (**a**) Clinical presentation. (**b**) Intraoperative exposition. (**c**) Nerve localized (marked with *black color*). (**d**) Tumor after excision.

Lipoma

When a whole human body is considered, this tumor is the most common benign soft tissue mass. Hand is one of the rarest localizations, which can be subcutaneous or intramuscular. Patient reports slow mass growth; no other symptoms can be noticed, but as in other benign tumors, local infiltration can result with inconvenient symptoms connected with nerve and vessel compression or muscle belly impairment. Even in the hand, underlying mass can grow to improportionally large dimensions – 20-cm lipomas have been described in the distal forearm region.[19] Tumors of this size (more that 5 cm) are treated as locally malignant, classified as well-differentiated liposarcomas, and need special care in diagnostics and treatment.

In imaging, ultrasound examination is of immense help; in specific cases, MRI can also be considered.

Treatment equals local resection, which sometimes can be difficult because of poor tumor borders that lead to incomplete resection, resulting in recurrence (Figure 39.5).

Fibroma

Fibroma is a benign tumor, often solitary and nonsymptomatic. In some situations, symptoms come from specific localization (e.g. fibroma of the tendon sheath; Figure 39.6).

It can be diagnosed by ultrasound and then excised.

Glomus Tumor

Hand is a favorite localization of this mass – 1–5% of all benign tumors. It derives from commonly found receptors regulating blood pressure and temperature.[62] The fingertip, especially close to the nail bed, is a region of the biggest concentration of these structures. Partial hypoplasia of the receptor leads to the formation of a glomus tumor, which troubles the patient with a specific triad: cold intolerance and rapid and strong pain in a precise localization.

The tumor is hard to find, and very often, there is no change in the shape of the fingertip, which delays diagnosis. Typically, tumors 5 mm in diameter and blue in color can bee seen through the nail.

Diagnosis is set based on clinical examination: pain testing with or without ischemia (symptoms disappear), and cold exposition can also be checked. Imaging can be additionally performed with high-resolution ultrasonography (US) or MRI.[61]

Resection can be done with or without nail removal. Former gives wide operating field, and the latter is more cosmetic (through fingertip approach) but can result in recurrence due to poor tumor visualization[34] (Figure 39.7).

Neuroma

It is a tumor-like mass that always forms as a result of nerve injury, at the site of lesion or in the place of reconstruction. The most characteristic

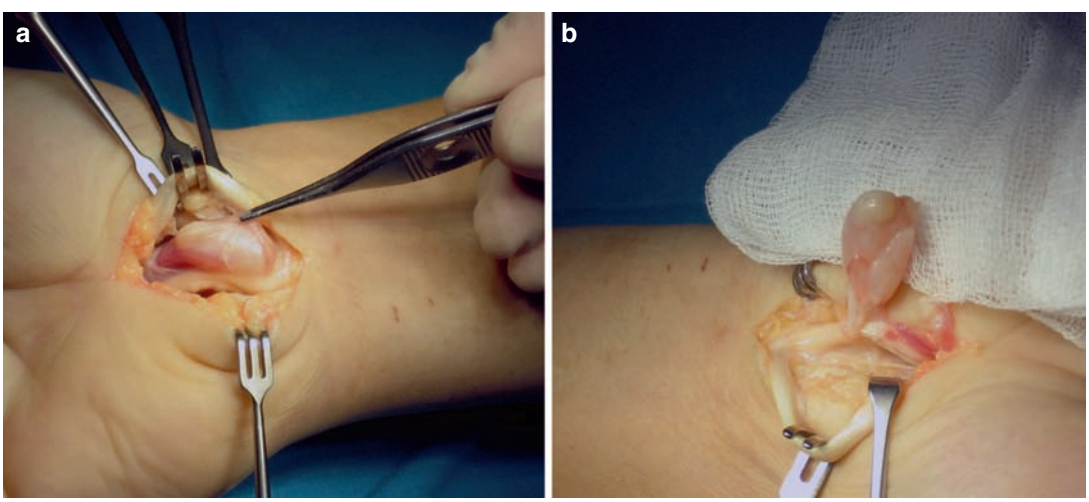

Figure 39.6. Fibroma of tendon sheath, causing triggering of fourth finger at the carpal tunnel.

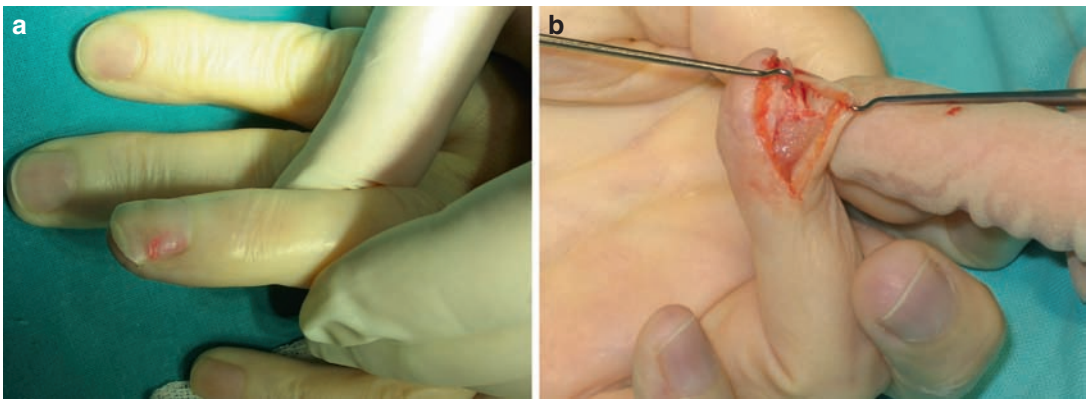

Figure 39.7. Glomus tumor. (**a**) Clinical presentation. (**b**) Resection technique.

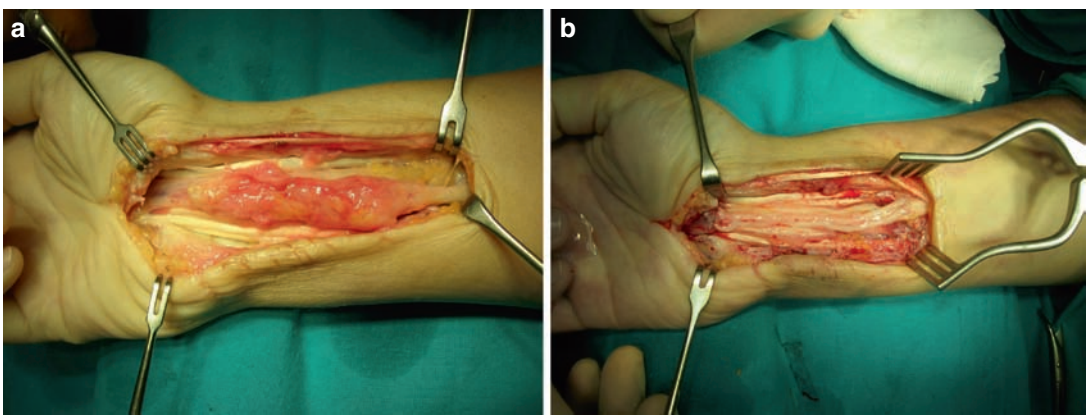

Figure 39.8. Large neuroma of median nerve. (**a**) Intraoperative view. (**b**) Resection with grafting.

and troublesome ones are painful neuroma of improperly formed finger stumps, which give precise, high pain, significantly decreasing finger function. Some of the neuromas can be a complication of surgical treatment of carpal tunnel syndrome when there is an injury to the cutaneous branch (palmar cutaneous branch of the median nerve [PCBMN]).[40]

To confirm the diagnosis, local anesthesia with lidocaine can be used – it relieves the pain completely in this pathology.

Neuroma must be removed, and the stump has to be buried in deeper layers of tissue to prevent its contact with skin or scar. When reconstruction neuromas are considered, patient complains of tenderness of the skin that covers the reconstruction site – nerve lies just below. Separational plasty can be performed with a local fatty flap, and if there are no regeneration signs, resection and another reconstruction should be considered (Figure 39.8). For the PCBMN neuroma, the technique of stripping has been described.[40]

Granuloma

A tumor-like mass can also form as a reaction to a previously injured or stained region. The tumor grows slowly with only a cosmetic defect. The symptoms may occur as a result of surrounding tissue pressure or limitation in the range of motion by the tumor mass.

X-rays are taken to check whether the bone is of tumor origin, because some of the granulomas

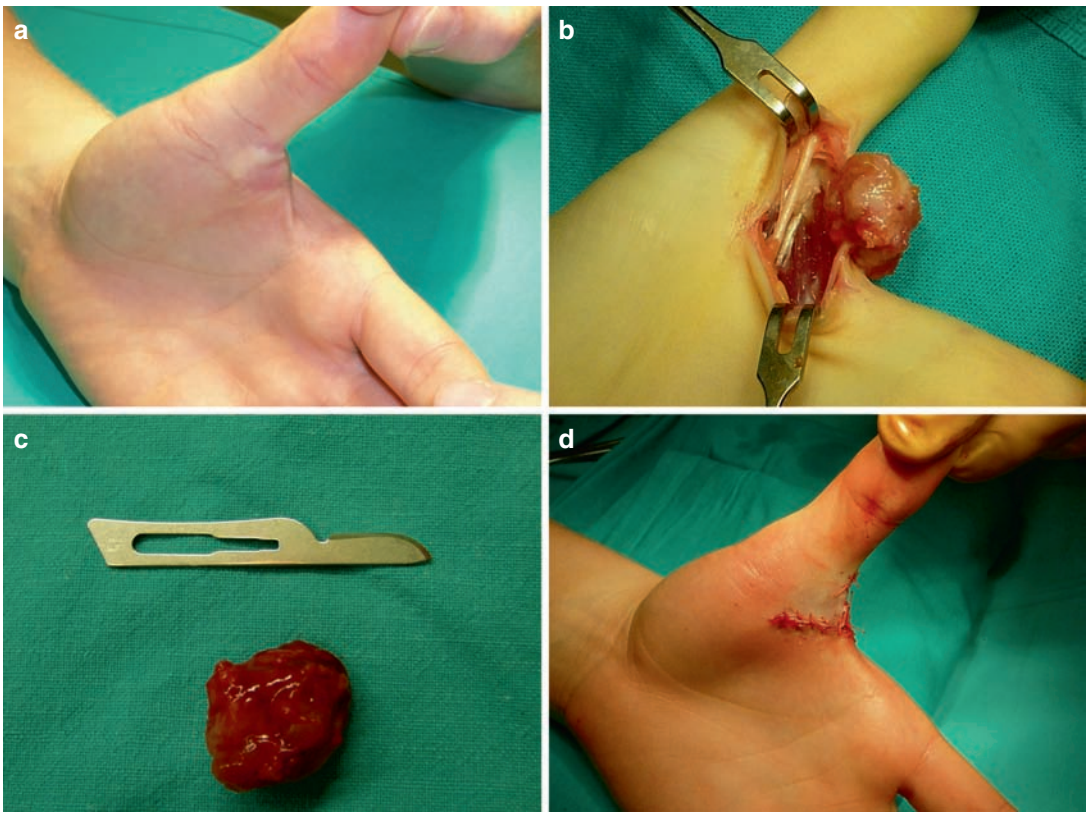

Figure 39.9. Granuloma. (**a**) Clinical presentation. (**b**) Intraoperative view with vessels inside the tumor. (**c**) Size after excision. (**d**) Skin closure.

can derive from the bony structure (giant cell reparative granuloma). US can determine the size and the location of the tumor.

Prognosis is very good – there are no reports of malignant transformation. Treatment is simple excision, but care should be taken due to a recurrence rate of 10–15% (Figure 39.9).

Hemangioma

When occurring in the hand, it is not seen after birth contrary to other locations. It appears later, looks like a blue or reddish mass, sometimes causes pain, and rarely bleeds (Figure 39.10). It can disappear until 7 years of age; if not, there should be diagnostics and treatment performed, especially when there are symptoms. This vascular anomaly is a result of benign growths of endothelial cells.

X-rays can show calcifications if the case history is long. The best technique is to evaluate blood flow in Doppler ultrasound, which is highly effective in confirming the diagnosis.

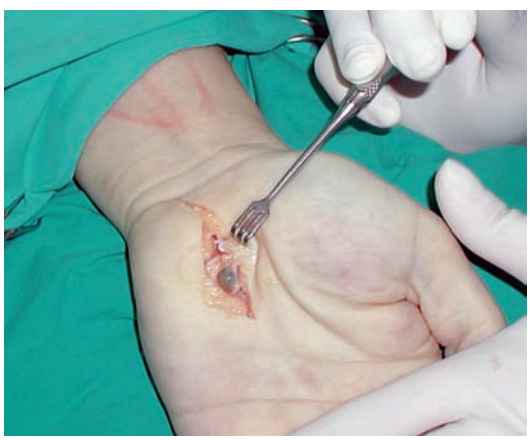

Figure 39.10. Hemangioma.

Resection of tumor should be performed precisely, and the pedicle should be ligatured. There are some options for laser ablation.[2,30]

Aggressive Fibromatosis (Desmoid Tumor)

Aggressive fibromatosis tumors are very rare in the hand and are mostly found in children and juveniles. They arise from aponeurotic or fascia; so they mainly affect the palm of the hand or the dorsal wrist. They are locally malignant tumors, and without a capsule, they grow aggressively through compartments surrounding tendons, nerves, and vessels.

Patients report a painless mass that mainly affects hand function (limitation of dorsal or palmar flexion).

Precise imaging can be done only in MRI – it should be used for setting of surgery margins and postoperative monitoring.

Surgery is limited due to the high risk of recurrence (up to 50%), which is a result of inability of complete removal.[13] Adjuvant radiotherapy should be considered in some cases, but it is not a choice for hand tumors[64] (Figure 39.11).

A summary of benign soft tissue tumors is presented in Table 39.1.

Benign Bone Tumors

Enchondroma

Enchondroma is the most common benign bone tumor of the hand (up to 90%) that affects patients between 10 and 40 years of age.[8]

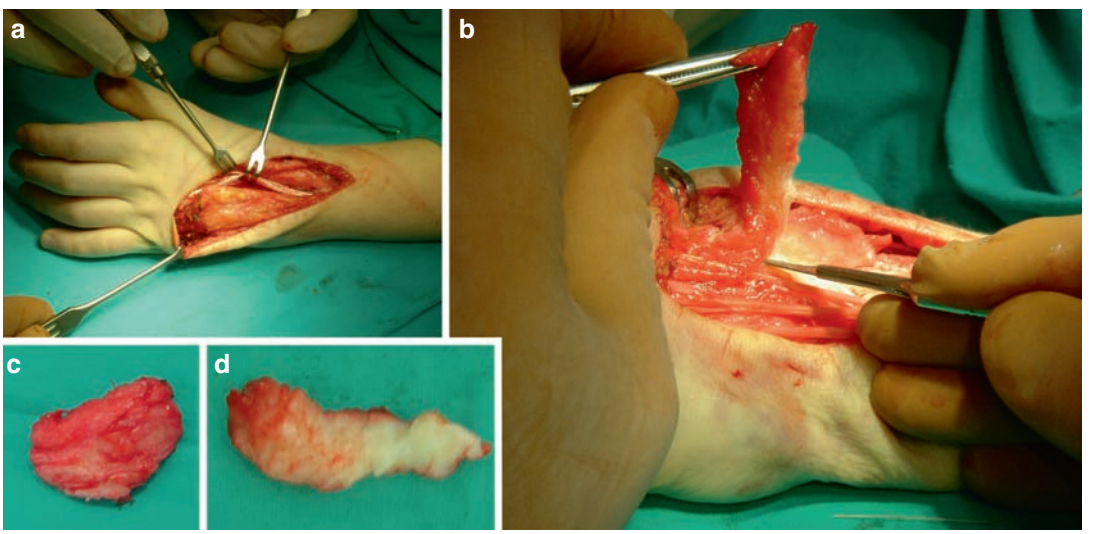

Figure 39.11. Fibromatosis aggressive in a 10-year-old boy. (**a**) Tendon infiltration. (**b**) Tumor excision. (**c, d**) Tumor mass.

Table 39.1. Summary of benign soft tissue tumors.

Tumor	Incidence (%)	Age (years)	Site	Classic treatment	Remarks
Ganglion	35	40–50	Dorsal wrist	Aspiration, excision	Possible steroid injection
TenosonovitisTenosynovitis nodularis	14	30–50	DIP joint, index and long finger	Excision	High rate of recurrence
Lipoma	13	40–60	Thenar	Excision	Possible multiple tumors
Fibroma	6	40–60	Fingers	Excision	Possible multiple tumors
Glomus tumor	4	30–40	Nail	Excision	Characteristic symptoms
Neuroma	1	30–50	Finger stumps	Excision, excision with grafting	Lidocaine test for diagnostics

Patients present with no symptoms until pathological fracture as a result of mild injury. In addition, a change in shape can be a signal of disease. In x-ray diagnostics, a well-defined bone lesion is seen. Some additional imaging such as CT or MRI is also possible but mostly unnecessary.

The distribution in the hand is specific: the ulnar side is mostly affected (31%); it also affects proximal phalanges (45%), middle and metacarpals (every 20%), and distal phalanges (14%).[28] Localization in carpal bones is extremely rare, and enchondroma is also found in the distal forearm.[24,55]

Malignant transformation is rare, but the risk increases to 30% in multiple tumors (Ollier disease).[26] The most serious combination is Maffuci syndrome where additionally hemangioma is present.

Treatment includes curettage with bone grafting; both autologous grafts and allografts can be used, especially for large or multiple lesions (Figure 39.12). Some authors consider filling the gap as unnecessary and not influencing the final result.[32]

New techniques employ a small invasive endoscopic control for exact curettage, filling with bone-like substance as prevention of recurrence in incomplete removal.[59]

Osteochondroma (Exostosis)

Although humerus is the most common localization, osteochondromas can also be found in the hand (mostly proximal phalanx of the thumb). They occur in the second and third decade.[45]

Symptoms are rare (pain and function impairment), and patients complain of cosmetic changes.

Treatment with excision is an effective method; intraoperatively, it combines bone and cartilage, so often it is bigger than in x-ray diagnostics. It should be removed from the base.

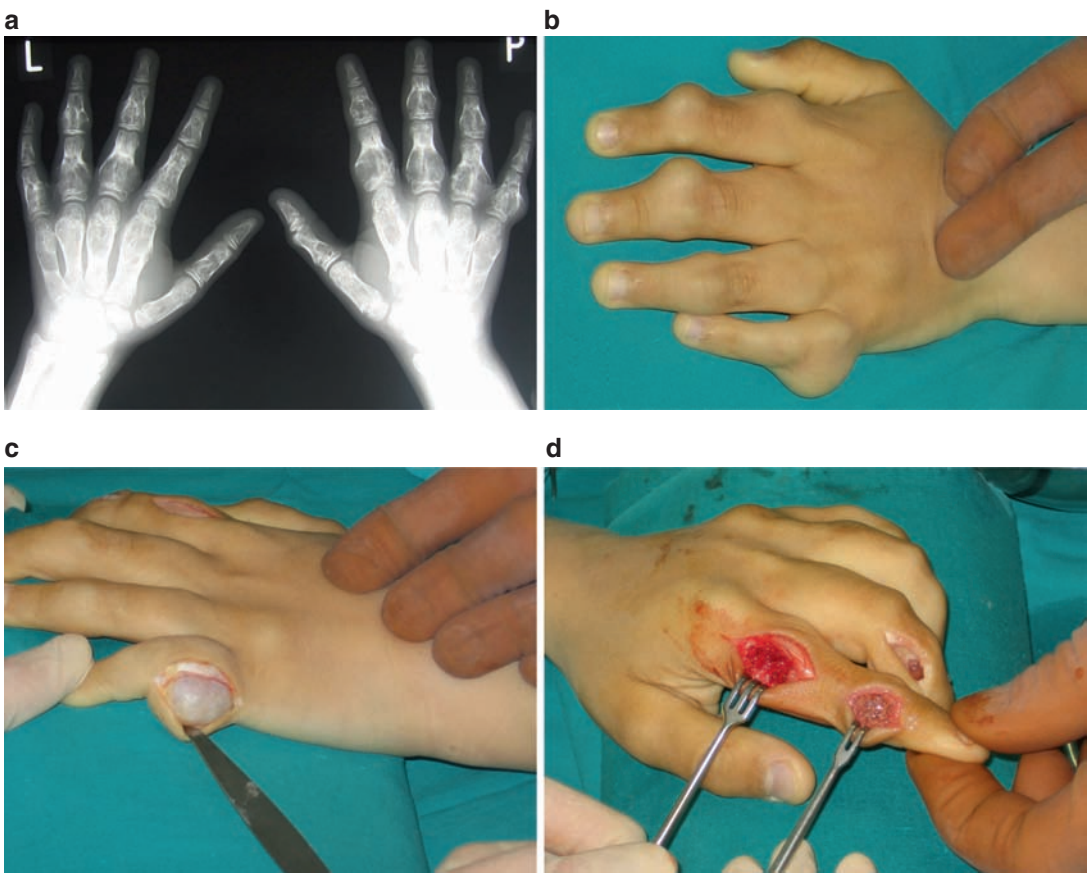

Figure 39.12. Multiple enchondroma. (**a**) X-ray of both hands. (**b**) Clinical presentation. (**c**) Intraoperative view. (**d**) Tumors curettaged and filled with autologous bone graft.

Osteoid Osteoma

Osteoid osteoma occurs between 10 and 30 years of age, more often in men (male to female ratio, 2:1). Patient complains of pain, also occurring at night, and change in shape; painless osteomas have also been described.[7,56] Although it localizes in lower limbs, hand is one of the more common sites (10% of all benign bone tumors).[43]

X-rays show a limited tumor with sclerotic margins (nidus; Figure 39.13). Some of the osteoid osteomas can be difficult to diagnose without CT or bone scan.

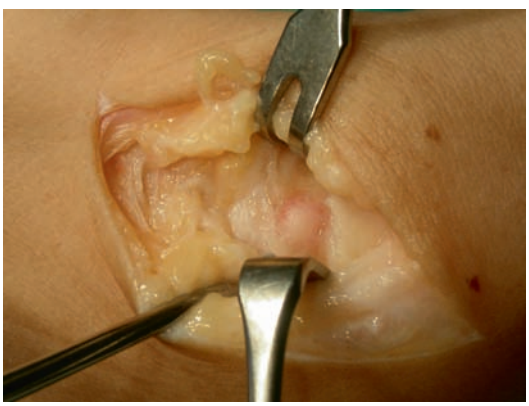

Figure 39.13. Osteoid osteoma of scaphoid; nidus in the center.

Tumor is excised en bloc or can be removed by curettage with bone grafting. Less invasive methods are also described – tumor resection by percutaneous thermal ablation.[6,20]

Aneurysmal Cyst

An aneurysmal cyst is a rare benign tumor that occurs in the hand in 3% primary benign bone tumor cases. Pathogenesis is unclear; some vascular malformations during bone formation are suggested to be an origin of this lesion. Patients do not complain, and similar to enchondroma, mild pain or pathologic fracture can be the only symptoms. An Aneurysmal cyst localizes in distal ends but usually does not cross the growth plate. Metacarpals are most often affected, followed by proximal phalanges; tumors in carpal bones are rare.[53]

Generally, there is a tendency for recurrence after surgery, but there were also spontaneous remissions described.[15]

Imaging starts with x-ray, but it should be extended with CT or MRI.

Treatment depends on aggressiveness – it usually starts with curettage and bone grafting; in more aggressive tumors resection with bone reconstruction is done (Figure 39.14). Due to frequent recurrence (60%), cryosurgery is performed in addition to curettage; filling with bone cement can also decrease the risk of recurrence.[4]

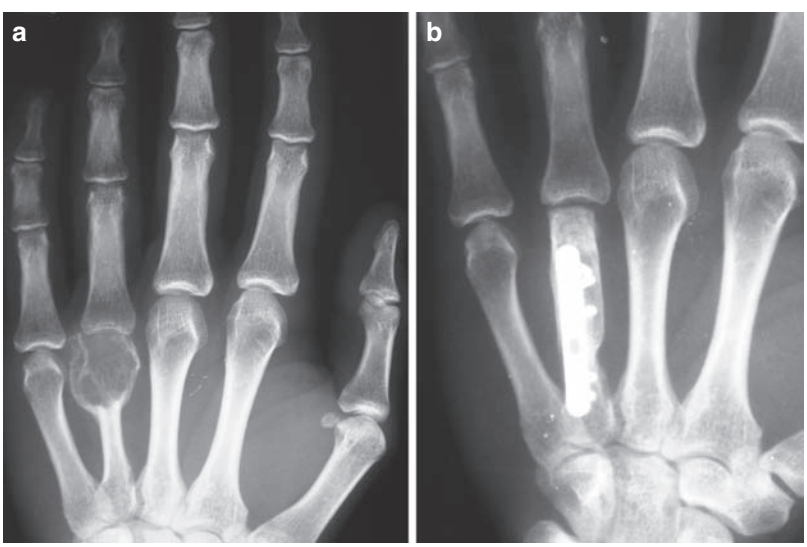

Figure 39.14. Aneurysmal cyst of fourth metacarpal. (**a**) Preoperative. (**b**) Postoperative. Resection with bone grafting and plate fixation.

Giant Cell Tumor

The incidence of giant cell tumor is similar to the aneurismal cyst (2–3%), and case history can be more serious with aggressive growth, malignant transformations, and metastases (5%) leading to death[57] It mostly occurs in the fourth decade of life, and the recurrence risk is high despite treatment (80%).[3,5]

X-ray findings are different from those in the aneurysmal cyst; the inside of the tumor is not filled, and margins are not sharp, with possible cortex erosion and expansion crossing the growth plate.

Before surgical removal, planned biopsy should be taken. Confirmed diagnosis leads to tumor resection or ray amputation. Less radical attempts including curettage, cryosurgery, and bone grafting increase the risk of recurrence (Figure 39.15).

A summary of benign bone tumors is presented in Table 39.2; the most common benign tumors localizations are summarized in Figure 39.16.

Malignant Tumors

Malignant Soft Tissue Tumors

Synovial Sarcoma

Synovial sarcomas are tumors that originate from the synovium or mesenchymal tumors, indicating synovial direction of differentiation.[11] They are one of the most frequent soft tissue malignant neoplasms of the hand and wrist.[36,39,56]

Clinically, a slowly growing nodule may appear, although in some cases the tumor grows rapidly.[11,63] The most popular location is the

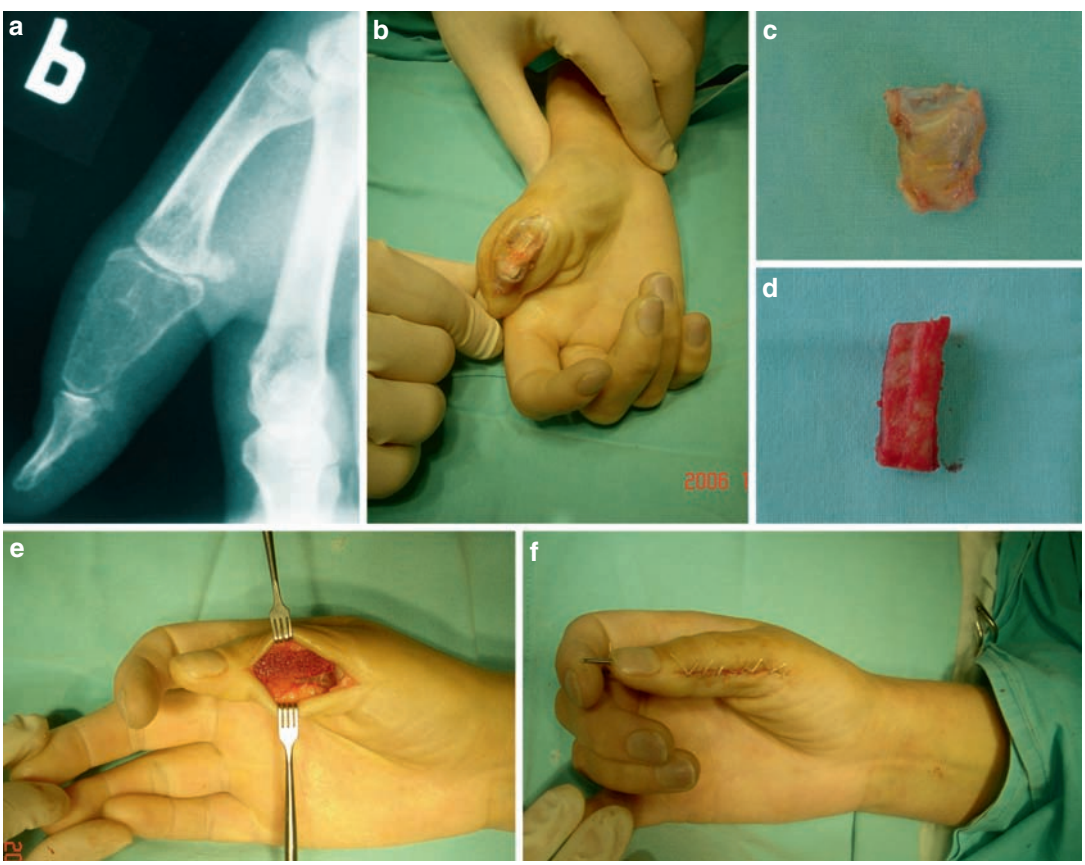

Figure 39.15. Giant cell tumor of thumb proximal phalanx. (**a**) X-ray. (**b**) Intraoperative view. (**c**) Excised tumor. (**d**) Bone graft. (**e**) Reconstructed phalanx. (**f**) Final appearance.

Table 39.2. Summary of benign bone tumors.

Tumor	Incidence (%)	Age (years)	Site	Classic treatment	Possible treatment
Chondroma	60–90	10–40	Proximal phalanx	Curettage with bone graft	No bone graft, arthroscopic assistance
Osteoid osteoma	4–7	10–30	Phalanges	Excision or curettage	Thermal ablation, cryosurgery
Exostosis	3–7	20–30	Proximal phalanx	Resection	None
Giant cell tumor (GCT)	6–13	40–50	Metacarpals	Resection, ray amputation	Curettage, cryosurgery, bone grafting
Aneurysmal cyst	3	20–40	Metacarpals	Curettage with bone graft	Cryosurgery, bone cement

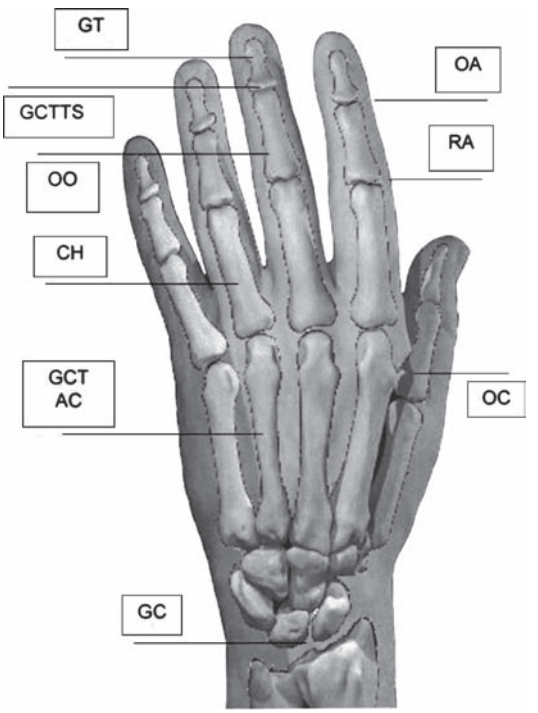

Figure 39.16. Most frequent localizations of benign tumors. GT: glomus tumor; GCTTS: giant cell tumor of tendon sheath; OO: osteoid osteoma; CH: chondroma; GCT: giant cell tumor; AC: aneurysmal cyst; GC: ganglion cyst; OA: osteoarthritis; RA: rheumatoid arthritis; OC: osteochondroma.

wrist, and sometimes they appear in the palmar and dorsal surface of a hand[36,44,56] and in the neighborhood of bursae and joints.[36,56] Locations within the medial nerve were also reported.[17]

Diagnostic methods include plain x-ray[11,56] and magnetic resonance imaging (MRI), also with contrast.[63]

For histologic evaluation, material might be obtained with a biopsy.[63] Intraoperative view is diverse – tumor can be hard and fibrous or soft and gelatinous, and most frequently it is white in color.[11] While performing primary diagnostics, one should remember the possibility of metastatic spread at the time of diagnosis. The commonest locations are the lungs.[11]

Treatment is based mainly on surgery.[39,63] Nowadays, if location of tumor, its growth, and size allow, limb-sparing resection is preferred over amputation. Reconstructive techniques may follow resection with microsurgery.[36,63] In some cases, more radical treatment is required like amputation (level depends on lesion extent).[36] If tumor is large, – preoperative chemotherapy and/or or radiotherapy may be used to decrease its size. They may also increase chances for limb-sparing procedure.[36,39,63] Preoperative radiotherapy is indicated for neoplasms bigger than 5 cm.[36] Postoperative radiotherapy is suggested to lower the risk of local recurrence in the site of previous treatment,[17,36,44] especially if sarcoma was either of size more than 5 cm or high-grade malignant.

After treatment, it should be remembered that disease may recur after a long time.[11,17] Positive prognostic factors are young age, size less than 4–5 cm and more mature histopathologic picture of the lesion.[17,44]

Epithelioid Sarcoma

Epithelioid sarcoma is considered as the most common malignant neoplasm of the hand. It originates from soft tissues,[35,36,56,58] comprising 15% of all malignant lesions of the hand.[35] It occurs more frequently in men, (in women, it

appears at a younger age). Average age of diagnosis is 31st year of life (10–35 years old).[22,35,58]

Symptoms of epithelioid sarcoma are not specific and often include a painless lesion within the hand. However, some patients sporadically complain of pain.[35] Usually it is single tumor of hard consistence[58] and of nodular appearance, developing along subcutaneous tissue, tendons, and fasciae.[14,22,36,39,58] It is yellowish-brown in color.[22] In some cases, ulceration with discharge may be present. Some patients describe an injury prior to lesion.[58] The most common locations are fingers and the palmar surface of the hand.[56] Clinical appearance may lead to misdiagnosis like infection or Dupuytren's contracture.[35,56] Great possibility of local recurrence and spreading to regional lymph nodes and to the distal locations (lungs) are characteristic for epithelioid sarcoma.[14,22] Metastases to regional lymph nodes appear more frequently than in other soft tissue sarcomas.[35]

Diagnosis is based on the histopathological evaluation of the specimen obtained during biopsy or surgical resection. Imaging techniques comprise magnetic resonance of involved location, plain x-rays, and computerized tomography (CT) scans of the chest. Diagnostic methods may be supplemented with ultrasound of the abdomen and bone scan.[14] Regional lymph nodes should also be examined[56] (peritrochlear and axillary groups, including sentinel nodes) with ultrasound and fine-needle biopsy.[14,22]

Surgery is the main treatment of epithelioid sarcoma.[14,22,35,36,39,56,58] Possible procedures are wide en block excision or amputation of ray of the hand, part of the hand, or even above it at a higher level. There are no unequivocal indications for primary resection of regional lymph nodes (axillary); some centers perform such a procedure as a treatment of choice.[39,56] In case of presence of lesions within regional lymph nodes, their resection is important because of the risk of the disease spreading.[35] In case of disease spread or presence of large tumor, chemotherapy and radiotherapy might be helpful.[35,56]

Radiotherapy might be used before the surgical procedure to decrease the size of a lesion. In addition, it might be considered in the treatment of local recurrences together with surgery.[56] For palliative patients, with lesions that cannot be treated with surgery, chemotherapy might be used, together with successive surgery or radiotherapy.[14,36]

It is hard to quote precise results of 5- and 10-year survival. Depending on the literature report, they vary – the former in the range of 25–78% and the latter, 25–74%.

Early diagnosis of lesions presenting features that suggest epithelioid sarcoma is important, because previous misdiagnoses and, more important, improper treatment worsen therapeutic results.[35]

Tumors of size less than 5 cm have a better survival rate. In addition, lack of local recurrence after primary surgery is a positive prognostic factor.[22] Negative prognostic factors are advanced age, male gender, total resection of the lesion not done, and more than one biopsy before final diagnosis.[35]

Liposarcoma

Liposarcomas are one of the more frequent saromas of soft tissues. Within the upper extremity, they constitute approximately 6–10% of those tumors, depending on the report.[52,56] There are five histopathologic subtypes, namely well differentiated, myxoid, round cell, dedifferentiated, and pleomorphic. Some authors discern a mixed subtype in place of the round cell type.[52,56] Different subtypes have different prognosis, 5-year survival, and risk of metastatic spread. Well-differentiated and myxoid types are thought to be low-grade sarcomas, whereas pleomorphic subtypes have the worst prognosis.[16,52] Liposarcomas are diagnosed mostly in the sixth decade, except myxoid and round cell types, which occur in younger adults. Among children, they are rare neoplasms. The most abundant subtype generally is myxoid liposarcoma.[16,50,52,56]

Disease occurrence in patients older than 45 years and/or the presence of necrosis within the lesion are negative prognostic factors.[56] Other prognostic factors include histologic subtype, neoplasm grade, size of the tumor, and excision with appropriate margin.[16,50]

Patients with liposarcoma usually seek medical help because of large tumor, and in some cases, painful tumor.[50,56] This neoplasm is capable of spread, both distant and local. Subtypes of low grade give metastases more rarely in comparison with those of high-grade malignancy.[16] Most commonly, distant metastases are located in lungs. The myxoid subtype, however, can spread to unusual sites such as the retroperitoneal space, mesentery of small and large intestine, pleura, pericardium, and soft tissues of the shoulder and back.[50,52] Because of that fact, it is important to

choose imaging methods wisely, both in primary diagnosis and in patients controlled after treatment.[52] They include local evaluation of tumor extent and possible metastatic spread. Computerized tomographic (CT) scan of the chest is performed together with abdominal CT scan in case of myxoid liposarcoma.[52] Magnetic resonance imaging (MRI) is used in diagnosis too. However, in MRI pictures, the liposarcoma may resemble lipoma. Therefore, treatment of lipoma should be undertaken cautiously, and always malignant neoplasm should be considered. Procedures that in future may hamper or prevent the proper treatment of liposarcoma are to be avoided.[19,30] The diagnostic scheme also includes biopsy.[19,52]

The mainstay of treatment is surgery. While performing resection, wide margins of healthy tissues must be obtained. Depending on lesion extent, wide resection or amputation on different levels is to be considered.[50,52,56] Additionally, adjuvant radiotherapy has application. In case of high-grade tumors and the presence of metastases, chemotherapy is introduced.[56]

A summary of malignant soft tissue tumors is presented in Table 39.3.

Malignant Bone Tumors

Osteosarcoma

Osteosarcoma is the most common primary tumor of the bones overall. It appears most frequently between 10 and 25 years of age, with prevalence among men.[23,42] Incidence of this disease is connected with periods of increased rate of bone growth.[12]There are different subtypes of osteosarcoma.[12,21,33]

Osteosarcoma of the hand appears very rarely, it constitutes 0.18% of all osteosarcoma cases,[23,49,60] and it appears more often between 40 and 70 years of life.[49] If it is the result of metastatic spread, then usually it affects younger population (2–3 decade).[60] There are divergent opinions on the most frequent location in the hand: second and third ray of hand[60] with predominance of metacarpal bones[49]; some reports suggest even spread of primary location within the hand.[27] Cases of osteosarcoma within the wrist are the rarest.[21,39,60] The most frqunt subtype of osteosarcoma within the hand is central, but some patients develop parosteal osteosarcoma.[38,49] It originates from the bone surface and is

Table 39.3. Malignant soft tissue tumors – summary.

Tumor	Incidence	Age	Site	Pretreatment	Local therapy	Posttreatment
Synovial sarcoma	8.5% of synovial sarcomas overall	No data	Wrist, palmar and dorsal surface of the hand, close to tendons and bursae	Consider radiotherapy, chemotherapy	Wide resection or amputation	Consider radiotherapy; chemotherapy
Liposarcoma	6–11% of upper extremity soft tissue sarcomas	Sixth decade except myxoid and round cell types which appear in younger adults	No data	–	Wide resection or amputation	Radiotherapy; in case of high grade lesion or metastatic spread chemotherapy may be of use
Epithelioid sarcoma	15% of all	Tenth to thirty-fifth year of life (31st most frequently)	Fingers and palmar surface of the hand	Radiotherapy	Wide resection or amputation; involvement of regional lymph nodes: resection; in palliative treatment, chemotherapy with successive surgery and radiotherapy	Radiotherapy if recurrence

characterized as intermediate malignancy.[12] The other primary location of osteoasarcoma with metastatic spread to the hand should always be excluded.

Symptoms of the osteosarcoma are not characteristic, and usual complaints are either pain or swell[23,27,42,49] or both simultaneously. Other, more rare, symptoms are pathological fractures, weakening of the hand, limited movement range, and venous stasis.[60] In many patients, the tumor quickly increases its volume.[23] Laboratory examination results may show increased levels of alkaline phosphatase, lactate dehydrogenase (LDH) (in 30% of cases), and erythrocyte sedimentation rate.

The set of imaging techniques in the diagnosis of osteosarcoma should consist of plain x-ray[23,42,49,60] of the involved extremity together with MRI with and without contrast, CT scan of the chest, abdomen, and pelvis, supplied with a bone scan.[27] Techniques mentioned earlier together with technetium isotope scan are used in the examination of metastatic spread. Most frequently, metastases locate in lungs.[42,60]

Open biopsy and fine-needle biopsy may be considered. However, fine-needle biopsy is usually not enough for histopathologic diagnosis.[42]

Treatment of osteosarcoma should be conducted in specialized centers. Main goals of a surgical procedure are removal of primary tumor and disclosed metastases, both together with appropriate margins of healthy tissues.[12] Surgical treatment depends on response to neoadjuvant treatment and also the presence of a wide enough safe margin of healthy tissues surrounding the lesion, enabling safe tumor excision within adjacent tissues.[31] Treatment of osteosarcoma within the hand can consist of en bloc removal, if a safe margin can be preserved. If not, amputation of the involved part should be considered; it can be, for example, a finger or ray of the hand. In case of en block excision, reconstructive surgery should be considered.[38]

Introduction of chemotherapy caused improvement in survival rate.[12] It is most effective together with surgical excision, because chemotherapy alone does not lead to the lesion's disappearance. It decreases the local recurrence rate and distal metastasis risk. Treatment consists of chemotherapy for a certain period of time before surgery (neoadjuvant therapy). It allows to judge lesion reaction to medicines that are enrolled in certain chemotherapy protocols. If tumor is not resistant, it will cause better demarcation of malignant tissues. It usually results in decrease in the local recurrence rate.[12,42] Chemotherapy consists of different medicine protocols.[12,33,42] Response to therapy is measured histopathologically, by percentage of malignant tissue destroyed by the medicine protocol.[12,31,60] Osteosarcoma is treated with radiotherapy only if the patient refuses surgical treatment. It is then combined with chemotherapy.[60]

Incidence of local recurrences worsens prognosis. Subsequent treatment is indicated, an important part of which is surgical removal of recurrence and or metastases. The negative prognostic factors are lack of good response for neoadjuvant treatment, metastases at time of primary diagnosis, large volume of tumor, high levels of alkaline phosphatase, and lack of wide enough healthy tissues margins enabling proper surgical treatment.[12,42] Some reports consider Paget's disease and previous exposure to radiation as negative risk factors[60] (Figure 39.17).

Chondrosarcoma

Chondrosarcoma is the most common malignant bone neoplasms of the hand; it constitutes 40% of all malignant bone tumors of hands and feet and 4% of all malignant neoplasms of a hand.[39,51] However, primary chondrosarcoma of this location is a rare lesion. More often, it is either metastasis from other primary sites or a result of malignant transformations of a previously benign neoplasm such as enchondromatosis and osteochondromatosis.[36,48]

The most frequent site of appearance of chondrosarcoma within the hand is the proximal phalanx.[48,51] Clinical symptoms are not characteristic and specific and usually mimic benign lesion.[48] Swelling is the most frequent sign; it can be accompanied by pain and, rarely, pathological fractures.[23,51] It is suggested that the lesion appears more often in older people – fourth to seventh decade of life depending on author.[51,54] Development of symptoms mentioned previously among patients older than 40 years, together with absence of chondrosarcoma located outside the hand that could result in metastases, may suggest a primary hand lesion.[23]

Diagnostic methods include histopathologic evaluation of specimen obtained by biopsy.[36,48,51] Clinical examination should be supplemented

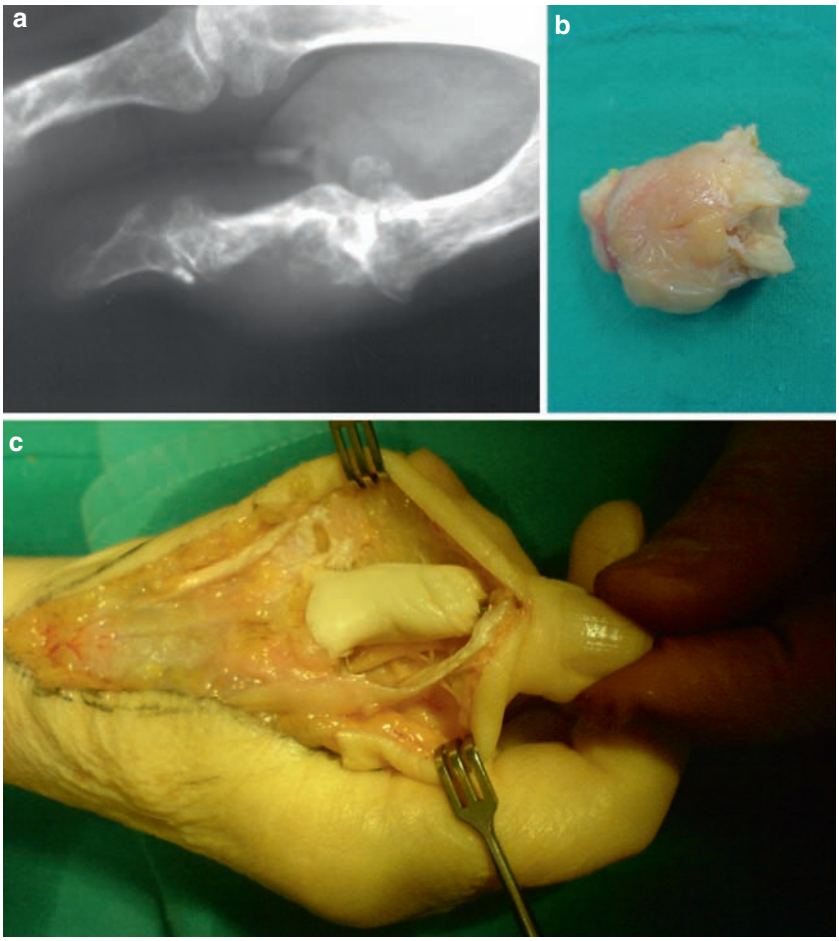

Figure 39.17. Osteosarcoma of the thumb. (**a**) X-ray image. (**b**) Tumor after excisional biopsy. (**c**) Temporary interposition with bone cement.

with radiological evaluation – plain x-ray. Another imaging diagnostic technique is bone scan and magnetic resonance.[23,36,48,51]

Treatment of chondrosarcoma is based mainly on surgery.[23,36] Procedures that are being performed are amputation of involved part, partial amputation if lesion is too advanced for safe excision, resection en block, and curettage (Figure 39.18). Amputation of the finger or ray of hand has the lowest risk of local recurrence and good results compared with other methods mentioned.[18,48,51] There are trials of removal of low-grade chondrosarcoma intralesionally with curettage or cryotherapy.[18,41] However, local recurrence was noticed after such procedures.[18] In case of local recurrence, each successive surgery must be more radical than the previous one.[51] Chondrosarcoma is a neoplasm resistant to chemotherapy. It is also not susceptible to radiotherapy. However, there are reports of trials of use of radiotherapy as the palliative treatment in unresectable lesions.[18,23,36]

The suggested algorithm of follow-up has been described previously in this chapter. According to the literature, chondrosarcoma of the hand has lower ability to result in distant metastases than other more proximal locations.[23,36,51]

A summary of malignant bone tumors is presented in Table 39.4.

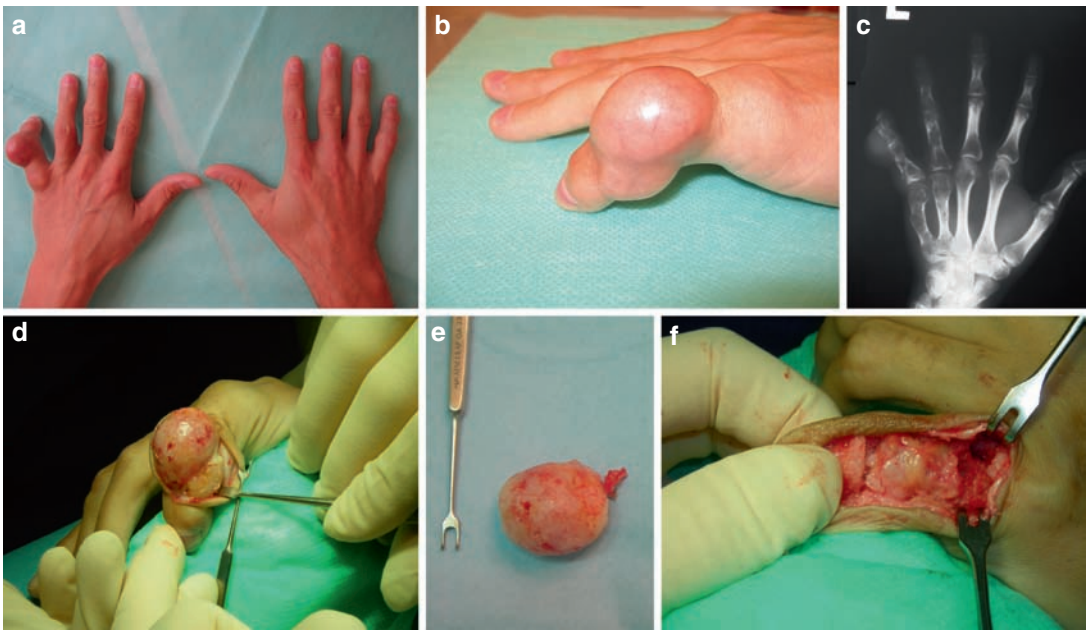

Figure 39.18. Chondrosarcoma of the little finger. (**a, b**) Clinical presentation. (**c**) X-ray. (**d**) Intraoperative view. (**e**) Excised tumor. (**f**) Site after tumor excision.

Table 39.4. Summary of malignant bone tumors.

Tumor	Incidence	Age of appearance	Site	Pretreatment	Local therapy	Posttreatment
Osteosarcoma	0.18% of all Osteosarcoma	Fourth to sixth decade of life	Metacarpal bones	Chemotherapy	Resection with wide margins or amputation; if surgery refused, radiotherapy	Chemotherapy
Chondrosarcoma	4% of all malignant lesions within hand	Fourth to seventh decade	Proximal phalanx	–	Surgical treatment: limb sparing or amputation; trials of radiotherapy in nonoperative tumors	–

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40

Chronic Arterial Ischemia of the Upper Extremity: Diagnosis, Evaluation, and Surgical Management

Mark F. Hendrickson

Summary

Chronic ischemia of the upper extremity reflects progression of occlusive disease, vasospastic disease, or both – vaso-occlusive disease. In contrast to the lower extremity, vasospastic disease is more common and critical in the upper extremity. Although less common in the upper extremity, atherosclerotic disease is functionally more problematic.

When nutritional flow is inadequate for local metabolic demands, chronic ischemia becomes problematic with pain, tissue loss, and persistent infection.

Abbreviations

DBI	Digital brachial index
PTA	Percutaneous angioplasty
PVR	Pulse volume recordings

Circulation: Flow and Pathology

Flow is directly related to the fourth power of the radius and indirectly related to segment length as noted in the Hagen–Poiseuille equation:

$$\text{Flow} = \frac{(\Delta\text{Pressure})\ (\pi)\ (\text{Radius}^4)}{(8)\ (\text{Viscocity})\ (\text{Length})}$$

Table 40.1 relates variation in radius to flow.

Both atherosclerosis and vasospasm limit perfusion. Worsening flow interference develops as fluid dynamics interacting with local functional anatomy yield high-risk areas for turbulent flow. These areas include bifurcations and local areas of vessel fixation. However, injury to the endo-thelium is the primary event for atherosclerosis. Along with the induced turbulent flow, other risk factors lead to progression of atherosclerosis. These risk factors include tobacco use, diabetes, radiation, hypertension, hyperlipidemia, and direct vessel injury. Collateral flow, as in the lower extremity, can finitely expand when the primary circulatory pattern is gradually obstructed. Although collateral flow can improve perfusion, chronic ischemia develops when these elements preclude adequate local, distal tissue oxygenation over time.

The two major forearm arteries continue as the respective arches: the ulnar artery to the superficial palmar arch and the radial to the deep palmar arch. The median artery is a significant contributing artery in about 5% of patients. In upper extremities, the superficial palmar arch is complete in about 80% and the deep arch is complete in about 95%. Complete indicates that the arch connects with an independent vessel. In the digital pulp, total blood flow includes both nutritional and thermoregulatory flow. Typically, 80–95% of total flow is thermoregulatory with the remainder being nutritional.

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Table 40.1. Variation in radius to flow.

Radius (cm)	Flow (L/min)	Baseline (%)	Baseline (fraction)
1	1	100	1
0.5	0.06	6.25	1/16
0.25	0.004	0.4	1/250

Surgical Exposure

Over the vessels, the incisions can be either longitudinal or a gentle zigzag. Incisions over joint creases must be angled. These joint areas include the shoulder, elbow, wrist, and digits (see Figures 40.1 through 40.5).[8] Along the interphalangeal joints, the incisions can be mid axial also. For adjacent digits, "Y" or "V" incisions can be used to approach the metacarpal phalangeal creases in the web spaces.

Clinical Evaluation

Chronic upper-extremity ischemia seldom develops in isolation. Although the focus of evaluation is the upper extremity, the other vascular systems

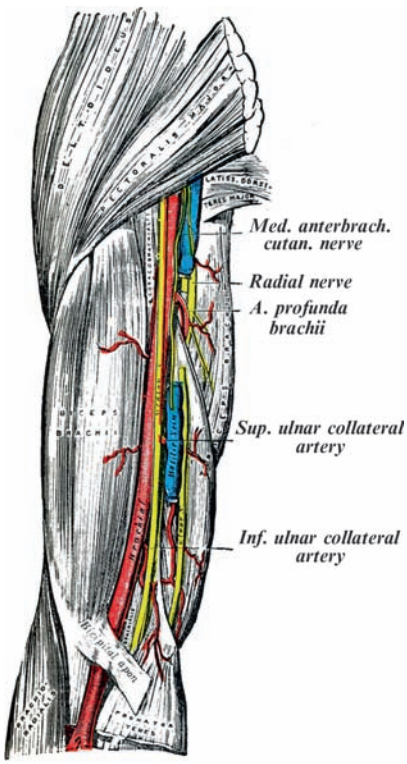

Figure 40.2. Brachial artery in arm. (Reprinted with permission from Gray.[8])

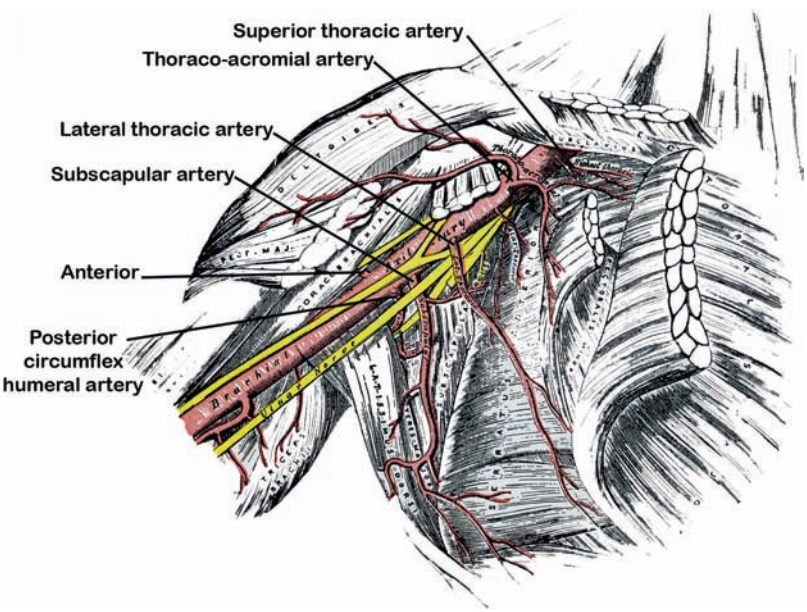

Figure 40.1. Axillary and brachial artery. (Reprinted with permission from Gray.[8])

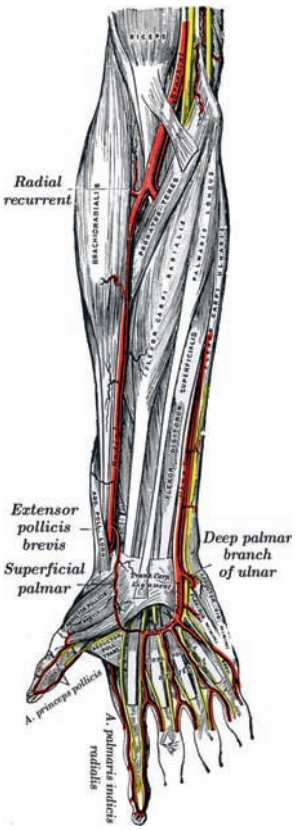

Figure 40.3. Superficial forearm and hand vascular anatomy. (Reprinted with permission from Gray.[8])

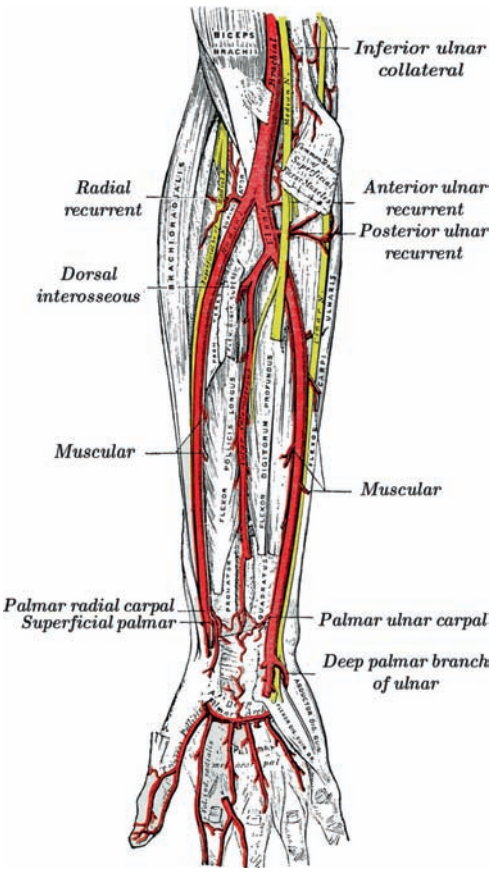

Figure 40.4. Deep forearm vascular anatomy. (Reprinted with permission from Gray.[8])

need to be reviewed. Cardiac disease includes significant coronary artery disease, myocardial infarction, congestive heart failure, cardiac arrhythmia, and abnormal electrocardiogram. Cerebrovascular disease includes transient ischemic attack and cerebrovascular accident. Although less common than in the lower extremity, intermittent claudication and rest pain can occur in the upper extremity. Effort results in crampy hand or forearm pain that resolves with adequate rest. Rest pain reflects the constant burning pain that is sometimes alleviated with dangling the extremity. Trauma, either penetrating or nonpenetrating, is questioned. The repetitive use of the hand as a hammer can occur. The patient history is also reviewed for impotence, transient ischemic attack, cerebrovascular accident, angina pectoris, myocardial infarction, and arrhythmias. The patient is evaluated for other risk factors. Smoking is one of the most common and powerful contributing factors to vascular disease.[10] Both severity and control of diabetes affect the extent of peripheral vascular disease. Hyper-tension and hyperlipidemia are also significant risk factors. Risk factors also include deep venous thrombosis, pulmonary embolus, and clotting abnormalities.

On physical examination, all extremities are evaluated. The entire upper extremity is examined from neck to digits. Detailed vessel examination of the upper extremity includes the subclavian, axillary, brachial, radial, superficial radial, ulnar, palmar arch, and digital arteries. A timed Allen's test is performed for the hand and digits. The presence of bruits, although uncommon, is noted. The extremities are evaluated for pallor, cyanosis, rubor, ulceration, wounds, gangrene, atrophy,

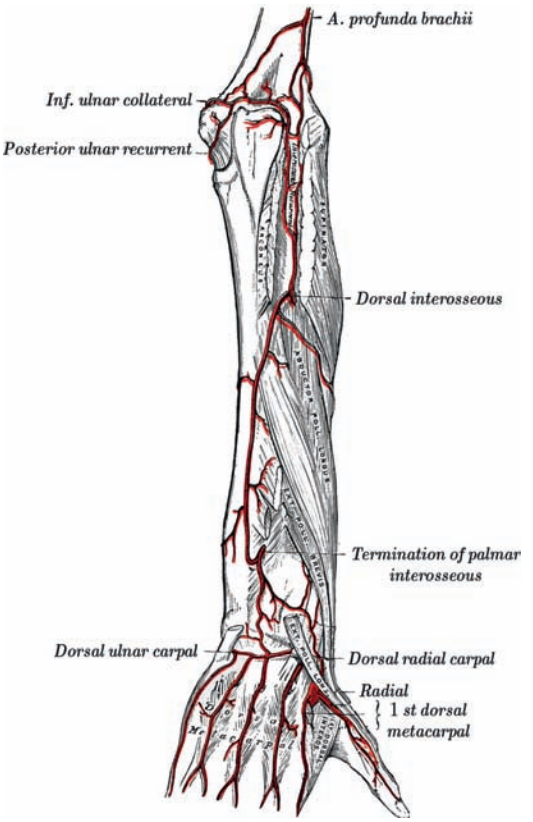

Figure 40.5. Dorsal forearm vascular anatomy. (Reprinted with permission from Gray.[8])

tissue loss, and temperature (seen in Figures 40.6 through 40.8).[14,37,43] Trophic changes are noted; these findings include hair loss, shiny skin, and thickened nails. Along with palpation, these arteries are evaluated with a Doppler probe as normal, turbulent, or absent. The Doppler probe can facilitate the timed Allen's test.[25,26]

More sophisticated testing includes laser Doppler fluxmetry and laser Doppler perfusion imaging. The systolic digital arterial pressure to systolic brachial arterial pressure, that is digital brachial index (DBI) is determined. The DBI is analogous to the ankle brachial index. A large gradient between segments indicates intervening obstructive disease. Adequate perfusion is indicated by a DBI > 0.7. DBI values less than 0.7 indicate inadequate digital arterial flow. Again, calcified vessels, as in diabetes, may yield a falsely normal DBI. Digital plethysmography (pulse volume recordings (PVR)) quantifies segmental flow. PVR usefully distinguish segmental or diffuse vasospastic occlusive disease. PVR can predict response to and document outcome from various interventions. Three-phase, isolated cold stress testing following digital temperature and laser Doppler cutaneous perfusion is likewise useful. Doppler ultrasound and color duplex imaging offer structural and functional information. Current angiographic techniques include standard contrast angiography, magnetic resonance angiography, and computed tomography angiography.[2,15,21,23,22,41]

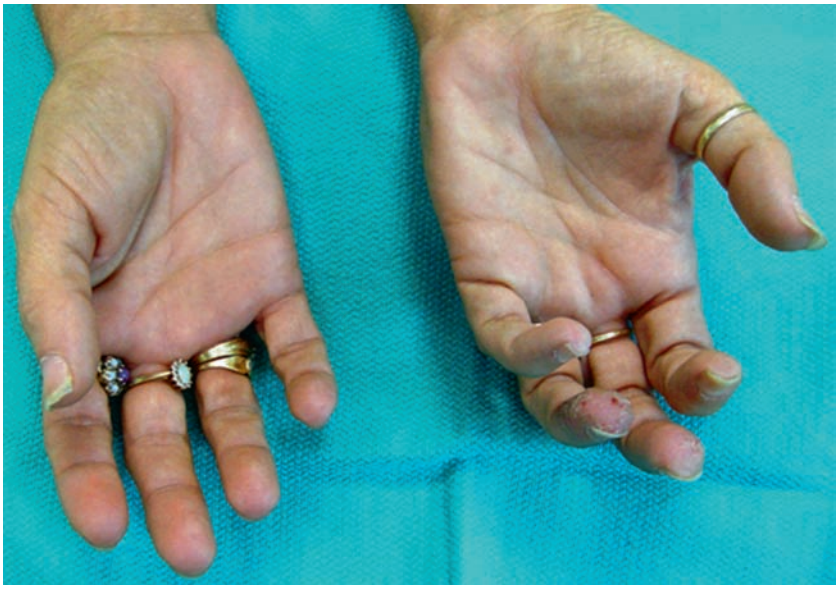

Figure 40.6. Chronic ischemia of hands.

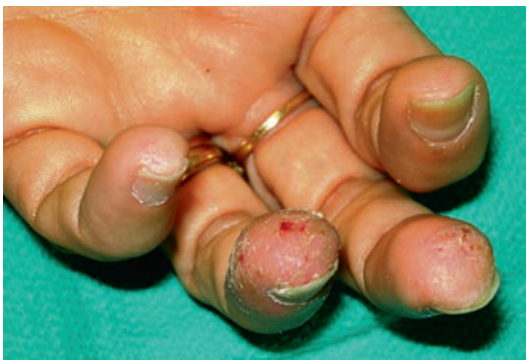

Figure 40.7. Chronic ischemia of hand with ulcerations.

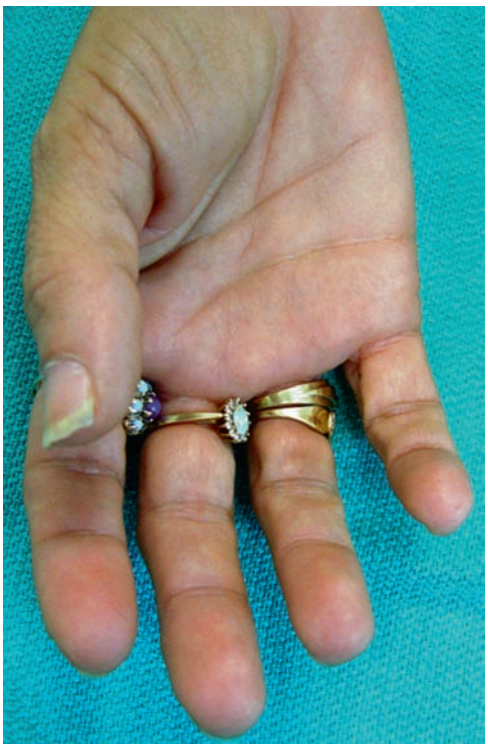

Figure 40.8. Chronic ischemia with pallor of fingers.

Surgical Management of Chronic Ischemia

The goal of surgical intervention is to improve local, distal arterial flow in order to alleviate pain, heal wounds, clear infection, preserve functional tissue, and improve quality of life (Figure 40.9). The techniques include periarterial sympathectomy, arterio-artery bypass, arteriovenous bypass with valvotomy, and free tissue transfer.

Sympathectomy

The Leriche sympathectomy is simply ligating the thrombosed arterial segment. With adequate collateral flow, the interrupted sympathetic overdrive is disrupted and vasospasm resolves.

Digital periarterial sympathectomy includes segmental sympathetic nerve branch to artery resection or division and adventitial stripping.[1,42] Sympathectomy is performed at three levels unless isolated vasospasm is present. At the wrist, the ulnar artery, radial artery, and superficial radial artery are stripped. In the palm, the superficial arch and origin of the deep arch are stripped. In the digits, the common digital arteries and the proper digital artery to the small finger are stripped. A 1–2 cm segment of adventitia is stripped at each location. The difficulty of adventitial stripping is removing the complete adventitia to the external elastic membrane of the media without damaging the intima or media.[5,6,16,19,20,24,31,35,40]

Arterioarterial Bypass

A DBI less than 0.7 indicates the need to consider arterial reconstruction. The success of reconstructing occluded arterial segments is dependent on several factors. Inflow and outflow are verified. The extent of inflow disease determines the proximal arterial source. Options include the ulnar artery, radial artery, or the dorsal superficial radial artery branch. Likewise, the distal anastomosis is determined by the extent of outflow disease. Options include the arch, common digital artery, or proper digital artery. If required and with the proper graft configuration, several digital arteries can be anastomosed to the distal graft. With severe, diffuse distal outflow, verification is difficult. The used conduit must be adequate. Conduit options include the lesser saphenous vein, the greater saphenous vein, cephalic vein, and the basilic vein. Interpositional grafting necessarily disrupts any existing collateral flow. Distal bypass grafting does not. Interpositional grafting with excision of intervening abnormal segment and distal bypass palmar or digital grafting is effective. Patency ranges from about 50% to 100% with varying ranges of follow-up. Radial side arterial reconstruction and

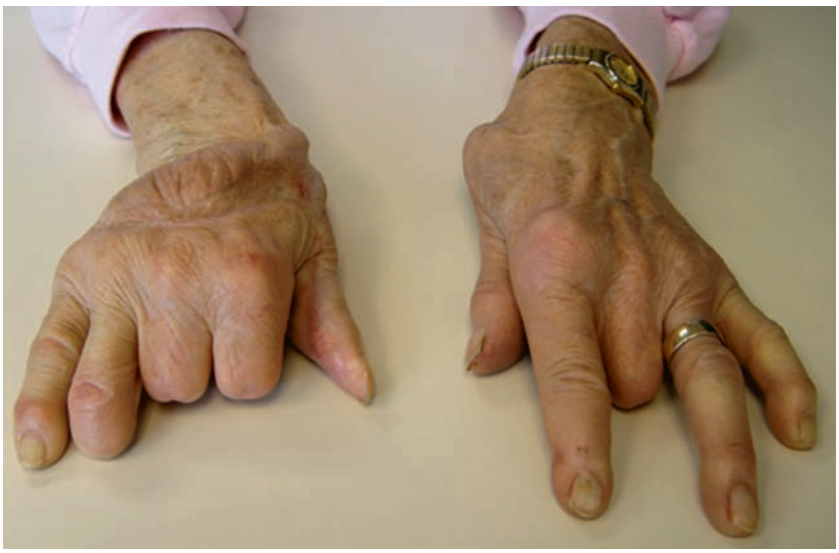

Figure 40.9. Long-term goals of surgical treatment.

distal bypass grafting appear to have greater and longer patency rates.[3,13,17,18,22,28–30,33,34,38]

Arteriovenous Bypass with Valvulotomy

When adequate inflow is present without evidence of adequate distal digital outflow, arterialization of the venous system is considered (Figure 40.10). The quality of distal digital outflow can be determined by clinical evaluation, including Doppler probe examination and several specialized imaging studies. In this bypass, the cephalic or basilic vein is anastomosed to the brachial artery about the elbow. A distal arterial anastomosis is not performed. Rather, the intervening valves are released with a valvulotome. This allows arterial flow into the distal venous system of the hand and digits.[9,34]

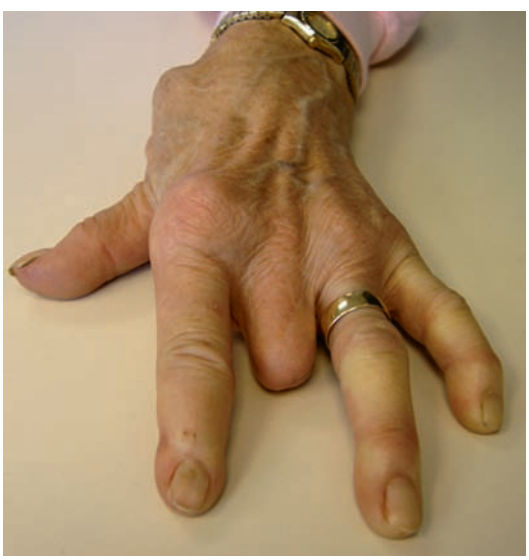

Figure 40.10. Long-term outcome of arterialization of the venous system.

Free Tissue Transfer

When chronic ischemia progresses to critical func-tional loss and more standard techniques are not possible or fail over time, free tissue transfer can be considered. Arterial inflow tends to be more proximal. Arterial outflow is usually inadequate due to diffuse, segmental digital disease. Successful fascial and omental free tissue transfers are documented (Figure 40.11). In such instances, free tissue transfer can salvage the hand and thumb in cases of severe chronic ischemia.[11,12,36,39]

Amputation

Amputation is indicated for necrotic, infected parts. Critical to successful outcome is delicate dissection and tissue handling and tensionless

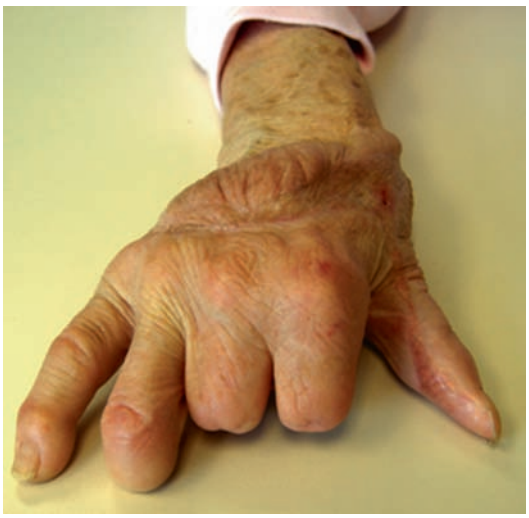

Figure 40.11. Salvage of hand and thumb with free omental transfer.

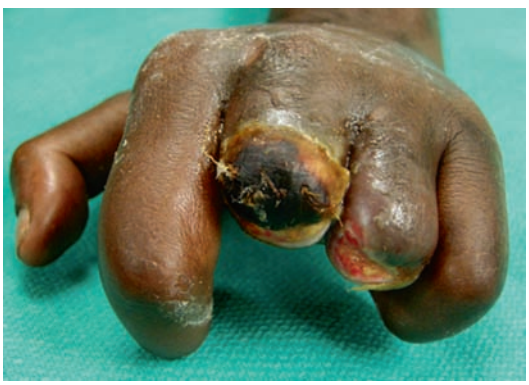

Figure 40.12. Outcome of digital amputation for severe chronic ischemia.

closure. Digital tourniquets can damage adjacent tissue. With the paucity of bleeding, tourniquets are often unnecessary. Possible levels include digit, hand, forearm, and arm (Figure 40.12).

Physiologic amputation is a consideration on rare occasion. If a patient is septic from an infected extremity, critically ill with multiple organ dysfunctions of failure, and therefore at prohibitive operative risk, physiologic amputation can be considered. Physiologic amputation requires an arterial tourniquet to obviate perfusion to the extremity and freezing the extremity. The arterial tourniquet must be failure-proof, applying a continuous high pressure. Dry ice can be used for freezing the extremity. All other body surfaces must be protected from the dry ice. If the patient survives, an open amputation can be performed 24–48 h later.

Future Trends

Percutaneous angioplasty (PTA) and stenting are well-documented options in many areas of occlusive arterial disease. The operative and postoperative benefits are similar to those in other less invasive procedures. PTA of more proximal upper-extremity occlusive disease results in outcomes comparable to, if not better than, conventional open procedures. Balloon angioplasty of the distal radial artery has been successfully performed with almost a 2-year outcome of salvage. Salvage of the hand by percutaneous angioplasty and stenting of the radial artery has also been reported.[4,7,27]

Two small studies suggest the effectiveness of botulinum toxin type A in patients with severe Raynaud's phenomena. Neither study was prospective, blinded, or controlled. In 2005, an Austrian group reported improved outcomes in four of five patients. A group of at least 16 patients from Southern Illinois University in Springfield reported significant pain relief, improved blood flow, and ulcer healing with treatment of botulinum toxin type A. The injections are around the neurovascular bundles in the web spaces. Initial injections are 30–200 units of botulinum toxin type A. Strength is transiently affected. The mechanism is thought to be by blocking the sympathetic nerves and the C fibers.

Rehabilitation

Fundamental to successful outcome is pain control and protection of the operated extremity. A bulky, soft dressing with splint rests and protects the operative area. Simple wound care will facilitate wound and ulcer healing. Protective positioning and gentle progressive motion are initiated soon after the procedure.

Summary

Chronic ischemia of the upper extremity is a complex problem with difficult surgical management and limited long-term successful outcomes.

Conservative treatment requires smoking cessation, cold avoidance, and appropriate medical management. Both oral and topical vasodilators may be useful. Sympatholytic agents can improve nutritional flow. By clinical evaluation, the extent of occlusive and vasospastic disease can be determined. When indicated, the appropriate surgical treatment can be instituted. Periarterial sympathectomy and distal arterial bypass are options. For salvage, arterialization of the venous system and free tissue transfer are options.

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41

Acquired Diseases of the Hand (Rheumatoid Arthritis and Dupuytren's Contracture)

Douglas C. Ross and Bing Siang Gan

Summary

In this chapter, two acquired diseases of the hand – rheumatoid arthritis (RA) and Dupuytren's disease (DD) – are reviewed. RA is a chronic, debilitating disease that affects the synovium at the cartilage–pannus junction (CPJ) and leads to joint destruction, joint laxity, and tendon dysfunction. Pathophysiology as well as treatment options for the wrist, metacarpophalangeal joints, and extensor tendon disorders, including rupture, boutonniere deformity, and swan neck deformity (SWD) is reviewed. DD is an acquired disease of palmar fascia. Epidemiology and postulated disease mechanisms are reviewed. In addition, pathologic anatomy, current treatment options, and treatment outcomes are reviewed.

Rheumatoid Arthritis

Rheumatoid arthritis (RA) is a chronic, debilitating disease characterized by an inflammatory arthropathy as well as a number of extra-articular manifestations. It has been shown to impose a greater cost burden on society than any other musculoskeletal disorder. For instance, in the United States, it is estimated that direct costs of the illness such as drugs and hospital admissions amount to $15 billion annually and that indirect costs such as time lost from work amount to approximately $50 billion per year.[50] This reflects nearly 2.5% of the gross national product. Such costs are similar across all industrialized countries.

Epidemiology

The prevalence of rheumatoid arthritis differs across different parts of the world. The prevalence in North America is estimated to be 10.7 per 1,000 population, whereas in southern Europe and in developing countries, the prevalence is estimated to be 3.5 and 3.3 per 1,000 population, respectively.[1] By far, the highest prevalence is among North American native populations.[56] Differences in prevalence among different geographical areas are thought to be caused by both genetic and environmental factors. The most significant genetic factor appears to be the presence of HLA DRB1 alleles. A number of environmental factors have been implicated in the development of RA, including dietary factors, breast feeding, infectious agents such as Epstein–Barr virus, and cigarette smoking.[56]

Diagnosis

The diagnosis of RA is based on the criteria published by the American Rheumatology Association in 1987.[5] The presence of four out of seven criteria for a period of 6 weeks or more is required to make the diagnosis (Table 41.1). These criteria have been criticized for emphasizing signs and symptoms associated with longstanding

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Table 41.1. ACR criteria (patient must have four of seven) for diagnosis of rheumatoid arthritis. First four criteria must have been present for at least 6 weeks.

Criteria
Morning stiffness > 1 hour
Arthritis of 3three or more of the following joints: MCP, PIP, wrist, elbow, ankle, knee, MTP
Arthritis of hand joints (wrist, MCP, PIP)
Symmetric involvement of joints
Rheumatoid nodules over bony prominences, extensor surfaces, or juxta-articular regions
Positive serum rheumatoid factor
Radiographic changes including erosions or bony decalcification located in, or adjacent to, involved joints

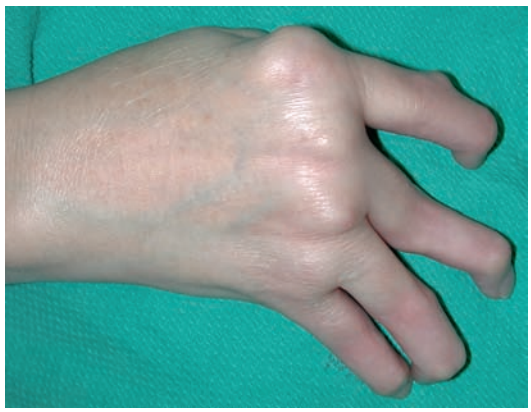

Figure 41.1. Typical clinical appearance of patient with RA. Ulnar drift and volar subluxation of the MP joints are evident. An SWD is present in the long finger.

disease and therefore being less sensitive for the diagnosis in patients with new onset symptoms. In practice, significant advances in the serological diagnosis of RA have been made. In particular, using antibodies to cyclic citrullinated peptides (anti-CCP) increases the sensitivity of diagnosis, particularly in patients with symptoms of duration less than 6 months.[34] Given that early pharmacologic intervention may significantly alter the course of the disease, early diagnosis is important, and sophisticated serologic testing is now frequently used to establish the diagnosis.

Pathophysiology of Rheumatoid Deformities in the Hand and Wrist

The etiology of RA is still unknown. Previous work has suggested that an autoimmune response to a relevant antigen may be the trigger to the disease. The resultant joint inflammation centered around the cartilage–pannus junction (CPJ)[62] remains the primary underlying pathology. The dominant cell types at the CPJ are fibroblast-like synoviocytes (FLS) and macrophages. FLS cells demonstrate invasive and proliferative properties as well as release proteolytic enzymes. This process leads to joint distension, capsular and ligamentous laxity, and resultant joint instability.

A number of biomechanical forces then act upon this system. At the level of the wrist, there is loss of carpal height and ulnar translocation of the carpus, resulting in radial deviation of the metacarpals. In addition, scapholunate instability, volar carpal subluxation, carpal supination, and dorsal subluxation of the distal ulna occur.[26] At the metacarpophalangeal joint, many activities of daily living such as grip and pinch activities tend to push the fingers into the classic ulnar drift deformity. Also contributing to this is the radial deviation of the metacarpals noted previously, which causes the extensor tendons to assume an ulnarly directed force upon the proximal phalanges. The classic picture of joint subluxation and ulnar drift is produced (Figure 41.1).

Surgery of the Hand and Wrist in Rheumatoid Arthritis

Indications

Surgery as part of the treatment of upper-extremity dysfunction associated with RA dates back at least 50 years.[11] Surgeons tend to assume that the prime indications and benefits of this surgery are improved function and pain relief. Alderman et al.[2] found that improved function and pain relief were the two most important factors for patients to choose surgery to correct MP joint disease. In a later study, Alderman et al.[3] found that although pain relief and improved function were equally prevalent as the most important factors for patients to choose surgery, a significant minority of patients valued hand aesthetics as the most important factor. In the same study, 30–40% of patients felt uncomfortable in social situations due to their hand deformities. Other studies and editorials have supported the importance of aesthetics in hand surgery.[7,36] These findings reinforce the idea that each patient may have a variety of reasons for seeking reconstructive rheumatoid surgery and that the surgeon must take the time to solicit

and understand these reasons to meet patient expectations.

Specific Surgical Procedures

The Wrist

Pathology of the wrist in RA includes the carpus as well as the distal radio–ulnar joint. The goals of surgery for the carpus include alleviation of pain, establishment of stability, and maintenance of as much motion as possible. In addition, if there is significant radial deviation of the metacarpals, it is best to re-align the wrist before undertaking MP joint arthroplasty. Otherwise, recurrence of ulnar drift is likely to occur quickly.

As for all joints, the potential surgical techniques include arthrodesis and arthroplasty. Wrist arthroplasty has evolved over the past decade, and newer implants have made arthroplasty an option for some patients. Historically, silicone hinge wrist arthroplasty enjoyed significant popularity.[61] However, concerns were raised by a number of authors because of particulate synovitis, which occurred a number of years postimplantation.[29,42] Most surgeons abandoned the use of these implants although some authors have documented good long-term outcomes. Kistler et al.[31] noted that patient satisfaction was good or excellent in nearly 70% of patients at a 15-year average follow-up.

A number of different modular wrist prostheses have been described[13,17] (Gellman, 1997). Surgery is technically demanding, and complication rates have been significant (Gellman, 1997), particularly distal implant loosening. Adams[17] published a report on a series of 22 wrist arthroplasties using the Universal Total Wrist Arthroplasty with 1- to 2-year follow-up (Figures 41.2 and 41.3). Mean flexion/extension arc increased from 48° preoperatively to 76° postoperatively, and disabilities of the arm, shoulder, and hand (DASH) scores improved an average of 24 points at 2 years. Three prostheses suffered instability requiring operative treatment, and all three had marked preoperative wrist laxity, suggesting that such patients may not be good candidates for wrist arthroplasty. In summary, wrist arthroplasty for RA is evolving with encouraging results with modern implant systems.

Arthrodesis remains a cornerstone of the surgical treatment of the wrist in RA. Total wrist fusion with a dorsal compression plate is a reliable procedure with excellent results in terms of pain relief[23,32] (Figure 41.4a, b). Although traditional teaching is to try and avoid bilateral wrist fusions to allow for such activities as perineal care, patients tolerate this well and note improved overall functioning once the wrist is stable and pain free.

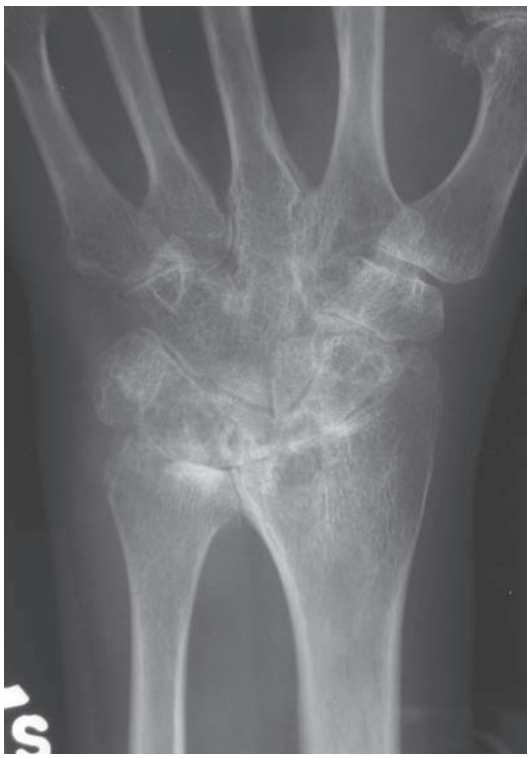

Figure 41.2. Patient with painful arthritis of wrist preoperatively. (Patient and photograph courtesy of Dr. B. Adams.)

Many patients may be candidates for radiocarpal (radiolunate or radioscapholunate) fusion [9,27] alone, which maintains motion through the midcarpal joint, which is often relatively spared in RA (Figure 41.5). Borisch reported on 91 wrists treated with radiocarpal fusion and noted a decrease in average flexion/extension arc from 74° to 46°. However, pain relief was excellent, and all but three patents stated that in retrospect, they would repeat the operation again. All patients requiring a wrist stabilization procedure should be considered for a partial wrist fusion.

Metacarpophalangeal Joints of the Fingers

Replacement arthroplasty of the MP joints is the most commonly done operation in the hand for dysfunction associated with RA. Various types of interposition or resection arthroplasty have

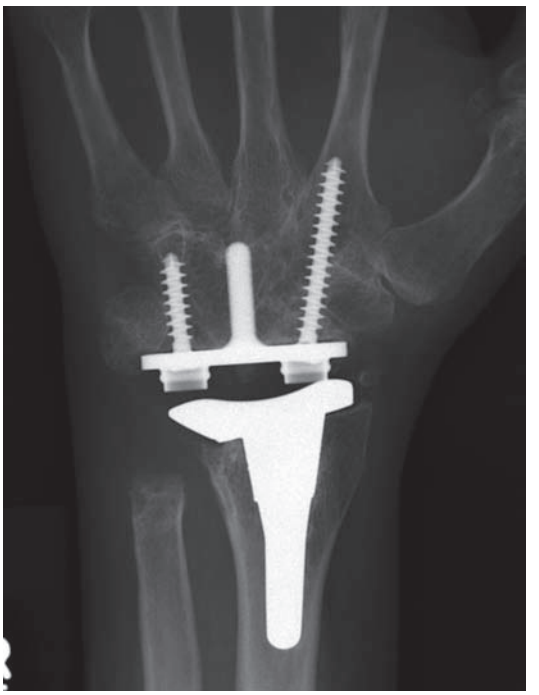

Figure 41.3. Patient after arthroplasty with Universal Total Wrist Arthroplasty. (Patient and photograph courtesy of Dr. B. Adams.)

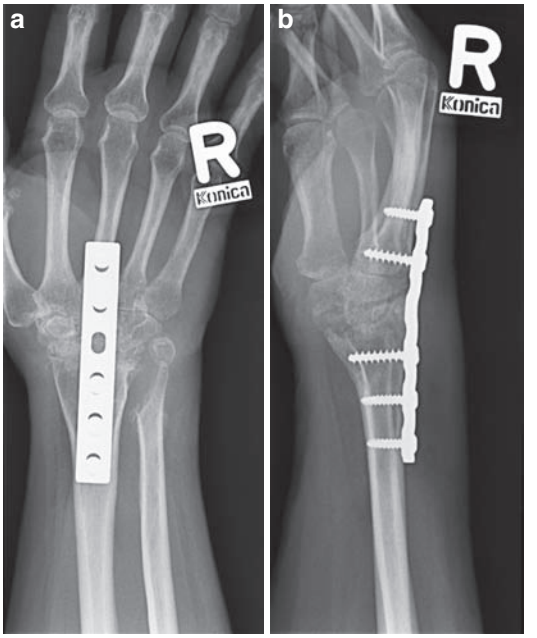

Figure 41.4. Patient after total wrist fusion for painful wrist secondary to RA.

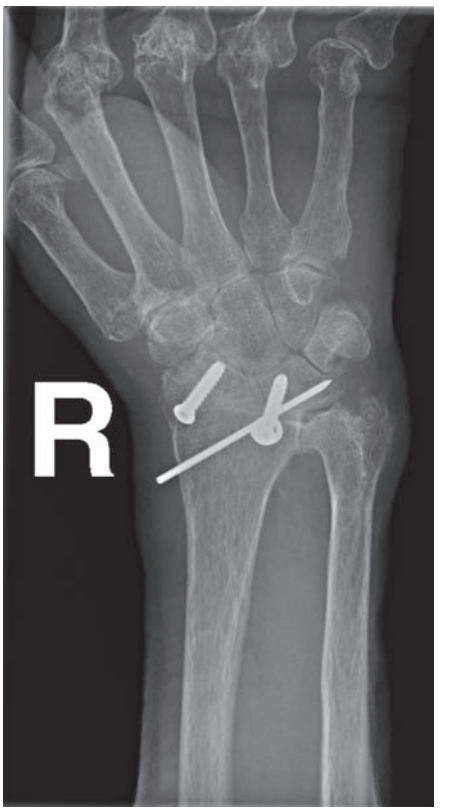

Figure 41.5. Patient with radioscapholunate fusion. Significant preoperative ulnar translation, and radial deviation of metacarpals is significantly improved while maintaining some motion through mid-carpal joint.

been described,[54] but with the advent of Swanson's silicone hinged arthroplasty in the 1970s, alloplastic replacement techniques have predominated. Swanson's series of papers[57–61] document a refinement of technique and materials that remains the standard against which all other MP joint arthroplasties are measured. The technique and the joints themselves have held up remarkably well.[12]

Several alternative alloplastic MP joint replacements have been described over the past decade.[47,49] The "Sutter" silicone MP joint arthroplasty is hinged volarly and has a more robust shoulder that interfaces with bone after insertion. The "Neufeld" joint is also a silicone hinged implant, which is prebent in 30° of flexion. This was to more closely mimic the natural position of the MP joint at rest as well as avoid loss of flexion over time as is commonly seen with

Swanson prostheses. However, in a prospective trial comparing both designs, Pettersson found no significant differences between the two.[49] In another well-done prospective trial, Parkilla found no significant differences between Sutter and Swanson prostheses.[48]

In the 1990s, Beckenbaugh et al. developed a pyrolytic carbon surface replacement arthroplasty for MP and PIP joints.[15,45,47] This is a nonconstrained device with a carbon core and pyrolytic carbon coating[15] and stems that sit within the medullary canals of the adjacent bones (Figure 41.6a, b). A limited number of articles have reported on the outcomes of these implants when used for reconstruction in RA patients.[15,45–47] Cook reported on 151 implants in 53 patients, 44 of whom had RA. Implant survivorship was 82.3% and 81.4% at 5 and 10 years, respectively. Recurrence of ulnar drift was modest (19°) at long-term follow-up. Parker reported on a mixed group of 61 patients treated with a more modern pyrocarbon implant, 42 of whom had inflammatory arthropathy. The RA group reported excellent pain relief and high patient satisfaction with hand aesthetics.

In summary, MP replacement arthroplasty for RA is associated with a high rate of patient satisfaction. Swanson silicone joints remain the most commonly implanted device for this indication. Yet, no prospective clinical trial has compared pyrocarbon implants with silicone implants, and therefore no conclusions may be drawn as to which, if any, provide superior results.

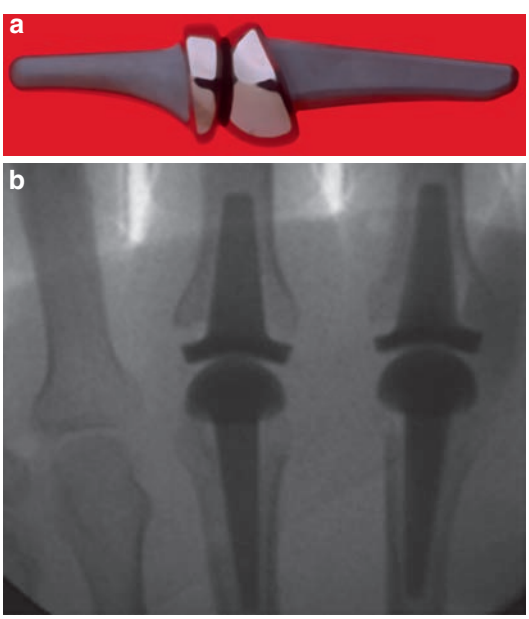

Figure 41.6. Photograph of pyrocarbon implant and RA patient with MP joint replacement. (Patient and photographs courtesy of Dr. R. Beckenbaugh.)

Extensor Tendons

Extensor tendons may be affected in several ways by rheumatoid arthritis including tenosynovitis and rupture, boutonniere deformity, and swan neck deformity (SWD).

Extensor Tendon Ruptures

Anatomically, the only location in which extensor tendons are surrounded by tenosynovium is subjacent to the extensor retinaculum (zone VII). It is in this location that extensor tendon ruptures occur. Factors contributing to rupture include synovitis and bony prominences, particularly dorsal subluxation of the distal ulna.[55] Techniques to identify patients at risk of rupture and thus candidates for prophylactic surgery have included clinical examination[69] as well as sophisticated imaging,[40] Unfortunately, it remains difficult to predict which particular patient is at risk. Many surgeons consider tenosynovectomy if active synovitis persists longer than 6 months while on appropriate medical therapy. The classic differential diagnosis in an RA patient presenting with a sudden inability to extend one or more fingers includes tendon rupture, posteriori interosseous nerve palsy (which may be partial), and subluxation of the extensor tendons into the ulnar valleys adjacent to the MP joints.

The pattern of rupture typically begins on the ulnar side of the hand (extensor digiti minimi, extensor digitorum communis to small finger) and then "marches" across the fingers in a sequential, radial direction. The extensor pollicis longus tendon also commonly ruptures. Treatment of ruptures depends on the number of tendons that have been disrupted. Most surgeons will combine reconstruction of the extensor apparatus with some type of distal ulna resection and tenosynovectomy of the extensor tendons.

There are a number of options to treat the ruptures specifically. Because of the attritional nature of the rupture, direct repair is not possible. Tendon

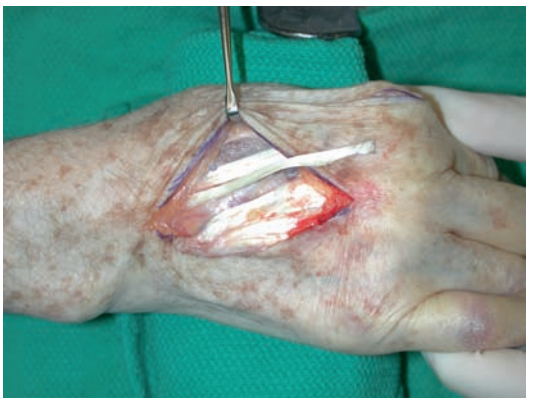

Figure 41.7. Patient with rupture of long and ring fingers with EIP tendon ready for transfer. EIP is ideal to reconstruct rupture of two tendons but insufficiently powerful to motor three fingers.

Table 41.2. Options for reconstruction after extensor tendon rupture.

Tendons ruptured	Preferred reconstruction	Alternative procedures
EDC(V), EDM	EIP transfer	Side-to-side transfer to EDC(IV)
EDC(V, IV), EDM	EIP transfer	Tendon grafts Side-to-side transfer to EDC(III)
EDC(V, IV, III), EDM	EIP to EDC(V, IV), EDC(III) side to side to EDC(II)	Tendon grafts FDS (long or ring) to two tendons
All finger extensors	FDS transfer (ring and long)	Tendon grafts FCR or FCU transfer
EPL	EIP transfer	Tendon graft

grafting is one option, and some authors have published encouraging results.[8,41] For rupture of one or two tendons (EDC to small +/− ring finger), side-to-side transfer to the adjacent, next most radial intact tendon is often recommended. The author has found that this procedure produces an excessively oblique pattern of pull on the transferred tendons, resulting in a limitation of effective tendon excursion and incomplete finger extension. Therefore, transfer of the EIP tendon to EDC is preferred (Figure 41.7). The EIP tendon is insufficiently strong to power extension for three fingers. Therefore, one must consider other transfers such as EIP to D5 and D4 plus end-to-side transfer of EDC(III) to EDC(II). Other options are available and are outlined in Table 41.2.

Table 41.3. Nalebuff's classification of boutonniere deformity with treatment options for each involved joint.

Stage	PIP joint options	DIP joint
Mild: Minimal extensor lag at PIP, limited passive flexion of DIP	Splinting Synovectomy if poorly controlled	Extensor tenotomy
Moderate: 30°–40° lag at PIP, DIP hyperextended	Shortening central slip, move lateral bands dorsally Transfer of lateral bands	Extensor tenotomy
Severe: PIP passively stiff	Arthroplasty fusion	Extensor tenotomy

Boutonniere Deformity

Boutonniere deformity in the context of inflammatory arthropathy differs from that of the deformity associated with trauma. Synovitis within the PIP joint leads to distension of the dorsal capsule and the overlying central slip tendon. Although the tendon may not be completely ruptured, it is effectively lengthened, and the classic pathomechanics of volar migration of the lateral bands occur leading to the deformity.

Early boutonniere deformities may be usefully treated with splinting to regain or at least maintain PIP extension. Splinting is also very useful preoperatively if it is able to produce sufficient passive extension to prevent the need for any type of volar joint release. Zancolli[70] has emphasized the importance of distinguishing between fixed and passively correctable boutonniere deformities. In joints that are stiff, the results of surgical correction are disappointing.[30] In these patients, arthrodesis is probably the best choice. Although PIP arthroplasty is an option, Takigawa et al.[63] found generally poor results for boutonniere correction in particular and RA patients in general. Nalebuff has proposed a useful classification leading to treatment choices[44] (Table 41.3).

The options for surgical correction of boutonniere deformities include anatomic reconstruction and tendon transfer techniques. All techniques require mobilization of the volarly subluxed lateral bands, and this in turn requires release of the contracted transverse retinacular ligaments. Direct reconstruction is done by resecting the attenuated portion of the central slip and then either advancing the proximal tendon to the dorsal base of the middle phalanx[10] or overlapping the remaining portion of the central slip in a "vest over pants" type of repair.

In patients with a hyperextension deformity of the DIP joint in association with boutonniere deformity, a tenotomy distal to the triangular ligament but proximal to the insertion of the spiral oblique retinacular ligament is a quick, easy, and effective solution. Although one is intuitively concerned about causing a mallet finger deformity, experience shows that this is essentially never a problem, and this procedure is a useful adjunct when correcting boutonniere deformities.

Achieving good results in boutonniere reconstruction is notoriously difficult. Several authors have pointed out that improvements with soft tissue reconstructive techniques are modest..[14,30] In joints in which the deformity is not passively correctable, arthrodesis is probably the most predictable procedure.

Swan Neck Deformity

The SWD is characterized by hyperextension at the PIP joint and a flexion deformity at the DIP joint (Figure 41.7). A number of factors may contribute to the development of an SWD in the setting of RA. These include PIP synovitis leading to volar plate laxity, intrinsic tightness leading to excessive extensor forces on the PIP, and DIP synovitis leading to attenuation.[70] The deformity may be fixed or supple, and this forms the basis of Nalebuff's classification of SWD; this is also useful in determining appropriate treatment choices[43] (Table 41.4).

Supple SWD (type I) may be treated with figure of eight splints, which keep the PIP joint in slight flexion and allow smooth, supple flexion of the joint from that position. These are extremely well tolerated by patients and can serve as a trial for operative techniques that rely on establishing a flexed position of the PIP joint to be successful. Nalebuff type I or II SWD may be treated surgically by either FDS tenodesis techniques, first described by Curtis,[16] or by lateral band translocation techniques, first described by Littler[64] and later modified by others.[20,68] The author has found that the latter may lose effectiveness over time, perhaps because of the reliance on the relatively weak Cleland's ligament acting as a pulley. Type II deformities usually require an intrinsic release as well; this can be determined by the preoperative physical examination.

Table 41.4. Nalebuff's classification of swan neck deformities and treatment options for each stage.

Type	Treatment options
I: PIP joint totally flexible	Splinting DIP fusion if mallet deformity significant
II: PIP joint flexion limited in certain positions	FDS tenodesis Lateral band translocation techniques Intrinsic release if necessary
III: PIP joint flexion limited in all positions	Dorsal release +/– FDS tenodesis or lateral band translocation
IV: PIP joint stiff with x-ray changes	Arthrodesis Arthroplasty

Curtis' technique is quick and effective. The divided FDS tendon may be sutured to the pulley system as described here rather than through a drill hole in the bone. A volar oblique incision is made over the proximal phalanx. A slip of the FDS tendon is divided proximal to A1 and the distal end passed through a window between A1 and A2. An oblique K wire is passed across the PIP joint at 30°, and the divided FDS slip is tensioned against this and reflected back along the outside of the A2 pulley and sewed to the pulley and to itself within the flexor tendon sheath. Care must be taken not to pick up the FDP tendon when suturing the FDS to itself. The K wire is removed 5 days postoperatively; the patient is placed in an extension block splint and begins active flexion exercises.

Type III deformities require an extensive dorsal release of extensor tendon, dorsal PIP capsule, and the dorsal portion of the PIP collateral ligaments. It is also necessary to release the dorsal connections of the lateral bands to allow them to slide volar to the axis of rotation of the PIP joint.

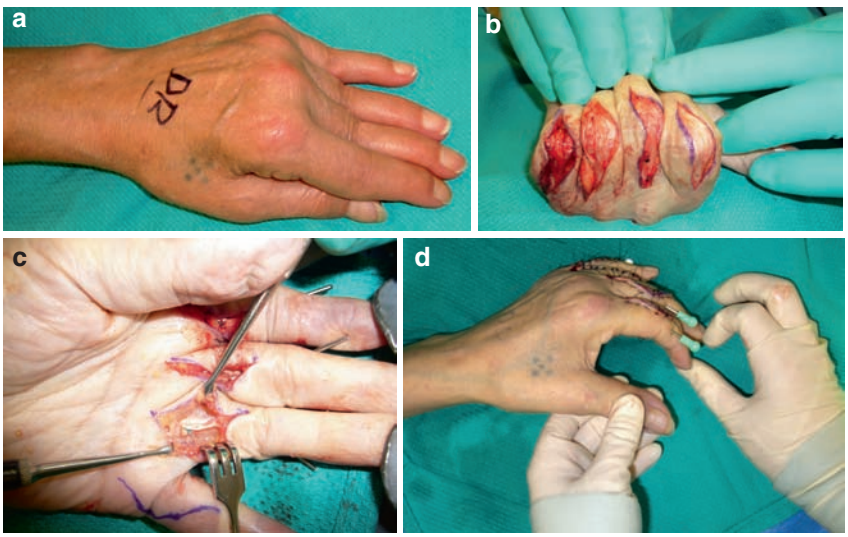

Figure 41.8. Reconstruction of type III SWD. Preoperative deformity (**a**) is corrected by dorsal release (**b**) and stabilized by a sublimes tenodesis (**c**). The final result is shown in (**d**). K wires are removed at 5 days postsurgery and active flexion commenced in an extension block splint.

If the lateral bands remain unstable, a sublimes tenodesis is done (Figure 41.8a–d). As might be expected with this extensive surgery around the PIP joint, results of surgery for type III deformities are often disappointing.[30] Because of the joint destruction in type IV deformities, arthrodesis is the best surgical option for these joints.

In summary, RA is a progressive disease with important functional implications for patients and their families. Surgery of the hand and wrist plays an important role in minimizing the effects of RA on patients' lives.

Dupuytren's Disease

History and Incidence

Dupuytren's contracture or Dupuytren's disease (DD) is a benign, but potentially debilitating, fibroproliferative disorder of the palmar fascia of the hand. The earliest reference to DD in the medical literature dates back as far as 1614; however, the disease received its eponymous name after the illustrious French surgeon Baron Guillaume Dupuytren (1777–1835), the chief surgeon at the Hôtel-Dieu Hospital in Paris who demonstrated the fasciotomy method of treating this disease in 1831.[22]

The disease is most common in males of Northern European descent between the ages of 50 and 70 years, but it equalizes between men and women after age 80 years.[37] DD usually affects the ring and small fingers and can occur bilaterally. It is rare that the thumb and index fingers are involved, and patients with DD of these digits may suffer from an increased genetic susceptibility for this disease, which has been termed the Dupuytren's diathesis (see below).[25] DD usually affects one hand more severely, a trend that is unrelated to hand dominance.[37]

Clinical Presentation

Luck classified the disease into three distinct stages.[35] The earliest *proliferative* stage is marked by the appearance of a small growth of hyperproliferative cells on the palmar fascia (the disease nodule). In the second *involutional* stage, the fibroblasts align along tension lines, and this subsequently develops into the third *residual* stage, when scar-like collagenous cords ultimately result in the fixed flexed contracture of the affected digit[51] (Figure 41.9). The early nodules are usually not painful, although some patients may have discomfort when gripping objects. The residual DD cords are also not painful; however, the resultant flexion contracture does affect hand

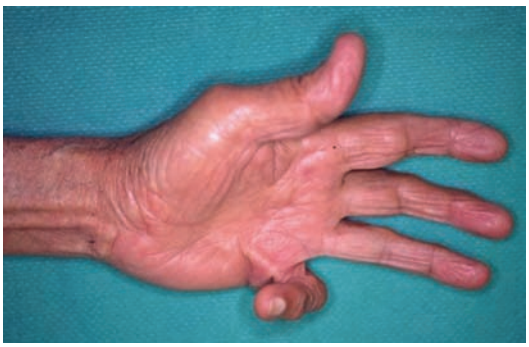

Figure 41.9. Clinical photograph of advanced Dupuytren's contracture of the small finger. Note the prominent precentral cord contracting the MP joint.

function. The progress of the DD nodule into a full-blown disease cord may occur at a variable rate, in some taking many months–years, in others mere weeks–months, and occasionally even in starts and stops. However, it is generally accepted that progress will always occur and that the cords never regress.

Risk Factors and Associated Conditions

A number of risk factors for DD have been described, the strongest of which are a positive family history and a genetic background from Northern Europe with ancestors from the Anglo-Saxon countries, including Scotland, Ireland, England, Scandinavia, and Iceland.[52] Other risk factors cited include alcoholism, diabetes mellitus, epilepsy, neurosyphilis, pulmonary tuberculosis, and smoking. The relationship with trauma and occupation remains controversial, and numerous reports detail the appearance of DD shortly after a localized trauma, including wrist fractures, burns, or crush injuries.[39] Hueston coined the term Dupuytren's diathesis to denote a constellation of factors that predisposed to DD and that may predict more aggressive recurrence after surgery.[25] He included a positive family history, bilaterality of the disease, and ectopic lesions such as knuckle pads and plantar lesions. Other factors compounding the presence of a diathesis are early age of onset and radial-sided disease – presence of associated illness such as diabetes mellitus and histologic factors such as proliferative index in the pathological samples.

DD occurs in increased incidence in conjunction with other fibroproliferative diseases, and patients exhibit an increased incidence of Peyronie's disease (fibrotic plaques on the shaft of the penis resulting in penile curvature), Ledderhose disease (nodules in the plantar instep area of the foot causing painful gait), and knuckle pads (nodules on the dorsum of the PIP joints). Moreover, recent epidemiologic evidence has suggested that patients with DD also suffer from increased incidence and total mortality of cancer, indicating that DD may carry a component of genetic susceptibility to other proliferative disorders of both benign and malignant nature.[21]

Molecular Mechanisms

Despite extensive investigation, the exact molecular mechanisms of DD remain unknown. Previous work has focused on the presence of the myofibroblast as a mechanism, whereby DD results in finger contractures. The myofibroblast is a specialized, highly contractile fibroblast that is also observed during the contractile phase of wound healing.[19] It is thought that myofibroblasts arise from the differentiation of normal fibroblasts, a progression marked by expression of a-smooth muscle actin (a-SM actin), the actin isoforms typical of smooth muscle cells.[19] Once differentiated, the myofibroblast can develop a prolonged contractile force. This force is transmitted from the actin cytoskeleton to the surrounding ECM, resulting in a local contraction of the matrix, which is then thought to be stabilized by deposition of collagen and becoming a permanent contracture.[19]

The presence and action of the myofibroblast and other similarities to the wound healing response, such as in increased collagen III content and upregulation of other wound healing related proteins, such as heat shock protein 47 (Hsp47),[24] and Fibronectin and its various isoforms,[24,65–67] have led to the hypothesis that DD may represent an exaggerated wound healing response.[19,33] Nonetheless, despite continued work on the molecular etiology of this disease, the exact aberrant biochemical pathways remain elusive.

Anatomy

The pathoanatomy of DD has been well studied, and numerous descriptions of the pathological cords in DD dating back to McFarlane's classical description of the affected fascia[38] are available.

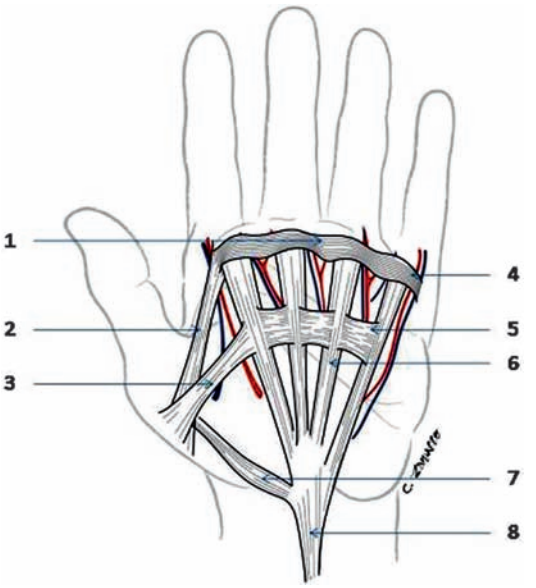

Figure 41.10. The main elements of the palmar fascia. (1) Natatory ligament. (2) Distal commissural ligament of the first web space. (3) Proximal commissural ligament of the first web space. (4) Distal transverse palmar ligament. (5) Proximal transverse palmar ligament. (6) Pretendinous bands. (7) Palmar fascial extension to abductor pollicis brevis. (8) Palmaris longus tendon.

The normal palmar fascia of the hand consists of three layers. The deepest layer (deep anterior fascia) is not involved in DD. The superficial layers contain transverse and longitudinal fibers (Figure 41.10). The most superficial longitudinal fibers insert into the dermis at the level of the distal palmar crease. When these fibers become diseased, they result in palmar pits. Intermediate fibers continue into the digits and contribute to the formation of retrovascular cords. Deeper fibers may bifurcate around the flexor tendon sheath and attach around the MPJ. The transverse fibers are at two levels. The most proximal transverse fibers run at the level of the distal palmar crease and on the ulnar side of the hand are not involved in DD. As these fibers are superficial to the neurovascular bundles, protection of these fibers during excision will aid the surgeon in avoiding damage to the nerves and blood vessels. On the radial side of the hand, the proximal transverse fibers of the fascia become the proximal commissural ligament of the first web space, and this ligament can be involved in DD. The distal transverse fibers run at the level of the web spaces and normally form the natatory ligaments. On the most ulnar aspect of the hand, these distal transverse fibers envelop the abductor digiti minimi and ulnar neurovascular bundle and contribute to the formation of the abductor cord on the ulnar aspect of the little finger. On the radial side of the hand, these distal transverse fibers course into the first web space and become the distal commissural ligament of the first web space. In addition to transverse and longitudinal fibers of the palmar fascia, there are also vertical fibers. These fibers form the vertical septa of Legueu and Juvara, which delineate the longitudinal compartments that contain the flexor tendons and common digital neurovascular bundles.

The digital fascia has also been well described and consists of Cleland's (dorsal to neurovascular bundle) and Grayson's ligaments (volar to neurovascular bundles). These two ligaments envelop the neurovascular bundles and arise from, respectively, the lateral aspect of the phalanges and the volar aspect of the flexor tendon sheath and attach to the dermis of the lateral skin of the fingers (Figure 41.11). The transverse and oblique retinacular ligaments of Landsmeer are not involved in DD.

Involvement of the normal structures of the palmar and digital fascia leads to the formation of pathological cords. These cords are responsible for the digital flexion contractures that are typical of DD. The precentral longitudinal bands transform into the precentral longitudinal cords and are responsible for MPJ contractures. Distally, the precentral cords may be continuous with the central cord, and these cords run from the distal palmar crease to the volar aspect of the proximal phalanx and the PIP joint. These central cords may contribute to PIP joint contractures. The precentral cord may also be continuous with the spiral band, the lateral digital sheath, and Grayson's ligaments. If these structures become diseased, they will give rise to the spiral cord that envelops the neurovascular bundle at the level of the proximal phalanx and can lead to displacement of the neurovascular bundle in a superficial, central, and proximal direction (Figure 41.11). The significance of this is that it places the neurovascular bundle at risk for iatrogenic injury in its abnormal position. The lateral digital sheath, the natatory cord, and the retrovascular structures of Cleland's ligaments may all become involved in DD, and retrovascular cord formation may contribute to PIP flexion and DIP flexion contractures. In rare circumstances,

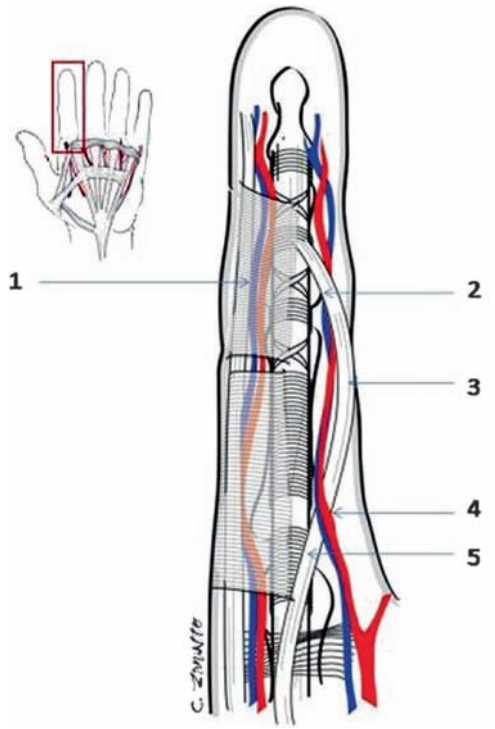

Figure 41.11. Fascial structures in the fingers. On the left side, the normal anatomical localization of the fascial elements and the neurovascular bundle. (1) Fascial elements envelop the anatomically normal neurovascular bundle. Grayson's ligaments are located on the volar aspect of the bundle, whereas Cleland's ligaments are located on the dorsal aspects of the bundle. The lateral digital sheath completes the fascial surroundings of the neurovascular bundle. (2) Distal part of spiral cord arising from involvement of Grayson's ligament. (3) Lateral part of spiral cord arising from the lateral digital sheath. Occasionally, the lateral digital sheath remains dorsal to the neurovascular bundle and involves Cleland's ligaments, leading to a retrovascular cord. Such a cord may lead to flexion contracture of the DIP joint or contribute to DIP joint hyperextension if the PIP joint is contracted. (4) The spiral cord, arising from the precentral bands, the deeper layers of the palmar fascia, or the natatory ligaments toward the lateral digital sheath and Grayson's ligaments, winds itself initially posterior to the neurovascular bundle. When this spiral cord contracts, the neurovascular bundle is displaced in a proximal, superficial, and central direction, putting it at risk for iatrogenic division during surgery. (5) The proximal part of the spiral cord can arise from deeper palmar fascia elements (spiral band) or from attachments of the natatory ligament that envelops the neurovascular bundle.

insertion of retrovascular cords into the extensor mechanism may lead to DIP joint extension deformities and occur simultaneously when severe PIP flexion contractures are present.

Current Treatment

Many patients with DD do not need treatment as their disease is limited and carries no functional consequences. Treatment is indicated for those patients who present with painful and disabling nodules in the palm without finger contractures and those patients who present with progressive finger contractures that interfere with hand function. A good rule of thumb is that contractures are significant when the hand cannot be placed flat on a tabletop. Urgency of any surgery is also modified by the rate of progression and the involvement of the various joints. In general, MPJ contractures are more readily correctable, and even long-standing contractures can usually be corrected by surgery. PIP joint contractures, however, lead more frequently to prolonged joint stiffness, and early intervention should be advocated. Treatment for DD can be categorized as nonsurgical and surgical.

Nonsurgical Treatment Options

Patients with painful nodules may be helped by injection with a corticosteroid. Steroid injection does not affect the progression of the nodule to cord formation, and this treatment should be seen as a means to reduce the discomfort associated with the process of nodule formation but not to halt progression of the disease. No other nonsurgical treatment options have been shown to be beneficial in the long-term. Alternative treatment options that have been tried include splinting, the use of radiation treatment, steroid injection, and injection with enzymes. The latter includes injection with collagenases, and encouraging recent results have shown that these enzymes can break down the disease cords.[6] Nonetheless, the safety profile and longevity of the treatment have not been firmly established, and until more clinical trials have been conducted, this treatment should be considered experimental. It is available only in a small number of academic medical centers.

Surgical Treatment of DD

The primary aim of DD surgery is to restore hand function when the patient experiences problems with daily activities. Usually the patient complains of an inability to put the hand in a pocket, put a glove on, or simply that the finger is in the way because of the flexed position of the

finger. A number of surgical options are available to restore normal finger extension. It remains controversial whether this is best achieved by simply incising the disease cords (fasciotomy) or whether removal of the disease cords (fasciectomy) leads to better long-term results. Several authors have pointed out that incising of the cords may lead to softening of the remaining cords and nodules and that alleviating the tension in the cords may lead to some form of regression of the disease. Others feel that recurrence after fasciotomy is inevitable and that fasciotomy is merely a temporary restoration of motion with almost guaranteed recurrence. The truth is probably somewhere in between, and both approaches have their relative advantages and disadvantages.

Fasciectomy

The most commonly accepted surgical treatment for DD is a partial palmar fasciectomy. This procedure entails the surgical removal of the disease cords with some margin of surrounding normal palmar fascia. Fasciectomy is achieved through a wide surgical exposure employing a combination of longitudinal, transverse, or zigzag incisions (Figure 41.12). This wide exposure allows good visualization of critical structures such as the neurovascular bundles to facilitate preservation of these while completely removing subtle disease elements such as retrovascular cords (Figures 41.13–41.15). Following fasciectomy, the skin can be closed primarily, transverse incisions can be left open, but longitudinal incisions should be lengthened with Z-plasties. Dermatofasciectomy entails a fasciectomy with complete removal of the skin and dermis overlying the disease cord and subsequent placement of a skin graft.[28] Dermatofasciectomy is thought to decrease recurrence rates in patients with Dupuytren's diathesis and may be indicated in patients with severe recurrences. Although recurrence rates have been reported from 0% to 80%, typical recurrence rates are in the range of 20%.[4,53] In patients with repeated recurrence, or when prior surgery has been complicated by nerve or vessel damage, amputations of the affected digits are sometimes necessary, albeit rarely.[28]

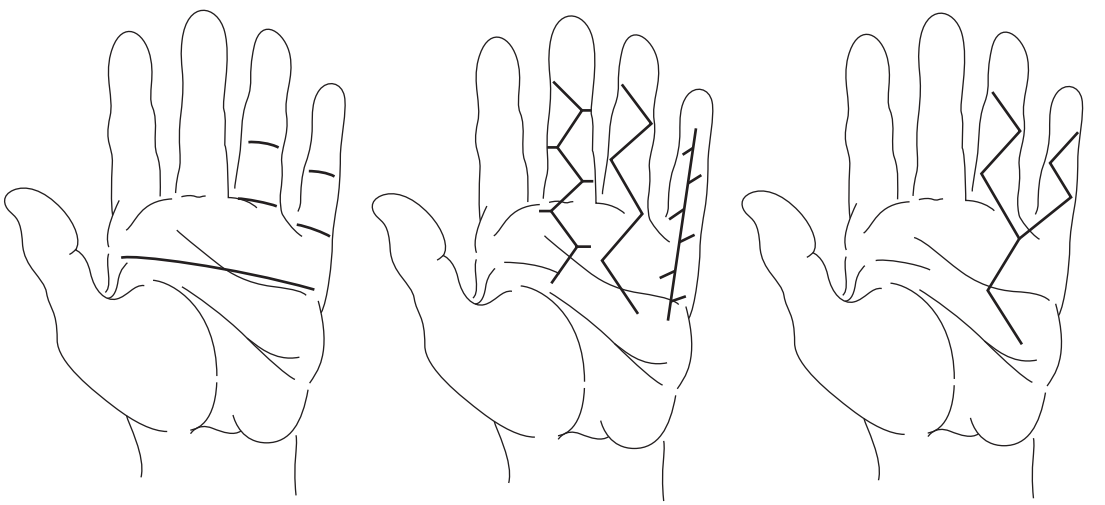

Figure 41.12. Possible incision used to perform Dupuytren's surgery. Left panel shows the use of multiple transverse incisions. This pattern of incisions has been criticized for the need for extensive undermining of the skin and the limited visualization of the critical structures such as neurovascular bundles. In addition, postoperative increased extension tends to open the incision. Some surgeons favor this incision pattern in the palm and leave the incisions to heal by secondary intention (open palm technique). Middle panel: Most commonly a combination of longitudinal (as drawn in the small finger), Bruner-type zigzag incision (as drawn in the ring finger) or a V-Y type of incision (as drawn in the long finger) is used to expose the structures in the digits. When longitudinal incisions are used, Z-plasties at appropriate intervals are required to prevent postoperative scar contractures. Right panel: Digital incisions can be extended into the palm and combined to expose the critical structures in the palm. An example of connecting two digital zigzag-type incisions to expose palmar disease in the ring and small rays is given.

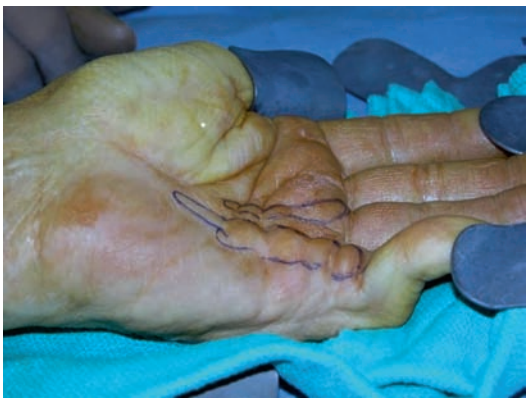

Figure 41.13. Clinical photograph of Dupuytren's contracture of the small and ring fingers. The small finger PIP joint is contracted approximately 80°.

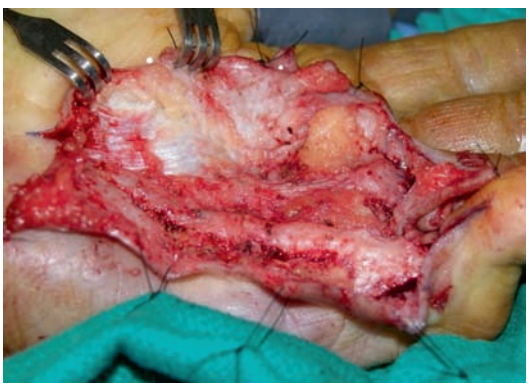

Figure 41.14. Wide exposure of the precentral cord, central cord, and abductor cord.

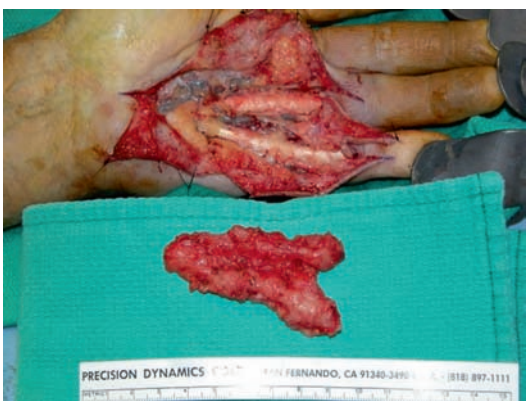

Figure 41.15. Removal of the diseased fascia with preservation of the flexor tendon sheath and the neurovascular bundles.

Fasciotomy

The less invasive fasciotomy can be performed as a minimally invasive procedure with the use of a needle (the so-called needle aponeurotomy) or as a minimally open incision of the cords or minimal resection of the cord.[18] Most surgeons who use this approach would reserve these less invasive variants for contractures that are less than 45° and limited to the MPJ. More distal cords are usually not addressed by needle aponeurotomy or subcutaneous fasciotomy owing to the uncertain relationship to the neurovascular bundles.

Outcomes and Complications

Complications arising from DD surgery are relatively common, and overall rates up to 17% have been reported. Damage to the neurovascular bundles is rare but possible, and an increased chance of this occurs in extreme contractures as well as in revision surgery. Hematoma, skin flap necrosis, and wound infections occur most commonly and can be minimized by meticulous surgical technique, leaving skin flaps as thick as possible, good intraoperative hemostasis, and meticulous closure. Occasionally despite complete resection of diseased tissue, full ROM of the joint cannot be achieved. Several authors advocate continuing with adjunct procures such as joint release, release of the flexor tendon sheath, and volar plate division; however, in our experience, under such circumstances, a relatively poor outcome is frequent. Other late complications include reflex sympathetic dystrophy and persistent loss of range of motion. Permanent sensory disturbances are rare, although most patients do complain of an early subjective loss of sensation. Recurrence of the disease, defined as new disease nodules and cords in an area previously operated, and extension of the disease, defined as new formation of diseased tissue in an area previously not involved in DD, are both common and in some series noted to be as high as 70% within 10 years after surgery.

Successful postoperative treatment for DD requires a combination of active ROM therapy and splinting to regain ROM while maintaining the extension achieved during surgery. Usually, this entails an episode of nighttime and daytime extension splinting, with the patient regularly removing the splint to do dressing changes and ROM exercises. In general, patients underestimate the intensity and duration of therapy that

is required after DD surgery. In our center, it is customary to splint 6 weeks day and night followed by 6 weeks of night splinting.

In summary, DD remains an enigmatic disorder with no known molecular cause. The disease can be debilitating, and surgery is indicated when flexion contractures interfere with daily hand activities. The most common treatment method for DD, the fasciectomy, is an invasive procedure that requires prolonged rehabilitation, the magnitude of which is often underestimated by patient and surgeon. Recurrences are common, and patients should realize that DD surgery at present does not provide a lifelong cure.

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42

Toe-to-Hand Transfers

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Summary

Toe-to-hand transplantation has revolutionized the reconstruction of hands with amputated or absent digits. Although a plethora of different techniques have been described in the literature reporting the transplantation of various combinations, sequences, and components of toes, they generally follow similar principles of harvest and inset and apply replacing like with like tissues. The types of toe transplant that will suffice in many situations are the trimmed great toe (for thumb reconstruction), the second toe variants, and the combined second/third toe transplantations. Although these provide the focus of this chapter, situations requiring other types of transplant are addressed. In addition, the initial surgical approach to the injured hand (that may or will require toe transplantation), the rehabilitation of the donor site and reconstructed hand, and the judicious use of secondary procedures are all often critical to success and are described.

Introduction

The purpose of this chapter is to provide a framework of principles that can be applied when faced with reconstructing non-replantable or absent fingers and/or thumbs with toe transplantation(s). They provide ideal like-for-like elements in terms of nail, pulp, tendons skeleton, and glabrous skin. When properly performed in correctly selected patients, they offer functional and cosmetic results that can be superseded only by replantation.

Initial Management of the Patient with Non-replantable Digital Amputations

For those patients who may undergo toe transplantation, excessive debridements and local flaps on the hand should be avoided.[26] By exercising the key principle of preserving viable tissue during the early stages of management, the future donor-site morbidity of toe harvest can be reduced, the available choice of toe transplant types can be maximized and, at least to some extent, the ultimate outcomes of toe-to-hand transplantations will be improved.[26,29] The general principles and the key reasons for preserving specific tissues and structures during hand debridements are outlined in Table 42.1.[9,24–26,29,]

Timing of Toe Transplantation

Primary toe-to-hand transplantations are being increasingly performed to expedite hand recovery, psychological recovery, and return to work.[17,43,47] Our center strictly reserves this approach for fit, intelligent, well-motivated, and cooperative patients who present with clearly demarcated

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Table 42.1. Principles applied to the preservation of tissues when debriding the injured hand.

Structure	General Pprinciple	Key Ccomments
Bones	5 mm of bone is enough for stable fixation of the transplanted toe.	Preserve native finger joints; never sacrifice their immediately distal, viable bone. Normal finger length can be restored if the amputation stump is not shortened beyond the middle of the proximal phalanx.
Joints	Preserve functioning joint surfaces, capsules, and collateral ligaments.	A proximal joint surface in the transplanted toe can articulate with a preserved distal joint surface in the finger to restore range of motion as well as length.
Flexor tendons	Preserve all tendon insertions and maximal length.	Flexor digitorum profundus provides stability in power pinch. The flexor digitorum superficialis insertion represents the functional length of a finger.
Extensor tendons	Preserve the extensor system.	The extensor apparatus and intrinsic muscle insertions maintain balance in extension for the transplanted toe.
Pulleys	Preserve especially the A2 and A4 pulleys.	Important to prevent bowstringing of the native or transplanted flexor tendons.
Nerves	Preserve viable sensory nerves.	Sensory reinnervation of the transplanted toe will be expedited if viable nerve length is not sacrificed.
Vessels	Preserve healthy vessels.	Good size match for donor vessels will more likely be available if vessel lengths are preserved in the finger.
Skin	Preserve all clearly viable skin.	Although a pedicle groin flap can import fresh uninjured skin, viable native skin is usually of superior quality.

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injuries that do not require additional debridement, soft tissue coverage, or bony reconstructions.[47] An analysis of a consecutive series of our own patients confirmed that primary toe transplantation can be performed within these parameters with as much success as secondary toe transplantation.[47] Whether performed primarily or secondarily, multiple toe transplantations can be completed safely in a single operation to expedite recovery without affecting the final result.[3,34,43] The duration of these operations can be reduced by recruiting additional microsurgical teams that work simultaneously at the hand(s) and foot (or feet).[3]

Assessing the Patient for Toe Transplantation

Choosing the correct toe(s) for transplantation depends on the following: (1) the characteristics of the digit(s) requiring reconstruction, (2) functional requirements, (3) cosmetic requirements, and (4) a consideration of donor-site morbidity. All these should be clearly discussed with the patient, as there is no uniform agreement on which technique should be used in any given situation.

Thumb Reconstruction

The thumb can be reconstructed with great toe variants or with the second toe. The great toe donor site is less pleasing esthetically and has more functional morbidity, but important modifications of the great toe transplantation have addressed these disadvantages.[16,29,33,39] Ultimately, a thumb reconstructed with a great toe tends to be superior to that reconstructed with a second toe.

The reader should be most familiar with the following great toe variants: the total, trimmed, and wraparound great toe transplantations.[16,33,39] In comparison to the thumb, the great toe possesses a broader nail and is fleshier and wider in anteroposterior and transverse dimensions of both soft tissue and skeletal components.[33] This means that total great toe transplantation will usually provide an overly bulky thumb; however, excellent thumb stability and good joint mobility

can usually be granted. The total great toe transplantation is therefore reserved for the patient with a more slender great toe, a severely injured hand, and for whom optimal thumb function is the only major concern.

A cosmetically improved thumb reconstruction can be achieved by reducing each of these differences. The great toe wraparound flap involves transplantation of the great toenail and the skin envelope wrapped around a conventional iliac crest bone graft, which provides the skeletal structure for the reconstructed thumb.[16] This method improves the cosmesis of the thumb and donor site and reduces the functional deficit at the foot but impairs the function of the reconstructed thumb in two ways. First, it does not provide a mobile interphalangeal joint and, secondly, the pulp is unstable as it may swing around the bone graft during prehension. In addition, bone graft resorption and significant donor-site morbidities from iliac crest harvest have been reported.[7,19,23,29,39] The wraparound flap (without the bone graft) is, however, an excellent alternative when reconstructing a degloved thumb that has an intact skeleton.[39] In addition, the wraparound great toe can incorporate part of the distal phalanx to replace a distal amputation of the thumb that has preserved interphalangeal and metacarpophalangeal joint motions, and it can be designed to replace avulsed proximal soft tissues as required.[6,7,23]

The trimmed great toe transfer combines the advantages of these two methods. It involves excision of excessive skeletal, nail, and soft tissues on the tibial side of the great toe for better thumb cosmesis.[33] Joint trimming causes the loss of approximately 10° of interphalangeal motion, but stability of the pulp and construct is maintained.[33] Thus, the trimmed great toe transplantation provides a better cosmetic result than the total great toe without excessively sacrificing functional outcome. This technique is described in detail later.

Use of the second toe for thumb reconstruction is indicated in a patient with one that is close to the thumb in cosmetic dimensions, who requests the minimum donor-site morbidity and does not require optimal thumb function (such as on the nondominant hand or in the elderly).[39] Second toe transplantation becomes a better choice for thumb reconstruction when transmetatarsal transfer is required to restore metacarpal length, because shortening of the great toe metatarsal causes unacceptable impacts on balance and ambulation due to its marginal position. The second toe is, however, structurally weaker than the great toe, it is narrower and has a smaller pulp surface area for opposition, it has a greater tendency to claw, and it can import less skin and subcutaneous tissues to the hand for reconstruction.

Finger Reconstruction

Few patients experience significant functional impairment from single finger losses.[8] Those who require specific digital functions, particularly musicians, or desire superior hand esthetics can benefit from single finger reconstruction by lesser toe transplantation.[28,38,44]

The insertion of the flexor digitorum superficialis tendon into the middle phalanx distinguishes between distal and proximal finger amputations and marks the functional length of a finger.[6,38] Distal finger reconstructions are designed according to one or more lost components: the fingerprinted pulp, the nail complex, its underlying phalanx, and the distal interphalangeal joint. Each can be closely replaced by its corresponding component on the foot. Options include vascularized nail, pulp, hemipulp, onychocutaneous, and wraparound flaps from a lesser toe.[4,6,10–12,36,38,44] The distal interphalangeal joint can be reconstructed with a vascularized joint from a toe.[6] Normal digital length can be restored for stumps as proximal as the middle of the proximal phalanx by including both interphalangeal joints in the lesser toe transplant.[24,25] The aim is to restore functional rather than normal digital length for more proximal amputations.

The principles applied to multiple finger amputations are similar but the reconstructive plan more critical.[14,24,28,36,38] One must thoroughly deduce the requirements of the debilitated hand according to the patient's most important manual skills, and a reconstructive plan with priorities and timings should be discussed and agreed upon.[24] Prehensile ability is always the most important function to restore, but the hand is a complex tool that has evolved numerous different grips and grasps that are suited to different activities. The individual patient's prehensile requirements need to be explored in detail so that a personalized repertoire of grips can be provided by the reconstruction and trained into reality during rehabilitation.[24]

Generically useful grips include pulp-to-pulp prehension that provides a precise pinch, tripod pinch (between the thumb and two fingers) that provides slightly less precision but more power, and the palmar grasp that is useful for handling larger objects. In most patients, the radial fingers are more important for global hand function and fine manipulation (such as writing), whereas the ulnar fingers provide broader hand span and stronger grip strength for cylindrical objects (such as the handle of a hammer).[24,25,28,29,38] At least two neighboring fingers should be reconstructed to provide a tripod pinch for handling precision, a strong hook grip, and lateral stability against the opposing thumb.[25,28,37]

Metacarpal Hand Reconstruction

A metacarpal hand is one that has sustained four proximal finger amputations with (Type II) or without (Type I) proximal thumb involvement.[14,15,29] This means that Type I metacarpal hands may have an intact thumb or thumb amputation distal to the interphalangeal joint, which is the minimum functional length required for precise prehension. Type I metacarpal hands are subclassified according to the level of finger amputations, and Type II, according to the adequacy of thenar function and condition of the basal joint.[14,29] The classification and guidelines for reconstruction take into account the involved digits, the level of amputation for each finger, and the competence of thenar musculature for facilitating thumb abduction.[14,29] Our most up-to-date guidelines have recently been published.[14]

Design, Harvest, and Inset of Different Toe Transplantations

Although several different toe harvests are possible, each shares certain principles. Many of these are also applicable to the composite flaps that can be harvested from the toes.

Choosing the Donor Foot

This consideration mainly regards the side of resultant donor-site morbidity and is influenced by the cosmetic desires and footedness of the patients, particularly if they are involved in competitive sports.[14,24,29] The donor side, whether ipsilateral or contralateral to the recipient site, bears little relevance to the dissection or outcomes for single or combined toe harvests. For combined or single toe harvests, the length of the reconstructed fingers can be suitably tailored by adjusting the position of the osteotomies in the amputation stumps and the transferred toes.[25]

Vascular Pedicle Identification and Dissection

The first step in toe harvest is to identify and trace the vascular pedicle. This is performed under standard tourniquet control. The great toe variants, the second toe variants, and the combined second/third toe unit are the most frequently used toe harvests. These single toes can be reliably based on a single dominant arterial inflow, which is either the first dorsal or first plantar metatarsal artery (FPMA).[25] Combined toe units are usually harvested with a supplementary backup artery and vein.[2] Usually, these backups are not required after the first pair of anastomoses and can be ligated.

In approximately 90% of cases, either the dorsal or plantar system will be clearly dominant in the web space.[40] Direct visual comparison is required for this assessment. Consequently, the most straightforward method for pedicle harvest is to identify the dominant arterial supply first by starting the vascular dissection in the respective web space (this immediately reveals the union between the two arteries); the lesser artery is then ligated, and the dominant supply is traced in retrograde fashion until the desired length is attained.[40] The vascular pedicle must be harvested cleanly without attached fatty tissues; this will ease flap inset later and improve cosmesis in the hand, particularly when performing partial toe transplantation for distal reconstructions.[41]

For a dominant first dorsal metatarsal artery (FDMA) toe harvest (approximately 70% of cases), extension vein grafts are rarely needed.[40] A dominant FPMA occurs in approximately 20% of cases.[40] Its dissection is relatively straightforward up to the deep perforator communicating between the two systems. It is easier to terminate the dissection at this point and extend the artery with a vein graft if additional pedicle length is required.[40] In approximately 10% of cases, neither system will be dominant in the web space.[40]

For these, the dorsal system should be chosen, as the dissection is less demanding.[25,40]

With retrograde pedicle dissection, any proximal anatomic variations are simply traced according to their dominant direction.[40] Routine preoperative angiography therefore becomes superfluous and is recommended only in patients who have a history of foot trauma, congenital foot anomaly, or significant peripheral vascular disease.[40]

Design of the Skin Flaps

The skin flaps and incisions need to be planned once the dominant arterial supply has been identified in the web space. Three principles need to be followed: first, adequate access must be provided for pedicle and toe harvest, second, they need to be designed so that the donor site can be closed primarily without undue tension and, third, incisions should not overlie weight-bearing areas.[3,35,38,41] The following design is applicable for single or combined toe harvests and accounts for each of these principles.

The first incisions are placed on the aspect of the foot that carries the dominant arterial supply to the toe(s). A distally based isosceles triangular flap is designed with: (1) its vertex located 10 mm proximal to the planned osteotomy level and (2) its base corners located at the midpoints of the web spaces either side of the toe(s).[335,38,41] Its apical point can then be extended as a lazy S incision for a dorsal pedicle or as a straight line for a plantar pedicle. The latter must be adjusted so that it does not pass over the weight-bearing areas of the foot, in particular the head of the great toe metatarsal.

Proximal Limit of Foot Dissection

Toe dissections need proceed only until the desired components are available. The level of finger/thumb amputation according to radiographs helps plan the level of toe osteotomy, but the final determinant is the intraoperative findings in the hand. As a rule, if 5 mm of good bone is available distal to an intact interphalangeal joint in the finger/thumb, then there is enough to secure the transplanted toe and preserve that joint.[45] With this in mind, the level of toe amputation should correspond to the level of finger ray amputation.

Specific Types of Toe Harvest

An ability to perform the trimmed great toe, the total/partial second toe, and the combined second/third toe unit will suffice for most reconstructions. Their principles can be applied to other lesser toe harvests, to the combined third/fourth toe unit, and other less common combinations; these are detailed elsewhere.[1,18,21,31]

Trimmed Great Toe Transplantation

This technique provides an adequately stable, strong, mobile, and esthetic thumb with improved donor-site appearance and function compared with the total great toe transplant.[33] The technique reduces the size discrepancy of the great toe to that of the normal thumb (Figure 42.1). Three circumferential measurements are taken from the contralateral (normal) thumb at the following levels: the nail eponychium, the widest girth of the interphalangeal joint, and the middle of the proximal phalanx. Each measurement is increased by 2 or 3 mm and then transposed onto the corresponding points on the great toe. The great toe dimensions will be larger to varying degrees; these excesses are placed on the tibial side of the great toe, and this skin, which is usually between 5 and 8 mm in breadth, will not be included in the transplantation. It should be tapered to the tip of the toe, leaving 2 mm of nail fold to facilitate skin closure. Previously, the nail and nailbed were also trimmed to match the normal thumb,[33] but this is no longer recommended as it risks subsequent nail growth and disrupts the specialized contours of the paronychium, the eponychium, and its vest to produce an unnatural, asymmetrically straight edge to the nail plate.[25]

The vascular dissection begins in the first web space and the skin flaps designed according to the principles described above.[40] Branches of the deep peroneal nerve are preserved. The extensor and flexor tendons are divided at the levels according to the recipient-site requirements and pulled out. The dissection returns to the medial skin flap that will remain on the foot. Dissection begins at the tip of the toe with the plantar-side incision deepened straight down until the periosteum and continues proximally in this plane until the medial collateral ligament of the interphalangeal joint is reached. Dissection continues just superficial to this so that the ligament is retained with the toe harvest. The dorsal-side

incision of the skin strip is then deepened straight down to bone, and the rest of the medial skin flap is elevated away proximally. This leaves a plantar-based flap of periosteum with overlying medial collateral ligament attached to the phalanges and interphalangeal joint. This is elevated as a single unit until the axis of the toe is reached on the plantar side to reveal the underlying skeleton. This flap is termed the perijoint flap and comprises the periosteum, joint capsule, and medial collateral ligament.[33] The bone is now reduced in width with a longitudinal osteotomy executed with an oscillating saw. This removes 4–6 mm of the medial joint prominence along with 2–4 mm of the phalangeal shafts (Figure 42.1a). The bone edges are smoothed with an electric burr. The perijoint flap is redraped over the raw bony surfaces (Figure 42.1b), the dorsal-side excess is excised, and the flap is secured into position with nonabsorbable interrupted sutures. This repair needs to be tight enough to maintain joint stability. The proximal phalanx of the great toe is osteotomized, leaving behind at least 1 cm of its base. This is important to maintain the span of the foot for appearance and the push-off function of the foot for ambulation.[33] The trimmed great toe is now attached only by its vascular pedicle, and the tourniquet can be released to perfuse the transplant for at least 20 min. When the recipient-site team is ready to receive the transplant, its pedicle is divided proximally and the donor site is closed primarily (Figure 42.1c), facilitated by the remaining medial skin strip if necessary. Skin grafts and drains at the recipient site are unnecessary.

Second Toe Transplantation

The second toe is particularly useful for thumb reconstruction when transmetatarsal transfer is required and for the reconstruction of individual fingers in specific individuals (see above). The vascular dissection for the second toe begins in the first web space, and the skin incisions are placed according to the principles described above.[40] When reconstructing adjacent fingers distal to the level of the web space, two separate second toes should be harvested instead of a combined second/third toe unit to

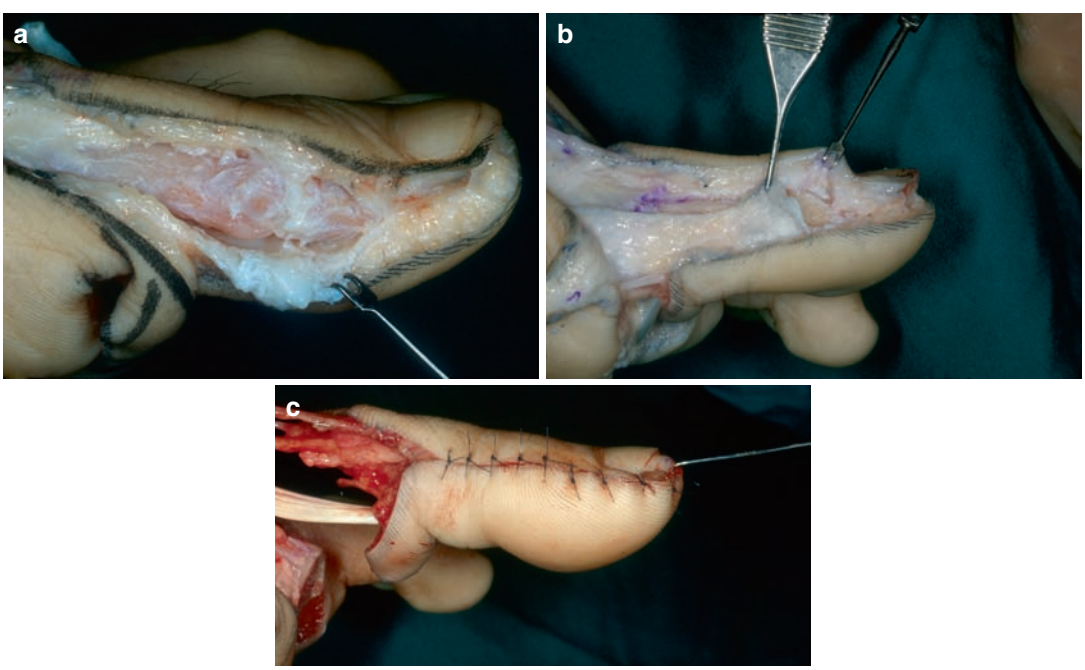

Figure 42.1. Harvest of the trimmed great toe transplant. (**a**) The phalanges, interphalangeal joint, and soft tissues of the left great toe have been reduced according to measurements taken from the uninjured thumb. (**b**) Re-draping of the perijoint flap. (**c**) Trimmed great toe ready for transplantation.

avoid a pseudosyndactylous appearance.[22,35] An alternative approach is to split the second/third toe unit into two separate toe transplants.[3] In addition, the third toe may be selected in certain circumstances where the second toe is not available, when the ipsilateral great toe is or will be used for thumb reconstruction, for nondominant bilateral metacarpal hand reconstruction, or due to a better size match.[31]

The wraparound second toe variant is a useful flap to have in one's repertoire.[36] It can also be designed on a different lesser toe, depending on the availability of toes and the match for the finger requiring reconstruction.[12,36] It is indicated for circumferential or dorsal hemi-circumferential loss of skin and nail distal to the middle of the middle phalanx, with intact skeleton, tendons, and proximal interphalangeal joint.[36] When designing this flap, it is better to place the incisions away from the radial aspect of the index or ring fingers and away from the ulnar aspect of the ring and little fingers. It is essential to skeletonize the neurovascular bundle for an esthetic distal finger. The distal phalanx is always included in the flap to prevent swiveling and instability of the donor pulp.[36] The toe is disarticulated at the metatarsophalangeal joint and the intervening skeleton discarded. This allows esthetic primary closure of the wound without tension and the creation of an inconspicuous web.

Dissection of the total third toe and closure of its donor site follows the same principles as for the total second toe, but since it is usually a reserve for when the second toe is not an option, it is often necessary to base it on the third common plantar digital artery to preserve the second metatarsal pedicle.[31]

Combined Second/Third Toe Transplantation

This option is used when reconstructing adjacent fingers that have been amputated proximal to the web space (Figure 42.2).[22,35] In this situation, it is superior to transplanting separate toes, because a normal looking digital web can be provided, only a single set of recipient vessels is usually required, only one donor site is required, and dissection time is reduced. The reconstructed fingers, however, will be shorter. The best global hand function and cosmesis can be attained when the remaining fingers are of relatively uniform length, approximating to the normal length of the little finger.[22,35] When the amputation is through the metacarpophalangeal joint but preserves the metacarpal head articular surface, the joint can be reconstructed by articulating the surface of the proximal phalanx of the toe and closing the joint with the donor capsule; this produces a range of motion of approximately 52°.[20] If the metacarpal surface has been damaged or is absent, transmetatarsal toe transplantation is performed for length. The vascular dissection, design of skin flaps and incisions, the levels of soft tissue harvests and osteotomies, and the closure of the donor site are according to the principles described above.

Recipient-Site Preparation

The hand is prepared under loupe magnification and standard tourniquet control. Any digital amputation stump should be opened with a cruciate incision centered on the digit's distal point to produce four equal proximally based mobile triangular skin flaps; these will interdigitate with the triangular skin flaps carried with the toe transplant.[41,44] Scarred and subcutaneous tissues should be trimmed to reveal the distal bone end. A minimum of 5 mm of good quality bone is required distal to the next available joint for toe fixation.[45] If there is not enough phalangeal bone available for toe fixation at the metacarpophalangeal joint, it should be disarticulated, preserving the capsule and cartilage, and the toe transplant disarticulated for composite joint reconstruction as described earlier. The integrity of the extensor system is essential for finger function; it should be identified distally and minimally disturbed. The flexor tendons need to be located if they have retracted proximally or left attached to their respective phalanx. The pulley system should be maintained as far as possible to obtain maximum tendon excursion. Tenolysis is performed if required. If no flexor is available, the adjacent flexor digitorum superficialis tendon can be transferred primarily.[25] The digital or common digital nerve stumps are identified next and debrided until normal looking fascicles are visible under the microscope. The recipient artery for thumb reconstruction is usually the radial (in the snuffbox) or the princeps pollicis artery.[25] The common digital or digital arteries are good options for fingers. Suitable recipient veins are usually abundant on the dorsal hand.

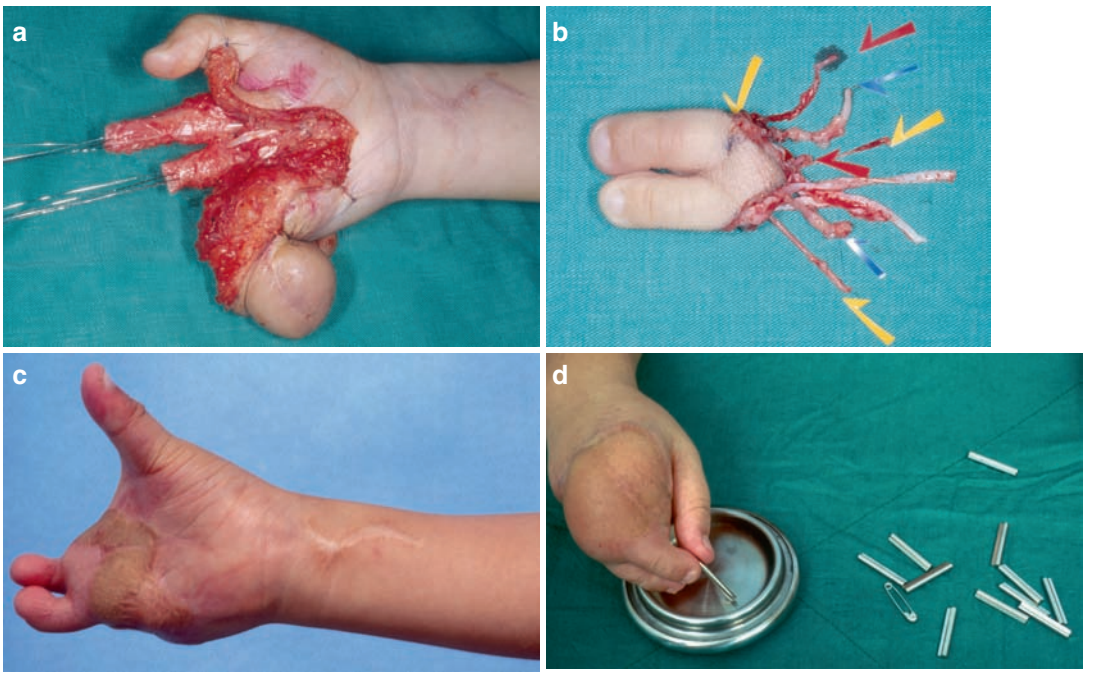

Figure 42.2. Combined second/third toe transplantation for Type I metacarpal hand reconstruction. (**a**) The amputation stumps of the radial rays have been prepared; note that the excess groin flap tissues (performed following initial debridements 9 months previously) are preserved and the positioning of the intraosseous wires. (**b**) Combined second/third toe transplant ready for revascularization in the hand; note that two arteries and two veins have been skeletonized. (**c** and **d**) Long-term result showing broad grasp and accurate pulp-to-pulp pinch of narrow objects.

Insetting Toe Transplants

Osteosynthesis is performed first with two interosseous wires in parallel per digit.[45] This is a straightforward inexpensive and quick method that provides adequate stability for early mobilization to minimize tendon adhesion formation. It also allows an initial slight mobility through splinting for correcting fine rotational or angular malalignments that may declare after swelling has subsided.[45] Extensor tendons are then repaired with all toe joints in maximal extension to reduce extension lag and flexion deformity followed by the long flexor tendon of the digit. For lesser total toe transplantations only, a single Kirschner wire is now driven across the distal and proximal interphalangeal joints in full extension.[6,29,41] The donor and recipient nerves are coapted end to end under the microscope and the vascular pedicle tunneled, if required, to the recipient vessels. The skin flaps on the hand and toe can be smoothly interdigitated when planning has been properly executed according to the presented principles. The arterial then venous anastomoses complete the transplantation procedure.

Postoperative Care and Rehabilitation

Like any free flap, toe transplantations are ideally cared for in an intensive care setting for the first several days. Specifically, the hemodynamic status of the patient is monitored invasively and kept optimized and the vascularity of the toes assessed regularly by specialized nursing staff. The digits are kept uncovered for monitoring, but the proximal palm and wrist are loosely wrapped and the limb kept slightly elevated to reduce edema. Digital motor rehabilitation commences as early as the second or third postoperative day and progresses in five distinct stages led by the dedicated hand and occupational

therapists. This regimen results in less stiffness, fewer tendon adhesions, and an early return to activities.[13,14] Sensory re-education begins once the patient can feel vibrations from a 30-cps tuning fork applied to the transplanted toe tip.[5,13] Later phase sensory re-education commences once touch sensation has begun returning.[13,30]

Specifically for toe to finger transplantations, the Kirschner wire placed across the two interphalangeal joints needs to be withdrawn from the proximal joint after 2–4 weeks and from the distal joint another 2 weeks later.[24] Night splints that position the finger into maximal extension should be used for at least 1 year.[6,29,41] These two measures are critical to reducing the development of lesser toe clawing.

The patient is allowed to walk on the donor foot after the second week but only with heel weight bearing and with absolutely no transfer to the anterior foot.[25] Skin sutures in the foot should not be removed earlier than 3 weeks postoperatively. After 4 weeks, the patient is allowed to walk with a normal gait and then with normal shoes after 6 weeks if the wound has healed.

Donor-Site Morbidity

Wound dehiscence, partial skin necrosis, and hypertrophic scarring on the donor foot are rare if tension-free closure is achieved and postoperative care follows according to the presented principles.[25] Clearly, the appearance of the foot will be altered according to the harvested toe transplantations. For great toe transfers, it is important to maintain at least 1 cm of the proximal phalanx for the reasons already explained.[33] The donor feet before and following second toe (in 36 patients) and combined second/third toe transplantations (17 patients) have recently been biomechanically analyzed during stance and walking in our center.[25] Both options are associated with minimal donor-site morbidity if harvested and closed according to the methods described herein.[25]

Secondary Procedures

Functional and/or cosmetic enhancement of the reconstructed digits with secondary procedures is not uncommon. In a previous study, secondary procedures for functional enhancement were performed in 19 of 133 toe-to-hand transplantations in our center.[46] An unfortunate problem that occurs when the triangular flaps on the toe and/or hand have been improperly designed is the so-called "cobra" deformity at the toe-finger junction.[41] It is not easy to correct and much better prevented by proper planning during the initial toe transplantation. Pulp plasty improves both cosmesis and pulp function and is now actively encouraged in this center.[32,41]

Conclusion

Toe transplantation has revolutionized the reconstruction of missing fingers and thumbs. They provide unique functional and esthetic advantages that are only superseded by replantation of the native digit(s). By respecting the several principles presented herein, with thorough patient consultation, and with a few key types of toe harvest available within one's repertoire, the results of single and multiple toe transplantations can be optimal and provide rewarding results for the patient and surgeon alike.

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43

Brachial Plexus Injuries and Repair

David Chwei-Chin Chuang

Summary

Closed traction injuries of the brachial plexus are always crippling. For somebody who has nothing, a little is a lot. The healing process, surgeon, patient, and the rehabilitation program are the main factors influencing the outcome of the treatment. The healing process involves age, degree, extent and location of injury, and the time interval between injury and surgery. The surgeon can directly influence the overall outcome, although assurance of functional restoration is unpredictable. A thorough knowledge of the anatomy of the brachial plexus, classification of the brachial plexus injury (BPI), types of brachial plexus injury, timing of surgery, techniques for nerve repair, postoperative management and rehabilitation, and familiarity with conventional hand reconstruction for palliative reconstruction for sequelae deformities are all basic requirements for the brachial plexus reconstructive surgeon. The patient's desire and cooperation are also important determining factors, especially for patients who receive nerve transfer. Microneurovascular transfer of functioning free muscles adds additional techniques to enhance the results. Throughout the understanding of the complexity of nerve and nerve reconstruction, outcome for the patients with brachial plexus injury and surgeons is more optimistic now.

Abbreviations

BPI	Brachial plexus injury
CC7	Contralateral C7 spinal nerve
CMB	Cervical motor branches
IC	Intercostal nerve
Ph	Phrenic nerve

The Anatomy of the Brachial Plexus

The "classical" constitution of the brachial plexus is formed by the anterior primary rami of the lower cervical (C5–C8) and the first thoracic (T1) spinal nerves, which give motor innervation to muscles of the shoulder, including all anterior and posterior chest muscles related to glenohumeral joint movement, muscles of the entire upper limb (extrinsic and intrinsic muscles), and sensation of the entire upper limb except the skin on some part of the medial aspect of the upper arm. In the so-called "prefixed brachial plexus," C4 provides a significant contribution to C5 (Figure 43.1). In the so-called "postfixed brachial plexus," T2 has significant contributions to T1. The adjoining of the ventral root and the dorsal root forms each spinal nerve. Each root is in turn formed by a number of rootlets. The dorsal roots carry sensory information to the CNS, whereas the ventral roots convey motor fibers to the muscles.

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The cell bodies (neurons) of the motor fibers locate in the anterior horn of the spinal cord, whereas the cell bodies of the sensory fibers reside in the dorsal root ganglion. The dorsal root ganglia are located within the intervertebral foramen, immediately outside the dura mater of the spinal cord. The dorsal and ventral roots unite a few millimeters distal from the ganglion to form a mixed spinal nerve between the scalene muscles (anterior and middle). Out of the scalene muscle, the postganglionic spinal nerves make a first union to form the three trunks: upper (C5 and C6 spinal nerves), middle (C7 itself), and lower (C8 and T1) trunk. Each trunk divides into anterior and posterior divisions just proximal to or directly under the clavicle. The nerves exchange fibers and form the second union, just distal to the clavicle and below the pectoralis minor muscle, termed "cord": the nerves from the anterior divisions pass anterior to the subclavian artery and form the lateral and medial cord, whereas the nerves from posterior divisions pass posterior to the subclavian artery to form the posterior cord. Each cord has two or more terminal branches to the peripheral. Numerous anatomical variations of the brachial plexus do exist and should always be kept in mind.[1–3]

Figure 43.1. Prefixed brachial plexus. C4 provides a significant contribution to C5.

Classification of the Brachial Plexus Injury

Brachial plexus injury (BPI) can be classified into four levels of injury[4] (Figure 43.2). Injury before the dorsal root ganglia is called "Level 1, Preganglionic Root Injury," localized at or inside the dura. Injury after the ganglion is called "Level 2, Postganglionic Spinal Nerve Injury," localized between the scalene muscles. In Level 2 injury, usually a layer of dense fibrous tissue appears

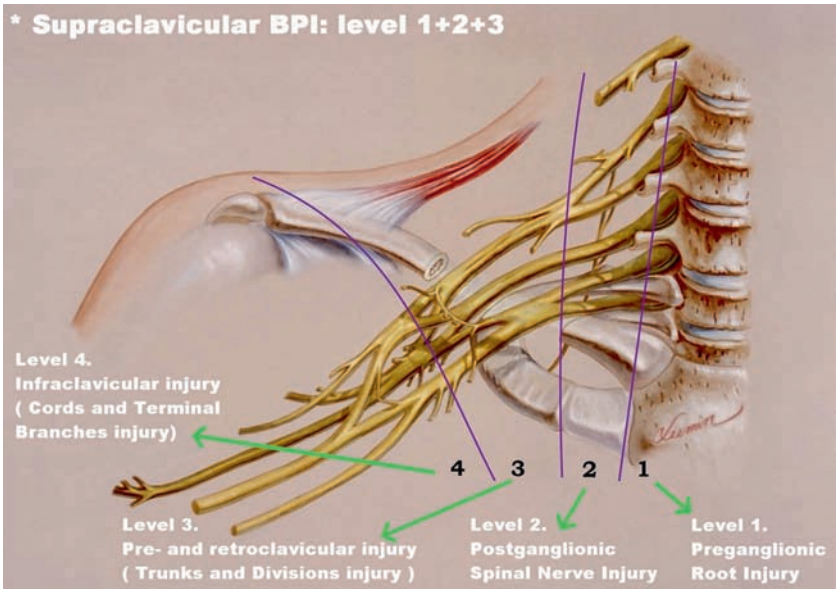

Figure 43.2. Classification of the brachial plexus injury.

over the injured spinal nerves, following removal of the scalene anterior muscle. Injury out of the scalene muscle is called "Level 3, Pre and Retroclavicular Injury." Injury below and out of the pectoralis minon is called "Level 4, Infraclavicular Injury." Level 1, 2, and 3 injuries are all called "Supraclavicular Injury." In supraclavicular injury, part of spinal nerve preganglionic root injury may associate with different levels of injury to other spinal nerves such as root avulsion of C7-T1 (Level 1) accompanying rupture of C5 and C6 spinal nerves (level 2) or rupture of upper trunk (level 3 injury). Level 4 injury may extend to Level 3 but rarely accompany Level 1 or 2 injury of other spinal nerves except in traction avulsion amputation of the arm.[5] Both infraclavicular and supraclavicular BPI have their own characteristically clinical pictures, different surgical approach, and different results. Clinical differential diagnosis is very important in respect of surgical procedure and prognosis. Supraclavicular nerve repairs or nerve grafts have a higher incidence of aberrant reinnervation than infraclavicular repair. Infraclavicular plexus lesions usually have better prognosis than supraclavicular lesions.

Types of Brachial Plexus Injury

There are, in general, three types of peripheral nerve injury: nerve division, nerve rupture, and nerve avulsion. Brachial plexus injury is the same. Nerve division means a nerve is transected into proximal and distal stumps by sharp instruments such as knife or scissors penetrating or during an iatrogenic injury. Nerve rupture means a break in continuity of a nerve by traction force, having two disrupted ends (proximal and distal) with a gap. Nerve avulsion means the tearing of a nerve by traction from the origin (proximal avulsion) or from its target (distal avulsion).

Nerve division does not have nerve deficit, but it may have nerve gap due to nerve retraction. Nerve rupture has a longer nerve gap due to nerve defect after stump trimming or neuroma resection and nerve retraction. In nerve avulsion, usually both stumps cannot be found simultaneously in one operative wound: proximal stump cannot be found in proximal avulsion, and distal stump cannot be found in distal avulsion. The second operative wound is usually required for missing stump exploration.

Brachial plexus injury may be caused by trauma (open or closed BPI), iatrogenic accident, compression, tumor, infection, toxin, inflammation, and irradiation. Closed traction injuries are the most common seen in traumatized adult patients. The majority of closed BPI in the adult population are preganglionic root injuries: avulsion or rupture. Nerves may be ruptured at preganglionic roots/rootlets or avulsion from the spinal cord. Both are called root injury. Rupture injury with neuroma formation is the most common finding in level 2, 3, or 4 injury. Distal avulsion is commonly seen in axillary, radial and musculocutaneous nerves.

Surgical Treatment

Surgery is the treatment of choice for adult BPI, either closed or open injuries.[6–10] Problems in surgical treatment algorithm include timing of surgery (primary or secondary repair), technique of exploration (supraclavicular, infraclavicular dissection, connection between supra- and infraclavicular, and other donor nerve dissection), selection of technique for repair (neurolysis, neurorrhaphy, nerve grafting, nerve transfer, functioning free muscle transplantation), reconstructive strategy (priorities of reconstruction) for treatment goals, and finally palliative reconstruction for the residual deformities.

Timing of Surgery

Immediate or Early Primary

In the case of an open injury in the neck region by a knife or other sharp objects, causing significant motor and sensory deficits, division of brachial plexus is highly suspected. Exploration and nerve repair immediately or few days after trauma are indicated. Direct end-to-end neurorrhaphy with 6-0 to 8-0 nylon for supraclavicular spinal nerve or trunk lesions or 8-0 to 9-0 nylon for infraclavicular lesions is highly possible, and excellent results can be expected (Figure 43.3a–d). The golden time for primary end-to-end repair is within 1 week for supraclavicular and 2 weeks for infraclavicular penetrating lesions. After the golden time, nerve grafts are usually required following neuroma resection.

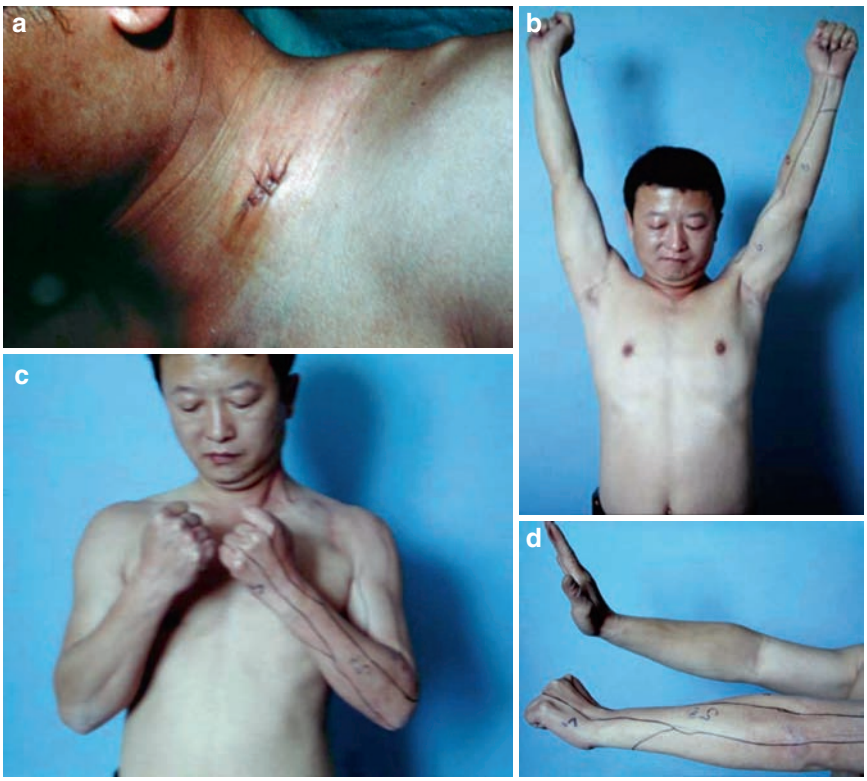

Figure 43.3. (a) A penetrating injury in the neck by sharp knife causes complete division of the upper trunk, middle trunk, and lower trunk. Immediate repair was directly done. (**b–c**) 2 years after nerve repair, complete recovery of shoulder elevation, elbow flexion and extension, and wrist and finger flexion and extension, except hand intrinsic muscles, extensor digitorum communis, and abductor pollicis longus.

Secondary Nerve Repair: Delayed Repair

There are three types of secondary repair: early delayed repair (nerve repair within 1 month for established diagnosis of open injury or within 5 months for closed injury); late delayed repair (repair more than 6 months after injury); and late repair (repair more than 1 year after injury). If the extent of brachial plexus injury was obvious during the primary exploration for vascular repair, then it is not necessary to wait longer for the secondary nerve repair. Early repair within 1 month is recommended because of less scar, easier dissection, and for performing nerve reconstruction straightly. However, for closed BPI, the main objective of the delay is to establish the diagnosis, including degree, site, and extent of the lesion. Management of these patients can be considered in six stages: Stage 1: Stabilization stage (the first month), including stabilization of vital signs, bone fracture, or joint dislocation. Stage 2: Diagnostic stage (the second month), including clinical examination and investigations to establish the diagnosis of the lesion condition; start physiotherapy to prevent soft tissue swelling, joint stiffness, and slow muscle atrophy by electric stimulation; begin psychological education before surgery. Stage 3 Surgery (the third to fifth month): If the patient fails to show a return of function at 3 months, surgical intervention is indicated. Nerve repair within 5 months seems to make no big difference when compared with surgery at the third month, in results of our series. However, results of muscle grading after nerve reconstruction more than 6 months after injury are usually significantly decreased compared with the denervation time within 6 months. It is not wise to perform brachial plexus reconstruction alone more than 6 months after injury. Local muscle transfer or functioning free muscle transplantation to enhance the results is often necessary.

Late Repair for Chronic Paralysis

In late repair for chronic cases (1 year after injury), the muscles have undergone long-time denervation and atrophy, which are replaced by connective tissue and fat. Late nerve repair in such circumstances is useless and usually leads to poor results. Good physical therapy and constant electric stimulation can only postpone the time of muscle atrophy but not save the muscle eventually. Nerve repair in such late cases is usually useless. Functioning free muscle transplantation,[11,12] or banked nerve grafts from ipsilateral or contralateral nerve transfer, followed by secondary functioning free muscle transplantation[13,14] is the option for chronic case reconstruction.

Technique for Nerve Repair

The available techniques in brachial plexus surgery include (1) neurolysis, (2) direct nerve repair, (3) nerve repair with nerve grafts (free or vascularized), (4) nerve transfer, and (5) functioning free muscle transplantation.

Neurolysis

Neurolysis is indicated only in lesions in continuity but preserved intraoperative nerve action potentials. It is to free the lesioned nerve from scar and to facilitate nutrition and axon growth. It might be epifascicular epineurectomy (external neurolysis) or interfascicular epineurectomy (internal neurolysis). For the supraclavicular BPI, external neurolysis is often adequate and there is no need for internal neurolysis. The relationship between neurolysis and recovery is difficult to prove and remains controversial among surgeons.

Direct Nerve Repair

End-to-end coaptation without nerve grafts is possible only in clear nerve division cases. It is essential that the coaptation is performed without tension. The techniques for direct coaptation without tension include (1) sufficiently mobilizing the respective nerve ends and (2) performing traction suture in the supraclavicular BPI with 6-0 nylon for the epineurium and 8-0 nylon for the internal group fascicular suture. Postoperative immobilization is required for at least 3 weeks.

Nerve Grafting

Nerve grafting is the predominant technique employed in brachial plexus repair. A tension-free nerve graft is better than a primary repair under tension. There are two types of nerve grafts: free and vascularized nerve graft. Sources of free nerve grafts are sural and saphenous nerves in most of the cases. Sometimes, the medial cutaneous nerves of the arm and forearm, lateral cutaneous nerve of the forearm, superficial radial nerve, lateral femoral cutaneous nerve, superficial peroneal nerve, or split ulnar nerve are used. The outcome of nerve grafting is influenced by the length of the nerve grafts, presence of scar tissue at the wound site, number of grafts used, and presence of healthy-appearing proximal stump available for grafting. Vascularized ulnar nerve graft is indicated in root injury of the C7-T1. A pedicle vascularized ulnar nerve graft based on superior ulnar collateral vessels is usually harvested from the wrist, including the deep motor branch, superficial volar digital branches, and the dorsal digital branches upward to the proximal arm level. A pedicle reverse vascularized ulnar nerve graft with the loop technique can be applied from the trunk or root (usually upper trunk or single C5) to the lateral and posterior cord and median nerve. A free vascularized ulnar nerve graft can be harvested segmentally and transferred, passing through subcutaneous tunnel or through prevertebral space. Vessel anastomoses are required during transplantation. Vascularized ulnar nerve grafts are used frequently in contralateral C7 elongation and transfer.[15]

Nerve Transfer

Nerve transfer (or neurotization) is a surgical option that intentionally divides a physiologically active nerve with low morbidity and transfers it to a distal, more important, but irreparable, denervated nerve. The procedure is better done within the golden time, which is defined as within 5 months of injury,[16] in order to reactivate the paralyzed muscle or muscle groups early (4–6 months postoperatively), effectively and successfully (M4 muscle strength). Neurotization can be broadly classified into four categories: extraplexus neurotization, intraplexus neurotization, close-target neurotization, and end-to-side neurorrhaphy neurotization.[13] Combined neurotization such as combined extraplexus and intraplexus for

treatment of rupture and avulsion of brachial plexus injury or combined extraplexus and close-target neurotization simultaneously for treatment of the upper plexus avulsed injury have now become an available option for a possible one-stage full reconstruction.

Extraplexus Neurotization

Extraplexus nerve transfer means transfer of a nonbrachial plexus component nerve to the avulsed brachial plexus for neurotization of a denervated nerve. The reported donor nerves in common use are mostly for motor reinnervation, including the phrenic nerve (Ph), spinal accessory nerve (XI), deep motor branches of the cervical plexus (CMB), intercostals nerve (IC), hypoglossal nerve (XII), and contralateral C7 (CC7) spinal nerve[13,16] (Figure 43.4a–d).

Intraplexus Neurotization

Intraplexus nerve transfer is used in cases of nonglobal root avulsion, in which at least one of the spinal nerves is still available for transfer, not to its original pathway but to other more important nerves. For example, in the case of C5 rupture and C6 avulsion or C5 and C6 rupture at the upper trunk but the C5 stump is more healthy than C6, the C5 fibers are transferred to C6 or the anterior division of the upper trunk for elbow flexion. The posterior division of the upper trunk or suprascapular nerve is then innervated by the injured C6, or combined extraplexus neurotization with Ph and/or XI nerve transfer for shoulder function is performed. Such intraplexus neurotization is individualized depending on the intraoperative findings and judgment, surgeon's philosophy, patient's condition, and requirements.

Close-Target Neurotization

Close-target nerve transfer is a procedure of distal nerve transfer,[17] providing a direct coaptation at a more distal site closer to the end organ targets, muscle or skin, thus achieving faster recovery of the motor and sensory outcome. In close-target nerve transfer, however, it could be extraplexus neurotization, such as XI nerve transfer to the suprascapular nerve through the upper back trunk approach or intraplexus neurotization, such as long head of triceps transfer to the axillary nerve through the posterior arm or axillary fossa approaches, partial ulnar nerve transfer to the musculocutaneous nerve through the medial upper arm approach, partial median nerve transfer to the branch of the brachialis in the arm or to the radial or posterior interosseous nerve in the forearm, and branch of the anterior

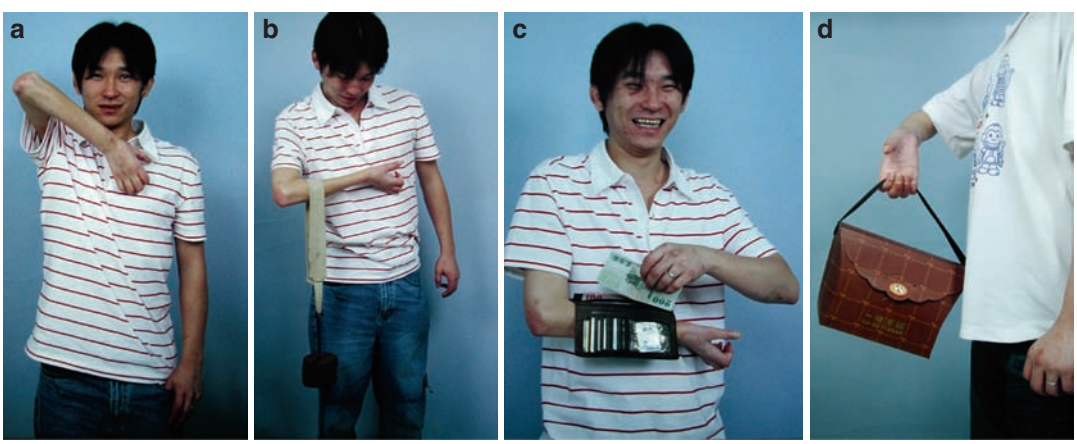

Figure 43.4. A case of total root avulsion (C4-T1) of the right upper limb. Results 5 years after the multiple nerve transfers: (1) spinal accessory nerve transfer to the suprascapular nerve, (2) hypoglossal nerve transfer to the posterior division of the upper trunk with a sural nerve graft; both 1 and 2 transfers are for shoulder function; (3) intercostal nerve (T3-5) transfer to the musculocutaneous nerve for elbow function; (4) contralateral C7 transfer to the median nerve with a vascularized ulnar nerve graft for the hand function. (**a–c**) The patient achieves improving shoulder elevation and elbow flexion functions; (**d**) The patient also achieves hand function after wrist fusion and tendon transfer from flexor carpi radialis and palmaris longus to the third to fifth flexor digitorum profundus.

interosseous nerve transfer to the deep motor branch of the ulnar nerve. The advantages of proximal nerve neurotization versus close-target neurotization are subject of much debate. Close-target nerve transfer has the advantages of direct nerve coaptation without the need for nerve grafting, a short operating time as dissection in the traumatized scar zone is avoided, and the nerve stumps are healthy with no scaring. The major disadvantage of the technique is the increased clinical or subclinical deficits from dividing the donor nerve in a more distal region. The proximal source nerves such as the spinal nerves or brachial plexus nerves are more powerful, easier for brain cognition, and are mixed nerves, with which partial division produces less deficits than close-target nerve transfer. Intentional neglect of the proximal, original, powerful mother nerve and performing only direct distal coaptation are not theoretically accepted. Close-target neurotization is appropriate in cases of confirmed irreparable proximal brachial plexus lesions, such as avulsion, neurofibroma (benign or malignant) excision, or intrinsic nerve palsy of the median or ulnar nerve or single terminal nerve palsy such as isolated axillary nerve palsy. It is not indicated in cases of rupture injury of the brachial plexus, where the proximal stump is still available for neurotization.

End-to-Side Neurorrhaphy Neurotization

End-to-side neurorrhaphy (terminolateral neurorrhaphy) neurotization is a technique to transfer the distal end of a paralytic and irreparable nerve to the side of an intact nerve with or without an epineurium window with no need for nerve transection. Its use has been reported for both motor and sensory reconstruction. The author has never used this technique for brachial plexus injury.

Postoperative Management and Rehabilitation

Immediate postoperative splinting for 3 weeks is required after nerve grafts or nerve transfer. Thereafter, retraining and rehabilitation should start, including physiotherapy (to avoid joint stiffness), muscle stimulation (to delay muscle atrophy), brain cognition, biofeedback, and occupational therapy. Patients should be followed up periodically every 3–4 months by their surgeon and the physical therapist (twice a month). Patients are encouraged to have electric muscle stimulation at home (twice a day) regularly. In cases of intercostal nerve transfer, passive shoulder elevation remains restricted to less than 90° for 6 months. Regular follow-up in the rehabilitation center and outpatient clinics is tremendously important and should be explained to the patients before and emphasized again after the surgery. "Induction exercise" is an important muscle exercise for patients with nerve transfer. For instance, after intercostal or phrenic nerve transfer, as soon as the reinnervated muscles start to move with deep breathing, the patients are directed and encouraged to run, walk, or climb hills to encourage numerous deep breaths, which can induce further stimulation to the reinnervated muscles. Similarly, tongue to palate push-up exercise in hypoglossal nerve transfer, shoulder adduction, or grasp exercise of the contralateral (or healthy) limb in contralateral C7 transfer all have the same effect. "Induction exercise" is indicated in all cases of nerve transfer and is started when the movement of the innervated muscles is palpable (M1).

Palliative Reconstruction for Sequelae Deformities

Palliative reconstruction can be considered when sequelae deformities persist after maximal recovery, either after spontaneous incomplete recovery or following nerve reconstruction. Palliative reconstruction procedures include tendon or muscle transfer, functioning free muscle transplantation, tenodesis, and arthrodesis. Alternatively, orthotics and prosthesis can be used for final assistance. Patients in this stage should be encouraged to recognize their disabilities and make use of the regained but not completely recovered limb. In a failed reconstruction, or if the patient perceived the injured limb as a useless dead weight, amputation might be considered. However, intolerant pain is rarely an indication for amputation since deafferentation pain is a central pain rarely affected by the peripheral amputation. Amputation of the injured arm does not cure the pain.

Conclusion

Closed traction injuries of the brachial plexus are always crippling. For somebody who has nothing, however, a little is a lot. The healing process, surgeon, patient, and the rehabilitation program are the main factors influencing the outcome of the treatment. The healing process involves age, degree, extent and location of injury, and the time interval between injury and surgery. The surgeon can directly influence the overall outcome, although assurance of functional restoration is unpredictable. A thorough knowledge of the gross and internal anatomy of the brachial plexus, an understanding of nerve pathophysiology, meticulous microneurovascular skills, and familiarity with conventional hand reconstruction are all basic requirements for the brachial plexus reconstructive surgeon. The patient's desire and cooperation are also important determining factors, especially for patients who receive nerve transfer. Intensive rehabilitation program should include intensive physiotherapy, treatment of pain, and direction in using the crippled limb to return to normal work and life or retraining for another occupation. Certainly, the outcome for patients with brachial plexus injury and surgeons is now more optimistic.

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Part VII

Trunk and Lower Extremity

44

Trunk Reconstruction

Mark D. Walsh, Michael R. Zenn, and L. Scott Levin

Summary

Reconstruction of the trunk requires a comprehensive understanding of anatomy and advanced techniques of reconstructive surgery. The trunk comprises three large subunits, including the sternum and chest wall, the abdominal wall, and the posterior trunk. Indications for trunk reconstruction generally arise from one of four clinical scenarios. They are infection, trauma, tumor resection, and radiation damage. Management of these defects should adhere to the well-established principles of reconstructive surgery, including wide debridement to healthy tissue and the use of well-vascularized tissue coverage. Commonly used methods of reconstruction include skin grafting, tissue rearrangement, local rotational flaps, and free tissue transfer. The use of prosthetic material may also be required. Methodical planning and multispecialty cooperation will enable reconstruction of even the most complex defects.

Abbreviations

VRAM	Vertical rectus abdominus myocutaneous
TRAM	Transverse rectus abdominus myocutaneous
TFL	Tensor fascia lata

Introduction

Reconstruction of the trunk requires an understanding of complex anatomy as well as advanced techniques of reconstructive surgery. The spectrum of defects can be extremely broad, ranging from loss of soft tissue to extensive full-thickness resections creating bony defects and exposed viscera. The etiologies of the underlying injuries are diverse, and the solutions necessary to obtain coverage may need to be quite creative.

The trunk may be subdivided into several regions, each with its own unique anatomy and reconstructive challenges. These areas include the anterior and posterior thorax, abdomen, and posterior trunk. A comprehensive approach to reconstruction may be initiated with a thorough understanding of the relevant anatomy and physiologic function of each region, adherence to the well-established principles of soft tissue coverage, and familiarity with a variety of prosthetic materials that may be necessary to supplement or reinforce available tissue coverage.

There are a few universal principles that should be considered preoperatively and intraoperatively for any surgical procedure. Beginning with the history and physical examination, the preoperative status of the patient should be evaluated. It is important to note any underlying medical or surgical history that may affect the patient's ability to withstand surgery. For example, in patients with underlying pulmonary disease, a large chest wall resection may compromise

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their respiratory status further. Similarly, in patients with a previous thoracotomy, flap design may be precluded by previous surgical injury.

Medications should be reviewed for drugs that may result in immune compromise or reduced wound healing. Anticoagulants and antiplatelet drugs should be noted. A history of smoking is also very important, as it may have implications for flap survival and wound healing. Laboratory values, including a complete blood count, coagulation evaluation, and markers of nutritional status, should also be considered. The preoperative nutritional status should be optimized before undertaking a large reconstructive procedure.

Previously radiated individuals comprise a unique patient population. Radiation damage creates fibrotic tissue with vascular compromise. Radiation damage is difficult to delineate and is frequently underestimated. Reconstruction requires well-vascularized flaps, and even under the best circumstances, complication rates are high.

The fundamentals of wound management should also be considered. The wound bed should be meticulously prepared and debrided of all devitalized tissue. Performing suboptimal debridement to limit the size of the underlying defect should be avoided. The wound should ideally be closed with two layers of healthy tissue – a deep layer of muscle or fascia and a nonradiated superficial layer of skin.

Finally, consideration should be given to the overall underlying physical and functional status of the patient. Future activity and longevity are important considerations that may help determine which of several reconstructive options will be the best choice for the patient. We consider each region of the trunk separately with the understanding that sound principles of flap design and surgical judgment are paramount to all scenarios.

Reconstruction of the Thorax

The thorax presents a unique challenge for the reconstructive surgeon. The underlying defects can be quite extensive, and bony defects of the rib cage and sternum present added complexity. In addition, significant bony loss not only compromises the cosmetic and protective aspects of the thorax but may also result in severe derangements in respiratory physiology. In the setting of underlying cardiopulmonary disease, this can have dire consequences.

The rigid chest wall consists of ribs bilaterally, which articulate with the vertebrae posteriorly and the sternum anteriorly. In the realm of rigid chest wall defects, disorders of the sternum deserve special attention. The advent of median sternotomy in 1957 was an important advance in the field of cardiac surgery.[1] However, with the increased use of median sternotomy came the finite but potentially debilitating and life-threatening complications of sternal dehiscence, deep sternal wound infection, and mediastinitis.

Deep Sternal Wound Infection

Sternal wound problems affect 1–3% of patients following median sternotomy.[2] Infection can range from a superficial cellulitis to a life-threatening mediastinitis. Prompt diagnosis enables some wounds to be managed with antibiotic therapy. Deep or persistent infection requires aggressive debridement and obliteration of dead space with vascularized tissue.

Pairolero and Arnold classify sternal wounds into three categories.[3] Type I wounds occur in the first few days and consist of serosanguinous drainage only. They typically respond well to antibiotics or minor debridement. Type II wounds occur during the first few weeks postoperatively and consist of purulent drainage, positive bacterial cultures, cellulitis, and mediastinal infection, which may include sternal osteomyelitis. Type III wounds occur months postoperatively and are typified by a chronically draining osteomyelitic infection. Type II and III wounds require sternal debridement frequently with flap coverage for obliteration of dead space.

Pectoralis Major Flap

Pectoralis major is a versatile flap for both head and neck, and thorax reconstruction. Owing to its proximity and bulk, its most valuable use may be for coverage following sternal debridement. It is classified as a Mathes–Nahai type V flap,[4] because it has a dominant pedicle of the thoracoacromial artery and veins, and secondary segmental pedicles from perforating branches of the internal mammary and intercostal arteries. The extensive blood supply to the pectoralis major allows multiple options for positioning the muscle. Maintaining the dominant pedicle,

the sternal attachments may be divided and the muscle advanced medially to fill a defect resulting from sternal debridement. This may be done bilaterally depending on the size of the defect. Division of the humeral insertion of pectoralis major allows further advancement. Preservation of the internal mammary perforators with division of the humeral attachment and thoracoacromial pedicle allows a turnover flap with folding of the pectoralis into the sternal defect to obliterate dead space. Dividing the muscle in parallel with its fibers enables creation of a "split turnover flap," increasing the surface area of the muscle for coverage (Figure 44.1).[5]

There are several considerations with pectoralis flaps. If bilateral internal mammary arteries are used for cardiac bypass, division of the thoracoacromial pedicle will result in insufficient muscle perfusion. Second, when mobilizing a pectoralis flap, consideration should be given to leaving a small portion of the lateral attachment of pectoralis to the humerus to preserve the appearance of the anterior axillary fold. This is for cosmesis only and has no functional significance as the bulk of the muscle is sacrificed. Finally, a pedicle of skin may be harvested with the muscle for coverage if extensive soft tissue debridement is required, though this may be technically difficult and not recommended with a large overlying breast. When pectoralis major is unavailable or potentially undesirable, rectus abdominus muscle, omentum, and free tissue transfer have also been described as second-line options for closure of sternal defects.

Sternal Dehiscence

An infrequent but debilitating complication of median sternotomy is sternal dehiscence. This clinical scenario may occur with or without infection. If unrecognized postoperatively, it may prevent bone healing and lead to chronic nonunion. The consequences of nonunion include debilitating pain and limitation of activity. Cardiac and pulmonary function are typically relatively unaffected.

Historical management of sternal nonunion includes sternal rewiring often using the Robicsek parasternal weave. Sternal plating is a viable alternative that is gaining widespread acceptance (Figure 44.2). Sternal plating has numerous advantages. It provides a well-approximated, stable closure allowing bone healing to occur. Application of the plates requires minimal dissection of the soft tissues of the mediastinum, reducing the risk of injury to bypass grafts or vital organs. Proper placement of the plates does not risk compromise of the blood supply to the sternum. Additionally, the plating mechanism is easily removed and does not impede overlying soft tissue closure.[6]

Chest Wall Reconstruction

Defects of the thorax are typically attributable to one of four clinical scenarios involving infection, tumor resection, trauma, and radiation injury. Reconstruction is critical for restoration of anatomic and physiologic function. The bony chest

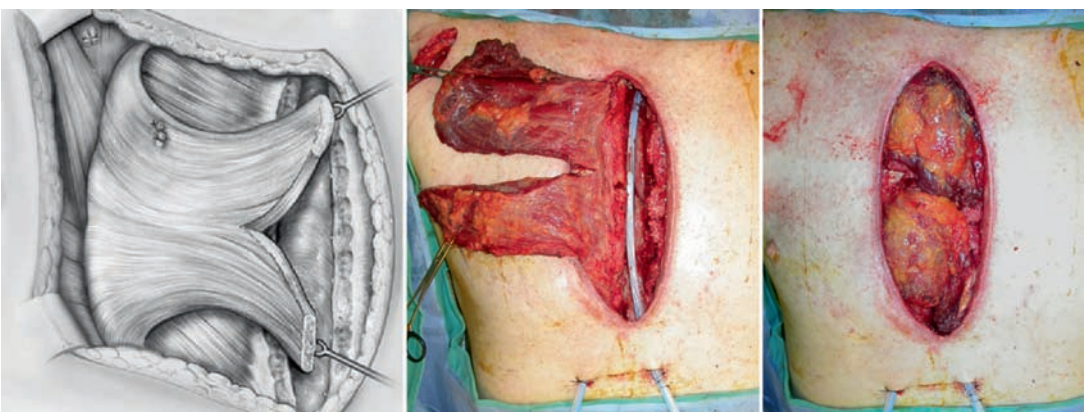

Figure 44.1. A 74-year-old man with sternal wound after CABG with secondary osteomyelitis. A split pectoralis major flap is shown in sketch form and after dissection, based on the perforating vessels of the IMA. It was transposed into the sternal defect. The skin was easily closed primarily.

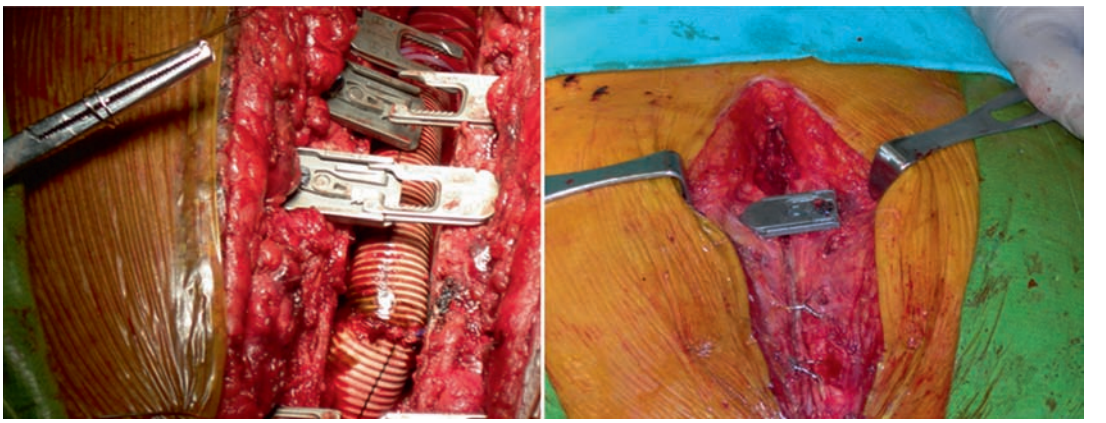

Figure 44.2. The use of sternal plating for sternal dehiscence. The plates are placed in the open configuration (*left*) and then tightened to secure the plates and reapproximate the sternum (*right*).

wall serves a multitude of functions. It provides a bony shell for protection of vital organs, while at the same time providing a flexible frame for respiratory movements. Expansion of the rib cage is crucial for facilitating the creation of a negative inspiratory pressure. In addition, muscles attached to the chest wall contribute to movement of the shoulder and arm.

There is a spectrum of reconstructive strategies depending on the underlying defect. These range from primary closure and skin grafting, to rotational and advancement flaps, to pedicled flaps, and finally to free tissue transfer. A separate but integral consideration is the need for bony reconstruction. Bony replacement becomes a consideration when there is a skeletal defect greater than 5cm or involving 4–5 rib segments.[7]

Historically, multiple methods of bony reconstruction have been used. Earlier methods used autogenous materials consisting of bone grafts from rib, fibula, or iliac crest. Fascia lata has also been used as an autogenous substitute.[8] More recently, synthetic materials have been increasingly used for their durability and ease of use.[9]

The ideal characteristics of a prosthetic material include rigidity, inertness, malleability, and translucency. Rigidity is important to preserve chest wall structure and support. Inertness is critical to minimize risk of infection and allow tissue ingrowth when possible to ensure durability. Malleability is helpful in allowing preservation of contour and improving ease of application. Finally, radiolucency prevents interference with radiographic monitoring of the underlying condition.[10] Mesh materials consisting of marlex, PTFE, and prolene have been used reliably. A combination of two pieces of marlex mesh flanking a layer of methyl methacrylate provides a composite "sandwich," which may be molded to restore a contour defect of the chest wall while providing rigid structure.

With reestablishment of the framework of the chest wall, soft tissue coverage becomes the next consideration. Fortunately, a variety of reconstruction options exist due to the extensive musculature of the trunk. Frequently employed rotational muscle flaps include latissimus dorsi, pectoralis major, serratus anterior, rectus abdominus, and external oblique (Figure 44.3).

Latissimus Dorsi Flap

The latissimus dorsi flap is considered the "workhorse" of trunk reconstruction. Its extreme versatility is due to its size, location, and relia-bility resulting from multiple sources of perfusion. It is a type V flap in the Mathes–Nahai classification. The dominant blood supply comes from the thoracodorsal vessels. Secondary sources include perforating branches of the posterior intercostal and lumbar vessels. This muscle has a large arc of rotation facilitating coverage of nearly any area on the ipsilateral torso. Division of the insertion on the humerus allows additional mobilization. Basing the flap on posterior midline perforators with division of the primary pedicle allows extended coverage of the posteroinferior trunk as well as the contralateral chest wall. A skin paddle may be harvested in conjunction with the muscle for improved soft tissue coverage (Figure 44.4).

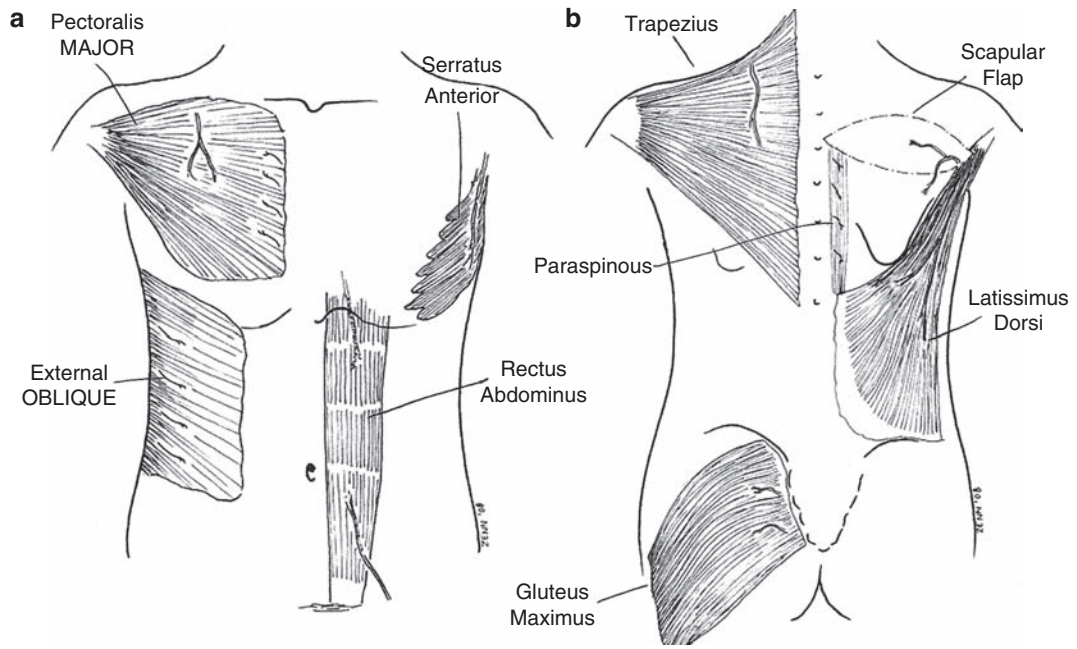

Figure 44.3. Commonly used flaps in trunk reconstruction. (**a**) Anterior view. (**b**) Posterior view.

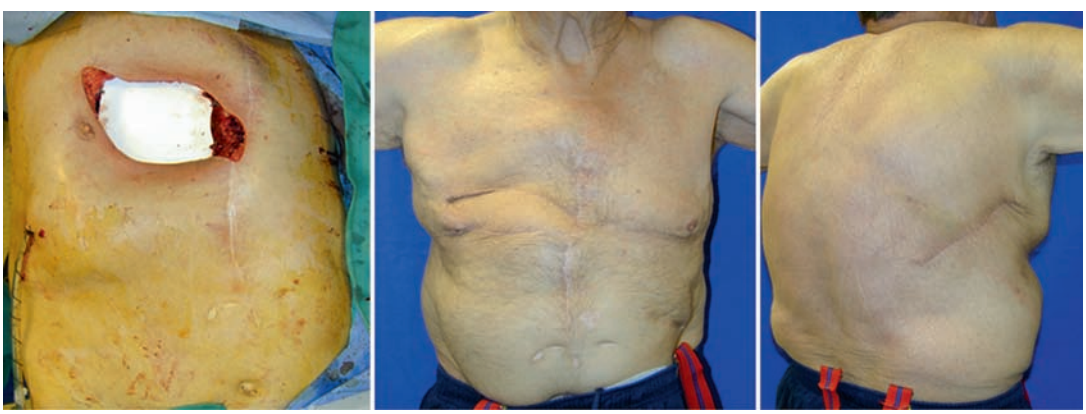

Figure 44.4. A 66-year-old heart transplant patient with a sarcoma of his chest wall. The defect was reconstructed with PTFE resurfaced with a latissimus flap. His 4-month postoperative result is shown.

Skin islands greater than 8–10 cm in width require a skin graft for closure of the donor defect.

Despite its versatility and reliability, sound surgical judgment must be used when employing a latissimus dorsi flap. In patients with previous thoracotomy incisions, the portion of latissimus inferior to the incision cannot be considered to have reliable perfusion and should not be transposed. Patients who have received prior radiation treatments or have had axillary dissections as part of their treatment may have unreliable perfusion through serratus branches.[11]

Disadvantages of the latissimus dorsi flap include the potential need for intraoperative repositioning, a high rate of seroma at the donor site, a readily apparent scar especially when a skin graft is employed, and a possible functional disability.

Rates of seroma may be improved by the liberal use of drains or application of fibrin sealant

before closure.[12] Functional disability in part depends on the preoperative activity level. In general, the other muscles of the trunk compensate very well for latissimus dorsi sacrifice, and it is surprising how little function is lost.

Serratus Anterior Flap

Serratus anterior is a thin muscle flap with origins from multiple ribs and insertion on the scapula. A Mathes–Nahai type III flap, it has two dominant pedicles, branches of the thoracodorsal artery, and the lateral thoracic artery. It has been characteristically used as an intrathoracic flap to fill dead space from an empyema or reinforce repair of a bronchopleural fistula. It may be harvested with latissimus dorsi as a chimeric flap for large soft tissue defects. This involves harvest of both the serratus and latissimus on one vascular pedicle, generating a large volume of muscle for reconstruction (Figure 44.5). Complete harvest of the serratus anterior will result in the classic "winged scapula" due to loss of the stabilizing function of the muscle on the scapula.

Rectus Abdominus Flap

A pair of rectus abdominus muscles flanks the midline of the abdomen. A type III Mathes–Nahai flap, each muscle is supplied by the superior

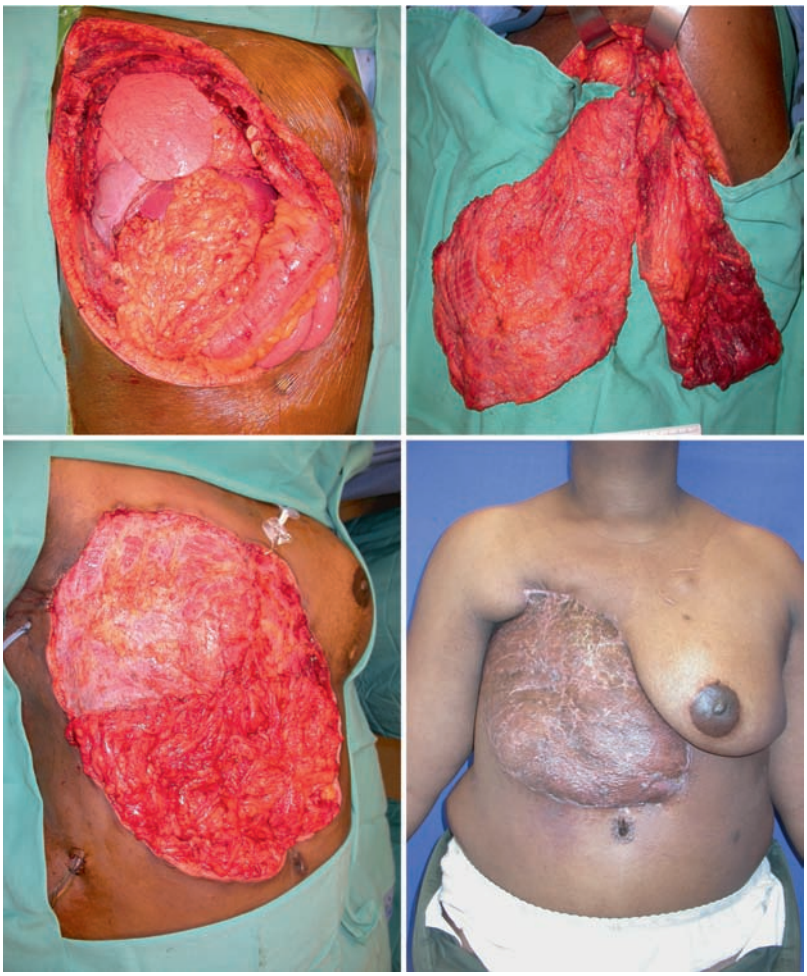

Figure 44.5. A 40-year-old woman with an aggressive sarcoma after previous breast cancer reconstruction with TRAM and irradiation. Her extensive defect required a chimeric free flap consisting of her entire latissimus and serratus muscles on one thoracodorsal pedicle for the upper two-thirds of her defect and a rotational omentum for the lower one third. Her 2-month follow-up is shown.

and inferior epigastric vessels. Best known for its use in breast reconstruction, it can naturally be used for chest wall reconstruction when based on the superior epigastric artery. It may be used as a muscle or musculocutaneous flap. When harvested with a skin paddle, the flap is named based on the orientation of the skin. Vertical rectus abdominus myocutaneous (VRAM) flaps have a vertically oriented skin paddle, whereas transverse rectus abdominus myocutaneous (TRAM) flaps have transversely oriented skin. Best skin viability will always be found with the VRAM variant, as all the harvested skin overlies the muscle (Figure 44.6). Some find the scar of the TRAM variant preferable, especially in cases in which only a small amount of skin is required. It may also be supercharged by preserving the inferior epigastric vessels for microvascular anastomosis if the superior circulation seems inadequate.

Disadvantages of the rectus abdominus flap are generally associated with donor-site morbidity. There is a degree of abdominal wall weakness following harvest of the rectus abdominus. In addition, the patient faces a future risk of abdominal wall bulges and hernias. A mesh may be required to achieve fascial closure to avoid undue tension at the donor site.

External Oblique Flap

The external oblique is a broadly based muscle that is frequently mobilized as a myocutaneous flap. In the Mathes–Nahai classification, it is a type IV flap with multiple segmental pedicles from perforators of the inferior eight intercostal arteries. The flap is elevated from the midline to the anterior axillary line and can be rotated as high as the third intercostal space and extended laterally 5 cm beyond midline.[13] It is ideal for lower one-third chest defects, especially when use of the rectus or latissimus is compromised by prior surgery or irradiation (Figure 44.7).

Omental Flap

Omental flaps are an option for chest wall defects and may also be valuable to salvage failed rotational flaps. The omentum provides no structural support but may be combined with prosthetic material to provide a vascularized bed for the adherence of a skin graft. Like pectoralis, serratus, and latissimus, omentum may be transposed to the intrathoracic space for treatment of empyema or bronchopleural fistula. Omentum adds the morbidity of an intra-abdominal procedure, and sometimes, transposition through the

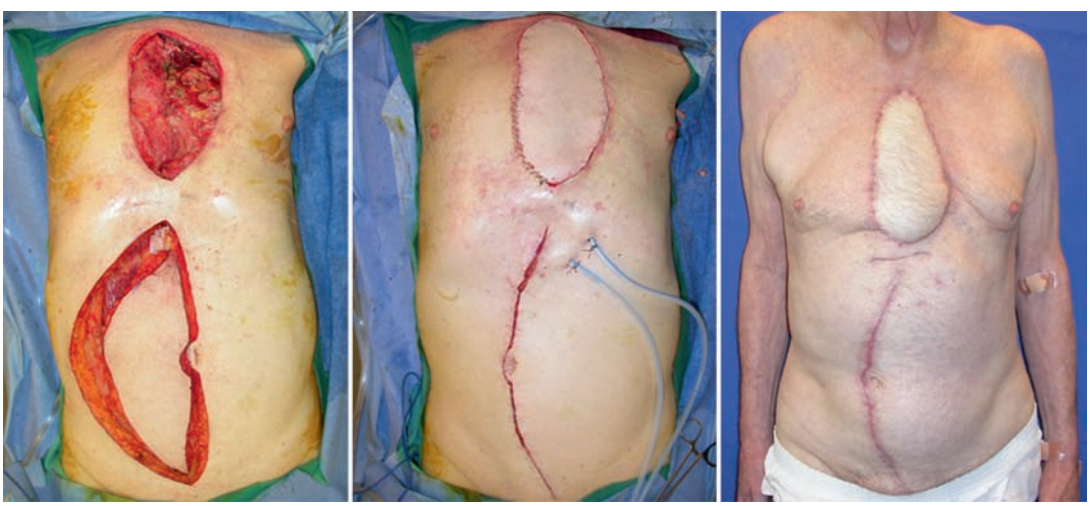

Figure 44.6. A 75-year-old man with recurrent mediastinitis and osteomyelitis of the left chest. After debridement to viable tissue, the wound was reconstructed with a VRAM flap based on the right-sided superior epigastric vessels. His 3-month postoperative result is shown.

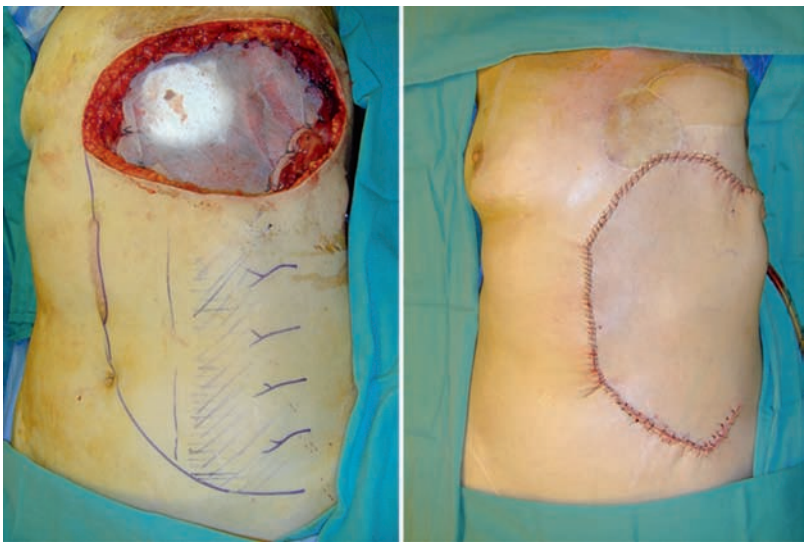

Figure 44.7. A 58-year-old woman with recurrent breast cancer with prior latissimus chest wall reconstruction. After resection, PTFE and an external oblique flap were used to reconstruct the defect.

diaphragm is the most expeditious option. Depending on the route of transposition, a hernia of the abdominal wall or diaphragm is possible.

Posterior Trunk Reconstruction

In the adult population, back wounds are typically acquired defects. Like chest wall defects, they may occur secondary to tumor resection, infection, trauma, and radiation. The breakdown of incisions for spinal surgery with or without exposed spinal hardware can also present challenging wounds requiring coverage.

The underlying principles are no different from those followed elsewhere on the body. This includes wide debridement of devitalized tissue, appropriate antibiotic use, and early tissue coverage.

Trapezius, latissimus dorsi, and gluteus maximus are three paired muscle groups that can be reliably used for most wounds requiring coverage. Other available flaps include scapular, parascapular flaps, and paraspinous muscle flaps (Figure 44.3).

The latissimus flap has been described in the section on chest wall reconstruction. To reiterate, it is an extremely versatile flap given its size and large arc of rotation, which enables coverage from the neck to the upper lumbar vertebrae. Lower back defects would use the paraspinous perforators as the primary blood supply to the muscle, as the thoracodorsal pedicle must be divided for adequate flap rotation and reach.

Trapezius Flap

The trapezius muscle is a large broad muscle of the back arising from the occiput through T12. It inserts on the clavicle, scapula, and acromion. In the Mathes–Nahai classification, it is a type II flap with a dominant pedicle off the transverse cervical artery and secondary perforators from the intercostal vessels and occipital artery.

As a consequence of its multiple attachments, it may be rotated, advanced, and turned over to provide coverage. In considering flap design, it may be useful to think of the trapezius as having two segments – an upper and a lower segment. The upper portion inserts along the lateral third of the clavicle and serves to elevate the scapula, while its bulk aesthetically forms the nape of the neck.[14] This portion of the muscle would be useful in posterior cervical defects. The lower portion is the more expendable of the two and allows use of the muscle for coverage while preserving a portion of its clinically important function. This lower portion is useful for upper thoracic defects.

Paraspinous Muscle Flap

The paraspinous muscle flap is a beneficial flap for the treatment of midline lumbar wounds usually as a consequence of lumbar spinal surgery. These defects may fall outside the arc of rotation of the latissimus dorsi flap. Disruption of local perforators during spinal surgery can also make a turnover latissimus dorsi flap unreliable. The paraspinous muscle flap is a Mathes–Nahai type IV flap with segmental perforators off the posterior intercostal artery. Preservation of the lateral perforators allows medial dissection and advancement to close small midline defects of the lumbar region.[15] Large defects or extensive local tissue destruction and edema will limit the use of the paraspinal muscles for coverage.

Scapular and Parascapular Flap

These flaps are based on the branches of the circumflex scapular artery, which divides to form a transverse scapular branch and a descending parascapular branch. They are fasciocutaneous flaps, which do not supply muscle used for deep wounds but are useful when there is a deficit of well-vascularized skin for coverage. There is great flexibility in design of these flaps as long as one of the perforating vessels of the circumflex scapular artery is included in the design. Flaps with widths of greater than 10 cm often require skin grafting of the donor site. An additional advantage of these fasciocutaneous flaps is that no muscle function is sacrificed.

Gluteus Maximus Flap

The paired gluteus maximus muscles are type III Mathes–Nahai flaps. The blood supply consists of two dominant pedicles from the superior and inferior gluteal arteries. The gluteus maximus originates from the posterior superior iliac crest and lateral sacrum and inserts on the greater trochanter of the femur and iliotibial tract.

For wounds of the lower back, the muscle flap is based on the superior gluteal artery. A muscle splitting incision is used to preserve the quantity of muscle that is not needed for bulk. Transposition of the flap can occur either in the plane of the muscle by rotation or sliding or as a turnover flap. The effective length of the flap can be augmented by several methods including inclusion of a portion of iliotibial tract with the muscle and conversion to an island pedicle flap. Additionally, performing an iliac ostectomy creates a groove through bone and minimizes the distance the flap must transverse. This flap has been used successfully in ambulatory patients with minimal loss of function when at least 50% of the muscle is left in place and the inferior gluteal nerve has not been injured. In some cases, the skin island of the gluteal flap can be raised based solely on the perforator of the gluteal artery (S-GAP flap), without any muscle sacrificed. This extends the pedicle length of the flap and is preferable in any ambulatory patient.[16–18]

External Oblique Turnover Flap

Another available option for coverage of lower back tissue defects is the external oblique muscle transposed as a turnover flap. When elevated to the level of its intercostal perforators and turned posteriorly, the external oblique muscle can cover the back from T10 to L4. Skin grafting is necessary for muscle coverage. Morbidity has not been demonstrated to be substantial. Preservation of the periumbilical perforators and associated nerves minimizes the risk of abdominal skin necrosis or considerable sensory loss. Unilateral harvest will cause an uneven abdominal wall contour.[19]

In summary, the posterior trunk is subject to developing large defects from a variety of etiologies. A systematic approach to the location of the defect and available flap options will allow successful reconstruction. Mathes et al. divide defects in the posterior trunk into cervical, upper thoracic, midthoracic, and thoracolumbar defects.[20] Cervical and thoracic defects can generally be closed with trapezius and latissimus dorsi flaps. Lower thoracic and lumbar defects can be approached with latissimus dorsi advancement or turnover flaps, paraspinous flaps, gluteus maximus flaps, or external oblique turnover flaps. Sometimes a combination of flaps is required for successful closure, and the option of a free flap is always available.

Abdominal Wall Reconstruction

Similar to the thorax, the abdomen is a cavity containing a number of vital structures. However, unlike the thorax there is no protective bony structure. Therefore, a working knowledge of the abdominal wall anatomy and function is

integral to the performance of an adequate reconstruction. Abdominal wall defects are multifactorial, and reconstructive options vary depending on the location and etiology of the wound.[21]

An understanding of the anatomy of the abdominal wall is of paramount importance for reconstruction. Skin and subcutaneous tissue form the external layer of the abdominal wall. The thickness of this layer can be quite variable depending on the degree of obesity of the patient. Lateral to the rectus muscles, the abdominal wall is three muscle layers thick. It comprises the internal and external oblique muscle and the transversalis muscle. The rectus abdominus muscles are paired midline muscles. The rectus sheath is formed by the aponeurosis of the fascial component of the external oblique, internal oblique, and transversus abdominus muscles. Above the arcuate line, the fascia of the internal oblique splits at the rectus abdominus, joining with the external oblique fascia to form the anterior rectus sheath, and the transversus abdominus to form the posterior sheath. Below the arcuate line, the internal oblique fascia passes anteriorly, resulting in a strong anterior sheath but leaving a relatively weak posterior sheath.

Abdominal wall defects can be categorized as midline or lateral defects. Midline defects are significantly more common. Acute defects arise typically from either wound dehiscence following laparotomy or deliberately delayed closure as a result of abdominal decompression for abdominal compartment syndrome.

With prompt recognition, acute wound dehiscence can often be managed with primary closure. However, in certain situations, for example, acute infection, debridement of large amounts of tissue, or a nutritionally depleted or otherwise immune compromised patient, primary closure may be difficult to achieve. Management should then consist of the debridement of devitalized tissue and appropriate measures to prevent evisceration.

With an infected or questionably infected wound bed, it is inadvisable to place a permanent mesh to retain the intra-abdominal contents. However, the use of an absorbable mesh or biologic materials, including porcine submucosa (Surgisis) or human acellular dermis (Alloderm), may help prevent evisceration. Skin defects can then be managed with healing by secondary intention, with or without vacuum therapy, or skin grafting. Once the acute events have resolved, the patient can then be closed definitively with a formal hernia repair at a later date if necessary.

When a primary closure of fascia is unobtainable and a mesh is not desired, one can consider a "components separation" as described by Ramirez.[22] The procedure releases the external oblique muscle and fascia off the internal oblique, providing mobility of the external oblique muscles toward the midline. Separation of components allows mobilization and advancement of the anterior rectus sheath and muscle approximately 10 cm at the waistline. With bilateral advancement, 20-cm defects may be closed primarily. Modifications of this technique include separate incisions for release of the external oblique muscle to avoid large skin flaps and performance of the procedure endoscopically.

Lateral abdominal wall defects are usually the result of acute surgical resection or traumatically created defects. Defect of skin and soft tissue may be managed in a variety of ways. For small or medium-size defects, tissue rearrangement or skin grafting may be adequate. Acutely, skin grafting may be used to close open wound with the plan of performing tissue expansion and definitive closure following complete expansion.

Tissue defects that include a loss of muscle and fascia should be approached with local rotational flaps when possible. The rectus abdominus flap, which has already been described, is a very useful flap for such defects. This flap has the option of being based on its superior or inferior pedicle. Use of the superior pedicle to supply the flap allows rotation to cover the upper two-thirds of the abdomen. Basing the flap on its inferior pedicle allows additional coverage for the lower one-third of the abdomen.

A second choice of flap that has also been proven to be very useful is the tensor fascia lata (TFL) flap (Figure 44.8). This flap has a large arc of rotation, which allows it to be transposed as high as the upper third of the abdominal wall. It is composed primarily as a fascial or fasciocutaneous flap, allowing its use for fascial replacement. Its main limitation is the donor site, which requires closure by a skin graft if a width of more than 8 cm of tissue is harvested.

Another option for lower abdominal wall reconstruction is the rectus femoris flap. It provides more fascia and muscle bulk than a TFL, and its arc of rotation allows coverage to the level of the umbilicus. The main disadvantage of its

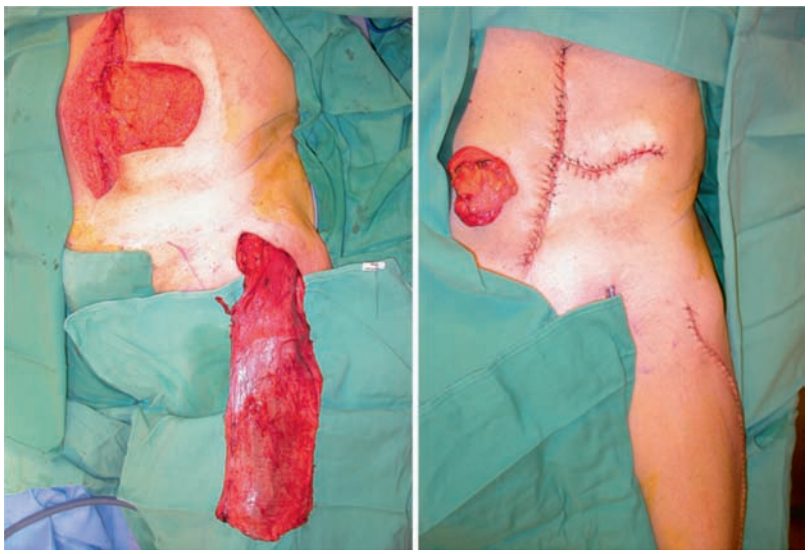

Figure 44.8. A 66-year-old man with a sarcoma of the abdominal wall requiring full-thickness resection, reconstructed with a rotation tensor fascia lata flap. No mesh was required.

harvest is donor-site morbidity, as it may cause some weakness of leg extension in the terminal 15° of knee extension.

Free Tissue Transfer

Although the majority of trunk defects may be managed with rotational flaps, there may be situations with extensive tissue destruction where a pedicled flap has failed, is unable to reach, or is too small to cover a given defect. With the availability of recipient vessels, free tissue transfer enables coverage of the most challenging defects, in salvage or as a primary procedure. Free tissue transfer is most useful in cases with large tissue requirements. Accordingly, transfers of latissimus, rectus, scapular, or thigh-based flaps have been most useful.

The spectrum of trunk defects is extensive, and reconstruction can be challenging if not daunting. The aims are to provide appropriate tissue coverage with minimal morbidity or loss of function. This can be achieved through methodical planning and adherence to the principles of sound surgical debridement. Application of the reconstructive ladder proceeding from primary closure to free tissue transfer will allow coverage of virtually any defect. Although the plastic surgeon is instrumental, trunk reconstruction is typically achieved through multidisciplinary cooperation, and continued collaboration among specialties is essential for optimal patient outcome.

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45

Lower Extremity Reconstruction Following Trauma and Tumors

Chih-Wei Wu, Christopher G. Wallace, and Fu-Chan Wei

Summary

Assessment and reconstructive approaches for post-traumatic and tumor-ablative lower limb defects are presented and discussed in this chapter. Addressed herein are the assessment and stabilization of the emergency trauma patient, the decision process surrounding attempted limb salvage or primary amputation, general debridement principles, soft tissue and fracture management options, timing of reconstruction, nonmicrosurgical and microsurgical reconstructive options, muscular and nonmuscular flap coverage options, useful composite flap designs, and recipient vessel selection and assessment techniques, including the debates and/or consensus surrounding these various issues. Specific attention is also given to the management of chronic problems such as post-traumatic osteomyelitis and stiff knee. Furthermore, advancements in the multidisciplinary treatment of patients with lower limb tumors and the reconstruction of postablative defects are outlined.

Abbreviation

MESS Mangled extremity severity score

Introduction

The management of lower limb injuries has evolved from simple wound care or amputations to technologically advanced dressings and reconstructive attempts. Development of the consensus on important issues and advances in wound care and surgical techniques have allowed for strategic reconstructions and the achievement of optimal outcomes.

The purposes of this chapter are to provide key principles, to present consensus, and to discuss the major options for lower limb reconstruction following trauma or tumor resection.

Functions and Special Features of the Lower Extremity

A clear understanding of anatomic structures and functions of the lower extremity is the basic requirement for its successful reconstruction. The increased incidence of atherosclerosis in the lower extremity is of great importance, as preexisting peripheral arterial disease can worsen the injury severity and complicate reconstruction of the affected limb. Particular attention should also be paid to the venous circulation, as venous problems are commonly encountered but often present obscurely and thus may be overlooked.

Bone, soft tissue (including vascularity), and nerve are the three major components of lower limbs and form the basis for lower extremity reconstruction themes. Weight bearing, the basic

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function of the lower limb, requires a sound and sturdy bony structure. With its thin soft tissue coverage, the tibia is vulnerable to injury; indeed, the fact that the degree of soft tissue injury correlates with outcomes forms the fundamental tenets of the Gustilo classification for open (compound) tibial fractures.[21,22] Gustilo Type III fractures are divided into three subtypes; Type IIIA injuries have adequate soft tissue coverage, Type IIIB have extensive soft tissue loss with periosteal stripping and bone exposure; Type IIIC injuries mandate vascular repair. Types IIIB and IIIC fractures generally require the importation of soft tissue coverage.

Protective sensation for the heel pad arises from the tibial nerve. It is widely accepted that a severely injured lower extremity with tibial nerve disruption and an insensate foot are better amputated than salvaged.[35,40] Some may still be amenable to salvage by nerve repair with grafts[26] or by providing a sensate glabrous cutaneous flap reconstruction.[28]

The basic reconstructive goal is to provide the patient with painless and stable ambulation and stance. This requires the functional restoration of all major components, namely rebuilding bony structural integrity, achieving satisfactory soft tissue coverage, and restoring protective sensation for a stable and durable heel pad.

Lower Extremity Reconstruction Following Trauma

To achieve optimal results, efforts should be made at every stage of reconstruction. Major phases include resuscitation and stabilization of the emergent trauma patient, initial debridement and/or fracture management, and definitive reconstruction and rehabilitation.

The Emergent Setting

Patient Stabilization and Injury Assessment

The key principle of management in the emergent setting is that patient salvage takes priority over limb salvage. International guidelines for acute trauma life support should be followed to stabilize the patient, and care should be taken to identify other associated injuries. Unrecognized or inadequately treated injuries may lead to failures of reconstruction or even threaten the patient's life.

Assessments of lower limb injury severity can be eased by considering its three major components as previously mentioned. A less optimal result can be expected when more components are involved.

Primary Amputation Versus Limb Salvage: Decision Making

Several scoring systems exist that aim to help surgeons decide between amputation and salvage of the traumatized lower extremity. The Mangled Extremity Severity Score (MESS) is widely used.[15,55,58] It was once generally accepted that a patient with a traumatized lower limb and a MESS of 7 or more was better served by an amputation rather than a reconstruction.[30] Through years of evolution, this threshold value has become more equivocal and is increasingly debated.[5,11,13] An important multicenter, large-scale study conducted by the Lower Extremity Assessment Project group prospectively compared the effectiveness of different trauma scoring systems in predicting outcomes following lower limb trauma[5]; a low trauma score was considered useful in predicting limb salvage; a high trauma score, however, did not preclude the possibility of a successful reconstruction. The wide variations in mechanisms and the extents of injuries make it difficult to devise a decision-making algorithm applicable to each circumstance. Therefore, such decisions should be made on a patient-by-patient basis rather than by applying guidelines and "scores" dogmatically. In general, our department suggests amputation if the following three situations coexist: (1) a MESS of at least 11; (2) severe disruption of the tibial nerve; (3) when the patient is elderly.

Principles of Initial Debridement

The impact of primary debridement on the final result of amputation or reconstruction cannot be overemphasized. Inadequate debridement in the early stage results in the need for repeated debridement(s) and thus it delays definitive reconstruction. Infection usually results from inadequate removal of devitalized tissue and is responsible for flap failures and chronic complications. In general, grossly contaminated tissue should be removed; injured skin should be conservatively trimmed to health and preserved as much as possible; devitalized subcutaneous tissue and muscle should be radically removed;

and efforts should be made to preserve vital structures such as neurovascular bundles and tendons. Whenever possible, revascularization of a degloved and devascularized skin flap should be attempted, especially for the heel pad, even through an arteriovenous anastomosis.[27]

Principles of Fracture Management

Fracture management constitutes the next step in the management of open fractures of the lower limb. An external fixator is generally a safe option, but it carries the risks of injury to the neurovasculature and pin tract infection(s). Nevertheless, an external fixator produces less violation to bone vascularity than internal fixation and is more timesaving. External fixation is recommended in the following conditions: (1) severe comminution (2) segmental bone loss, (3) severe osteoporosis, (4) severe soft tissue injury, and (5) severe contamination.

In contrast, internal fixation provides better convenience for wound care and facilitates future reconstructive procedures. However, internal fixation is less feasible for severely comminuted fractures and inappropriate when there is severe contamination. Studies have shown that intramedullary nailing produces more tissue damage and may compound a systemic inflammatory response; this is, therefore, less desired in patients with poor physical reserve or associated major injuries. There is also a growing sense that long bone fractures in severely injured patients should be managed with damage-control orthopedics, consisting of early external fixation followed by definitive internal fixation 5–7 days after injury.[54,64]

Timing of Definitive Reconstruction

The best timing for definitive reconstruction has been a debate for decades. Godina was credited for demonstrating the advantages of early free flap transplantations within 3 days of injury in terms of less infection rate, less flap failures, and shorter bony union time.[19] This approach has evolved with cumulative experiences in lower extremity trauma and microsurgical reconstruction. Nowadays, once debridements have converted the injury to a clean wound, definitive soft tissue coverage can then be performed.[25]

Bearing in mind that key factors in determining the timing for definitive reconstruction are the general condition of the patient and the wound, there is little benefit in performing a primary definitive reconstruction in a patient with poor physiologic reserve. The incidence of infection and flap failures rises with wound contamination; a more contaminated wound carries higher risk of infection and is better served by a delayed reconstruction following repeated and extensive debridements.

Another issue worthy of discussion is the emergency free tissue transplantation. In the authors' opinion, a free flap can only be defined as emergent when it is *mandatorily* performed immediately for revascularization purposes; this can be either as a flow-through free flap or as a vessel repair (direct or with vein graft) plus free flap soft tissue coverage. Others define it as a flap transplanted "either at the end of primary debridement or within 24 h of injury".[38] Some authors suggest that an emergency free flap is indicated when there is a need for coverage of certain vital structures, such as a major artery, which may otherwise necrose and significantly affect the outcomes of reconstruction.[7,25] The benefits of performing free flaps in the immediate post-trauma period should be weighed against the risk of a prolonged initial operation and the depletion of resources of the trauma team.

Soft Tissue Reconstruction

Achieving soft tissue coverage is the primary goal and cornerstone in post-traumatic lower limb reconstruction. Its importance can be demonstrated in several ways: the Gustilo classification of open tibial fractures is mainly based on the extent of soft tissue injury, which influences outcomes; vital structures such as nerves, vessels, and tendons will become ischemic and necrose without coverage; bone deprived of periosteum will devitalize in an unhealthy or inadequate soft tissue environment. The extent of soft tissue loss depends on many factors, including the mechanism and magnitude of the traumatic insult, the circulation status of the affected limb, the adequacy of debridements of devitalized tissue, and the presence or absence of infection. Generally, the involved soft tissue circumference has more influence on the result than its length; an injury involving a greater circumference of the affected limb has a higher incidence of primary or secondary amputation and chronic sequelae.[40]

An ideal solution for soft tissue coverage should shorten wound healing time, reduce wound complications, provide satisfactory function,

minimize chronic sequelae, and, if possible, provide better cosmesis. Though some wounds are amenable to closure by secondary intention or by skin grafting, these methods may yield less optimal functional results. A deep defect is best served by muscle flaps for dead-space obliteration. A defect of the dorsum of the foot is best served by a thin cutaneous or fasciocutaneous flap. Management of lower extremity wounds, therefore, involves complex decision-making trees other than the traditional reconstructive ladder.

Advances in wound care have greatly enhanced the management of some lower limb wounds that might have required a flap for coverage in the past. Vacuum-assisted closure may be considered as a cornerstone in wound care and is effective in decreasing tissue edema and wound circumference and increasing granulation tissue. It is also effective in stimulating granulations over exposed bone and thus facilitates wound closure by secondary intention or with skin grafting.

Regional Tissue Transfers

The soleus and the medial or lateral head of the gastrocnemius, regarded as traditional muscle flaps, provide reliable coverage for the middle and the upper thirds of the tibia, respectively. Fasciocutaneous flaps spare the underlying muscle and thus reduce the functional deficit. A better understanding of vascular anatomy has greatly increased the number of regional fasciocutaneous flaps available in the lower extremity and cumulative experiences in flap design have allowed for improved flap survival and final outcomes, in turn decreasing the need for free tissue transplantations. Meanwhile, conventional muscle flaps have gradually decreased in popularity. A reconstructive surgeon should be familiar with the application of regional fasciocutaneous flaps in any given anatomic region.

Free Style Flaps

Experience gained in dissection of perforator flaps has allowed the surgeon to harvest a skin island based on any good quality and sizable cutaneous vessel in a free style manner, as originally described by Wei et al.[42,69] With this technique, a cutaneous or fasciocutaneous flap of small to moderate size can be elevated with a far extended degree of freedom.

An average of 374 perforators with a diameter of 0.5 mm or greater in a human body was demonstrated in the study by Taylor and Palmer,[66] and the reliability of Doppler ultrasonography in locating cutaneous vessels has been established.[31,67] Therefore, in many instances, a skin flap designed according to a robust Doppler signal can be successfully elevated. The flap dissection can be carried out in a suprafascial plane to allow for a thinner flap and less donor-site morbidity or in a subfascial plane with more convenience and same accuracy. Once an appropriate cutaneous vessel is identified, it is dissected in retrograde fashion to its mother vessel, and then the flap is islanded and transferred to cover the defect. As the exact orientation and length of proximal vessels can be fully disclosed only after dissection, the main drawback of the free style flap may be its unpredictable pedicle length; a design that places the vessel eccentrically allows for increased flap reach (Figure 45.1). The maximal surface area that can be supplied by a single

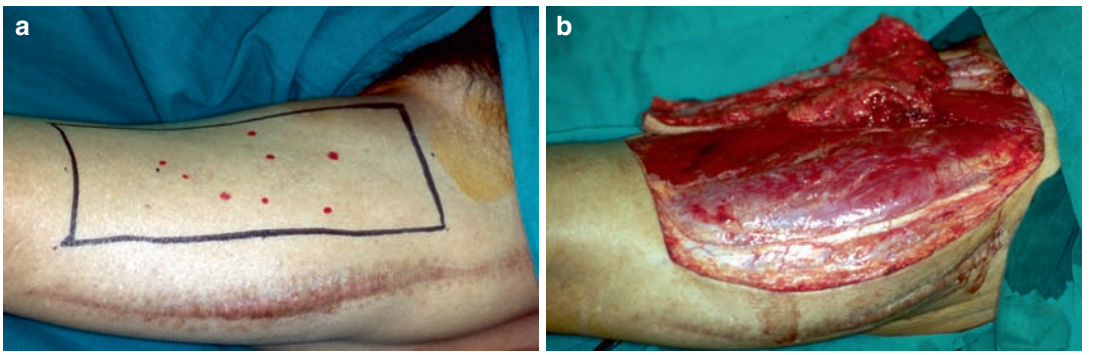

Figure 45.1. (**a**) Multiple arterial signals have been mapped by handheld Doppler over the anteromedial thigh in preparation for a free style flap harvest. Note the scar from the previous use of an anterolateral thigh flap. (**b**) A fasciocutaneous flap has been harvested in a free style manner based on the most proximal arterial Doppler signal to increase distal flap reach.

cutaneous vessel varies with the size and quality of the vessel. Though this can easily exceed 8 × 20 cm, as demonstrated by several studies, the circulation at the skin edge should always be ascertained before pedicle transection.

It should be noted that the cutaneous vessels must be carefully inspected for the existence of both arteries and veins before dissection. In a cadaveric dissection by Ghali et al., 3 of 40 distal medial perforators of the lower leg did not have any accompanying veins.[18]

Microvascular Free Tissue Transplantations

Soft tissue coverage with regional tissue transfers is generally possible in low-energy injuries. In the setting of high-energy injury, however, tremendous force during trauma may produce extensive soft tissue damage and preclude the possibility of regional tissue transfer. In addition, regional tissue may be inadequate for resurfacing larger defects or cause unacceptable donor-site morbidity.

As advances in microvascular techniques greatly enhanced free flap success rates and concomitantly significantly decreased complications, free tissue transplantations have become a most reliable option in many lower extremity trauma scenarios. Microvascular free flap transplantation is particularly indicated when regional tissue is unfeasible or inadequate and when there is a concern for better cosmesis or fewer functional deficits. Several characteristics of the free flap make it ideal for management of most post-traumatic defects in the lower extremity as it is able to (1) deliver abundant soft tissue and thus allow for radical debridement; (2) achieve simultaneous soft tissue coverage and bony reconstruction; and (3) correct functional deficits (e.g., functioning muscle transplantation). Donor-site morbidity can be minimized by proper flap selection and careful dissection techniques.

Selection of Soft Tissue Free Flaps

Basic options of soft tissue free flaps include fasciocutaneous flaps, muscle flaps, and musculocutaneous flaps. In the past, open fractures were considered better served by muscle-containing flaps rather than by nonmuscular flaps. As several studies have demonstrated satisfactory results for coverage of open tibial fractures or chronic osteomyelitis with fasciocutaneous flaps, in terms of flap survival, infection rate, chronic osteomyelitis, bone union, and stress fractures, this concept has gradually changed.[59,75] It is believed that the adequacy of wound debridement and dead-space obliteration is even more important than the selection between muscular and cutaneous flap types.[75] Considerations of soft tissue free flap selections include the required pedicle length, the volume of the deficient tissue, and the surface area of the wound.

The muscle flaps, with bulky and pliable soft tissue and great vascularity that allows for better resistance to infection, are the primary options when there is a need for dead-space obliteration. When there is a shallow defect with large surface area, as commonly presented in the lower third tibia and the dorsum of the foot, fasciocutaneous flaps are first considered. Fasciocutaneous flaps tend to better tolerate subsequent procedures, such as tendon transfers, tenolysis, hardware removal, or debulking procedures.

The anterolateral thigh flap is the desired option when a fasciocutaneous flap is indicated. It is of great versatility and reliability, and additional tissues, such as muscle in particular, can easily be incorporated.[70]

Recipient Vessel Selections in Free Flap Transplantations

In free tissue transplantations, the identification of healthy recipient vessels is of paramount importance before flap elevation. Algorithms are available for choosing better suited recipient vessels in microvascular lower extremity reconstruction.[47] In general, the anterior tibial artery is easier to approach than the posterior tibial artery; end-to-end or end-to-side anastomosis can be considered when using the anterior or posterior tibial artery. Use of recipient vessels distal to the injury zone can be an option provided distal pulses are present and a flow problem is not encountered intraoperatively.[32,47] Other than the major arteries, the branches of the deep femoral, superficial femoral, saphenous, genicular, and medial plantar arteries are all available options. Recipient vessel selections will be discussed according to different anatomic sites.

Assessment and Preparation of Recipient Vessels

Methods for preoperative assessment of lower limb arteries include physical examination,

Doppler ultrasonography, and angiography. Examination of pedal pulses by palpation or Doppler ultrasonography is mandatory and, in most instances, suffices to ensure the quality of arteries.[39] Angiography remains useful in (1) identifying the injured arterial site when both pedal pulses are absent[39] and (2) evaluating the peroneal artery, which is difficult to assess by palpation or Doppler ultrasonography. It should be noted that an artery with normal angiographic findings is not always suitable for anastomosis; conversely, abnormal angiographic findings usually correlate well with abnormal intraoperative findings.

During recipient vessel preparation, dissection should be carried out proximal to and outside the injury zone, and robust arterial spurting pressure must be ascertained. Fibrotic tissue surrounding the vessels is suggestive of an injury zone and a possible vascular injury. It is not uncommon to change the planned recipient vessel(s) intraoperatively; consequently, familiarity with the vascular anatomy is required to overcome unpredictable findings.

Soft Tissue Coverage in Different Anatomic Sites

Patella and Peripatellar Region

Skin in the patella and peripatellar region is characterized by its pliability to allow for knee flexion and its durability to serve as a kneeling surface. Adequate range of motion of the knee joint is the primary consideration for soft tissue coverage here; therefore, healing by second intention or skin grafting is usually less desirable, as the subsequent contracture will limit knee flexion.

Traditionally, the medial or lateral head of the gastrocnemius is used to cover defects around the knee; the medial head is more commonly used because of its wider arc of rotation and broader belly. The functional deficit after division of a single head of the gastrocnemius is minimal except for altered jumping ability, but the aesthetic results are less favorable. Other valuable muscle flap options include the distally based sartorius and vastus lateralis.

Fasciocutaneous flaps should be considered when the defect is shallow or when there is a desire for better cosmesis. The saphenous artery is a constant vessel over which a fasciocutaneous unit can be designed and elevated as an axial pattern flap. The distally based anterolateral thigh flap is a reliable and valuable option provided the distal connection between the descending branch of the lateral circumflex femoral artery and the lateral superior genicular artery or the deep femoral artery remains intact.[46] A free style flap can easily be elevated here as a number of cutaneous vessels are available near the patella.[44]

Free tissue transplantations are indicated when there is an extensive defect. Recommended recipient vessels are the superior medial genicular artery for posterior defects and the descending genicular artery or the deep femoral artery for anterior defects. These vessels are deeply seated, and thus a longer vascular pedicle is usually required.

Reconstruction becomes more complex if the patella tendon is involved. Chiou et al. used the lateral gastrocnemius–Achilles tendon complex to provide coverage of the patella together with patellar tendon reconstruction.[8] When there is an extensive soft tissue defect, the primary option is a tensor fascia latae myocutaneous flap; the tensor part provides coverage of the patella, and the fascial part reinforces or replaces the patella tendon.[68]

Upper and Middle Thirds of the Tibia

Traditionally, a single head of the gastrocnemius is used to cover upper third tibial fractures, whereas the proximally based soleus is used to cover middle third tibial fractures.

Fasciocutaneous flaps applicable to cover the patella, as aforementioned, can usually be employed to cover upper third tibial fractures; those for coverage of middle third tibial fractures are usually based on medial or posterolateral perforators.

Free tissue transplantations are indicated when the defect is too extensive, when there is a need for both soft tissue and bone reconstruction, or when there is a concern for better cosmesis or lesser donor-site morbidity. Defects in this region tend to extend more deeply and more irregularly; thus, the preferred flap type is usually a muscle or musculocutaneous flap, because the muscle portion provides better dead-space obliteration. Recipient vessels in this region are also deeply seated, and thus a free flap with a longer vascular pedicle is usually required.

Distal Third of the Leg, the Ankle, and the Dorsum of the Foot

Management of defects over the lower third tibia and ankle remains a great challenge to reconstructive surgeons. In selected cases with smaller defects, employment of regional tissue may be attempted. The proximally based soleus can provide coverage for the upper portion of the lower third leg; variations in the location of the tenomuscular junction and the width of the distal part soleus significantly affect the distal limit it can reach and the defect size it can cover. Scoring of the soleus epimysium can be performed to increase the reach of this flap. The distally based soleus can be employed when the defect location and dimension are favorable. When transected at its mid portion, the soleus can be elevated and transferred based on its distal perforators arising mainly from the posterior tibial artery.

Numerous fasciocutaneous flaps are available for coverage of defects in this region. Of importance is the distally based sural flap, alternatively termed as the reverse sural flap or the sural neurocutaneous flap. This flap relies on retrograde blood supply from the distal peroneal artery perforator, located 4–7 cm above the malleolus, and the accompanying vascular plexus of the sural nerve and lesser saphenous vein.[16] Lower third tibia and hind foot defects can be easily covered with a reverse sural flap; however, partial or total flap necrosis is a potential problem, and the reported incidence in the literature is not low.[16] As it is thought to be related to venous congestion, one can consider performing an anastomosis between the proximal end of the lesser saphenous vein and any venous stump available at the defect.[65] Studies have shown that the complication rate of the distally based sural flap rises significantly with (1) a patient age of greater than 40 years; and (2) existence of one or more of the following three comorbidities: diabetes mellitus, venous insufficiency, and peripheral arterial disease.[3]

Islanded fasciocutaneous flaps can be elevated based on distal cutaneous vessels from the anterior tibial, posterior tibial, and peroneal arteries. These vessels are constant and can be found 4–7 cm proximal to the medial malleolus and 4–10 cm proximal to the lateral malleolus along the course of the corresponding artery. Flaps can be harvested to include the skin distally or proximally, skeletonized, and transferred to cover the defect.[4]

As regional tissue may be injured in the event of trauma, in some instances, the microvascular free tissue transplantation becomes the only option; it is also the desired option by virtue of its reliability and unparalleled cosmesis. Defects located in the pretibial region, ankle, or foot dorsum usually have more of a surface area than volume requirement. Therefore, the fasciocutaneous flap is the recommended flap type. Flap pedicle length is of little concern when choosing the anterior tibial artery due to its superficial location and easy accessibility in this region.

The skin in the foot dorsum is characterized by its thinness and great pliability, which allow the foot to fit in normal footwear. Defects of the foot dorsum, therefore, mandates thin soft tissue coverage (Figure 45.2). The recommended flap type is a fasciocutaneous flap that provides thinness and better tolerance of subsequent procedures, including tenolysis and debulking. Primary thinning of the flap has been advocated by some; however, in the author's experience, even with thin flap coverage, a minimum of two debulking procedures is usually required to make the foot suitable for normal footwear.

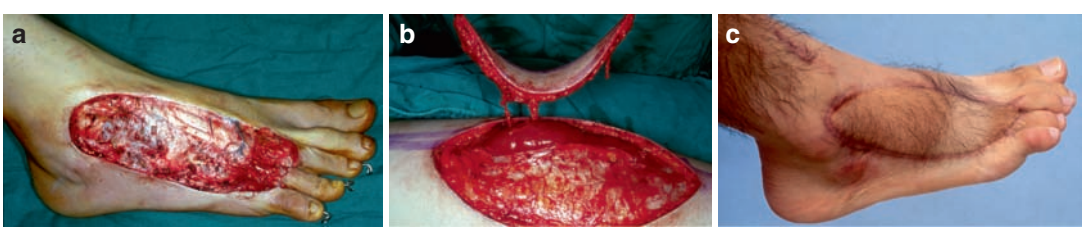

Figure 45.2. Foot dorsum coverage. (**a**) Foot dorsum defect following radical release of severe soft tissue contractures of the lateral three toes. (**b**) Thin suprafascial anterolateral thigh free flap. (**c**) Good foot contour with no toe contractures, permitting the use of normal footwear, shown 18 months following reconstruction.

Plantar Area

The skin in the plantar area is the thickest of the human body, reaching up to 3.5 mm. Unique fibroseptal structures together with dense subcutaneous tissue serve as a cushion and provide great resistance against shearing force during walking. In general, skin grafts suffice to resurface non-weight bearing plantar surfaces. Weight-bearing surfaces, whenever possible, should be replaced by similar plantar tissue; one should be aware that heel coverage with tissue other than plantar skin always carries a risk of breakdown. Plantar skin can be raised based on the medial plantar artery, the lateral plantar artery, or both. That based on the medial plantar artery is also termed the instep flap. It can be either pedicled or free, from the ipsilateral or the contralateral unaffected limb, respectively. The pedicled instep flap is well suited for resurfacing a plantar skin defect in the calcaneal weight-bearing area and is not too extensive.[61] Its donor site is located in the non-weight bearing area and can be covered with a skin graft without significant morbidity.

For coverage of a larger defect where an instep flap is not feasible, concerns regarding durability, stability, and sensation arise in selecting different flap options. In general, the former two are more critical as studies have shown that innervated flaps and noninnervated free flaps yield similar results in terms of protective sensation and walking ability.[53,60] Debates between skin-grafted muscle flaps and fasciocutaneous flaps continue. A well-inset muscle flap with skin graft produces stable interfaces between the underlying bone and the muscle and between the muscle and skin graft, and the fact that the muscle tissue will undergo fibrosis and contracture is the main contributing factor to its stability. However, its durability suffers. A fasciocutaneous flap is, conversely, of better durability and less stability; the thicker the subcutaneous fat, the greater the shear force generated. Stability and durability should be balanced to provide an optimal reconstruction.

In the author's opinion, a fasciocutaneous flap with subcutaneous fat of moderate thickness is the primary option, as it provides optimal durability and does not lose too much stability; a skin-grafted muscle flap is employed when an appropriate fasciocutaneous flap is not attainable.

Bone Reconstruction

Traumatic bone loss in the lower extremity is more crucial than that in the upper extremity, as the lower limbs are responsible for weight bearing and a length discrepancy will affect walking and exacerbate the development of osteoarthritis of the hip and knee. The bone defect may occur as a result of primary bone loss in the event of trauma and/or secondary bone loss following infection, surgical debridements, or devascularization (due to periosteal stripping or comminution). Key factors for successful bone reconstruction in the lower extremity are rigid bone fixation and adequate blood supply. An ideal bony reconstruction should achieve timely and stable bony union, reduce time to full weight bearing, and produce less chronic sequelae such as nonunion and chronic osteomyelitis.

One-Stage Versus Two-Stage Bone Reconstruction

An unhealthy soft tissue environment is responsible for bone graft failures or post-traumatic osteomyelitis; therefore, radical debridement is mandatory before bone grafting. A one-stage reconstruction of both soft tissue and bone defects is justified if the wound is clean and all dead space can be obliterated by the soft tissue component of the composite vascularized bone flap.[74] Otherwise, a more conservative approach that provides soft tissue coverage in the first stage followed by bone grafting in the second stage is recommended with a minimum of 6 weeks between the two stages.

Bone Reconstruction Options

The principal methods of bone reconstruction are conventional (nonvascularized) bone graft, vascularized bone flap, the Ilizarov technique (distraction osteogenesis), and their combinations. The selection depends on the location and length of the bone defect and the quality of the surrounding soft tissue.

Conventional (Nonvascularized) Bone Grafts

The nonvascularized bone graft, classified as cancellous or cortical, is a well-established clinical procedure. The typical healing process of nonvascularized bone grafts, termed "creeping substitution", consists of graft resorption and

bone remodeling by osteoclasts and new bone formation by osteoblasts. Cancellous bone grafts differ from cortical bone grafts in terms of shorter incorporation time and better efficacy in promoting union of unhealed fractures. Although the cortical bone graft can provide immediate mechanical strength, the required initial skeletal support can instead be obtained with bone fixators. In addition, the strength of a cortical graft will decrease and will be weakest at 6–12 months after implantation.[14] Cortical bone grafting is therefore of little value in lower extremity bone reconstruction.

Nonvascularized bone grafts suffice to bridge a bone defect up to 6 cm in length if the soft tissue envelope is healthy and adequate. A more lengthy bone defect warrants the employment of a vascularized bone flap or distraction osteogenesis. Shorter segmental defects, however, are usually still better reconstructed with vascularized bone flaps by virtue of improved healing and resistance to infection. Whatever technique is employed, the average time to complete bony union usually exceeds 8 months[49,74]; a protective splint is always needed for a period after removal of fixators to prevent stress fractures.

Vascularized Bone Flaps

With preservation of blood flow to the bone, the vascularized bone flap serves as a good solution to bridge bone gaps in a poorly vascularized environment. Unlike nonvascularized bone grafts, the incorporation of a vascularized bone flap undergoes a similar healing process to that in fracture healing, allowing faster and more reliable union. Indications for vascularized bone flaps for long bone reconstruction include (1) segmental bone defect of more than 6 cm in length, (2) segmental bone defect of any length without healthy and adequate soft tissue coverage, (3) repeated failures of conventional bone grafts, (4) septic or nonseptic nonunions, and (5) congenital pseudoarthrosis.[6,37,45,48] A careful assessment of the general condition of the patient, the wound condition, and the lower limb circulation are crucial before vascularized bone transplantations, as failed bony reconstruction can result in an amputation. The authors recommend that a skin paddle should always be included when transferring any bone flap for (1) simultaneous soft tissue coverage, (2) replacement of unhealthy or infected soft tissue envelope, (3) easier postoperative monitoring, and (4) to facilitate wound closure.

Commonly used vascularized bone flaps are the fibula, iliac crest, serratous anterior rib, and scapula. The free fibula osteoseptocutaneous flap, with its great versatility and reliability, is considered as the primary option for lower extremity long bone defects (Figure 45.3).[37] Several characteristics of the fibula make it ideal for lower extremity long bone reconstruction, including a high-density cortical bone, adequate length, a reliable skin paddle, and insignificant donor-site morbidity. It is possible to increase the size of the skin paddle when the soft tissue defect is extensive, to incorporate part of the soleus muscle for dead-space obliteration, and to osteotomize and fold the fibula into a double-barrel configuration to increase mechanical support.

Special Considerations: The Femur

With its abundant soft tissue envelope that provides more protection and better vascularity, a traumatic bone defect of the femur is far less encountered than at the tibia. The femur is a large-caliber bone and under severe stress during weight bearing. Whether reconstructed with vascularized bone flaps or nonvascularized bone grafts, significant hypertrophy of the transferred bone is required to achieve full weight bearing. The process of hypertrophy can last more than 1 year and yet be insufficient to allow full weight bearing. A fibula osteoseptocutaneous flap in double-barrel configuration is best suited for this situation by virtue of better mechanical strength and shortened time to full weight bearing; it is the preferred option when reconstructing segmental femur defects.[48]

Management of Chronic Complications and Functional Deficits

Post-traumatic Osteomyelitis

Chronic osteomyelitis is defined as an osseous infection persisting for more than 6 months. It may be latent and evolve over months or years and may derive from one or more foci containing pus or infected granulation tissue. Clinical presentations may be with low-grade inflammation, pus drainage, and/or a compromised soft

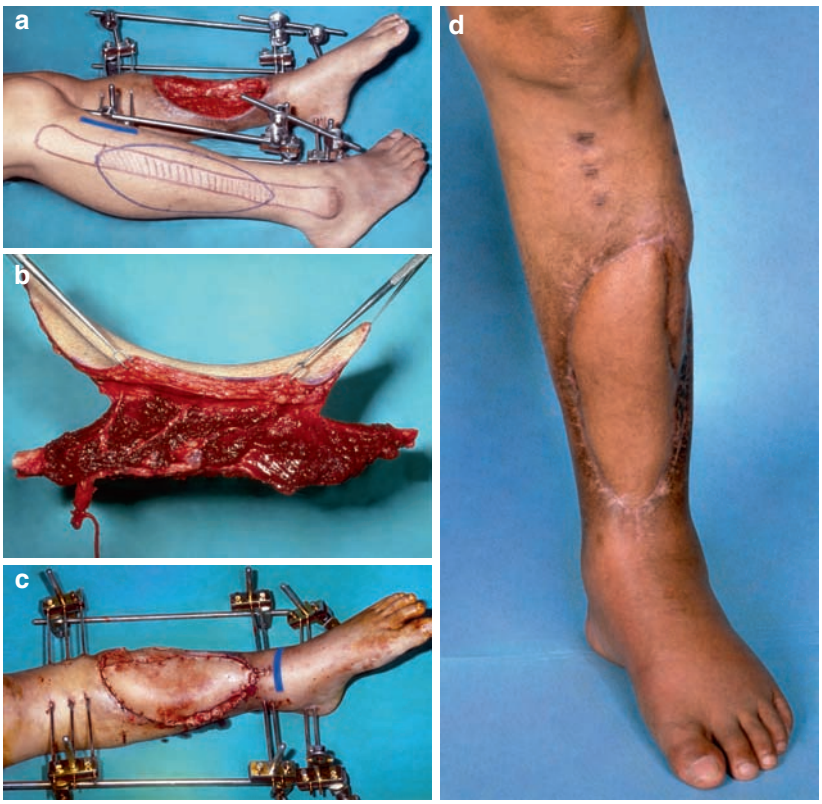

Figure 45.3. Fibula OSC flap. (**a**) Composite 15-cm tibial segmental defect from resection of chronic post-traumatic osteomyelitis. A fibula osteoseptocutaneous free flap has been designed accordingly on the contralateral limb. (**b**) Harvested fibula osteoseptocutaneous free flap ready for inset. Note the presence of two septocutaneous vessels to the skin paddle and the inclusion of a larger-than-usual muscle cuff for complete dead-space obliteration in the recipient site. (**c**) Composite bony and soft tissue reconstruction achieved with a single free flap based on a single pair of microvascular anastomoses. A small skin graft was required to release tension for the skin paddle. (**d**) Outcome, 18 months following reconstruction, with full weight-bearing ability after removal of bony fixation.

tissue envelope. Causes of post-traumatic osteomyelitis can be classified into wound and surgical factors; causative microorganisms may be introduced to the wound during trauma due to delayed wound coverage; inadequate surgical debridement of the devitalized bone or soft tissue may leave an environment favorable for bacterial growth. Regardless of etiologies, its diagnosis is based on bacteriologic and radiological analysis. Identification of causative microorganisms is essential for proper antibiotic treatment. Plain radiographs usually provide little diagnostic information but are helpful for follow-up and may be useful in demonstrating bone loss or sequestra. Bone scan is of greater sensitivity for earlier diagnosis of osteomyelitis.

Chronic osteomyelitis is primarily a surgical rather than a medical disease. Definitive treatment depends on radical removal of devitalized or infected bone and soft tissue. The resultant bony defect should be filled with bone grafts; indications for different bony reconstruction methods are as aforementioned. If the soft tissue overlying and around the infected bone is unhealthy and infected, it must be excised as well; the resultant soft tissue defect may require a flap transfer for coverage.

Post-traumatic Stiff Knee

Post-traumatic knee stiffness causes marked disability to the patient and is characterized by

limited knee joint range of motion, especially in flexion. Management of this problem requires an interdisciplinary collaboration of physical therapists, orthopedic surgeons, and plastic surgeons.

Skin and soft tissue contracture, adhesion of the extensor mechanisms (the patella ligament or quadriceps tendon), delayed rehabilitation, and prolonged immobilization are all contributing factors. In the setting of proximal tibial or distal femoral fractures, the extensor mechanism will usually be injured and, as a result, adhesion between the tendon/ligament and the underlying bone occurs. In addition, the knee will be immobilized for some time, because an external fixator is usually applied across the knee joint. Passive range of motion should be commenced immediately after external fixator removal; if rehabilitation fails to restore adequate knee flexion for daily activity, surgical intervention is indicated. Release procedures, which include scar excision/revision, V-Y quadricepsplasty, lengthening of the patellar tendon, and/or capsulotomy, must be thorough to allow adequate knee flexion.

The need for free flap transplantation after release procedures is not uncommon, as the native skin may be inadequate for wound closure when the knee is in flexion or may contract in the longer term. Lin et al. reported a series of nine patients with post-traumatic stiff knee successfully treated with release procedures and free tissue transplantations[68]; in their series, the mean knee range of motion increased significantly from 20° preoperatively to 100° postoperatively. The advantages of free flaps for stiff knee management include well-vascularized tissue for replacement of scarred soft tissue as well as abundant soft tissue to facilitate more aggressive release procedures.

Lower Extremity Reconstruction Following Tumor Resection

Prior to the 1970s, tumors of the lower extremity were treated almost invariably by amputation. Currently, however, lower extremity cancers can mostly be treated successfully with limb-sparing operations. Critical to this shift towards limb preservation are (1) increased basic scientific understanding of the biology and behavior of specific cancers; (2) advancements in the combined use of radiotherapy, chemotherapies, and surgery; (3) the availability of higher-resolution imaging modalities that define the extent of tumor invasion more precisely; and (4) improved surgical ablation and reconstruction techniques. Limb-sparing wide tumor resection followed by adjuvant chemotherapy and radiotherapy has allowed limb salvage without an increased risk of recurrence.[20,36,63] Amputation provides no significant survival benefit over limb-sparing surgery with adjuvant treatments for sarcomas, but approximately 5% of tumors still cannot be resected by a limb-sparing operation.[2,50,56,71] In addition, tumor shrinkage by preoperative radiotherapy and/or chemotherapies may facilitate resection.[10,51]

Tumors affecting the lower extremity are highly heterogeneous. Apart from metastases from distant sites, they include numerous types of primary bone tumors and soft tissue tumors, including melanoma and other skin tumors. Approximately 46% of soft tissue sarcomas affect the lower limb, with malignant fibrous histiocytoma, liposarcoma, and leiomyosarcoma being the most common subtypes.[62] According to recent data mined from the National Cancer Data Base of the American College of Surgeons, the largest proportion of each of the most commonly reported bone tumors occurs in the long bones of the lower extremity: (1) osteosarcoma (57.6% occur in the lower extremity long bones); (2) chondrosarcoma (31.3%); and (3) Ewing's sarcoma (27.9%).[9]

Limb-sparing resections for cancer can leave massive defects involving any combinations of tissue losses. Four basic types of excision exist for the various sarcomas; each is classified according to the relationship of the excisional dissection plane to the tumor and its pseudocapsule.[62] An *intralesional excision* is performed within the tumor mass usually for palliative intent; curettage and burr drilling, however, can be an adequate treatment for some benign bone tumors. In a *marginal excision*, the dissection passes through the pseudocapsule of the tumor. In a *wide* (*en-bloc*) *excision*, the tumor is removed with its entire pseudocapsule, together with a cuff of uninvolved tissue peripheral to the tumor/pseudocapsule in all directions. A *radical excision* involves removal of the tumor, its pseudocapsule, and the entire anatomical compartment within which it resides to account for skip lesions. A sarcoma resection generally demands a *wide excision*; although the "safe" thickness of the uninvolved tissue cuff remains a matter of controversy, it is generally believed to

be a few centimeters.[62,72] Guidelines for the safety margins for melanoma and other cancers are provided elsewhere. An amputation may occasionally be required to satisfy a clearance margin and can therefore be classified similarly as above. It should be particularly noted that there is no justification for limiting a limb salvage procedure based only on a patient's prognosis, even if he or she has only a short life expectancy. Indeed, some of the most valuable information regarding prognosis is unavailable until after the primary operation has been performed.[1,24,29,33,34,62] Segmental vascular or sciatic nerve defects can be reconstructed and are no longer absolute contraindications to limb salvage.[43]

Although limb-sparing surgery appears to offer the possibility of better psychosocial functioning and a more intact body image, the operation is often more complex and demanding than amputation and may therefore be associated with more morbidity and complications.[12,17,57] Amputees are prone to specific problems also, however, including phantom limb pain, bony spurs, joint contractures, requirements for revisional surgery, and progressive difficulties ambulating with or without their prosthesis in later life. Feelings of unattractiveness and embarrassment about their prosthesis may lead to patients having difficulties developing close relationships and restricting social activities.[23,52]

The reconstructive goals are to provide adequate wound coverage and timely wound healing so that adjuvant treatments are not delayed, to restore skeletal structure, to preserve and/or replace function, and to optimize aesthetic outcome. Microvascular free transplantation of vascularized composite tissues has been increasingly used to reconstruct these defects. In particular, vascularized bone-containing free flaps, in contrast to nonvascularized bone grafts, resist radiotherapy and tend to be osseointegrated satisfactorily. Successful wound healing despite radiotherapy (that alters vascularity) and/or chemotherapy (that causes immunosuppression) is vitally important for these patients and can usually be offered by microvascular free tissue transplantation. Furthermore, muscular free flaps can be transplanted with their motor nerve intact (for coaptation to an appropriate recipient motor nerve) if a functioning reconstruction is required; tendon transfer may complement such procedures or sometimes suffice alone, depending on the deficit. Since the administration of external beam radiation and/or chemotherapy requires that wound healing be complete, microvascular free tissue transplantation has proved an attractive reconstructive option for even smaller defects, as they provide fresh tissues from a distant site and can reinstate a sharp oxygen gradient to initiate wound healing. Many centers, therefore, prefer primary microsurgical reconstructions over local/pedicled flap reconstructions in such cases. Advantages also include reduced hospitalization time, decreased morbidity, increased rate of limb salvage, and higher patient satisfaction.[2]

Microvascular free transplantation of various composite tissues can complement the plethora of available orthopedic techniques to reconstruct massive bony and/or joint defects, including prostheses and megaprostheses, allografts, alloprosthetic composites, distraction osteogenesis and others.[41] An understanding of the availability and execution of these techniques is important for the reconstructive surgeon, but patients with lower extremity tumors are best treated by a combined approach involving all the necessary subspecialists working together in an organized multidisciplinary team.[73]

Conclusion

Advances in lower extremity reconstruction have allowed limb salvage following trauma and limb preservation during cancer resection. Microvascular free tissue transplantations deliver abundant soft tissue and bone with great reliability, facilitate aggressive debridements or resections, and provide better cosmetic and functional outcomes with acceptable donor-site morbidity.

The basic goal of lower extremity reconstruction following trauma remains to provide the patient with painless and stable walking and stance; however, there is a growing sense that an ideal reconstruction should also provide better cosmesis and function. Development of the multidisciplinary team approach has been critical to the improved management of patients in both trauma and cancer settings. Particular efforts should be made to minimize the occurrence of chronic complications and functional deficits following any reconstruction.

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46

Abdominoplasty

Peter A. Kreymerman and Raymond Isakov

Summary

Abdominoplasty is a common aesthetic operation that addresses a patient's abdominal skin excess, lipodystrophy, and wall laxity. The pertinent anatomy of the abdominal wall is introduced. Discussion includes patient evaluation and selection of the most appropriate operation for a patient's specific deformity. Techniques for traditional, limited, "mini," high lateral tension, and Fleur-de-lis abdominoplasties are highlighted. It is important to know the complications commonly associated with abdominoplasty and understand their management. Postoperative care of the patient is also discussed.

Abbreviations

ASIS	Anterior superior iliac spine
BMI	Body mass index
DVT	Deep vein thrombosis
IV	Intravenous
PDS	Polydioxanone

Introduction

According to the American Society for Aesthetic Plastic Surgery, U.S. surgeons performed more than 1,85,000 abdominoplasties in 2007. It was the fourth most common aesthetic operation. Its history dates back to nearly a century, when the first cases of abdominal fat and skin excision were reported.[5] Over the years, the operation underwent many improvements and modifications. The preservation of the umbilicus was an early improvement, and many different types of incisions were suggested as the operation evolved.[16] More recent variations to the operation and the expanding role of liposuction in body contouring have given the surgeon more options that can be personalized for the specific requirements of each patient (Table 46.1).

Indications

Patient evaluation and selection of the appropriate operation are critical. The patient deformity can be broken down into three issues that need addressing: skin excess, lipodystrophy, and abdominal wall laxity.

Women with multiple pregnancies typically present with mild to moderate skin excess, minimal to moderate amount of lipodystrophy, and significant abdominal wall laxity. Body mass index (BMI) greater than 30 is associated with increased complications and a less satisfactory aesthetic outcome. Well-placed incisions and umbilical appearance are very important to this patient population. These patients are typically addressed with a traditional, limited, or "mini" abdominoplasty depending on skin laxity, extent of diastasis recti, and umbilical position. A traditional

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Table 46.1. Abdominoplasty options.

Traditional
Limited
"Mini"
High lateral tension
Fleur-de-lis

(also known as full) abdominoplasty is performed for patients with moderate skin excess plus upper and lower diastasis. Instead, a limited abdominoplasty can be preformed for patients with mild skin excess and a high umbilicus. A good candidate for a "mini" abdominoplasty is one with mild lower skin excess and only lower abdominal diastasis. Liposuction can be used as an adjunct to simultaneously address epigastric or flank lipodystrophy.[9] Patients with moderate horizontal, in addition to vertical, skin excess can be treated using a high lateral tension technique. The method was described by Lockwood and challenged many of the principles of a traditional abdominoplasty.

Weight loss patients, either by gastric bypass or diet/exercise, can pose a different set of challenges. Excision of the majority of redundant truncal skin is the primary goal. Patients without an overwhelming amount of redundant horizontal (anterior and/or posterior) skin can be addressed with a traditional or a high lateral tension abdominoplasty. Weight loss patients with significant anterior skin excess, in the horizontal plane, get the best contour when treated with a Fleur-de-lis. Significant excess posterior skin is an indication for a circumferential lipectomy and is covered in Chapter 48: Postbariatric Reconstruction.

Preoperatively, patient history should be thoroughly reviewed, specifically focusing on history of significant comorbidities, continued fluctuations in weight, and smoking. Extensive physical examination should be performed to evaluate for any abdominal scars and/or hernias.

Anatomy

Knowledge of the layers of the abdominal wall and blood supply is essential in planning a successful operation. The layers of the abdominal wall can be broken down into skin, fat, fascia, and muscle. Understanding cutaneous innervation and the umbilicus is important.

The skin and fat overlying the rectus muscle are primarily supplied by perforators originating from the deep epigastric system. Secondary blood supply to this area is via the superficial epigastric system, intercostal, subcostal, and lumbar vessels (Figure 46.1). The deep epigastric system perforators are interrupted during flap elevation, and flap survival is dependent on the secondary supply.

The superficial and deep fat is divided by the superficial fascial system (Scarpa's fascia). The rectus sheath and muscle form the central portion of the anterior abdominal wall, whereas the external oblique, internal oblique, and transversalis muscles form the lateral aspect.

Innervation of the anterior abdominal wall is by intercostal nerves T7 to T12 traveling lateral to medial. The lateral cutaneous branches run within the subcutaneous plane. The anterior cutaneous branches travel between the internal oblique and transversalis muscles, then pass through the rectus muscle, and then enter the skin (Figure 46.2).

The position of the umbilicus and preserving its blood supply are important. The ideal position of the umbilicus is 12–14 cm from the pubis. Subdermal blood supply is interrupted by transposition, but perforators from the deep epigastric system and vessels within the stalk should be preserved.

Description of Techniques

Traditional Abdominoplasty

The ideal location of the incision is dictated by the patient's usual style of undergarment and the limitations imposed by the specific deformity. Patients are evaluated for marking in the standing and sitting positions (Figure 46.3). The lower incision line is marked from one anterior superior iliac spine (ASIS) down to just above the pubic hair and then up to the other ASIS. If the patient has a low transverse scar from a cesarean section, an attempt is made to incorporate it (Figure 46.4). The marking must account for some elevation of this incision, especially medially, due to cephalic pull from the elevated anterior abdominal wall flap. There is minimal elevation laterally, because the ASIS area is a zone of adherence. The possibility that there may be a small midline incision because the umbilical site

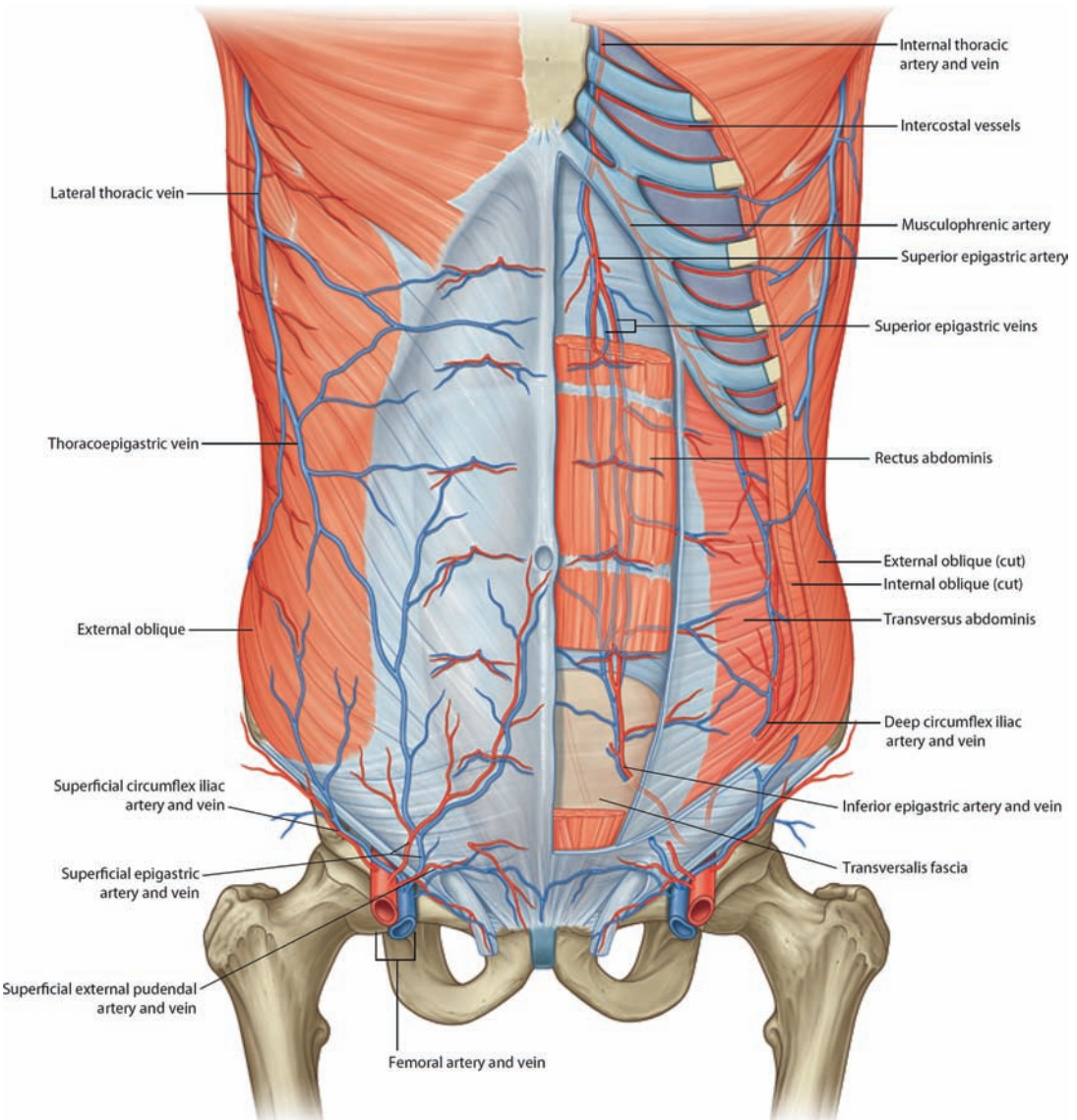

Figure 46.1. Abdominal wall vascular supply. (Reprinted with permission from Drake R., et al., (2008). Gray's Atlas of Anatomy. Copyright Elsevier 2008.)

could not be excised must be discussed with the patient.

The skin and subcutaneous tissue are incised to the anterior abdominal wall, and a flap is raised up to the subcostal margins laterally and the xiphoid medially (Figure 46.5). During flap elevation, a circumferential incision is made around the umbilicus, the stalk is dissected down to the fascia, and a stitch is used to orient the superior aspect. Careful hemostasis is maintained. Attempt is made to preserve as many intercostal, subcostal, and lumbar perforators as possible. In an attempt to decrease seroma formation, a thin layer of subcutaneous/lymphatic tissue is preserved attached to the anterior abdominal wall. The laxity of the midline abdominal wall is evaluated, and the rectus diastasis is plicated using #1 or 0 Ethibond, Prolene, or Polydioxanone

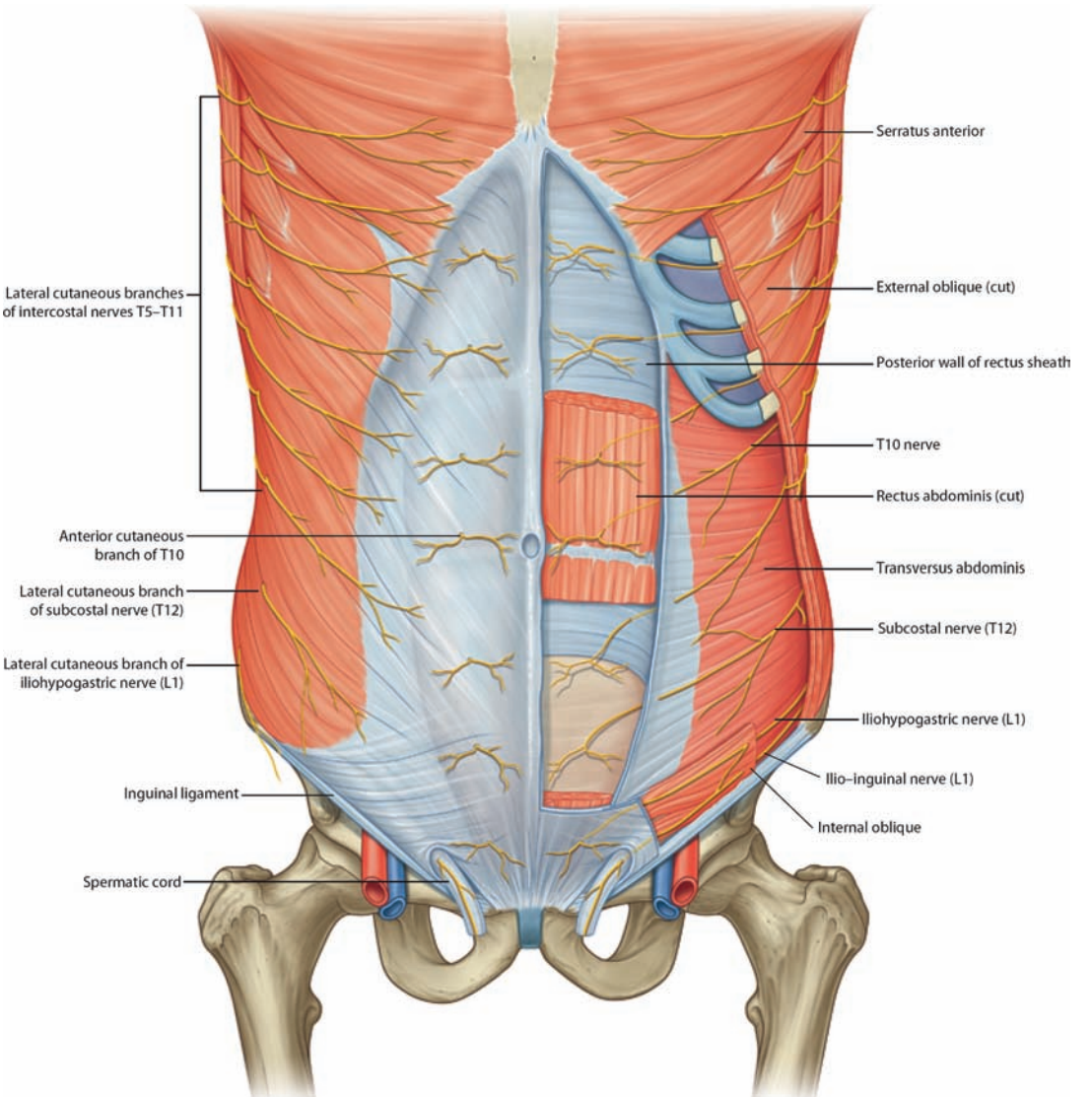

Figure 46.2. Abdominal wall innervation. (Reprinted with permission from Drake R., et al. Gray's Atlas of Anatomy. Copyright Elsevier 2008.)

(PDS) suture. This is done in two layers. Separately first above and then below the umbilicus, buried figure-of-eight sutures are placed. This is followed by a superficial running suture that will reinforce the deep layer (Figure 46.6). Lateral plication can also be added if a significant amount of lateral laxity remains, but it is associated with increased postoperative pain. The redundant portion of the elevated skin/subcutaneous flap is excised. The amount is determined by bringing the flap down caudally with the patient in mild flexion (Figure 46.7). Ideally, the site of the original umbilical insertion is excised, but it can always be sutured closed if there is too much tension on the flap or the mons pubis is pulled too cephalad. At this point, any planned liposuction of the flanks can be performed[11] (Figures 46.8 and 46.9).

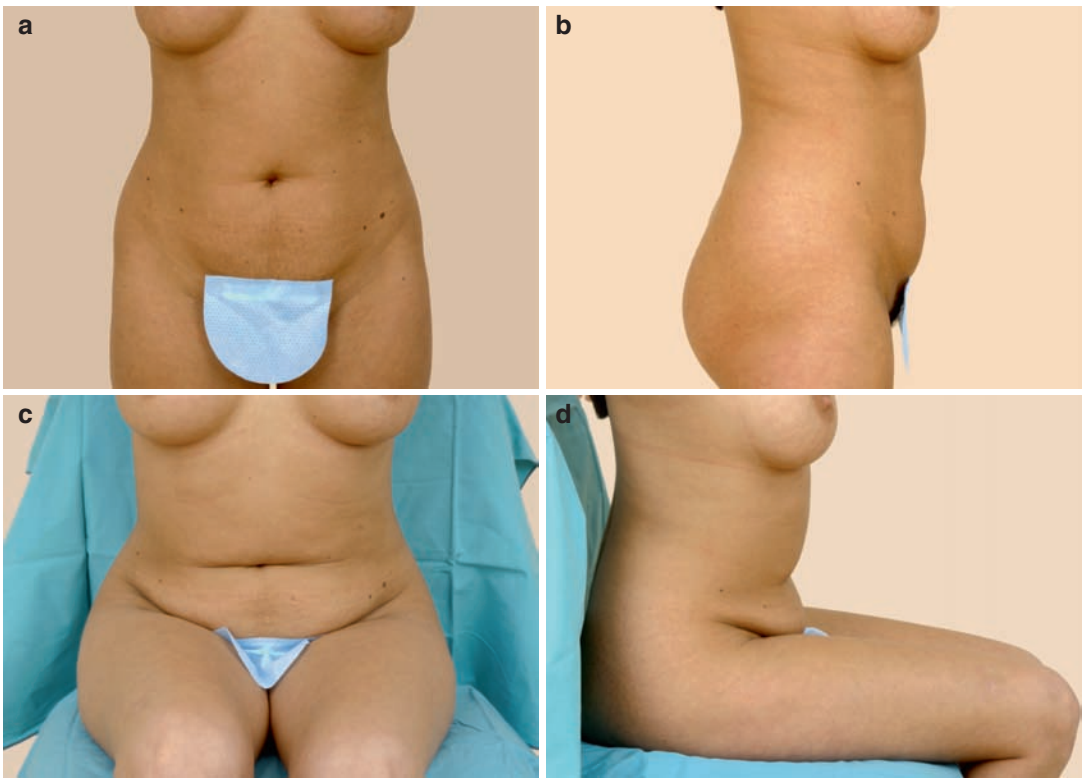

Figure 46.3. Thirty-one-year-old patient presenting for abdominoplasty consultation. (**a**), (**b**) Routine upright photographs. (**c**), (**d**) Sitting views better demonstrate her deformity.

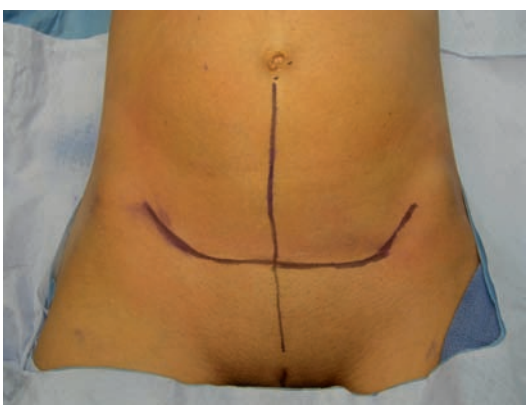

Figure 46.4. Intraoperative marking. Caesarian section scar included in incision.

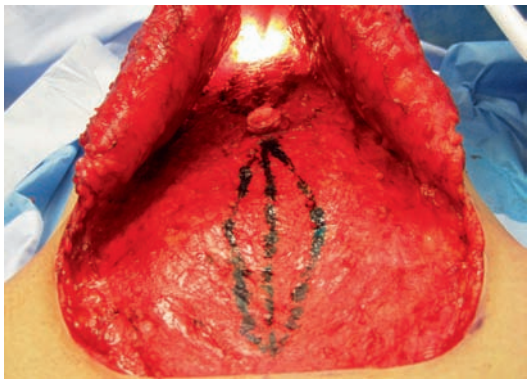

Figure 46.5. Dissection and elevation of skin/subcutaneous flap. Planned amount of plication is marked.

Two closed suction drains are placed through separate stab incisions, and the flap is advanced inferiorly. Quilting sutures when combined with drain placement have not shown a benefit in seroma reduction.[1] During closure, first the midline is reapproximated and the location of the umbilicus is marked. Suturing of Scarpa's fascia is with 2-0 PDS and the dermis with one or two layers of Monocryl. The umbilical stalk is

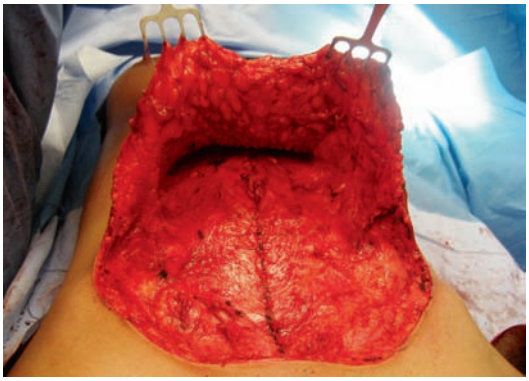

Figure 46.6. Midline plication with buried figure-of-eight Ethibond sutures followed by a running superficial suture line.

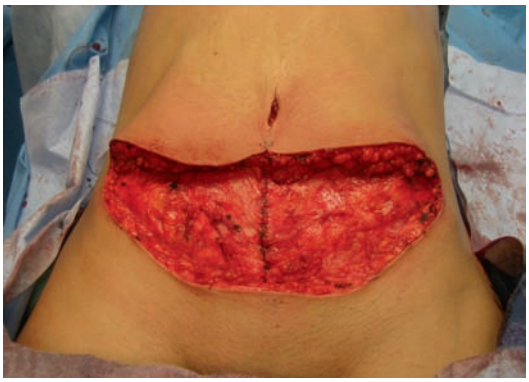

Figure 46.7. Prior to closure, the amount of skin/subcutaneous resection can be noted.

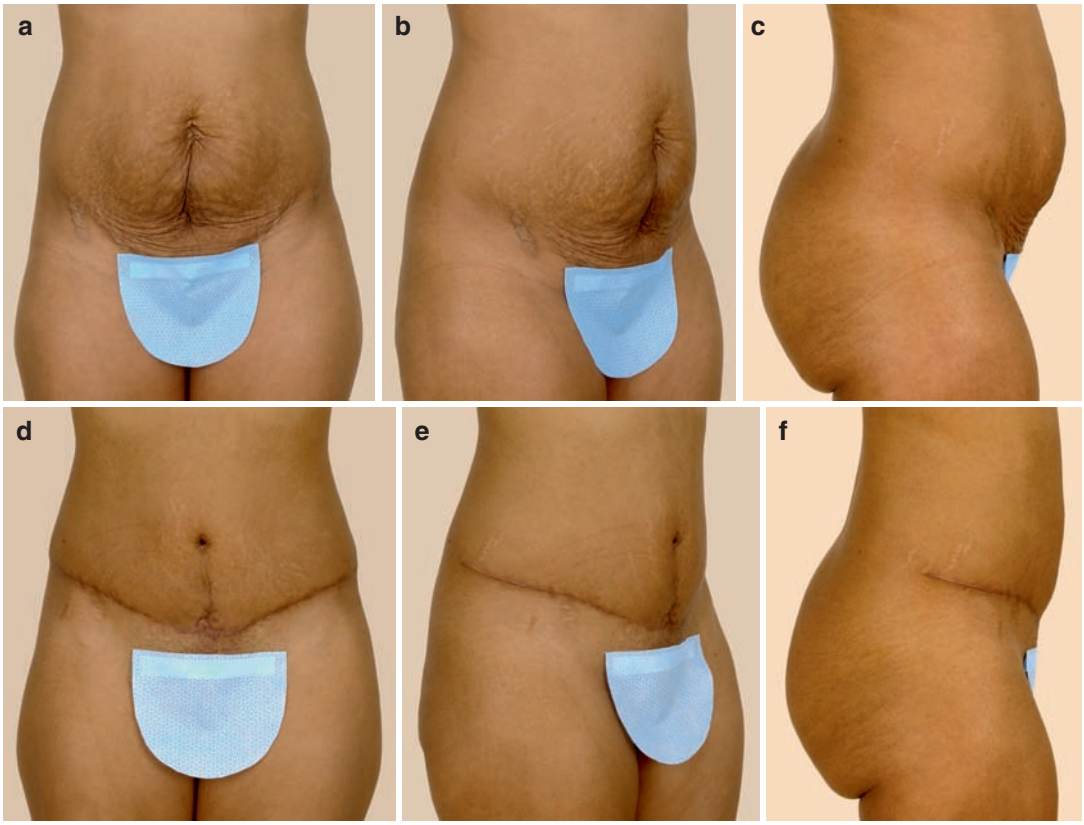

Figure 46.8. Thirty-two-year-old woman who underwent traditional abdominoplasty with flank liposuction. She had midline closure of the umbilical incision and developed a small eschar that was treated with local wound care. (**a**–**c**) Preoperative; (**d**–**f**) 4 months postoperative.

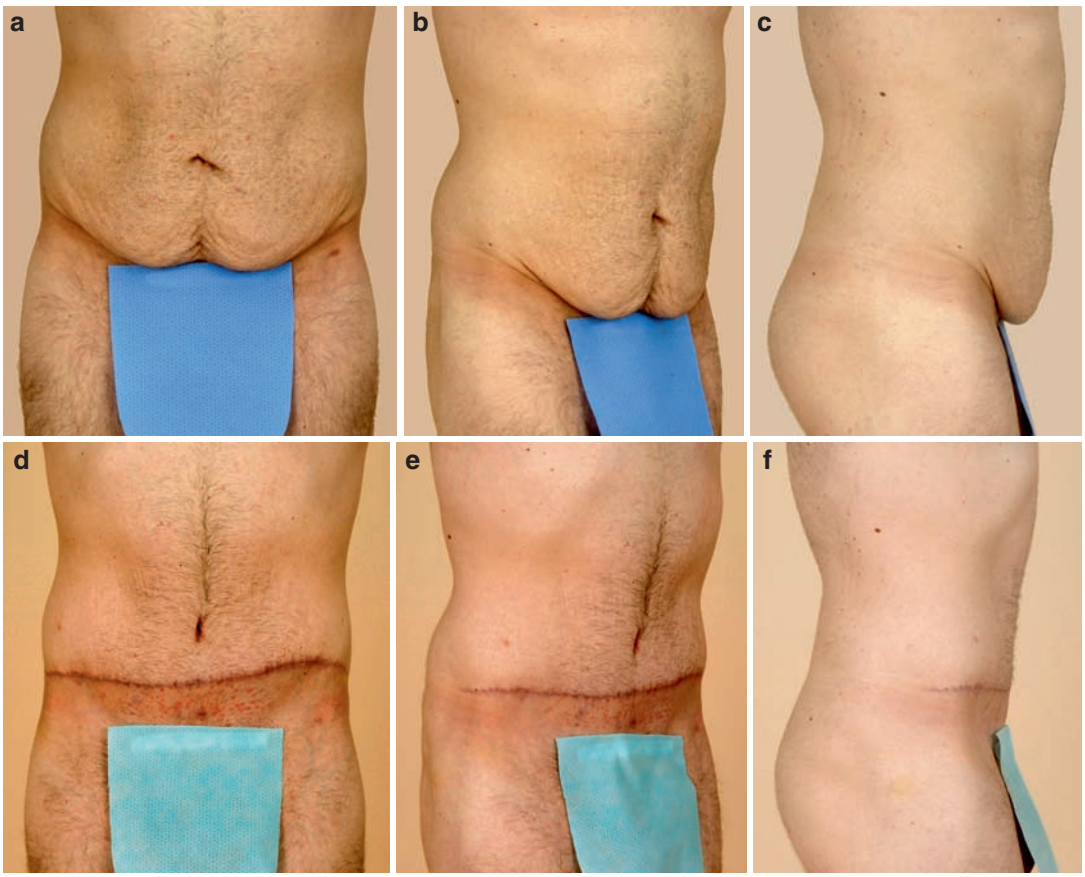

Figure 46.9. Twenty-two-year-old man with 150 lb. diet and exercise weight loss who underwent traditional abdominoplasty. (**a**–**c**) Preoperative; (**d**–**f**) 6 months postoperative.

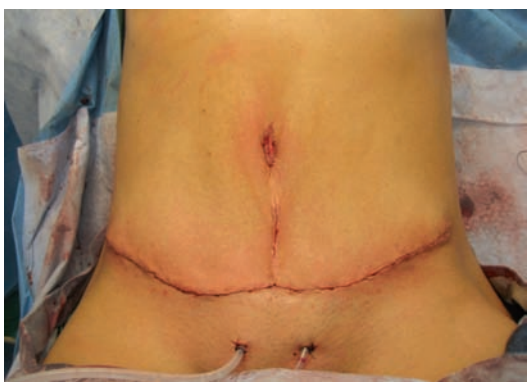

Figure 46.10. Intraoperative closure and drain placement.

brought out through a slit or triangular incision (Figure 46.10). Multiple variations of umbilical recreation have been described.[2,8]

Limited Abdominoplasty

The markings and operative technique are identical to the full abdominoplasty until the flap dissection reaches the umbilicus. Instead of excision, the umbilical stalk is transected at the base, and the umbilicus is "floated." Dissection to the costal margins and xiphoid is more limited, and only enough is performed to allow plication of the upper and lower midline. The flap is then pulled down inferiorly to determine the amount of excess skin and subcutaneous tissue that can be removed. The amount to be removed is restricted by the downward shift of the "floating" umbilicus. After excision of the inferior aspect of the flap, the closure is identical to the full abdominoplasty. If the umbilicus drops below the level of the ASIS, the appearance is unnatural. This procedure is ideal for an individual without a large amount of excess skin, good skin quality,

and with a high umbilicus. It predominately addresses an abdominal laxity problem.[10]

"Mini" Abdominoplasty

Patients with a small amount of lower excess skin and abdominal wall laxity that is isolated to below the umbilicus are strong candidates for this procedure. The markings are similar, but the incision can be made significantly shorter depending on the amount of extra skin that needs to be excised. The skin and subcutaneous flap are elevated to the level of the umbilical stalk. The lower rectus is plicated with two layers of suture. The flap is advanced inferiorly, the excess is removed, and the incision is closed by reapproximating Scarpa's fascia and skin. The downward traction of the flap might pull down the umbilicus a centimeter or two. Drains are again used. Liposuction can be used to address any flank or upper abdominal lipodystrophy.[10] Less lateral flap dissection allows safe epigastric liposuction.

High Lateral Tension Abdominoplasty

The Lockwood philosophy (Table 46.2) to abdominoplasty challenged the classic methods.

The markings are similar to a traditional abdominoplasty. The ideal location of the eventual scar is determined. Careful planning is important, because the incision will rise with flap elevation, excision, and closure. The periumbilical and low transverse incisions are made, dissection is carried down to the abdominal wall fascia, and the skin/subcutaneous flap is elevated. The dissection is restricted laterally, and only enough is performed to allow rectus plication. This is performed using large buried figure-of-eight sutures and is followed by running a more superficial suture to reinforce the deep figure-of-eight layer. Additional release and mobilization of the flap can be performed by conservative dissection. This is done bluntly with vertical scissor spread or even a finger. It is important to maintain as many perforators as possible. The elevated flap is split up the middle until the midlines of the flap and lower transverse incision can be approximated without significant tension. Then, the lateral aspect of either side of the flap is grasped and pulled at an inferior-lateral vector, whereas the lateral aspect of the cut abdominal wall on that side is pushed at a superior-medial vector under the elevated flap. The amount of flap excess is marked. This procedure in repeated on the opposite side, and the marked tissue is excised. During closure, tension will be directed laterally. Tumescent liposuction of the flap, pubis, and flanks is then performed. Drains are placed, and closure is initiated by thoroughly reapproximating the superficial fascial system. In this technique, typically not all the tissue between the umbilicus and transverse incision is excised. With the tension on the fascial layer, the dermis is closed tension free. The previously marked new umbilical site is incised, and then the umbilicus is delivered and sutured.[6,7]

Table 46.2. Lockwood philosophy.

Minimal medial dissection
Conservative central resection
Aggressive lateral flap resection (tension placed laterally)
Additional flap undermining with liposuction
Meticulous closure of the lateral superficial fascial system

Fleur-de-Lis Abdominoplasty

This technique not only directly addresses the vertical abdominal excess but also deals with the horizontal. Often patients are willing to trade a midline scar for improved contour. If the patient has a previous midline incision, it is incorporated. The operative dissection can be thought of as an extension of the high lateral tension dissection, because lateral perforators are preserved. Marking of the horizontal and the vertical excisions should be approached independently. The excess tissue is pinched, and the amount of planned excision is marked. It is important to be conservative with the initial markings.

An inferior transverse and two paramedian incisions are made. Dissection is carried down to the abdominal wall fascia, and the midline wedge of tissue is excised. The umbilicus can be spared and brought out during the midline closure. The skin flaps are minimally raised to allow some vertical excision, while preserving lateral blood supply. Undermining is discouraged. The rectus plication is performed in two layers with Ethibond, Prolene, or PDS. "Tailor tacking" is used to assess the vertical redundancy, and the excess tissue is removed. Closure is typically performed in three layers.[3,13] Many body contouring patients will receive an adequate

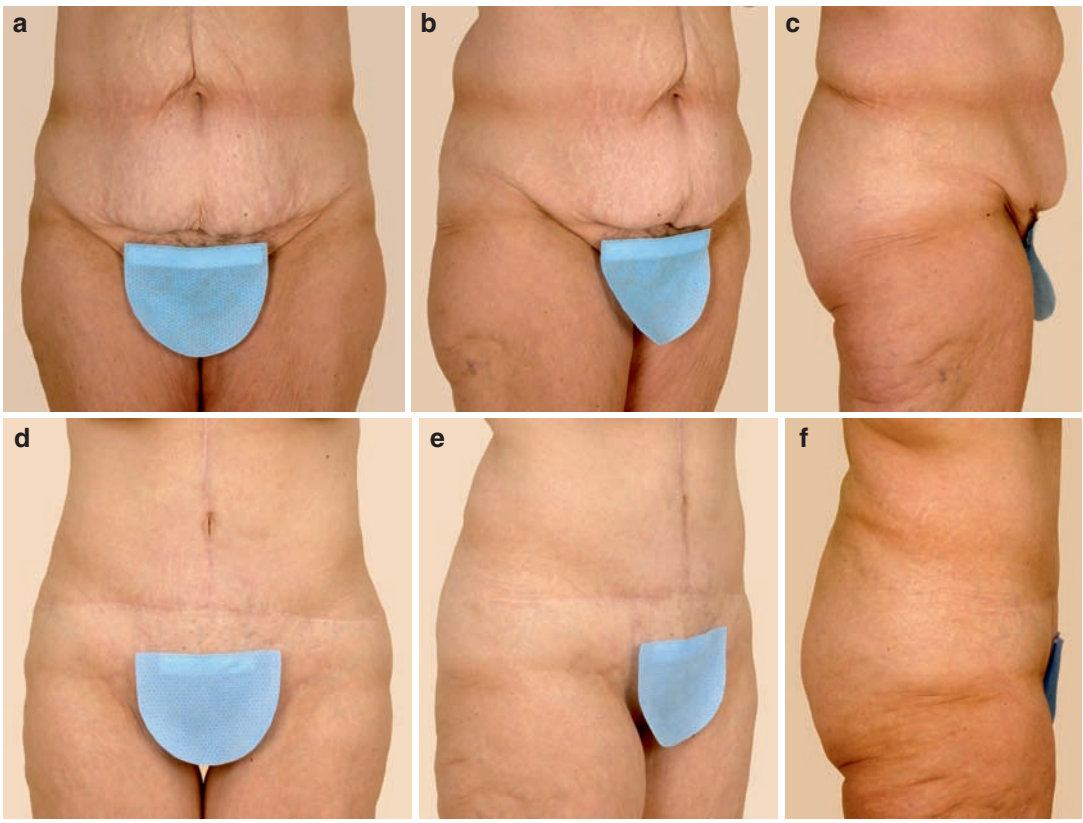

Figure 46.11. Forty-seven-year-old woman with massive weight loss after gastric bypass who underwent a Fleur-de-Lis abdominoplasty. (**a**–**c**) Preoperative; (**d**–**f**) 1 year postoperative.

result with the Fleur-de-Lis abdominoplasty and will avoid having a posterior resection (Figures 46.11 and 46.12). The epigastric fullness is sometimes a problem secondary to a "dog ear" created with upper midline closure. This method of abdominoplasty is more frequently associated with flap necrosis, usually at the inverted-T intersection.

Complications

Patients with a higher BMI and procedures with more undermining are at a greater risk for having a complication. Seroma is the most common complication and can usually be treated with drainage. Combining quilting sutures with drain placement has not been shown to decrease seroma formation.[1] Persistent seroma is treated with percutaneous drainage and rarely with sclerotherapy.

Wound healing problems are not rare. Skin tension, vascular compromise of the flap, infection, and/or marginal nutrition are usually responsible. In the traditional and Fleur-de-Lis abdominoplasties, flap skin loss and wound breakdown typically occur at the midline, because that is the point of most tension and vascular insufficiency. Any necrotic tissue should be debrided, and local wound care is usually sufficient (Figure 46.8). Aggressive lateral dissection combined with epigastric liposuction increases the wound complication risk.

Cellulitis is usually adequately treated with antibiotics, but abscesses must be drained and the wound packed. Scar revision is sometimes requested and should be done 1 year postoperatively after full maturation. Hematomas can be large due to the extensive "dead space" created during flap elevation, and these patients can become symptomatic. The incidence of atelectasis, pneumonia, and deep vein thrombosis (DVT) is

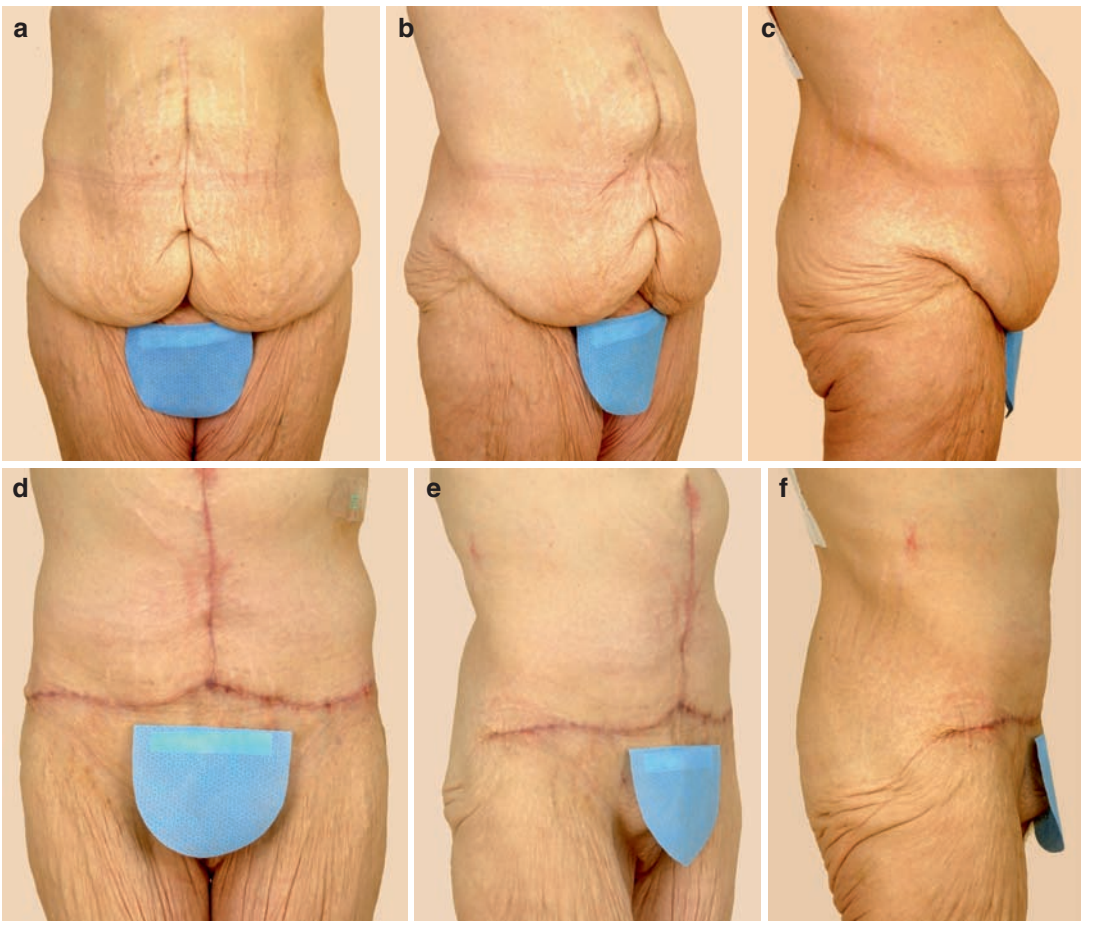

Figure 46.12. Forty-nine year old woman with 110 lb. weight loss after gastric bypass who underwent a Fleur-de-Lis abdominoplasty. (**a**–**c**) Preoperative; (**d**–**f**) 3 months postoperative.

reduced with early ambulation. DVT and subsequent pulmonary embolism are uncommon complications, but prophylaxis with compression stockings and appropriate vigilance are a must.[4,12]

Postoperative Care

Recovery is dependent on the patient and the type of procedure performed. Patient disposition ranges from spending a few hours in the recovery room to spending a few days in the hospital. Younger and healthier patients do well when recovering at home.[15] A patient with a borderline BMI and several comorbidities will benefit from at least overnight observation.

Oral narcotics are typically sufficient for pain control, but intravenous analgesia may be required for some hospitalized patients. The addition of Valium for muscle spasm can be helpful. Postoperative antibiotics are not necessary.[14] The patient spends a week sleeping in the "beach chair" position, early ambulation is encouraged, and an abdominal binder is optional. The drains are usually removed in 1–2 weeks, and patients typically return to full activities in 4–8 weeks.

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Lymphedema of the Extremities

R.G.H. Baumeister

Summary

Lymphedemas of the extremities are a worldwide problem. Whereas in tropical countries damage to the lymphatics by filarial diseases is the major source of lymphedemas, in developed countries, lymphedema due to a lymphatic blockade in the course of treatment or trauma is the most common cause. Conservative treatment plays a major role in the treatment of lymphedemas. However, lifelong therapy is advocated. Surgical therapy can be divided into three categories: resectional, diverting, and reconstructive procedures. In the case of a locally interrupted lymphatic pathway, bypassing the defect using the patient's own lymphatic vessels may normalize the lymphatic outflow of an extremity and therefore provide the chance for a cure and for becoming free from lifelong therapy.

Introduction

The lymphatic network is often underestimated. It can be damaged, often to a great extent, without causing trouble. The vessels are small, and the content is almost invisible. The system has the ability to repair itself to a remarkable extent. If, however, a lymphatic disorder occurs, secondary changes develop in the affected tissue, and the treatment becomes difficult. Therefore, interference with the lymphatic system should be restricted to the necessary extent, especially in oncologic surgery. In other circumstances, the surgical technique should respect the main lymphatic pathways.

Pathophysiology

Lymphatic physiology can be described as the balance or imbalance between the lymphatic load and the lymphatic transport capacity in a defined region of the body, for example, an extremity. The lymphatic load describes the amount of fluid and material that has to be cleared by the lymphatic system. The material is taken by blind ending lymphatic capillaries with their openings to the interstitial space and transported via lymphatic vessel and lymph nodes back to the venous system at the venous angulation.

The lymphatic transport capacity describes the clearance of the lymph fluid. It is dependent on the number and functioning of lymphatic vessels and lymph nodes.[11,12]

Iatrogenic medical interactions such as lymph node resections, transections of major lymphatic channels, followed by scar formation due to radiation or infection, are a major source affecting the transport capacity.

Secondary tissue changes, such as a supply of adipose tissue and fibrosis[26,27] occur, especially in a long-persisting imbalance with a predominance of the lymphatic load,

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Modern surgical strategies focus on the pathophysiology, trying to improve or normalize the lymphatic transport capacity, and to reduce surplus adipose tissue, if necessary.

Treatment of Lymphedema of the Extremities

Imbalances due to diminished transport capacity should be respected to avoid additional increase in the lymphatic load. Therefore, the possibility of getting scratches during gardening or getting injured walking barefoot can be minimized by wearing gloves or shoes. Medical intervention at the affected extremities should be restricted to the extent that is absolutely necessary.

Taking notice of the beginning of a swelling, therefore, is the first step to inducing treatment.

Thereafter, conservative treatment with manual lymphatic drainage, physiotherapeutic exercises, and application of elastic stockings will be the first step of therapeutic procedures. The question is, how long this therapy should continue. Since transient lymphedemas are described with a duration of about 6 months, this period should be reserved as a minimum for noninvasive therapy.

As surgery in former times was equivalent to resection procedures, this treatment was reserved for the final stage of the disease. Often, surgery was condemned by protagonists of conservative treatment.

However, because reconstructive procedures using autogenous lymphatic grafts to bypass a localized blockade of the lymphatic system could show a measurable improvement in lymphatic transport capacity, early reconstructive surgery should be indicated to prevent the sequelae of secondary tissue changes.[4,32]

Surgeons have three options to deal with lymphedemas.

- They can resect the surplus tissue.
- They can lead the lymph to a route different from the normal one with the help of flaps, for example, to a nonedematous area or to the venous system at peripheral sides.
- They can reconstruct interrupted lymphatic vessels to restore the lymphatic system itself.
- They could also combine the different approaches.

Resectional Methods

As edemas are characterized by an increase in epifascial tissue with a surplus of fat and fibrose tissue, partly associated with skin alteration, resection of the altered tissue is one possibility of treatment. This type of surgery is mostly reserved for the advanced stages.

The extent of resection varies within the treatment groups.

A local surplus of tissue at different areas may result in wedge resections. Cystic areas that are emptied by conservative therapy may also be resected locally.

Resection of large areas of subcutaneous tissues needs plastic surgical techniques creating flaps for covering the resected areas.

Finally, resection of all skin and subcutaneous tissue together with the fascia and covering the defect with split-skin grafts are part of the most radical procedure reserved for extreme, mostly tropical elephantiatic deformations.

Resection of prominent bulky tissue via wedge resection was described by Sistrunk.[29]

Modern forms of resectional methods are liposuction with reduction of the subcutaneous tissue[7,18] To keep the results stable, continuous support using a strong garment is necessary.

Resection of greater amounts of tissue needs plastic surgical covering. Resection of defects with the flaps from the same area was described by Anchincloss,[1] Fontaine and Fontaine,[13] Homans,[17] and Servelle.[26–28]

Circumferential resections of all the tissue together with the fascia, a method originally described by Charles,[9] need covering by split-skin grafts and are often performed in two stages to reduce the trauma.[2]

The main problem of resectional procedures is that the transport capacity is not changed. Therefore, relapse of edema or increased edema in parts of the extremity peripheral to the restricted area is common.

Diverting Methods

Leading the fluid away from the edematous area outside of the normal lymphatic route is the common principle of the diverting methods.

The first attempts to drain lymphfluid away from the epifascial compartment consisted of making holes in the fascia or resecting the fascia,

leading the lymph into the muscular department. Implantation of flaps of the greater omentum or segments of ileum in the edematous tissue should enhance spontaneous lympholymphatic anastomoses between the edematous tissue and the flaps. Implanting of threads or tubes to lead free lymphatic liquids away has also been attempted

Resection or fenestration of the fascia should allow the lymph to escape from the superficial compartment.[20,23]

A combination of resecting and diverting was proposed by Thompson. After resection of subcutaneous tissue and fascia, a slim flap is created, and the rim of the flap is de-epithelialized to open lymphatic channels. Thereafter, the flap is transposed deep close to the vessels to facilitate possible communication with the deep lymphatic vessels.[31]

Drawing off the lymph has also been attempted by inserting different tissues in the lymphedematous extremities, promoting spontaneous lympholymphatic anastomoses.

Different flaps have been described by Gillies,[14] Smith, Conway,[30] and Wynn.[33]

The greater omentum[15] and segments of ileum[19] have also been used in a similar manner.

The most popular method is to create peripheral lymphonodule or lymphovenous anastomoses.[10,22,24] One of the most significant experiences was reported by O'Brien.[25]

One of the major problems is reflux of blood due to adverse pressure gradients and enhanced thrombosis of the anastomoses, in peripheral lymphovenous connections.

Reconstructive Methods

Thanks to the progress of microsurgery, a locally interrupted lymphatic pathway can now be bridged by the patient's own lymphatic vessels. They are harvested from the medioventral bundle in the thigh. Direct lympholymphatic end-to-end or end-to-side anastomoses are created to allow the lymph to flow in its own appropriate vessel.[5,6]

Before the transplantation of lymphatic vessels, venous grafts were used[16] and promoted.[8] Experimental comparison between lymphatic and venous grafts have revealed the superiority of the lymphatic vessels.[34]

Indication

Secondary lymphedemas due to a locally interrupted lymphatic system are the main indication for lymphatic grafting. In western countries, armedemas due to operative interactions at the axillary lymph nodes or the inguinal or pelvic region are commonly seen.

As one leg has to serve as the harvesting site in leg edemas, only unilateral edemas of the lower extremities can be treated.

In primary lymphedemas, a selected group with unilateral atresias of the lymphatic system can be treated by lymphatic grafting as well.

In cases with a history of malignancies, the patients must be tested to be tumor free.

Since the burden of surgery is comparable to venous interventions in the subcutaneous tissue, there is almost no known general restriction for this type of surgery.

Each patient should report on adequate conservative treatment before surgery with a duration of at least 6 months; therefore, before reconstructive surgery, all patients have to get at least a complete conservative therapy, including manual lymphatic drainage, elastic stockings, and physical therapy for at least 6 months.

Preoperative Diagnostic Procedures

The harvesting side has to be checked by lymphoscintigraphies to show that there is no preexisting lymphatic disorder.

The edematous extremity is also checked by lymphoscintigraphy to clarify the lymphatic outflow and to give a baseline for postoperative controls.

Especially in primary lymphedemas, the situation of the lymphatic system in the periphery is checked, either by indirect lymphography using water-soluble contrast medium or by MR Lymphography, demonstrating possible variation of the lymphatic vessels.

In the case of a malignancy, the patient has to be shown as tumor free.

Operative Technique

The grafts are harvested from the medial aspect of the thigh. As many as 16 lymphatic vessels can be found here, within the ventromedial bundle.[21] About one to three vessels are used as grafts, to avoid the narrowing of the lymphatic system at the groin and at the knee region.

To facilitate the preparation, about 15 min before the incision, Patent blue® is injected subdermally in the first and second web space. The joints are moved to improve the transport of the dye.

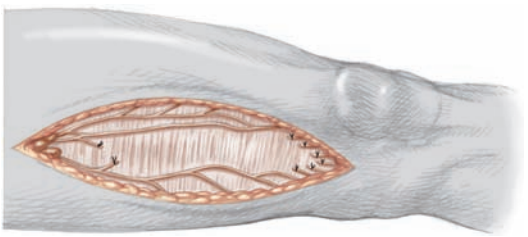

Figure 47.1. Harvesting lymphatic vessels from the patient's thigh. (Reprinted from Berger A, Hierner R, eds. *Plastische Chirurgie: Extremitäten*. Heidelberg, Germany: Springer; 2008.)

The incision is started medially to the palpable vessels beneath the inguinal ligament. The incision is extended distally step by step following the direction of the stained vessels.

Also the ramifications of the main lymphatic collectors can be used for anastomosing purposes. Therefore more lympho-lymphatic anastomoses can be performed at the affected extremity as the equivalent of the number of the harvested main collectors. For safety reasons, it is necessary that stained lymphatic vessels also remain untouched.

Depending on the length of the thigh, the grafts can be harvested up to a length of about 30 cm (Figure 47.1). The grafts are secured at the proximal end with 6-0 sutures and transected proximally and distally. On the distally transected side, the incoming lymphatic vessels are ligated to avoid lymphatic leakages. In armedemas starts the description of the procedure at the edematous extremities.

In armedemas, an oblique incision is performed at the inner aspect of the upper arm. Under the microscope, the tissue is searched for lymphatic vessels. Since the transport of dye is disturbed in lymphedema, no staining is performed.

At the neck, an oblique incision is made at the dorsal rim of the sternocleidomastoid muscle. Prior to that, a dye injection is performed cranial to the ear to enhance the chance of dyeing the lymphatic vessels at the neck. Behind the muscle up to the lateral border of the internal jugular vein, thin-walled lymphatic vessels can be found. Often, it is easier to prepare several lymph nodes.

In between the incisions at the upper arm and the neck, a tunnel is created by blunt dissection, and a silicon tube is temporarily inserted (Figure 47.2).

In the tube, a thread is inserted, and with its help, the grafts are pulled through. Finally, the tube is removed, and the grafts lie in the subcutaneous tissue without tension.

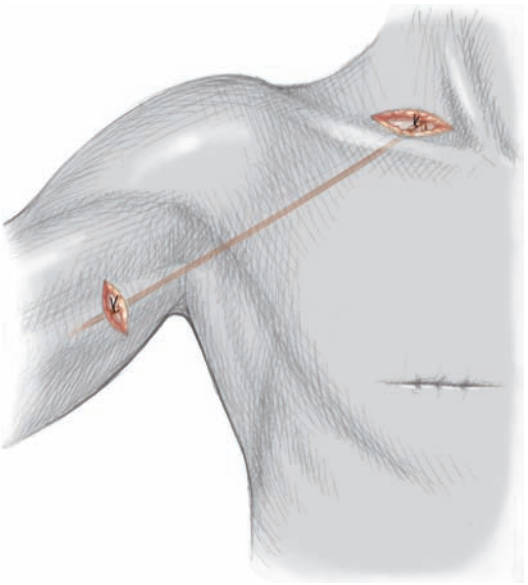

Figure 47.2. Bridging a lymphatic gap at the axilla with autogenous lymphatic vessels, lympholymphatic anastomoses at the upper arm and the neck. (Reprinted from Berger A, Hierner R, eds. *Plastische Chirurgie: Extremitäten*. Heidelberg, Germany: Springer; 2008.)

The anastomoses are performed under the so-called tension-free anastomosing technique in an end-to-end or end-to-side fashion with 10-0 absorbable suture material.[6] In the neck region also, lympho-lymphonodular anastomoses can be performed.

In unilateral lymphedemas of the lower extremities, the grafts remain attached to the inguinal lymph nodes at the harvesting side. Ascending lymphatics are dissected via an incision below the inguinal ligament at the affected side. The grafts are placed in a technique similar to that in arm edemas. After microsurgical lympholymphatic anastomosing, the lymph flows via the grafts to the healthy side (Figure 47.3).

Postoperative Procedures

The limbs are elevated, and bed arrest is recommended for 3 days. For about 5 days, antibiotics are given, and infusions of low molecular dextrane or HAES are administered. Elastic bandaging is applied, and elastic stockings should be worn for 6 months. In addition, a prophylaxis against erysipelas is recommended for the same time period. Thereafter, we try to discontinue the additional therapy. About one-third of the

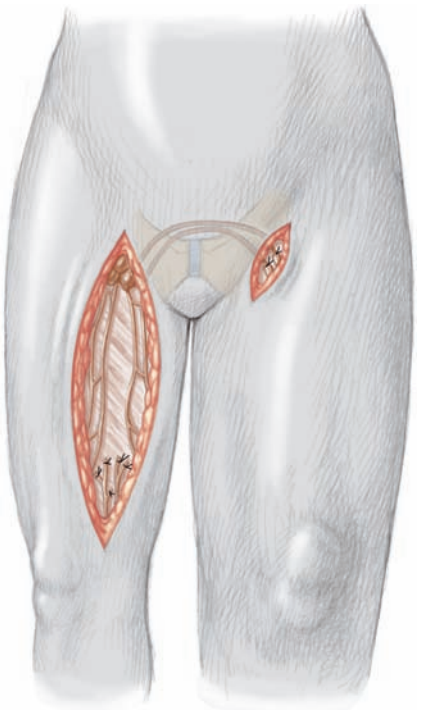

Figure 47.3. Lymphatic grafting in unilateral lymphedema of lower extremities; the grafts remain attached to the inguinal lymph nodes. (Reprinted from Berger A, Hierner R, eds. *Plastische Chirurgie: Extremitäten*. Heidelberg, Germany: Springer; 2008.)

patients do not need any additional therapy, one-third use the garments during heavy work, and one-third of the patients also need additional manual lymphatic drainage.

Postoperative measurements show a significant reduction in volume even after a follow-up period of more than 8 years in arm edemas.[3] Lymphoscintigraphic studies show a significant improvement of the lymphatic outflow. In a subgroup of patients with clearly visible lymph transport along the graft, a normalization of the lymphatic transport index was also measured.[32]

Long-time patency for more than 10 years of the grafts could be seen using indirect lymphography.

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Postbariatric Reconstruction

Jeffrey A. Gusenoff and J. Peter Rubin

Summary

Postbariatric reconstruction begins with proper evaluation and selection of the massive weight loss (MWL) patient. Patients with a lower body mass index (BMI) can be offered a wider scope of safe surgical procedures. Traditional abdominoplasty techniques must often be modified for the MWL patient, as deformities vary significantly. The circumferential lower body lift (LBL) has a powerful effect on the lateral thighs, buttocks, and anterior trunk in a single operative setting. Because of opposing vectors of pull, the vertical thigh lift is best performed in a separate operative setting as the LBL. Vertical thigh lift, performed as a staged procedure relative to the LBL, will complete the aesthetic contouring of the thighs. Vertical thighplasty performed without an LBL in the staged plan will provide adequate results less commonly. Brachioplasty techniques must address the axilla to achieve satisfactory results. In addition, careful segmental resection of arm skin will avoid over-resection. Mastopexy in the MWL patient can be performed using a dermal suspension technique with selective autologous tissue augmentation. Several techniques for upper body lift can be used concomitantly, melding scars, depending on the deformities. Staging multiple procedures of the upper and lower body can maximize safety and aesthetic outcomes.

Abbreviations

MWL	Massive weight loss
BMI	Body mass index
LBL	Lower body lift
SFS	Superficial fascial system

Evaluation of the Massive Weight Loss Body Contouring Patient

Massive weight loss (MWL) is a major life-changing event for patients. Although weight loss success may be achieved, self-esteem may not be fully restored. Thus, all patients should be congratulated on their weight loss success to acknowledge their accomplishments. This also serves as an introduction to obtaining a detailed history including weight loss (mechanism of weight loss, highest weight and body mass index (BMI), lowest weight since bariatric surgery or other weight loss method, current weight and BMI, how long weight has been stable, and goal weight); pregnancy; prior surgery (general and plastic); DVT, PE, or coagulopathy; nutritional status (protein intake (50–70 g), vitamins (calcium, B12, iron), nausea, emesis, dumping syndrome); and other medical comorbidities (i.e., hypertension, diabetes, sleep apnea, cardiac disease, smoking, mental illness).

Consultations with other specialists are ordered as indicated to evaluate and optimize medical

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issues before surgery. Establishing patient goals and identifying main areas of concern along with setting realistic expectations are key components of the initial body contouring discussion. Patients must be prepared to trade in excess skin and fat for new contours and new scars.

An overall physical assessment takes into account the distribution of skin laxity, remaining adiposity, rolls, folds, skin tone, integrity, scars, abdominal wall structure (rectus diastasis, hernias, thickness), and overall constitution (i.e., poor mobility, chronic pain, stigmata of malnutrition). Asymmetries should be demonstrated to the patient and documented. Standardized photographs of the patient should be taken from multiple angles to document preoperative deformities.[1]

Patients typically present for postbariatric reconstruction at least a year after gastric bypass surgery and should be weight stable for at least 3 months. Current literature suggests that a BMI greater than 35 may increase the risk of surgical complications.[2,3] Patients with a BMI greater than 35 should generally lose more weight before opting for surgery. Exceptions include a true giant disabling pannus or chronic panniculitis, conditions for which a functional panniculectomy would be indicated. A favorable BMI, however, does not imply that the patient is a good surgical candidate. Nutritional status, medical and psychological issues, expectations and goals, financial concerns, time off from work, and support systems (family, friends) all need to be assessed prior to surgery. For patients who are not ready for surgery, encouragement and reassurance can motivate them to work on these issues and follow up for another consultation.

Hernias are common in patients who have had previous open abdominal surgery. Consideration should be given to a team approach with the bariatric surgeons. Postbariatric reconstruction can be performed at the same time as hernia repairs; however, operative time for lengthy procedures should be considered.[4] Preoperative bowel preparations may decrease the risk of intraoperative contamination and postoperative discomfort.

The only absolute contraindication is the presence of systemic medical disease that precludes safe general anesthesia and significant surgical trauma. The authors consider tobacco use a strong relative contraindication and make all patients stop the use of tobacco products at least 1 month before and after surgery. Other relative contraindications include active intertrigo at the operative site, BMI greater than 35, coagulopathies, collagen diseases and other disorders that would interfere with wound healing, and severe pulmonary, cardiac, or renal dysfunction.

Variations of Abdominoplasty for the Weight Loss Patient

The abdomen is perhaps the area of most concern for patients who seek postbariatric reconstruction.[5–7] For the MWL patient, the abdominal pannus is often the source of functional problems, with difficulty ambulating or intertrigo. In general, patients with a higher BMI and/or those with higher medical risk will undergo a functional panniculectomy. The umbilicus is usually sacrificed, no plication is performed, and undermining is limited.

A true giant pannus is not a very common entity and is most often seen in patients who start the bariatric surgery process at an extremely high BMI and, despite significant weight loss, may still have a BMI that is in the severely obese range. The functional impairment of the giant pannus can warrant the risk of surgery. To facilitate the operation, the pannus is suspended from ceiling bars using orthopedic pins and traction bows. This allows venous blood to drain from the pannus, prevents the pannus from resting on the patient's chest (which can impair ventilation), and enables better exposure and control of the impressively large blood vessels that will be encountered.

A vertical scar may be necessary to eliminate additional horizontal excess in the MWL patient. If not done at the initial operation, it may be added as a second stage if a patient is not satisfied with the results of a transverse only abdominoplasty.[8,9] Undermining should be limited beyond the area of resection to preserve perforators. Great care is taken to avoid excessive tension on the "triple point" where complications are common. Tissue in the horizontal axis is resected first followed by the vertical resection. When insetting the umbilicus, minimal, if any, cutout should be created. With lateral tension and time, this will naturally widen. If a circular incision is made at the site of the umbilicus inset, this will widen to an undesirable horizontally elongated shape.

During the abdominal contouring procedure, the incision for the lower margin of resection should be placed 6 cm above the anterior vulvar commissure (just above the pubic symphysis)

with the tissues on upward stretch. Both thinning of the mons with direct excision deep to the superficial fascial system (SFS) and suspension to the abdominal fascia may be necessary to avoid an unsatisfactory result. Liposuction may result in prolonged edema and an unpredictable result. Three to five sutures are placed from the deep layers of the mons SFS into the abdominal wall fascia using 0-braided nylon. This technique has resulted in durable results with a high degree of patient satisfaction. Patients are often so pleased to have this region rejuvenated that they report an improvement in their sexual function. Patients are warned that the angle of the urine stream may be changed temporarily because of the pull on the mons tissues.

Lower Body Lift

Correction of the abdomen alone is often not enough to restore appropriate contour for many MWL patients. Many procedures have been devised to affect a circumferential correction of laxity in the buttocks, lateral thighs, and abdomen with elimination of rolls and festoons.[10–14] Lockwood popularized the lower body lift (LBL) and introduced many important contributions in this field including the repair of the SFS.[15] Further enhancements in outcomes have come from autologous augmentation of the buttocks with LBL procedures.[16,17] Fat can be preserved from the posterior resection based on gluteal artery perforators, de-epithelialized, and rotated into pockets over the gluteal muscles to give shape to a region that otherwise becomes routinely flattened. For the abdomen, the fleur-de-lis can be added concomitantly if laxity remains in the horizontal vector.

The original description of the body lift by Lockwood involved correction of the buttocks and lateral thighs with the anterior scars merging into the groin crease (Type I lift). This procedure was later modified by Lockwood to be a combination of a thigh/buttock lift and an abdominoplasty (Type II lift). Although Lockwood described this operation as a three-position procedure (lateral decubitus, contralateral decubitus, and then supine), the authors advocate a two-position procedure (prone followed by supine). Either technique will allow intraoperative thigh abduction to result in less tension on the closure.

Markings are dependant on the specific body type: a higher (more superior) circumferential resection will directly excise flank rolls and emphasize the waistline, while a lower (more inferior) resection will effect a stronger elevation of the lateral thigh tissues and provide a greater ability to contour the buttock region using autologous tissue flaps. It is the author's preference to keep the resection as low as possible to maximize the correction of saddlebag deformities and optimize buttock shape. Abdominal wall plication and vertical abdominal skin resection, when indicated, will provide adequate waist definition. The markings begin with the patient in the supine position. With upward stretch on the lower abdominal tissues, a mark is made approximately 6 cm superior to the anterior vulvar commissure. The markings continue with the patient in the standing position facing away from the surgeon. The first critical decision is to select the superior anchor line. The superior anchor line is extended from the mid-axillary line to the posterior midline bilaterally. Vertical reference marks are drawn at 6 cm intervals. A pinch test estimates the amount of resection. As the lateral tissues are estimated, the patient is asked to slightly abduct the legs. The inferior line of resection is then drawn. The lateral margin of resection is then selected to connect the superior and inferior lines of incision. This is usually at the mid-axillary line and will be a transition zone between the posterior and anterior resections. At this point, estimation is made about the amount of adipose tissue that will be preserved to shape the buttock region. This can take the form of either an island of adipose tissue or an actual fasciocutaneous flap that will be undermined in its lateral region and transposed into the inferior buttock region. The patient is then asked to turn and face the surgeon. With upward tension exerted on the tissues on the patient's right hip, a line is drawn from the lower margin of resection and connected to the point above the mons. The same maneuver is then repeated on the contralateral side. The line just drawn will represent the line of incision for the abdominoplasty part of the operation (Figure 48.1). Routine intraoperative care includes warming blankets, Foley catheter, appropriate padding, and sequential compression devices. The legs are prepped in a circumferential fashion. The Lockwood discontinuous undermining device (Byron Medical, USA) is passed in the subcutaneous tissue of lateral thighs allowing mobilization during closure. To facilitate tension-free closure in the lateral aspects of the posterior

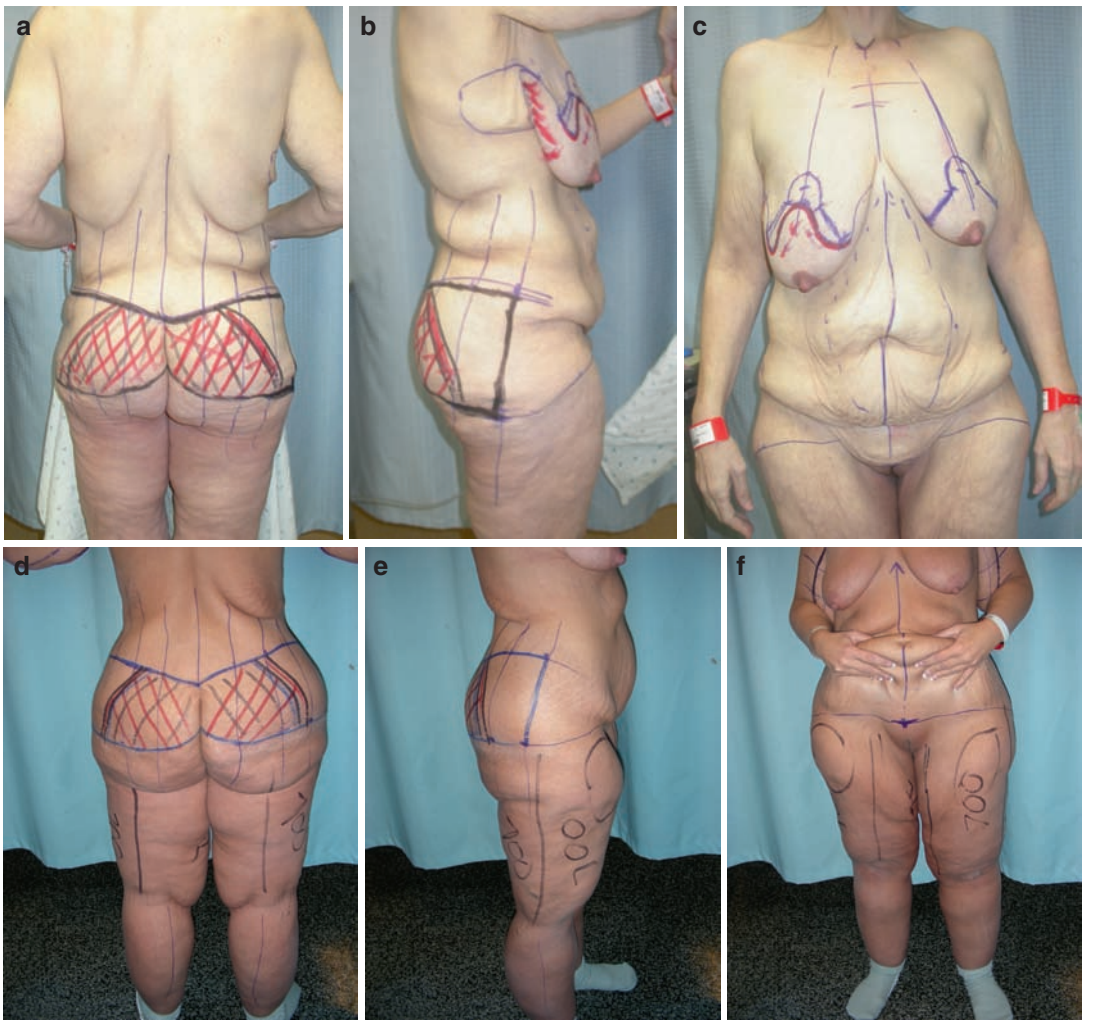

Figure 48.1. Examples of two patients marked for circumferential lower body lift. On the posterior views (**a**, **d**), the *red hashed area* indicates the gluteal fat tissue that will be preserved for buttock shape. Note that these patients have very different body types, but both will benefit from preserving gluteal fat. On the lateral views (**b**, **e**), the extent of lateral skin resection is noted. On the anterior views (**c**, **f**), the low position of the transverse scar is observed. With the tissues on upward stretch (**f**), the final scar location is suggested. The second patient (**d**, **e**, **f**) is also marked for extensive debulking liposuction concurrent with the lower body lift. This will facilitate better results for the second-stage vertical thigh lift.

wound, the legs are abducted onto extended arm boards that are placed in the lower region of the bed. The wound is secured with towel clips to approximate the skin edges and closed over drains with a two-layered closure and skin glue. The large lateral dog ears are stapled shut and are resected when the patient is supine. An occlusive plastic dressing over gauze is placed on the wound for the remainder of the procedure. The patient is then flipped and prepped. At this point, the procedure is essentially the same as any abdominoplasty. If the posterior resection is set low/inferior, flexing the waist in the supine position will not put much tension on the posterior closure. If, however, the posterior resection is high/superior on the trunk, flexing the waist may apply pull on the posterior closure, and a less aggressive resection should be planned. Two closed suction drains are placed in the abdomen. SFS sutures, using absorbable braided suture, are placed in the lateral high-tension areas. For postoperative care, the patient is kept

in a "beach chair" position, flexed at the waist. The patient is instructed to be out of bed with assistance by the following morning or the same night, if possible. The average inpatient stay is two nights for this procedure. An example of a body lift without fleur-de-lis abdominoplasty is shown in Figure 48.2. An important adjunct to the LBL, especially in staged cases, is debulking liposuction of the thighs. Figure 48.3 shows a patient with significant adipose tissue on her thighs who underwent bilateral circumferential thigh liposuction concurrent with her abdominoplasty. This deflates the thigh tissues and facilitates a good aesthetic contour during a second-stage vertical thighplasty. In patients with or without a previous abdominoplasty, a Lockwood "Type I"

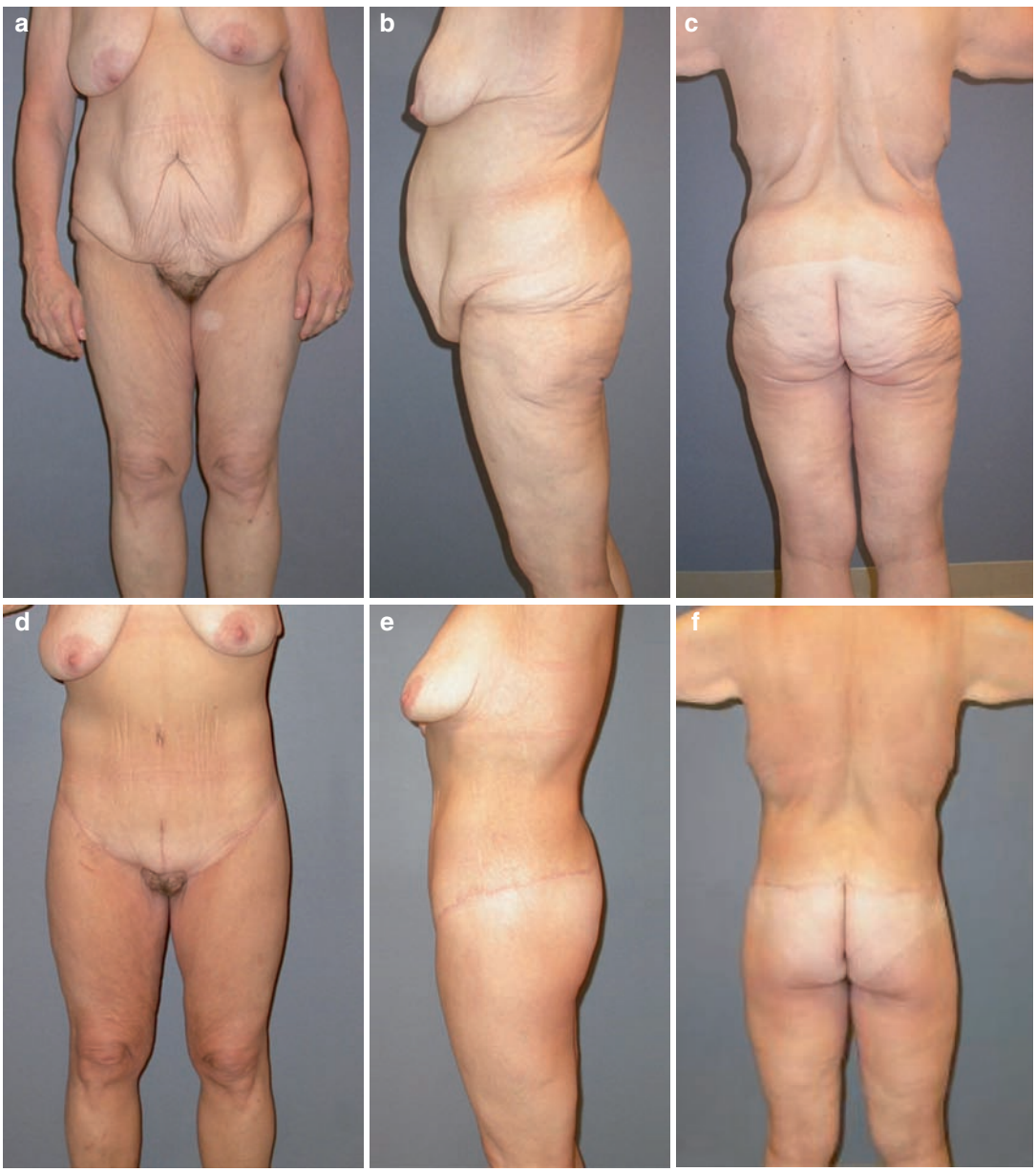

Figure 48.2. Forty-seven-year-old woman following 52-kg weight loss and lower body lift. (**a**, **b**, **c**) Pre- and (**d**, **e**, **f**) postoperative views shown at 1 year.

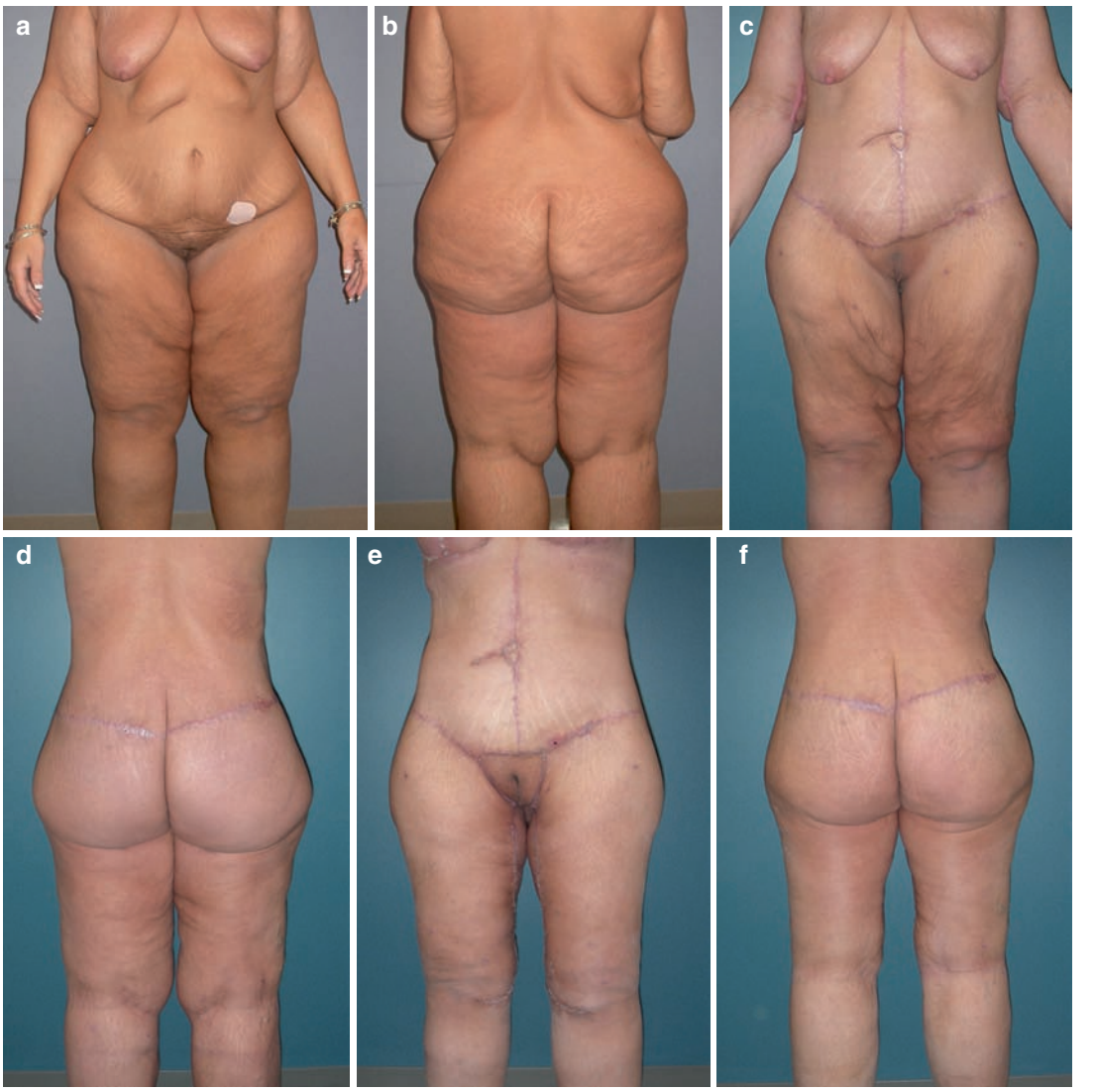

Figure 48.3. Thirty-nine-year-old woman following 45-kg weight loss (**a**, **b**) and a first-stage lower body lift with fleur-de-lis abdominoplasty, monsplasty, and 9 l debulking liposuction of the thighs (**c**, **d**). Six months later, the patient underwent a second-stage vertical thighplasty (**e**, **f**).

LBL can be employed, with the anterior scars veering into the groin crease bilaterally. This will provide a moderate degree of pull on the medial thigh tissues. A key point is to keep the scars very close to the mons region so that they do not descend below the level of the groin crease.

Vertical Thigh Lift

The medial thighs present many technical challenges for the plastic surgeon.[18,19] On a practical level, patients with significant skin laxity on both medial and lateral thighs can rarely be treated satisfactorily with a single procedure. The LBL has minimal impact on the medial thighs.

Similarly, a vertical thighplasty will affect the medial thighs while leaving notable deformities on the lateral thighs unchanged. There are well-selected patients with reasonable tone on the lateral thigh and buttocks who can benefit from an isolated vertical thighplasty. However, most of the MWL patients will require both LBL and vertical thighplasty. It is the authors' preference to stage LBL and vertical thighplasty, with the LBL serving as the initial "cornerstone" procedure. This is beneficial for the following reasons: (1) The LBL, especially when done with the abdominoplasty, will have a greater impact on the patient's body shape and lead to an immediate improvement in body image; (2) The LBL will tend to relax over time, allowing the thigh tissues to rotate inferiorly and medially. Staging the vertical resection compensates for this relaxation and achieves better contour, whereas a revision could be necessary if the procedures are performed concurrently. (3) The patient will have a more arduous recovery if the procedures are performed together. (4) Performing both procedures together can be taxing on the surgeon.

A simple crescenteric, transverse, medial thigh excision is very much underpowered and only improves laxity in the upper third of the medial thigh, not to the knee. Transverse scars are also prone to descent from the vertical vector of gravity. This can be mitigated by keeping the scars close to the mons (actually superior/medial to the groin crease) and anchoring the deep SFS tissue to Colle's fascia.[20] This procedure also tends to result in pleating along the transverse scar that takes time to resolve. This operation does, however, avoid a visible longitudinal scar and is of much less magnitude than the full vertical thighplasty. Additionally, this version of the thighplasty can be safely combined with an LBL. Achieving patient satisfaction with this procedure depends on selecting a patient with primarily proximal medial thigh deformities and properly conveying the limitations of the operation.

The markings and surgical technique are demonstrated in Figures 48.4 and 48.5. The dissection over the femoral triangle is superficial to avoid injury to underlying lymphatic structures. For postoperative care, ace wraps are secured on the legs. The patient is instructed to keep legs elevated in the early postoperative period, and compressive hose are used for 2–4 weeks. Figure 48.6 shows the results of a vertical thighplasty in the absence of an LBL. Complications of leg swelling and temporary lymphedema may be observed in the first 2–6 weeks following surgery. Another variant to this procedure is a short vertical scar thigh lift. There is often a noticeable step-off in contour at the transition between the end of the scar and the lower portion of the thigh. Candidates for this operation tend to have good skin tone in the lower or distal thigh and obvious festoons in the upper thigh that cannot be corrected with a transverse thigh lift.

Brachioplasty

Many variations exist for correction of excess skin of the arms with regard to location of the incision either in the brachial groove or more posteriorly. In addition, the axilla may be managed with the inclusion of Z, W, or L-plasties to mitigate scar contracture.[21–24] Permanent sutures securing the SFS to the axillary fascia help to retain the axillary fold after skin excision.[25] The surgical goals for correction of redundant skin on the arm include proper scar placement, elevation of the axillary fold, avoidance of uneven resection, avoidance of over resection, and correction of laxity on the lateral chest wall.

Liposuction serves as a useful adjunct for thinning the posterior aspect of the arms before brachioplasty; however, resultant swelling may make skin closure more difficult. One should infiltrate with tumescent fluid and move immediately to aspiration to avoid edema. The authors promote a cautious step-by-step excision that allows concomitant closure with resection to avoid the inability to close the arm wound.

The scar from a brachioplasty is very prominent and visible. Moreover, this scar can stay thick and red for many months before maturing and becoming less noticeable. This element of preoperative counseling is vital with brachioplasty candidates. Silicone sheeting or paper tape, along with massage, can help with scar maturation. Although there is controversy over the ideal location of the scar in brachioplasty procedures, the authors prefer the bicipital groove. In this position, the scar is more difficult to see in most situations. A posterior scar may be quite visible to other people.

When marking, the patient should have the arms bent 90° at the elbow and 90° at abduction

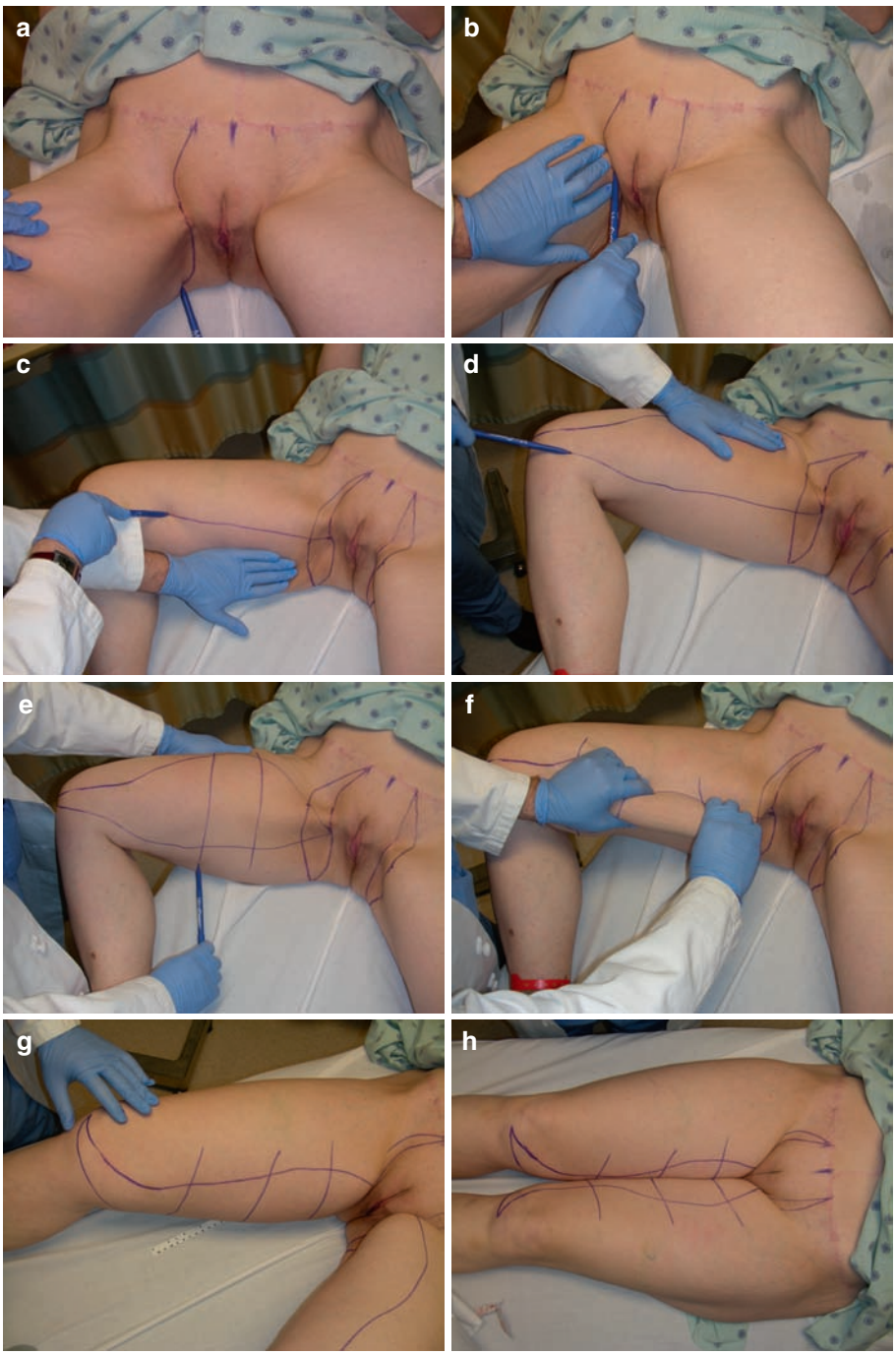

Figure 48.4. Markings for vertical thighplasty. (**a**) Line of incision drawn in groin crease starting 4 cm lateral to midline of mons region; (**b**) pinch test to estimate the vertical component of skin resection; (**c**) anterior line of resection drawn by placing posterior traction with left hand and marking along mid-thigh. This will show estimated scar position. (**d**) Posterior line of resection drawn by applying anterior traction with left hand and marking along mid-thigh. (**e**) Cross-hatching is performed to assist with wound closure. (**f**) A pinch test is performed to verify markings. (**g**) Note that distal pattern of resection crosses knee in mid-axial position and curves under patella. (**h**) Symmetry of markings checked when legs are approximated – patterns should be mirror images if legs are symmetrical to start.

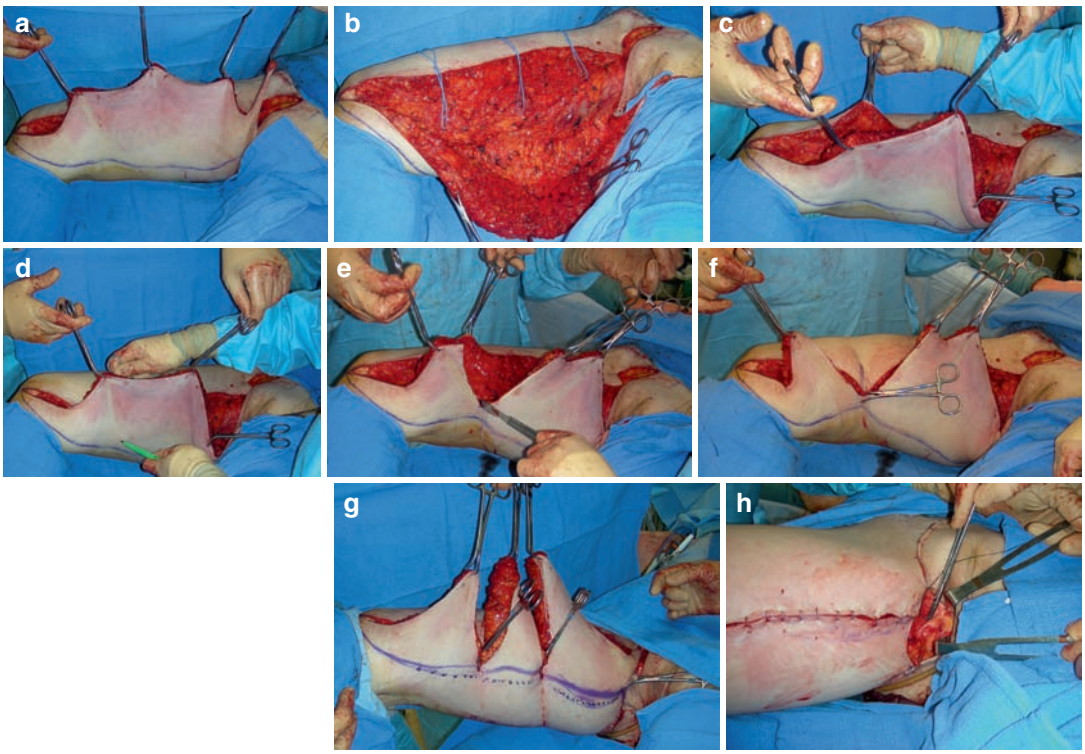

Figure 48.5. Operative technique for vertical thighplasty. (**a**) Anterior line of resection has been incised and the flap undermined to the posterior estimated line of resection. (**b**) Saphenous vein has been identified and preserved (course marked with vessel loops), with the plane of dissection just above the vein. (**c** and **d**) Sharp towel clamps are used to hold the dissected flap, and an everted towel clamp placed at the edge of the anterior incision line is used as a flap marker. (**e** and **f**) The flap is incised at intervals along the thigh and secured with sharp towel clamps. (**g**) After multiple segmental incisions into the flap, the posterior line of resection is remarked between the towel clips and incised. (**h**) 3–5 Braided nylon sutures are used to approximate the SFS of the thigh tissues to Colles' fascia on each thigh.

of the shoulder. The bicipital groove is marked and represents the intended scar placement. Next, a superior line of incision is marked by pulling tension downward on the skin of the arm to estimate skin excursion under stretch. In general, the superior line of incision is 2–3 cm superior to the bicipital groove mark. Under the tension of wound closure, the superior line of incision will be pulled downward to the level of the bicipital groove. The distal extent of the resection is usually at the level of the elbow. By placing the resection along the bicipital groove, any scar will cross the elbow in the midaxillary position relative to the joint, allowing one to extend the incision distally without fear of joint contracture. The proximal extent of the superior incision line is set high into the dome of the axilla. Extending inferiorly and perpendicularly from the dome of the axilla is the extension of the incision onto the chest wall. The length of that incision is determined by the relative skin laxity on the lateral chest wall. Next, a point in the axilla is selected that will reach the dome under tension and will correct the decent of the axillary fold. This mark is usually 4–5 cm from the dome. Next, the estimated inferior line of incision is determined by simple pinch test. This line is only an estimate and will not be incised at the start of the procedure.

The operative technique is shown in Figure 48.7. Securing the SFS at point A prime to a clavipectoral fascia at point A with a permanent 0-braded nylon suture will help keep the tissue

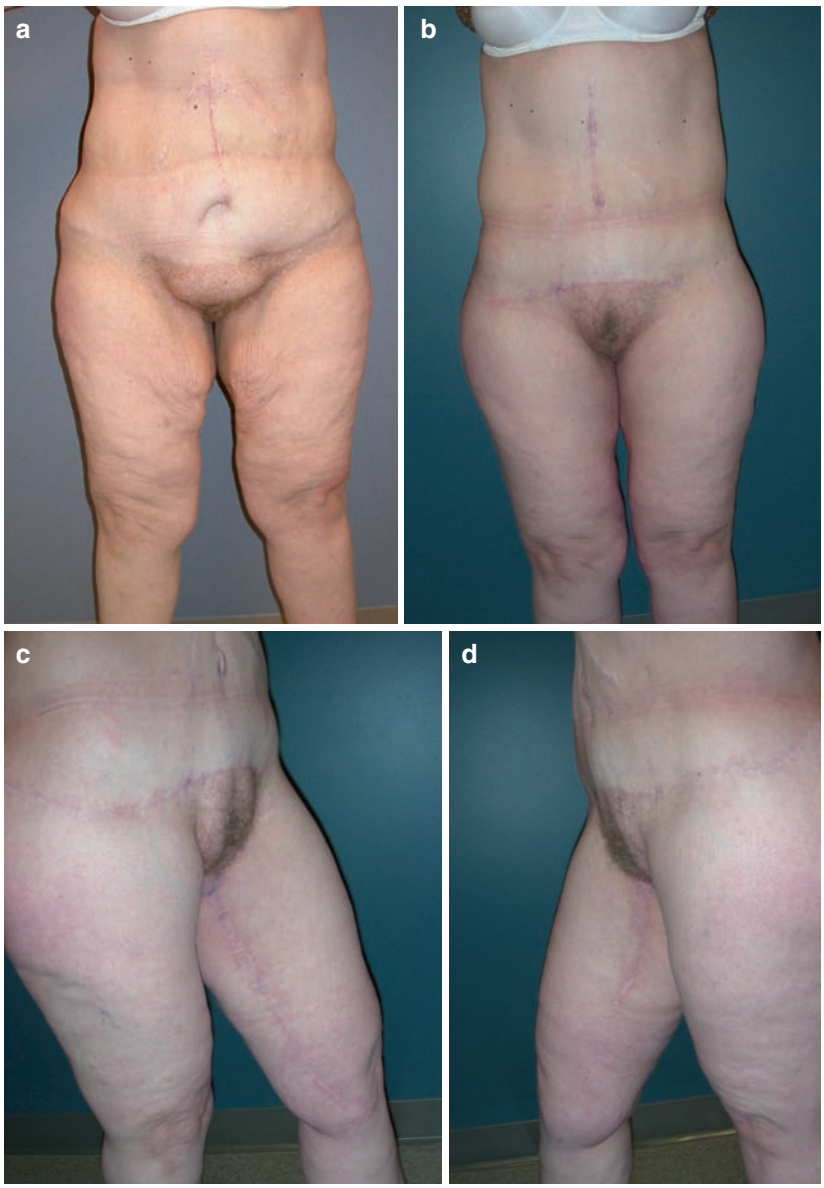

Figure 48.6. (**a**) Fifty-one-year-old woman s/p 59 kg weight loss and previous abdominoplasty. (**b**, **c**, **d**) Postoperative views 1 year after revision abdominoplasty and vertical medial thighplasty without having had lower body lift.

suspended in the axilla. Following wound closure, a sterile compressive wrap is placed on the arm. The patient is advised to avoid heavy lifting for at least 2 weeks. In addition, the patient is advised not to raise the arms and abduct the shoulders above 90° for the first week. After two weeks, the patient is prescribed gentle active range of motion exercises to fully abduct the shoulder. Figure 48.8 shows a representative case with maturation of scar over time.

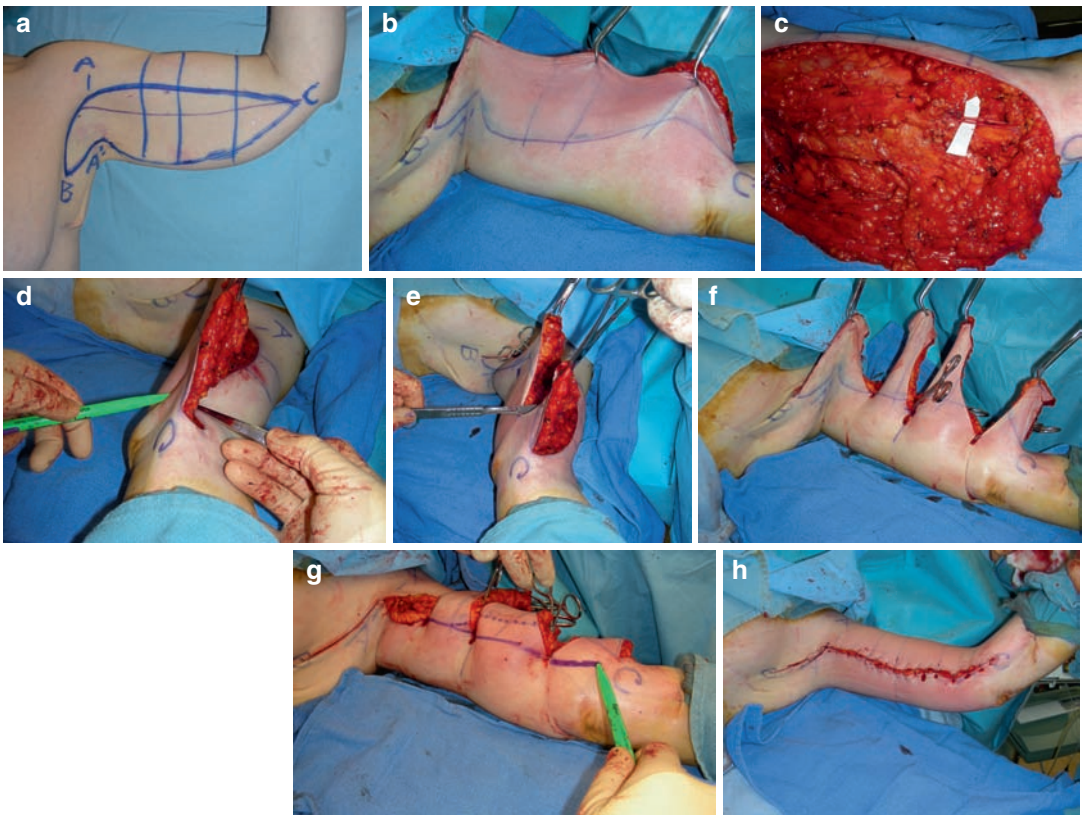

Figure 48.7. Brachioplasty technique. (**a**) Markings; (**b**) elevation of flap without to level of estimated margin of resection; (**c**) medial antebrachial cutaneous nerve (MABC) adjacent to basilic vein at the elbow; (**d**) flap marking technique using heavy forceps; (**e**) incision of flap to point of resection determined by flap marking; (**f**) multiple tissue segments isolated based on flap marking at three intervals along upper arm; (**g**) new, more exact margin of resection drawn in between interval incisions; (**h**) tissue segments removed to complete excision and wound approximated. Note offset angle of scars in axilla, which prevents scar contracture at the joint, similar to a Brunner incision on the hand.

Specialized Mastopexy Technique in the MWL Patient, Including Upper Body Lift

Breasts deformities after MWL are characterized by deflation and distortion. In particular, there is significant volume loss, elasticity loss, asymmetry, medialization of the nipples, and continuation of laxity to the lateral chest wall and to rolls in the back. Traditional techniques of breast reduction and mastopexy, especially with short scars, tend to be insufficient solutions for this deformity. The senior author has developed a technique using principles of dermal suspension and total parenchymal reshaping to fundamentally create an internal brassiere and provide lasting shape to the breast.[26] A modification of the traditional Wise pattern provides control of the skin envelope, and a central dermoglandular pedicle is well vascularized and supports the nipple. Volume is maintained, and autoaugmentation using excess tissue from the side of the chest lateral to the breast extending to the mid-axillary line can increase volume substantially and reliably. Approaches to the breast should take into consideration excess skin of the lateral chest wall

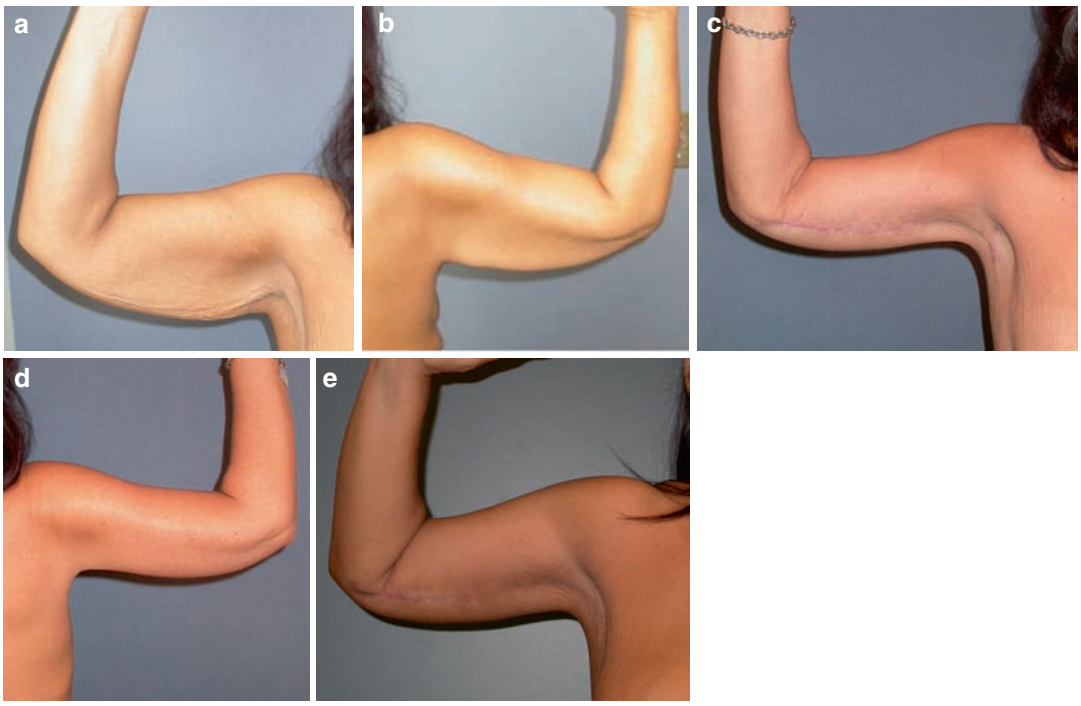

Figure 48.8. Forty-four-year-old woman s/p 83-kg weight loss. Preoperative views (**a**, **b**), and postoperative views shown at 6 months (**c**, **d**) and 2 years to demonstrate scar maturation (**e**).

as well as the back. If rolls are prominent in a horizontal fashion on the back, their excision can be incorporated into the mastopexy or brachioplasty for a complete upper body lift. Rolls that have a more vertical orientation can be excised vertically along the mid-axillary line and may be in continuity with brachioplasty scars. Improving the arms, breasts, and back can help create a more harmonious appearance to the upper body aesthetic unit.

In marking the patient, the nipple position is referenced to the inferior mammary fold and moved to a more lateral position along a symmetrically drawn breast meridian. The lateral portion of the Wise pattern is extended posteriorly to encompass the axillary skin roll and provide additional autologous tissue for breast volume. Markings and surgical technique are demonstrated in Figures 48.9 and 48.10.

Restoration of breast shape and symmetry can be achieved in difficult cases with this technique. Patient satisfaction has been high in all cases. Pre and postoperative results are shown in Figure 48.11. Figure 48.12 demonstrates the markings and technique for an upper body lift.

Specialized Gynecomastia Correction Technique in the MWL Patient

The severe skin deformities of the male chest following MWL are difficult to correct without a major skin excision. Moreover, periareolar techniques do not work well in this population. The authors advocate an elliptical excision of chest skin with preservation of the nipple on a thinned broad-based dermal pedicle. The scar position is in the inframammary fold. Figure 48.13 shows the results of the technique and scar progression.

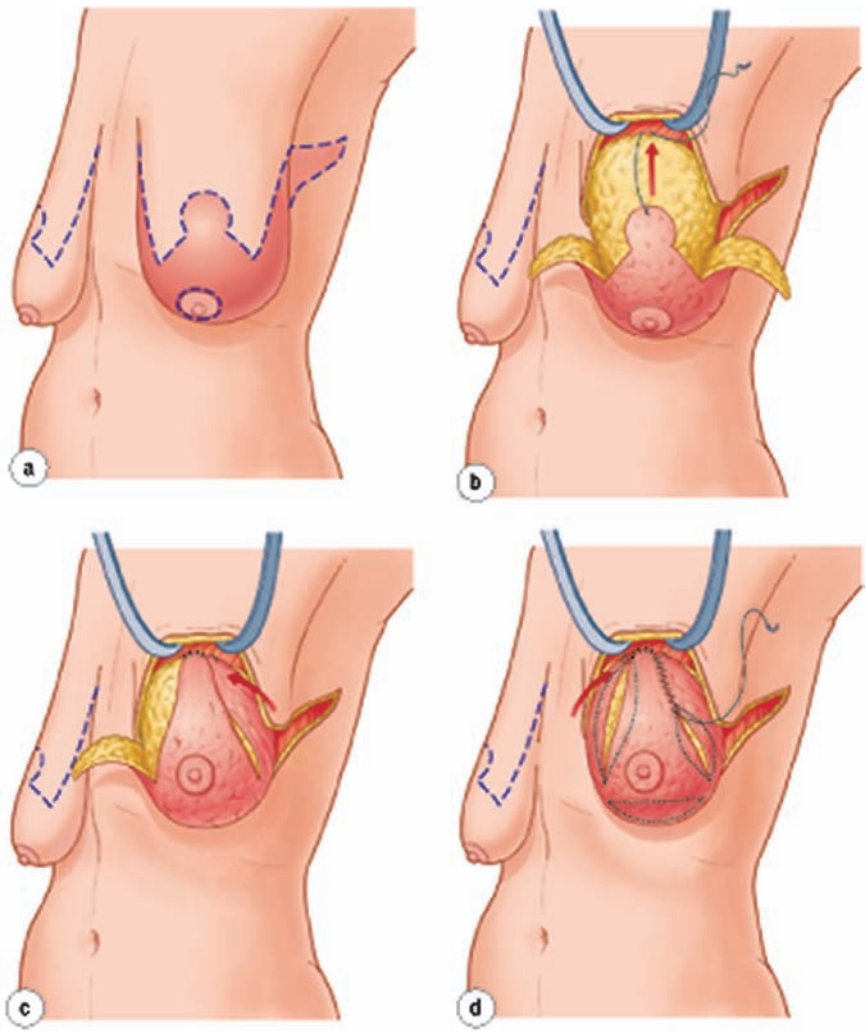

Figure 48.9. (**a**) The patient is marked with an extended Wise pattern that extends laterally to include the lateral chest roll. (**b**) The breast parenchyma is degloved by raising a 1 cm thick flap that continues superiorly along the pectoralis fascia to the level of the clavicle. Medial and lateral dermoglandular flaps are raised. The central dermal extension is secured to the chest wall (usually the second rib periosteum) using braided nylon suture. (**c**) The lateral breast flap is elevated and secured to the chest wall near the central dermal extension (usually the third rib periosteum). The lateral flap can be extended posteriorly on the chest wall to provide extra tissue for selective autoaugmentation. (**d**) The medial dermal flap is elevated and secured to the chest wall. Interrupted and running absorbable braided suture is used to plicate the parenchyma medially, laterally, and inferiorly, forcing projection centrally. (Reprinted from Rubin JP, O'Toole J, Agha-Mohammadi S. Approach to the breast after weight loss. In: Rubin JP, Matarasso A, eds. *Aesthetic Surgery After Massive Weight Loss*. New York: Elsevier; 2007:37–48.)

Multiple Procedures and Staging

Multiple procedures may be combined, but providing safety along with appropriate operative setting (e.g. inpatient facility) is the overriding principle. When combining procedures, the authors avoid combinations that will result in opposing vectors of pull. An example would be combining LBL and upper body lift. Additionally, the magnitude of recovery, given the patient's age and medical condition, should be considered. The experience of the operative team,

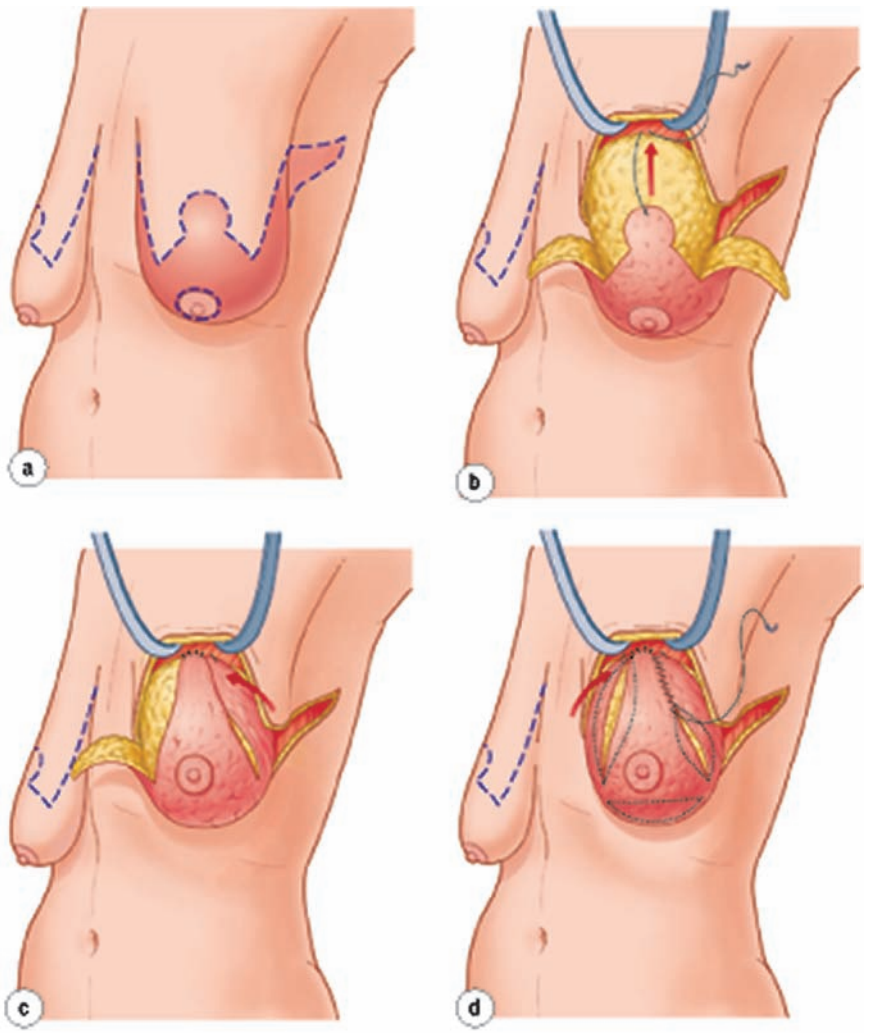

Figure 48.10. (**a**) The inferior placation shortens the lower pole of the breast so that the distance from the areola to the inframammary fold is approximately 5 cm. (**b**) Permanent sutures are placed along the lateral border of the lateral chest flap to the chest wall fascia (not rib periosteum) to enhance the lateral curvature of the breast. (**c** and **d**) The skin flaps are redraped to check the aesthetic contour of the breast. Once parenchymal reshaping is completed, the incisions are closed over drains in two layers with absorbable suture. If the nipple is tethered and pointing in an inappropriate direction, the dermis adjacent to the nipple can be scored to release tension. (Reprinted from Rubin JP, O'Toole J, Agha-Mohammadi S. Approach to the breast after weight loss. In: Rubin JP, Matarasso A, eds. *Aesthetic Surgery After Massive Weight Loss*. New York: Elsevier; 2007:37–48.)

number of operating surgeons, and potential for surgeon fatigue should also be taken into account. In general, the authors will do as much as a circumferential LBL and one upper body procedure in a single operative setting for well-selected patients. When planning staged procedures, the authors allow at least 3 months between stages. Well-planned stages can result in a safe total postbariatric reconstruction (Figure 48.11).

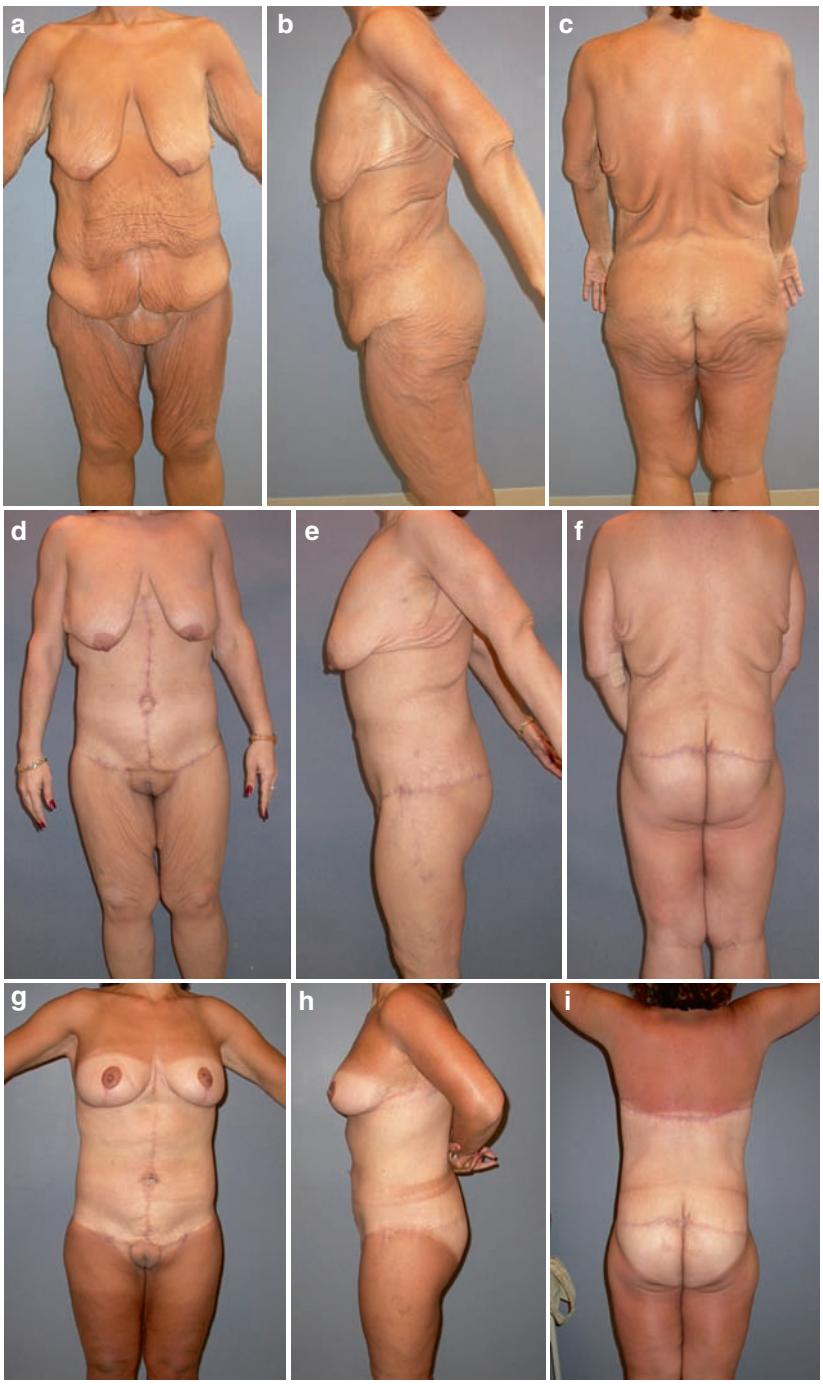

Figure 48.11. Forty-six-year-old woman s/p 95-kg weight loss, planned for two-stage total body reconstruction. (**a**, **b**, **c**) Initial preoperative photographs; (**d**, **e**, **f**) s/p first stage circumferential lower body lift, vertical abdominal resection, and brachioplasty; (**g**–**i**) 6 months s/p second-stage upper body lift, mastopexy, and vertical thigh lift.

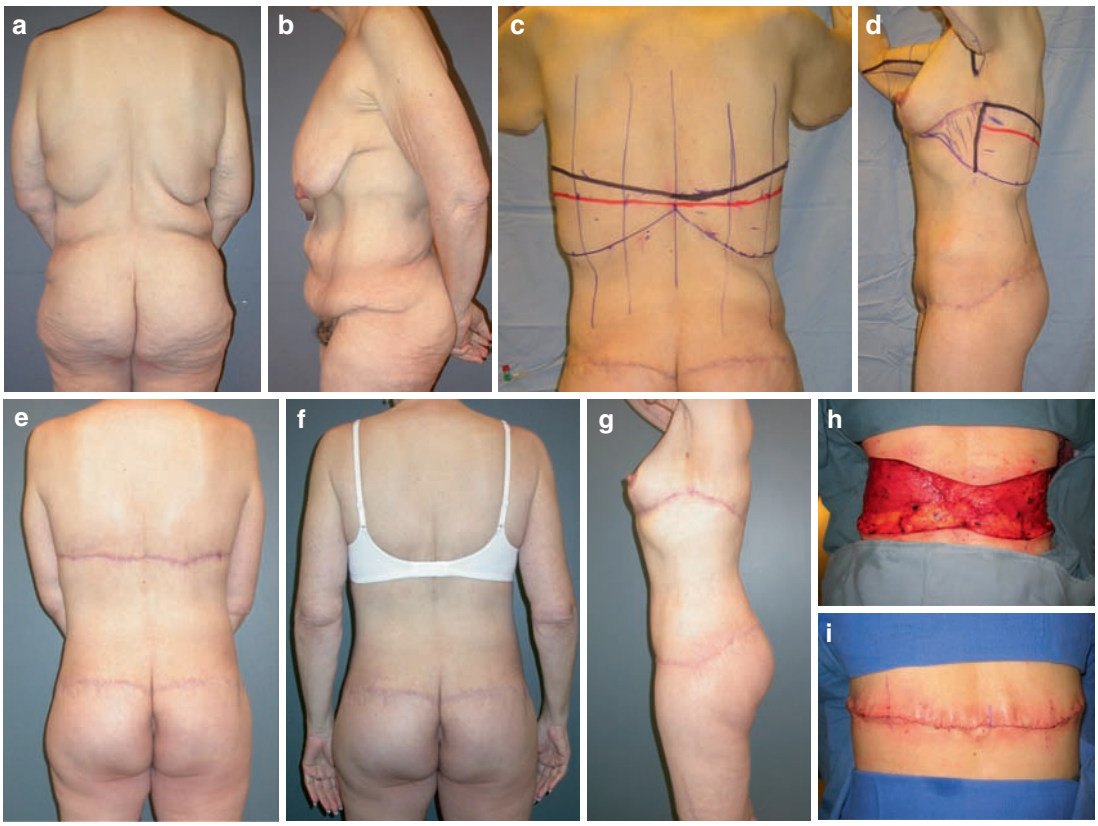

Figure 48.12. Fifty-six-year-old woman s/p 80-kg weight loss. (**a**, **b**) Initial preoperative views; (**c**, **d**) patient shown 3 months following lower body lift, mastopexy, and preliminary debulking liposuction of back rolls. She is marked for transverse upper body lift, with red line indicating intended scar position and pattern of resection designed so that scars will merge with the mastopexy scars. (**e**, **f**, **g**) Patient shown 7 months after upper body lift, with correction of back rolls and scar hidden by bra. Additionally, the lower body lift scars can be seen in relation to the upper body lift scars. (**h** and **i**) Intraoperative photographs showing defect on back after resection and closed wound.

Complications Inherent to Postbariatric Reconstruction

An overwhelming majority of complications with body contouring procedures consist of wound healing complications (mainly dehiscence) and seroma. One notable phenomenon in this population is recurrent skin laxity weeks to months following surgery. This can be severe enough to require revision. A host of less common complications are seen in this population, including infection, bleeding, skin necrosis, and thrombotic complications. Attention to venous thromboembolism prophylaxis is an important safety measure.

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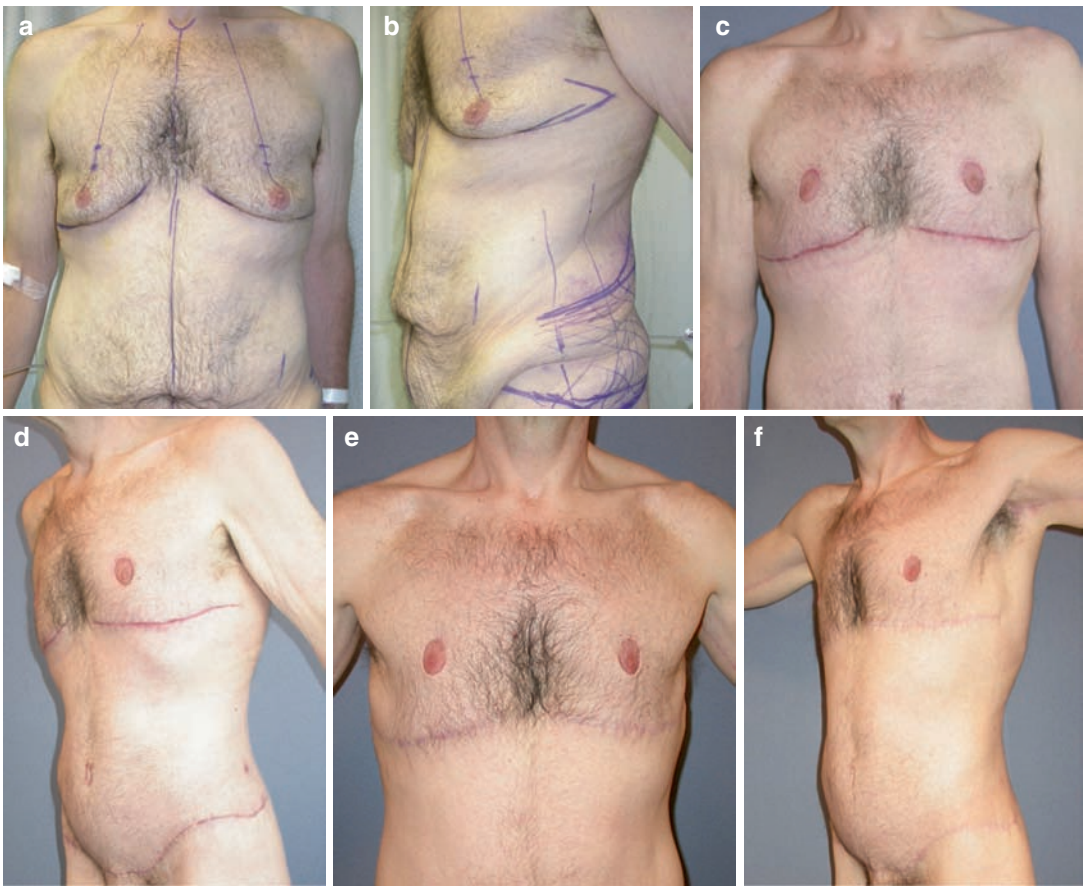

Figure 48.13. Forty-five-year-old male patient s/p 89-kg weight loss. (**a**, **b**) Inframammary fold marked as well as anticipated nipple position and superior extent of elliptical excision; (**c**, **d**) results at 6 months, with scars still red and thick; (**e**, **f**) results at 1 year, with scars matured.

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Reconstructive and Aesthetic Surgery of the Genitalia

Daniel A. Medalie

Summary

Plastic surgery of male and female genitalia is complex and frequently confusing for the novice practitioner. The genitalia act as urinary conduits, are vital for normal sexual function and have obvious aesthetic value. Abnormalities of the genitalia are both functionally and psychologically debilitating for the patient. Congenital anomalies, infection, trauma and cancer are all reasons for plastic surgery in the pelvic region. More recently, transgender surgery and aesthetic labial and vaginal surgery have become common place. It is incumbent upon the plastic surgery practitioner to understand the variety of problems that might arise in operating upon male and female genitalia, and to have the skills sufficient to handle some of the more basic procedures. Since these topics easily encompass an entire book, this chapter will focus mostly on adult reconstructive and aesthetic surgery.

Congenital Anomalies

Syndromes

A variety of syndromes can result in congenital anomalies of the male and female genitalia. Some of these include gonadal dysgenesis, Turner's syndrome, Klinefelter's syndrome, testicular feminization, female pseudo-hermaphroditism (ambiguous genitalia), and male pseudo-hermaphroditism. In the male, problems can result in testicular atrophy and failure of descent, complete failure of formation of the external genitalia, hypospadias, and epispadius. One of the most devastating syndromes is testicular feminization. The patients are genetically male but lack responsiveness to normal levels of fetal androgens due to receptor dysfunction. Frequently, the syndrome is not noticed until the patient fails to menstruate. Early attempts at forcing these patients to continue in the role of females after it has been determined that they are genetically male have resulted in severe psychological problems for the patients. It is currently recommended to attempt phallic reconstruction and have the patients make a transition to a male role. In the female, ovarian, uterine, and vaginal agenesis can occur. Conversely, adrenogenital syndrome (congenital adrenal hyperplasia) resulting in an excess production of adrenal androgens can result in masculinization of the female genitalia.[10]

Hypospadias

Hypospadias encompasses a variety of disorders of the external urinary meatus. The meatus is located on the ventral surface of the penis, between the perineum and the ventral aspect of the glans. The location can be glandular, distal

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penile, penile, at the peno-scrotal junction, scrotal, or perineal. Chordee, downward bending of the penis, can occur with or without hypospadias. Preoperative evaluation is crucial, and circumcision should never be performed before the hypospadias reconstruction. This is because the prepuce may be needed for the reconstruction. For distal defects, meatal advancement glansplasty incision (MAGPI), urethral advancement, or flip-flap techniques are useful. For more proximal or mid-shaft defects, skin grafts or vascularized prepuce flaps are used.[10]

Adult Genital Reconstruction

Phallus Reconstruction

The goal of reconstructive phallic surgery is the same regardless of the surgical indication. Whether the lack of a penis is congenital or acquired, the end result should be a phallus that allows standing urination, sexual intercourse, erogenous sensation, and acceptable appearance. It is very difficult to perform a one-stage procedure that achieves all of these goals. Frequently, multistage operations are performed. The introduction of microsurgery to the plastic surgeons' armamentarium has radically transformed methods of penile construction and reconstruction. The most common free tissue transfer procedures performed for phallic reconstruction include the radial forearm free flap and the free osteocutaneous fibula flap.

The advantage of the radial forearm free flap is the extreme pliability of the skin and the ability to form a tube within a tube, facilitating urethral reconstruction. The medial and/or lateral antebrachial cutaneous nerves are used to provide cutaneous and erogenous sensibility to the flap. The disadvantage of the flap is its postoperative flaccidity, requiring a separate prosthesis to achieve rigidity. A portion of the radius can be included with the flap, but, by necessity, it is thin, unicortical, and subject to fracture. Long-term maintenance of bony integrity has not been thoroughly studied. Another disadvantage is the large forearm donor site, which is difficult to disguise. Prefabrication of the flap by tissue expanding the forearm before the definitive reconstruction can help to minimize the donor defect. Alternatively, tubed groin flaps with a sensitized radial forearm flap to serve as the urethra and glans can minimize the donor site while maximizing flap size.

The free osteocutaneous fibula flap has the advantage of intrinsic rigidity and size. The superficial peroneal nerve can be used to neurotize the flap. The donor site is below the knee and can be easily disguised by wearing high socks. Disadvantages include the requirement for an internal skin graft urethral reconstruction, which is not as well vascularized as the radial forearm "tube within a tube." The skin paddle has more hair, is thicker, and less amenable to being wrapped. It is unclear whether the sensitivity of the fibula flap is equal to that of the radial forearm. Both the fibula and forearm flaps should undergo hair removal before transfer.

It is the author's preference to use the free fibular flap for phallus reconstruction. The reconstruction is typically multistage. The first stage is a delay procedure that involves harvesting a full-thickness skin graft from the groin and burying it underneath the planned skin paddle of the fibula flap (Figure 49.1a). The skin paddle (15 × 13 cm) is completely elevated to the septum, and the graft is applied to the underside of the flap. A sheet of Gore-Tex is then interposed between the muscle and graft, to prevent adherence of the flap and graft to the underlying muscular bed. About 7–10 days later, the free tissue transfer is performed. The fibula is harvested with the peroneal vessels (Figure 49.1c). Prior to transection of the vessels, the bone is cut to length, and the flap is tubed upon itself. The full-thickness graft is wrapped around a large-diameter silicone Foley catheter (Figure 49.1d, e). The recipient urethra is prepared with parachute sutures, and the superficial femoral artery and vein are prepared for end-to-side anastomosis. Because the flap can be centered distally over the fibula, the peroneal vessels can be dissected away from the proximal fibula, allowing for a long vascular pedicle (Figure 49.1b–f). This is another advantage of the fibula flap over the radial forearm flap. Depending on the type of surgery performed, the nerve to the penis or clitoris will have to be dissected. No nerve branch is divided. Instead, an epineural window is created, and the nerve to the fibula skin paddle is sewn end to side to the window. Drill holes are used in the fibula to pass permanent sutures anchoring the bone to the pubic periosteum. This allows the fibula flap to hinge on the pubis. The tubed, full-thickness skin graft is then sewn to the native

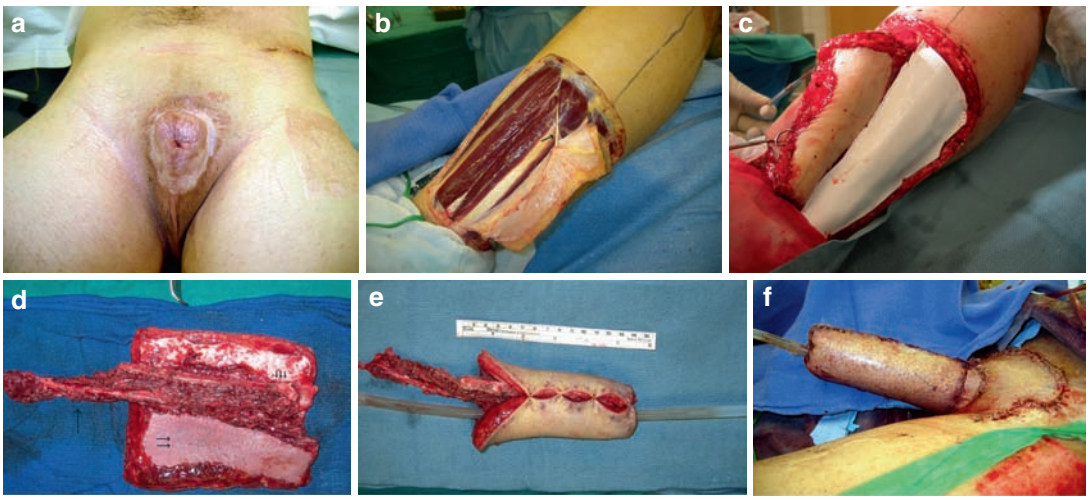

Figure 49.1. (**a**) The patient sustained a traumatic amputation of the penis and testicles. (**b**) The first stage of the osteocutaneous fibula flap reconstruction is the elevation of the skin paddle with identification of the superficial peroneal nerve (*arrow*). (**c**) A full-thickness graft is sewn to the underside of the flap, and PTFE is interposed between the flap and underlying muscle. (**d**) One week later, the flap is harvested with the fibula (*triple arrows*), peroneal vessels (*single arrow*), and a well-vascularized graft (double arrow). (**e**) The flap is tubed, and the graft is also tubed around a Foley catheter. (**f**) The flap is inset, the fibula is attached to the pubic periosteum, the nerve is attached end-to-side to the pudendal nerve, and the pedicle is anastomosed to the femoral vessels.

urethra with the previously placed sutures. The biggest concerns are fistula formation and urethral stricture. By making sure that there is bulky, healthy fibular skin paddle over the urethral anastomosis, fistula formation can be controlled. At a later date, minor revision procedures such as glansplasty can be performed. Sadove et al. (1999) report good long-term maintenance of bone stock in the fibular flaps and feel that the fibula is at least as suitable a flap for penile reconstruction as the radial forearm flap.[3,9,14,15,23,24]

Perineal and Vaginal Reconstruction

Vaginal reconstruction is required for patients with congenital partial or complete agenesis of the vagina, loss of the vagina due to infection, trauma, or cancer, and male to female transgender patients. The goal is to create an epithelial-lined tube that has sufficient width and depth to allow for sexual intercourse. Many of the techniques described for vaginal reconstruction can also be used for local perineal and pelvic reconstruction.

Options for reconstruction include split- or full-thickness skin grafts, local or regional fasciocutaneous or musculocutaneous flaps, free tissue transfer, and intestinal recto-sigmoid flaps. Due to severe problems with stricture, it is recommended that split-thickness skin grafts be used only for perineal reconstruction and not for vaginal reconstruction (Figure 49.2a–c). Even with full-thickness grafts, stenting and dilation are frequently required. Regional flaps are thus the recommended modality for vaginal reconstruction. The gracilis myocutaneous flap and rectus abdominis myocutaneous flap are the two most common muscle-based flaps used for partial or complete vaginal reconstruction. Bilateral pudendal thigh flaps are the most common fasciocutaneous flaps used in reconstruction. Posterior thigh flaps can be used to cover very large defects of the pelvis and perineum.[5,8,12,13,18,21,25–28]

The gracilis flap was the first muscle flap described for vaginal reconstruction. The muscle serves as one of the adductors of the thigh and is dispensable. Its dominant blood supply derives from branches off of the medial femoral circumflex artery approximately 8–10 cm from the pubic tubercle. The proximal two thirds of the muscle can support a fairly large skin paddle centered directly over the muscle. The distal

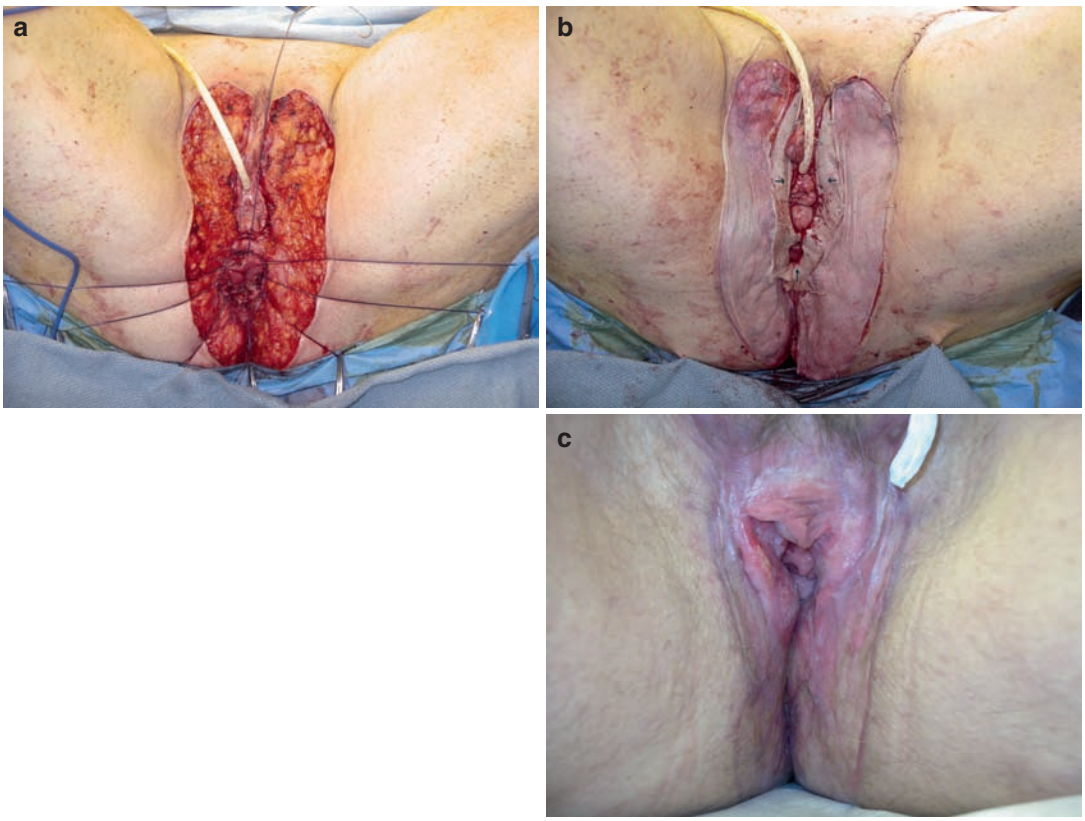

Figure 49.2. (**a**) Resection of Paget's disease of the perineum and vulva results in a large external defect. (**b**) A full-thickness graft is applied around the anus and vagina (*single arrow*), and a split-thickness graft is applied to the surrounding perineum. (**c**) The grafts demonstrate good take.

third is unreliable. Perforators from the vessel have been shown to travel both through the muscle and around it; thus the practitioner must be careful to incorporate some of the muscle fascia to the sides of the gracilis in order to capture the perforators traveling around the muscle (Figure 49.3a–d). Tacking the skin paddle to the muscle is advocated to prevent shearing of the skin during the passage of the flap to the vaginal defect (Figure 49.3b). A tunnel is created between the proximal incision used to harvest the flap and the vaginal defect. The flap should be completely islandized, and if extra pedicle length is desired, the short branches of the vessel to the adductor longus can be divided. After the bilateral muscles are transposed through the tunnels, they are sutured to each other and then inset into the vagina (Figure 49.3c, d). The donor sites can be primarily closed for skin paddles up to approximately 10 cm wide, depending on the laxity of the medial thigh skin. The length of the skin paddles should be at least 12–15 cm, and the widths of the paddles combined should be at least 15 cm to create an inner diameter of at least 5–6 cm. Problems with the flap include unreliability of the distal portion of the skin paddles and tethering of the pedicle.[5,18,21,27]

The rectus abdominus myocutaneous flap pedicled on the deep inferior epigastric artery is the most versatile flap used for vaginal reconstruction. The skin paddle can be based anywhere proximally or distally over the muscle and has an excellent blood supply. The flap can be tunneled over the pubic bone to the vagina but is better used intra-abdominally during pelvic exenteration and vaginectomy. Both partial and complete vaginal defects are amenable to reconstruction (Figure 49.4a–c). Advantages of the flap include its flexibility and nearly unlimited quantity of skin. The skin paddle can be oriented in any direction and tubed without difficulty. The bulk of the flap also fills the pelvic defect during an

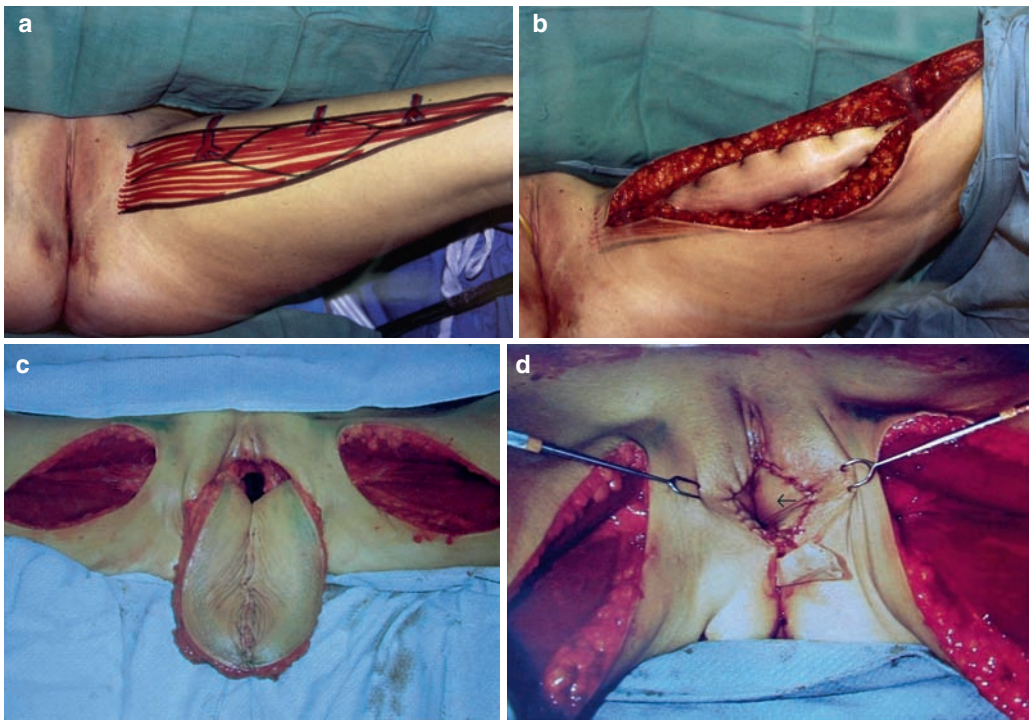

Figure 49.3. A total vaginectomy for cancer will be reconstructed with bilateral gracilis myocutaneous flaps. (**a**) The left flap is outlined on the leg. (**b**) The flap skin paddle has been harvested and tacked to the muscle in preparation for transfer. (**c**) Both flaps have been tunneled to the midline defect and sewn to each other. (**d**) The vagina has been reconstructed.

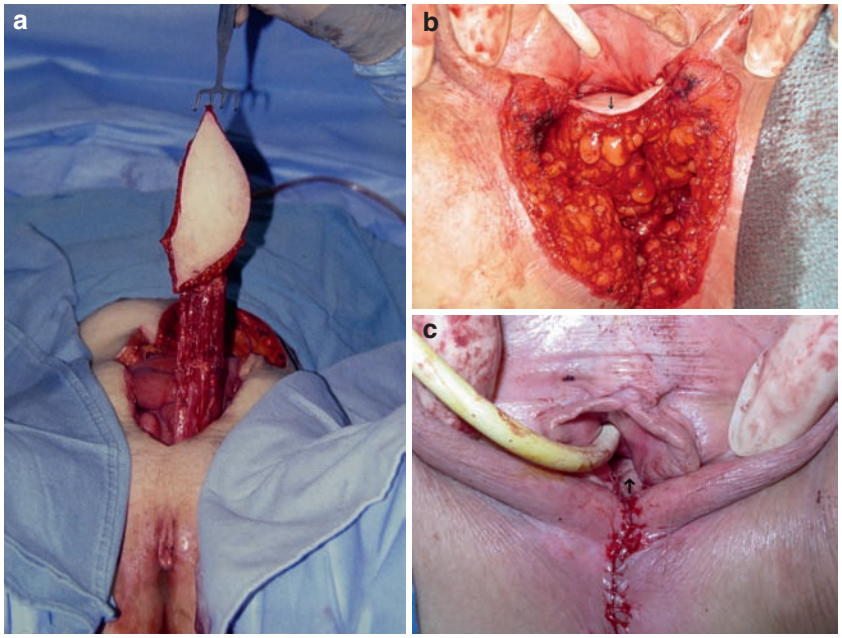

Figure 49.4. (**a**) A pelvic exenteration and posterior vaginectomy for cancer has been performed, and a left vertical rectus myocutaneous flap has been elevated to reconstruct the floor and apex of the vagina. (**b**) The flap has been transposed to the vagina (*arrow*). (**c**) The defect has been closed and the flap inset (*arrow*).

exenteration and reconstitutes to some extent the pelvic floor, preventing descent of the bowel into the pelvis postoperatively. Disadvantages of the flap include bulkiness if the patient is obese and abdominal bulging from the complete loss of one of the paired rectus muscles. If performed in conjunction with a pelvic exenteration, the reconstructive surgeon must carefully coordinate with the extirpative surgeon. Frequently, one or more stoma through the rectus muscles is required, and preplanning must be performed to allow 1 healthy rectus to remain free for the vaginal reconstruction. If a midline incision is not part of the resection procedure, then the use of the rectus creates an unattractive midline scar.[5,25,27]

The pudendal thigh flap is a fasciocutaneous flap based on perforators from the posterior labial branch of the perineal artery, which is a continuation of the internal pudendal artery. The flap is designed with its base adjacent to the posterior extent of the vaginal introitus. The medial edge parallels the outer border of the labia majora, and the apex of the flap points toward the femoral triangle. Frequently the donor site can be closed primarily by mobilizing the medial thigh tissue. Paired flaps are passed underneath the labia to meet in the midline much like gracilis flaps. They are then sutured to each other to create an epithelial-lined tube that can be inset into the vagina. The main disadvantage of this flap is that the medial border may be infected with a disease and be removed. Radiation before the surgery can negatively impact the vascularity of the flap, since it is more local than the gracilis muscle or rectus muscle. It is there-

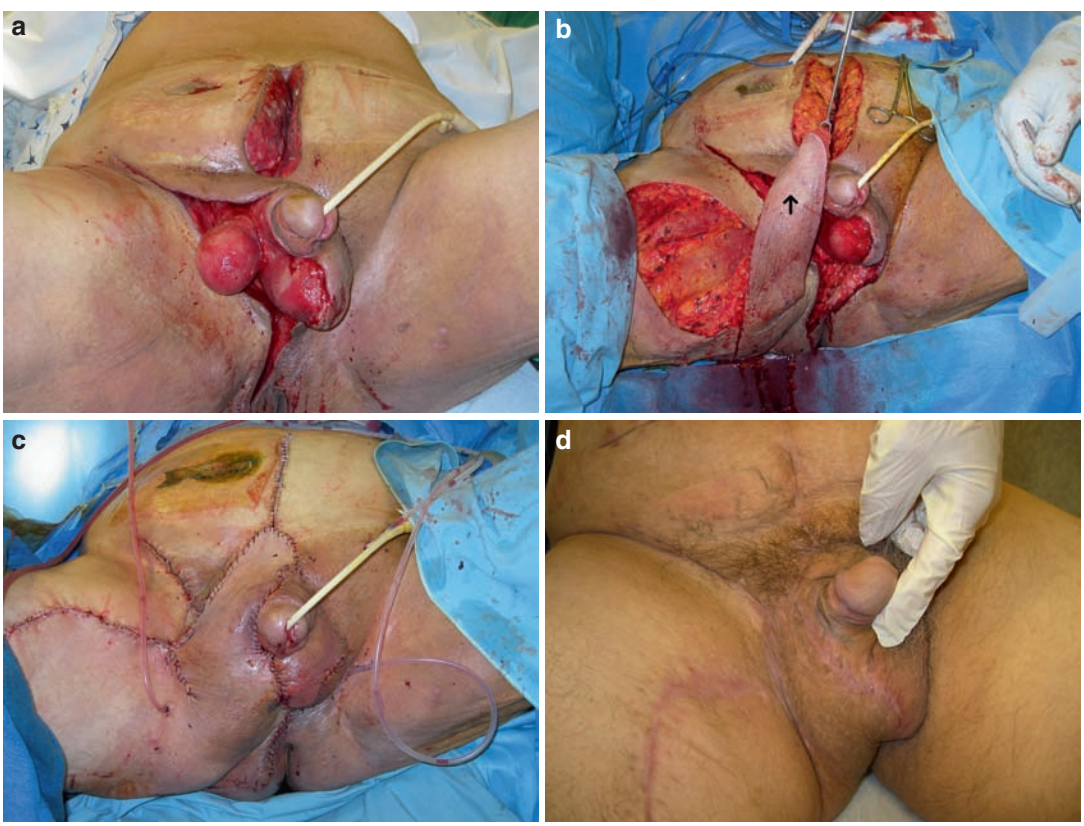

Figure 49.5. (**a**) A partial defect of the penis and scrotum after necrotizing fasciitis. (**b**) A right pudendal thigh flap has been elevated and transposed to the defect (*arrow*). (**c**) The flap has been inset with an opposite side scrotal flap and primary closure of the donor site. (**d**) The wound has healed well.

fore recommended to use this flap primarily for reconstruction of vaginal agenesis, smaller circumferential defects, or unilateral defects. In the context of pelvic exenteration, the paired flaps may not have sufficient bulk. Their advantage is their excellent pliability and aesthetic donor-site scar, which is hidden in the inguinal crease.[8,13,27,28]

Rectosigmoid transplantation for vaginal creation is a relatively new technique. A 15-cm loop of upper rectum and lower sigmoid colon is transposed into the vaginal cavity to provide a mucosal-lined tube that mimics normal vaginal mucosa. The advantages of the flap are its natural lubrication, healthy blood supply, and anatomic similarity to the vagina. Disadvantages include the need for a laparotomy if not already present and the requirement for a bowel anastomosis. Stricture can also form at the mucosal/vaginal junction. There are some reports that mucous production can be excessive, though skin-lined reconstructions may have similar problems with excessive sebum production.[27]

The pudendal thigh flap as well as the previously described muscle flaps can be used equally in men for reconstruction of oncologic or traumatic defects. Since coverage of a defect is the primary concern, these flaps can be used in many combinations to achieve the goals of stable reconstruction.[19,27] Because of the many well-described local and regional methods for reconstructing this region, free tissue transfer is almost never needed (Figure 49.5a–d).

Aesthetic Female Genital Surgery

Aesthetic surgery of the female genitalia is an increasingly common subset of surgeries performed by plastic and reconstructive surgeons. The procedures have arisen from modified techniques developed to reconstruct vulvar and vaginal defects created by disease and trauma. Some physicians and patient interest groups maintain that the surgeries are controversial. However, if done for appropriate reasons and with technical skill, these operations have a high success rate and are perfectly appropriate. The most common procedure performed is labia minora modification and reduction (labiaplasty). Vaginal tightening (perineorraphy) with restructuring of the vaginal introitus is a more involved procedure and is subsequently less common. It is nevertheless rising in popularity, perhaps as a result of the frequency of routine episiotomies performed by obstetricians during normal vaginal delivery. Episiotomies hit their peak in the 1980s and 1990s and are now no longer recommended as a routine procedure. Attention brought to aesthetic vaginal surgery by the Internet and lay press, particularly women's magazines, has also driven demand. Finally, the advent of multiple plastic surgery "reality" shows has increased the number of real patients asking for and receiving these types of operations. Clitoral unhooding and hymen reconstruction are much less common procedures and correspondingly more controversial. There are valid aesthetic and functional reasons for performing labia minora reduction and vaginal introitus tightening. Clitoral unhooding is occasionally warranted but should not be performed routinely. It is this author's opinion that hymen reconstruction does not have legitimate aesthetic or functional value and is requested more frequently by women who have confused beliefs about their own sexuality or are trying to conceal their sexual history. This falls under the category then of body dysmorphic disorder and should not be treated surgically.[1,2,4,6,7,11,16,20,22]

Labiaplasty

Enlargement of the labia minora is usually congenital in nature but can also result from childbirth, advancing age, and weight gain with subsequent weight loss. There is no specific rule as to what constitutes an aesthetic labia minora. Most patients, when queried, feel that the shape of the labia is less important than the overall size. They state a desire to have the labia recessed below the level of the labia majora. A patient typically does not notice small differences in appearance that may be noticed by a surgeon. This author has performed a retrospective analysis of elective labia minora reduction with regard to operative technique, outcomes, and patient demographics. The study was designed to evaluate the author's experience performing labia minora reduction over the last 5 years.

The operations were evaluated by chart review, and the results were evaluated by before and after photograph review paired with patient response to a questionnaire. Factors assessed included patients' reasons for the surgery, patient satisfaction with the procedure, operation duration

and location, operative technique, and outcomes. The study was also designed to evaluate typical patterns of labial hypertrophy and outcome based on starting classification. Because the review is retrospective, its conclusions cannot be taken as definitive. Nevertheless, several useful classifications and trends have emerged from the data.

Over the last 5 years, the author has performed 118 labia minora reductions. Most of these operations were performed in the last 3 years, as the procedure has grown in popularity. The author currently performs 4–6 procedures per month. Thirteen percent were performed in combination with vaginoplasty to tighten the vaginal introitus. Twenty-four percent were performed in combination with other surgeries such as bladder suspension (dual surgery with a urogynecologist), abdominoplasty, liposuction, and breast augmentation. Patient ages ranged from 16 to 57 years. Reasons given for undergoing the surgery included irritation or discomfort with physical activity (aerobics, bike riding etc.) or intercourse and embarrassment at the appearance of the labia. Many patients noted that they were self-conscious wearing form fitting clothes or swimsuits. Although some patients mentioned that their sexual partners had commented on the appearance of their labia, most maintained that they were doing the operation for themselves and their own personal comfort and satisfaction. One hundred percent of patients who responded to the questionnaire stated that they were happy that they had undergone surgery. Since many patients did not return their questionnaire or could not be found, it is certainly possible that this satisfaction rate is artificially elevated. A prospective study would be ideal.

Historically labiaplasty consisted of straight transection of the redundant tissue with oversewing of the raw edge. Although technically simple, the surgery is not aesthetically optimal. The labia, which should form a gentle arc from anterior to posterior, instead take on the appearance of a plateau with retained redundancy of tissue except in the region of transection. The raw edge is also along the length of the labia, which can be very irritating. This author uses a "v" wedge resection of the central portion of the labia as previously described[1,2] (Figure 49.6a–d). If performed correctly, this procedure addresses the majority of the redundant tissue (since most labia hypertrophy occurs centrally) and then bowstrings the remaining tissue to minimize the appearance of redundancy in the remaining anterior and posterior segments of the labia. The whole labia are thus recessed and tightened, and most importantly, the tissue nestles within the folds of the labia majora as opposed to projecting above and over the outer labia. At the time of the main procedure, smaller modifications to complex folds in the tissue can also be performed without changing the overall end result. Because the labia are recessed after the procedure, they are less visible, and small variations in size or appearance from one side to the other are usually not of concern to the patient. The visible portion of the scar is only several millimeters in length and only faintly perceptible. More complicated variations of this surgery have been described, but they are not recommended by this author for the casual practitioner. Simple wedge resection will produce good results with minimal morbidity. The use of small local flaps and Z-plasty in the labia can result in occasional tissue loss without a demonstrable gain in aesthetic result.[7, 17, 20]

Initially the surgeries were uniformly performed in surgery centers with either general anesthesia or deep sedation. In the last 2 years, most of the isolated labiaplasties have been performed in a well-equipped clinic procedure room with local anesthetic (9 cc 1% lidocaine and epinephrine mixed 50:50 with ½% marcaine and 1 cc Bicarb) and mild or no oral sedation (Ativan 2 mg by mouth 1 h before surgery). Operative time is routinely less than 1 h. All wounds are closed with 5-vicryl or monocryl for deep tissues and 5-0 chromic for the outer skin and mucosal layer. Postoperative care includes 5 days of antibiotics (Bactrim) and 2 weeks of topical Bacitracin ointment as well as frequent washes. No bowel prep, vaginal packs, or urethral catheterization are performed. The patients are told to refrain from intercourse for 1 month postoperatively.

Labia minora hypertrophy can be classified into three main subtypes: anterior, central, and diffuse (Figure 49.7a–f). Most of the observed hypertrophy follows a central pattern, with the excess tissue located roughly equidistant from the anterior and posterior extent of the labia.

All subtypes were treated with a similar "V" wedge central excision with occasional anterior perpendicular extensions to eliminate multiple folds in the tissue. Only four simultaneous clitoral unhooding procedures were performed. Results were most favorable with the central hypertrophy

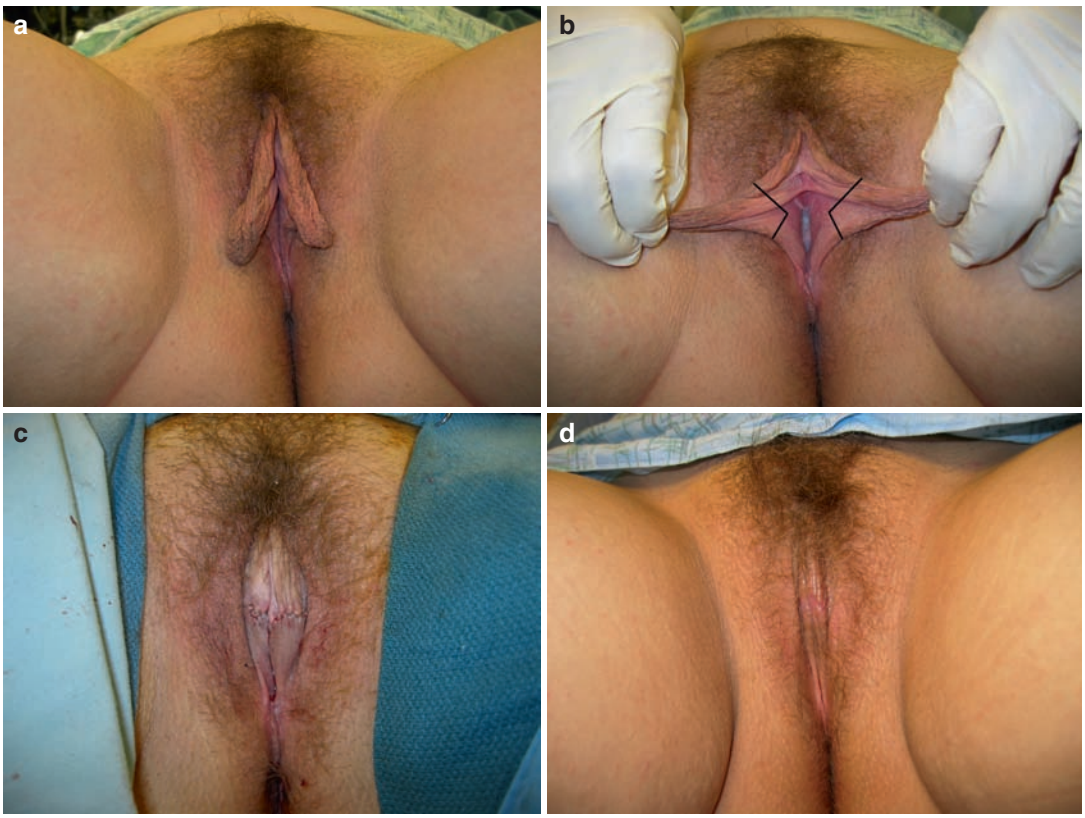

Figure 49.6. (**a**, **b**) A patient with congenital hypertrophy of the labia minora. (**B**) The resection design is "V" shaped. (**c**) The resection has been performed and the labia repaired. (**d**) The labia at 3 months reveal good position and contour and minimal scarring.

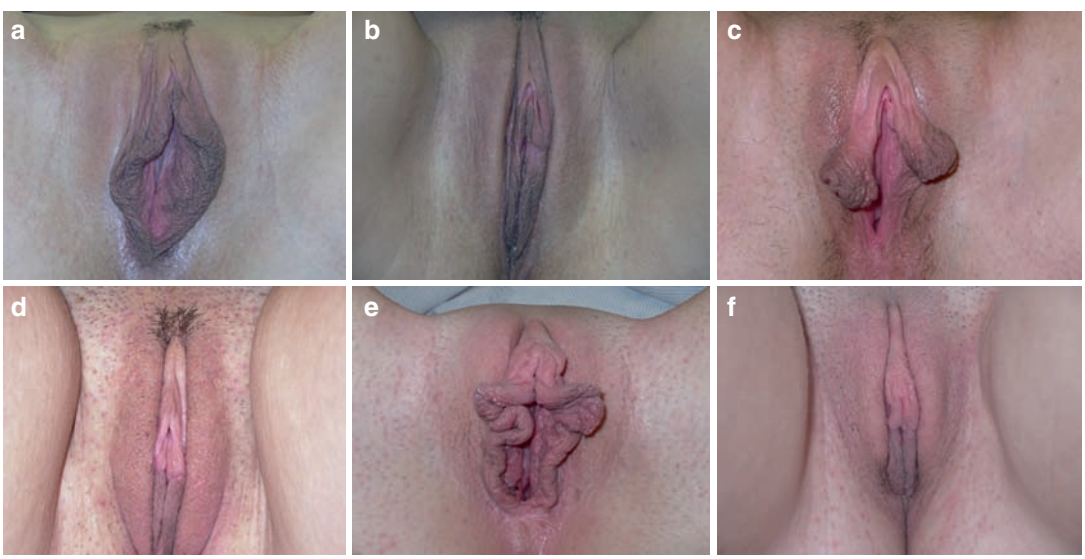

Figure 49.7. Labial hypertrophy falls into three broad categories: (**a**) diffuse hypertrophy, (**c**) central hypertrophy, and (**e**) anterior hypertrophy. The postoperative results achievable with "V" resection are demonstrated for each of these patterns, respectively (**b**, **d**, **f**).

pattern and least favorable with the diffuse hypertrophy, but all patterns responded fairly well to the basic operation (Figure 49.7a–f).

Complications were classified as minor (no intervention necessary), medium (minor intervention required or prolonged recovery), and major (serious medical intervention required or chronic patient complaints). There were no major complications. Minor complications included local irritation lasting longer than 1 month and suture breakage that was allowed to heal secondarily. Medium complications included infection requiring antibiotics, prolonged irritation (up to 3 months), or poor scar or asymmetry requiring minor surgical correction.

Infections were rare and limited to yeast infections from perioperative antibiotics and occasional stitch abscesses from deep vicryl sutures. Seven revisions were performed to correct asymmetry, persistent hypertrophy, or uneven scars, five of which were performed in the clinic. The longest reported chronic irritation and pain with intercourse was 4 months in one patient and 3 months in four patients. This seemed to correspond with the duration of the deep vicryl sutures. Since many patients could not be followed up after the initial postoperative care, it cannot be stated that these figures are truly accurate. It is presumed, however, that if a significant complication was present, the patient would have contacted the office.

Surgical reduction of the labia minora is a quick and well-tolerated procedure that can frequently be performed in a well-equipped clinic or office procedure room. Complications are rare and correctible. Even patients with complications express satisfaction with the procedure. The scars are small and minimally apparent and there appear to be no long-term chronic problems that arise from this operation. In the appropriately selected patient, labiaplasty is a safe, effective, and noncontroversial procedure.

Vaginoplasty

Vaginal tightening addresses several patient concerns. As opposed to labiaplasty, the patient population is older and almost always parous or multiparous. Some of the women have enlargement or senile changes of the labia minora as well and seek combination labiaplasty and vaginoplasty. Typical changes that occur in the vagina with aging and childbirth include diastasis or frank disruption of the pubococcygeal musculature (resulting from poor episiotomy repair), rounding of the posterior aspect of the vagina with shortening of the distance from the anal verge to the introitus, mucosal redundancy, and blebs that can protrude beyond the boundaries of the introitus. Large quantities of weight gain and subsequent loss can lead to similar vaginal and labial changes. Associated problems include rectocele, cystocele, and stress urinary incontinence. Discussion of these problems is beyond the scope of this chapter, but it is very important for the practitioner to be aware of them. Prior to scheduling surgery, a thorough examination must be performed, and the patients should be referred to a urogynecologist if they have complaints of the aforementioned disorders. Vaginoplasty can be performed in combination with bladder slings and other minor procedures such as labiaplasty, but it should be deferred until more serious complaints such as rectocele are addressed. Patients should be made aware

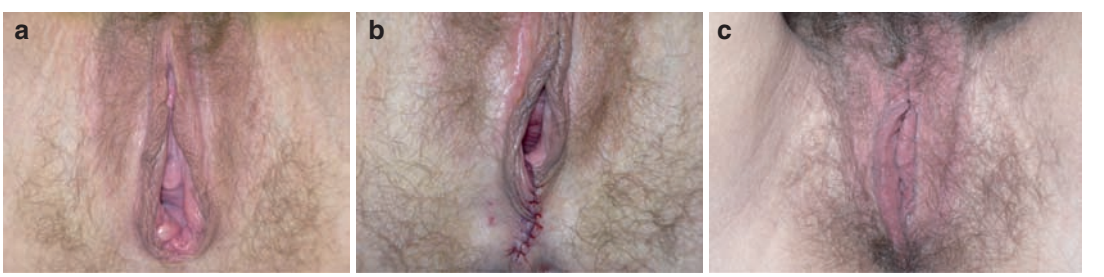

Figure 49.8. (**a**) A patient seeking vaginoplasty demonstrating typical blunting of the posterior aspect of the vagina with mucosal excess and deficient muscular tone. (**b**) The operation removes the redundancy of skin and mucosa and tightens the muscle. (**c**) The vagina at 3 months reveals decreased diameter with restoration of the posterior angle.

that the vaginal procedure addresses only the introitus and outer ring of tissue.[6]

The surgery is always performed in lithotomy under general anesthesia or sedation with local or regional block. A mild bowel prep is performed the day before the procedure, and the patients are given a 5-day course of antibiotics. Immediately following surgery, lomotil is prescribed for 5 days to allow healing to take place with few or no bowel movements. Colace is recommended to prevent constipation after the lomotil is discontinued. Frequent sitz baths are recommended.

The surgery starts by infiltrating local anesthesia and epinephrine into the tissues. Fifteen minutes are allowed for hemostatic effect. The procedure involves the resection of an asymmetric diamond shape of perineal skin in combination with vaginal mucosa. Approximately 2 cm of perineal skin is removed (measured from the introitus to the anal verge), and a 4-cm-long triangle of vaginal mucosa is removed (again measured from the introitus) (Figure 49.8a–c). Once the skin and mucosa are removed, gentle dissection with a fine hemostat is performed to reveal the musculature. Extreme care must be taken not to enter the rectum or anal canal. Repair and centralization of the musculature are performed with 2-0 vicryl sutures. The deep mucosa is repaired with 3-0 vicryl, and the vaginal mucosa is repaired with a running 3-0 chromic suture. The perineal skin is repaired with deep 3-0 vicryl and superficial 4-0 chromic interrupted horizontal mattress sutures. Closure is to an arbitrary tightness of 2–3 fingerbreadths depending on the size of the practitioner's hand. Care should be taken not to overcorrect, especially in the context of simultaneous labiaplasty (which further tightens the vaginal introitus). Foley catheters are not used during the procedure. Bacitracin ointment is applied to the area for 2 weeks postoperatively and then Vaseline. Sexual activity is restricted for a minimum of 1 month.

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Part VIII

Future Directions in Research

50

Anesthesia and Pathophysiology of Microcirculation

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Summary

Microcirculation is defined as the blood flow through the terminal portion of the cardiovascular system. The capillary network and primary draining site – a postcapillary venule, are the critical sites of microcirculation. Interactions between blood cells occur at the border of their lumen and endothelium. Multiple studies investigating the effect of anesthetics on peripheral and systemic microcirculation indicate that the choice of anesthetic during anesthesia affects basic mechanisms of microcirculation regulation. This issue concerns surgical procedures lasting many hours and microsurgical procedures of organ and tissue transplantation in particular. Authors also present the literature data on factors controlling microcirculation, such as neural, humoral, and muscular regulation and prostaglandins and kinins, oxygen free radicals, and nitric oxide (NO). The major part of the chapter is devoted to the effect of volatile and intravenous anesthetics of endothelial function and regulation. These considerations indicate that anesthetics are responsible for the course of the therapeutic process to an equally high degree as the surgical procedure itself and the surgical technique. Thus, anesthesia, which is simultaneously a therapeutic process itself, can significantly influence the final result of the therapy, and its proper selection can play a key role in obtaining ultimate therapeutic success.

Abbreviations

EDHF	Endothelium-derived hyperpolarizing factor
EDRF	Endothelium-derived relaxing factor
EMF	Electromagnetic flowmeter
ET-1	Endothelin I
GMP-140	140-kD granule membrane protein
LDF	Laser Doppler flowmeter
L-NOARG	L-NG-nitro arginine
MAP	Mean arterial pressure
MBF	Microcirculatory blood flow
NO	Nitric oxide
NOS	Nitric oxide synthase
PAF	Platelet activating factor
SVR	Systemic vascular resistance
TNF	Tumor necrosis factor

Introduction

In organ- and tissue transplant-related situations, surgery is often extended up to 12 h or more; therefore, it is important to have extensive knowledge about the effects of anesthesia on the perioperative period.[67,150] Reconstruction and microsurgery procedures involving free tissue transfer in orthopedic, trauma, and plastic surgery are also exceptionally time consuming and expose patients to many unfavorable conditions.[142] Such surgery was first reported by Daniel and Taylor.[36] Since then, many articles have been

M.Z. Siemionow and M. Eisenmann-Klein (eds.), *Plastic and Reconstructive Surgery*, Springer Specialist Surgery Series,

published on free transfer of tissues such as bones and muscles, as well as transfer of fingers and entire joints in animal experiment conditions. Unfortunately, surgeries that were successful in experimental conditions failed completely in clinical conditions, and the transferred tissues and organs became necrotized in the postoperative period. The imperfect microsurgical technique, incompetent use of anticoagulants or agents that facilitate vessel perfusion in joint pedicle, and other agents were believed to be the key reasons for these failures.[2,42,59,141]

The effect of volatile and intravenous anesthetics and muscle relaxants on the dynamics of circulation in the brain and other organs has been researched for many years, showing that the choice of drugs used during surgeries may affect the final outcome of these surgeries.[33,34] However, the effect of these drugs on the maintenance of peripheral and organ microcirculation is still not fully understood.[100,101,121,143]

Microcirculation

Anatomy, Physiology, and Pathophysiology of Microcirculation

The primary role of the cardiovascular system is to supply tissues and cells with the required amount of oxygen and nutrients. However, the final process of utilization of oxygen and substrates begins at the microcirculation level. The course of the process is normal only when three fundamental conditions are met: (1) resistance vessels function properly, (2) vessels responsible for transfer processes are in appropriate condition, and (3) vessels that are responsible for eliminating the products of metabolic transformations that occurred in cells are present.[1,69] Optimal blood flow also directly and indirectly depends on systemic blood pressure and blood viscosity.[73,162]

In the past, Zweifach proposed a straightforward definition of microcirculation as the flow of blood through the distal section of the cardiovascular system.[162] The anatomical structure of this system at the discussed level is strictly ordered. Starting from the large artery and ending at a metarteriole, each of these vessels is accompanied by a vein and a venule. The arteries typically give off smaller arteries, usually half in size, and branch off at right angles. At the level of the arterioles, the vessel diameter is only 50–100 μm. This vessel system begins the vascular network that comprises the microcirculation. At this level, the arterioles have more than one layer of smooth muscle and are always accompanied by a nerve. Owing to another ramification, the end arterioles of diameters smaller than 50 μm are created. They are surrounded by only one layer of smooth muscle with a closely connected nerve. In this section, the vessels can be categorized into two types. Besides the vessels described above, the other type of vessels are classified as metarterioles, with a smaller diameter within the range of 10–20 μm, with vessel walls also lined with smooth muscle tissue periodically interrupted.[13,65] At this point, only the precapillary sphincter separates the vessel from the spreading network of capillaries. It is believed that the sphincter is the structure that controls, among others, the appropriate blood flow within the capillary network.[13,26,45,46,66,72] Both the capillary blood pressure and the capillary blood flow are of oscillating nature, which is also to some degree dependent on the activity of precapillary sphincter. Two well-known mechanisms are used in attempts to explain this phenomenon. The first hypothesis claims that the sphincter is responsible for the act of regulation of the flow volume between extravascular and intravascular space. Opening of the sphincter should result in the increase of hydrostatic pressure in the capillary lumen and migration of fluid into the extravascular space, as in filtration; the sphincter constriction results in a drop of the hydrostatic pressure in the capillary lumen and leads to reabsorption of the fluid into the intravascular space. The other hypothesis involves local metabolic processes. The state of constrictive activity of the sphincter is associated with the supply of oxygen and other required primary substrates, as well as with the level of ingredients related to metabolic transformations, such as CO_2 and lactates.[8,13,20,37,38,40,50] The distribution of the capillary network in organs and tissues is varied. In those of high metabolic activity, such as myocardium and skeletal muscles, the network density is high, whereas in tissues of low metabolic activity, the density is significantly lower. Average erythrocyte flow rate in a capillary is $1\,mm \times s^{-1}$ and may drop to zero, but it may as well exceed several millimeters per second.[99] Capillaries are built of a single layer of

endothelial cells covered with the basement membrane. They are not surrounded by smooth muscle cells. Capillaries comprise the anatomical junction between the arterial and venous system; the latter begins with the so-called venous capillaries. These, in turn, are connected with postcapillary venules up to 30 μm in diameter. Then, in turn, there are the collecting venules 30–50 μm in diameter, with walls composed of one or two layers of smooth muscle tissue. The confluence of these venules leads to the first collective veins, 300 μm in diameter.[90,105,112]

The arterial and venous system within certain organs includes arteriovenous canals, a specific form of which is Hoyer canals. They are absent in skeletal muscles.[38,50,70]

Direct transport of fluid through the capillary endothelium is determined by the balance between osmotic and hydrostatic pressures. The hydrostatic pressure within capillaries is a result of the pressure and resistance in precapillary and postcapillary vessels. Oncotic plasma pressure opposes the hydrostatic pressure, allowing the fluid to be retained within the lumen of the vessels. Outside the capillaries, the hydrostatic pressure of the fluid contained in the tissue counteracts filtration from the capillary lumen, whereas the value of the oncotic pressure within the interstinum promotes inflow of fluid from the capillary lumen.[10,12,13,19,26] The above phenomena are illustrated by the hypothetical Starling equation.[100]

$$\text{Fluid transfer} = K\,(Pc + Oi) - (Pi + Oc)$$

Where

Pc – hydrostatic pressure in capillaries
Oi – oncotic pressure in the interstinum
Pi – hydrostatic pressure in the interstinum
Oc – oncotic pressure in the capillaries
K – capillary endothelium filtration constant

Therefore, anesthetics affecting the intravascular pressure and contractility of the smooth muscle comprising the vessel walls may significantly disturb the balance associated with the precapillary and postcapillary resistance indicators, influencing the filtration processes within the microcirculation limits.[76,84]

Capillaries may be capable of constriction due to special properties of endothelial cells and constriction of myofibroblasts, which are adjacent to the capillary walls. At the same time, this is another factor promoting utilization of substrates at the capillary level, and the capillary endothelial metabolic functions are the direct object of the anesthetic agent's activity.[112–114,119]

Mechanisms That Control Microcirculation

In general, the mechanisms controlling microcirculation can be divided into two categories: distant and local ones. Distant mechanisms include control by elements of the nervous system and humoral factors. Local regulation factors originate from active cellular transformations occurring within the region supplied by the microcirculation system in question.[102,103]

Neural Factor

Neural regulation at the peripheral microcirculation level depends on the sympathetic activity of autonomous nervous system, with fiber endings located in the vessel walls. These adrenergic nerve fibers cause constriction of vessels, in particular the resistance vessels. As a neurotransmitter, noradrenalin causes constriction of the vessel lumen by activating the vessel alpha receptors. The vascular response to sympathetic adrenergic stimulation is not uniform within the peripheral circulation due to both different nerve fiber densities in individual regions of the cardiovascular system and nonuniform number and nature of the receptors.[105,106,109,114] Arterioles are accompanied by the richest adrenergic innervation, whereas the postcapillary venules are poorer with respect to this innervation. Larger veins and the great veins are poorly innervated. Similar gradation of response to adrenergic stimulation is observed in the pre- and postcapillary peripheral circulation vessels. In consequence, adrenergic stimulation causes a twofold majority of the vascular resistance value within the precapillary region compared with the postcapillary region.[43,46,47] The precapillary sphincter is also subject to sympathetic adrenergic stimulation, although local factors are clearly predominant in terms of vascular control at the microcirculation level, especially at the capillary level.[37,40,50]

Humoral Factors

Release of catecholamines from adrenal medulla and noradrenaline from the sympathetic nerve endings into circulating blood is a factor controlling the tension within the peripheral vascular bed.

In the case of skeletal muscle, the increase in adrenalin level shows a two-phase activity on the muscle arteriole. Physiological levels of this hormone cause dilation of these vessels by influencing the presence of a greater density of beta receptors compared with alpha receptors. Rapid increase in adrenaline levels is responsible for higher activity of alpha-receptors and vasoconstriction. However, more recent studies suggest that the presence of adrenaline and noradrenaline, even if administered onto the surface of a denervated muscle, results in arteriole dilation. Primary response to noradrenaline is always expressed by an explicit vessel constriction. The only exceptions are the skeletal muscle bed and the liver bed.[49,50,77,115,129]

A particular situation takes place within the skeletal muscles and the vessel response to circulating catecholamines. Direct observations and indirect conclusions may suggest that beta-adrenergic stimulation caused by circulating catecholamines clearly alleviates the alpha-adrenergic increase in regional vascular resistance, thus, preventing the muscle from the blood flow reduction. In addition, as beta-adrenergic stimulation within the muscle dilates the peripheral arterial part of the system, that is, the arterioles, and the venous part, that is, the venules, it increases the capillary surface area, making the capillaries more accessible for the flowing blood, thus, affecting the course of local filtration processes.[106,134,136,151] During a bleeding episode, the mixed adrenergic reaction caused by high levels of circulating catecholamines (adrenaline and noradrenaline) may significantly affect fluid transport within the skeletal muscle, which, in turn, facilitates an increase in the volume of the circulating blood.[4,21]

Local Factors Affecting Microcirculation

Local factors regulating peripheral microcirculation function can be divided into two major categories: metabolic and myogenic regulation.[55,69,77] Metabolic regulation is achieved by changes in levels of certain substrates and metabolic end products and intermediates such as O_2, CO_2, H^+, and K^+.[40,42,44]

Myogenic regulation of peripheral circulation is closely associated with the persistent mechanism pertaining to small vessels, consisting in vessel constriction upon hydrostatic pressure increase and dilation upon hydrostatic pressure reduction.[37,134] Upon perfusion of resting muscle with both arterial and venous blood, it shows that venous blood has a vasodilating effect within the perfused part of the muscle. Numerous excess active compounds were identified in the muscle subjected to the experiment. Among those, one should mention the increase in potassium (K^+) ion levels, increase in osmolarity, excess of H^+ ions, adenosine, phosphorus, magnesium, and an increase in partial CO_2 pressure or reduction of partial O_2 pressure. With time, when oxygen consumed by the muscle starts to exceed the oxygen supply, excess amounts of H^+ ions, adenine nucleotides (ADP and AMP), and adenosine are released from muscle cells. These factors, combined with the increase in osmolarity during the experiment, are responsible for the increase in muscle perfusion.[116,119,134]

In their research, Duling and Pittman placed great emphasis on the determination of the significance of the influence of oxygen on the microcirculation status. Two components of microcirculation are largely responsible for oxygen availability in cells – vascular resistance and capillary density within a strictly defined study region. According to Duling and Pittman, vascular resistance is determined by the tension of arterial vessels, whereas the density of capillaries participating in the flow (number of active capillaries per volume unit) depends on the activity of the precapillary sphincter.[40]

The progressive drop in partial oxygen pressure in microcirculation, starting from the proximal section toward the distal section and ending at the capillary level, irrefutably indicates that oxygen diffuses outside the vessel lumen, starting at the arteriole level. Partial oxygen pressure in the capillaries of 15–25 μm diameter equals 30–40 mm Hg, whereas it ranges between 85 and 100 mm Hg in vessels of 50–100 μm diameter. The direct relationship between partial oxygen pressure and arteriole wall tension cannot be established, since even in tissues, where the pressure values are in the range of 10–12 mm Hg, arteriole wall tension is maintained and often remains unchanged even at lower oxygen pressures. The lower limit value of cellular oxygen pressure is 2 mm Hg.[4,16,19,119,135]

In 1968, Honig proposed a hypothesis regarding the mechanisms that control the behavior of precapillary sphincter cells depending on oxygen supply. When the sphincter is open, its cells are supplied with O_2 by the blood flowing

directly along these cells. When the sphincter is closed, O_2 supply originates from the interstinum, and the oxygen pressure in the muscle drops to a level approximating the lower limit for the upkeep of the oxidative phosphorylation mechanism. The muscle becomes relaxed, the blood flow and the oxygen pressure start to increase, and the entire cycle is repeated.[8,77]

Myogenic Regulation of Microcirculation

The role of myogenic factors in the control of blood flow resistance, exchange, and the condition of capacitance vessels was studied by Mellander and Johannson.[114] This control scheme includes the assumption that the increase in transmural pressure, being the origin of the vessel wall tension, is responsible for the tension of the smooth muscles in this wall. In consequence, the vascular resistance is inversely proportional to the transmural pressure. The myogenic response to transmural pressure changes is present both in arterioles and in precapillary sphincters.[13,15,114,115]

It is commonly known that the changes in arteriole and metarteriole diameter directly affect changes in the systemic vascular resistance (SVR). Isoflurane is the agent that in experimental and clinical trials was responsible for the reduction of mean arterial pressure (MAP) via a reduction of the systemic vascular resistance.[33,34,158] The values dropped in proportion to increasing levels of the anesthetic in the breathing gas mixture. In an experimental model, a significant increase in vessel diameter was observed during 4 h of anesthesia using halothane concentration of 2 MAC. This phenomenon was not observed during isoflurane anesthesia at values of 2 MAC. These findings may suggest that during halothane anesthesia in a denervated muscle, the anesthetic has a direct effect on the vascular wall, whereas a vasodilating effect of isoflurane on the resistance arterioles must have consisted in isoflurane's inhibiting the sympathetic nerve endings of the autonomous nervous system. The mechanism regulating the systemic vascular resistance at the microcirculation level depends primarily on the behavior of precapillary sphincters. However, neither the presence of precapillary sphincters in the cremaster muscle in rats is well documented nor have they been definitely recognized.[66,104–106] According to one of the hypotheses, this role may be played in the muscle by the third-order or the fourth-order arteriole, A3 or A4.[66] It is the behavior of one of these arterioles and the changes in its tension, which may indicate that it is a sensitive indicator of blood flow resistance in peripheral microcirculation.[91] Figures 50.1 and 50.2 illustrate the characteristics of these changes.

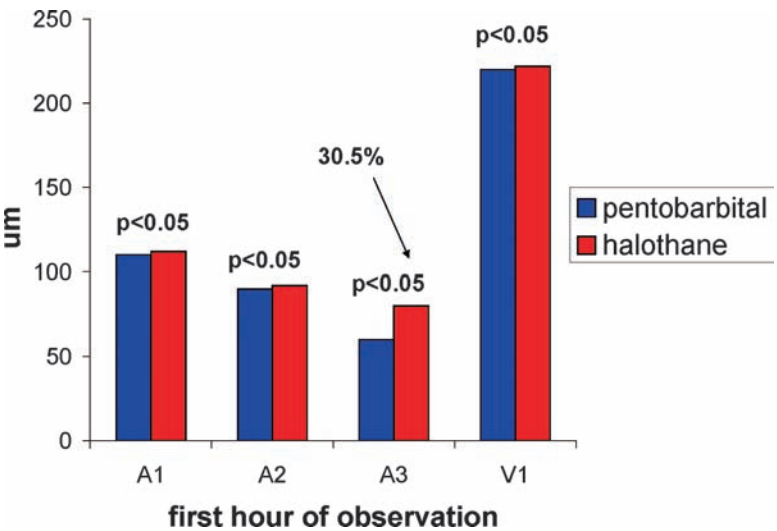

Figure 50.1. An increase in the diameter of arteriole A3 was observed in the halothane group, whereas the diameters of the remaining arterioles are comparable with those of their counterparts in the pentobarbital group. (Data from Kusza K, Nalbantoglu U, Siemionow M. Halothane anesthesia improves flow hemodynamics of the muscle flaps. *Surg Forum*. 1996;XLVII:785–787.).

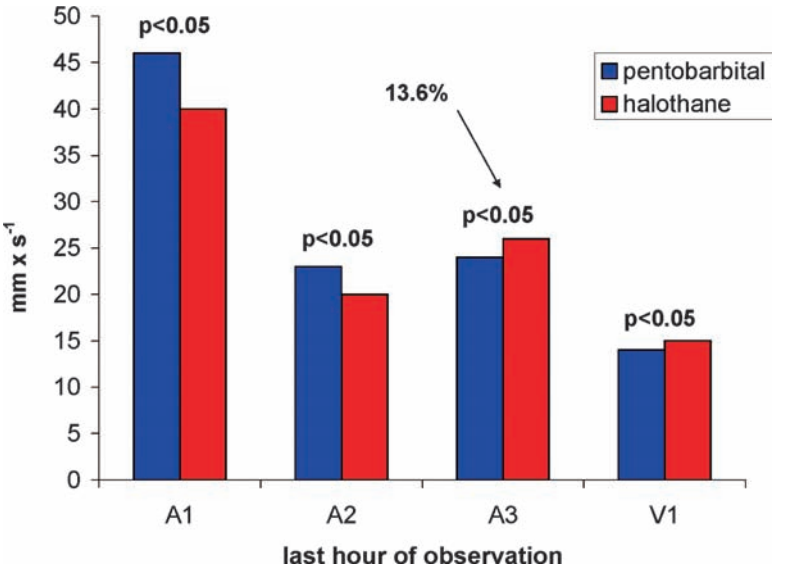

Figure 50.2. An increase in the erythrocyte flow rate in arteriole A3 was observed in the halothane group, whereas the flow rate in the remaining arterioles is higher in the pentobarbital group. (Data from Kusza K, Nalbantoglu U, Siemionow M. Halothane anesthesia improves flow hemodynamics of the muscle flaps. *Surg Forum*. 1996;XLVII:785–787.).

Other Factors Affecting Microcirculation

Prostaglandins and Kinins Versus Microcirculation Regulation

Other important regulators of microcirculation are prostaglandins and other arachidonic acid-related compounds. Prostaglandins are originally synthesized in the arteries and veins by the endothelial cells.[152,153,155]

Prostacyclin is the major prostaglandin synthesized by arteries and veins; however, prostaglandin E_2 is synthesized to the largest extent in microcirculation. Stimulation resulting in the release of endogenous arachidonic acid reserves or arachidonic acid administration results in a blood flow increase as a result of arteriole dilation.[130,148] This effect can be effectively cancelled out by the use of cyclooxygenase inhibitors, such as indomethacin.[130] Prostaglandin activity is always accompanied by the presence of bradykinin, which was proven to be responsible for the increase in prostaglandin synthesis in vivo and in vitro.[98,148] In addition, bradykinin selectively affects prostaglandin synthesis and causes an increase in PGE levels in arterial vessels and PGF levels in venous vessels, which clearly explains its effect on the dilation of arteries and constriction of veins.[140,153]

Chury et al. proved that the absence of endothelial cells leads to suppression of the response leading to prostaglandin release following stimulation with bradykinin.[30] Forstmann et al. have somewhat confirmed these observations by proving that bradykinin mediates the reduction of arteriole wall tension in the presence of simultaneously released prostacyclin and prostaglandin E_2, originating from the subendothelial intima of the vascular wall. The above studies were conducted in rabbits. Arterial vessels in dogs and humans relax under influence of bradykinin action as well; however, it is the vascular endothelium that is responsible for their production.[51]

Free Oxygen Radicals and Microcirculation

Owing to the activity of cyclooxygenase enzyme, the synthesis of prostaglandins becomes the source of oxygen metabolites, which are also present in the microcirculation. These oxygen species cause dilation of arterioles in the vascular bed by direct action and by activation of nitric oxide (NO).[25,27,63,133]

Free oxygen radicals are formed in situ in Fenton's reaction (hydrogen peroxide + iron sulphate).[48] Free oxygen radicals are characterized by short half-time and high biological activity.

Oxygen radicals express chemotactic activity and are the cause for multinuclear leukocyte accumulation.[86] Oxygen radicals released by these leukocytes start a cascade that initiates further formation of endoperoxides.[127] The time between leukocyte accumulation and the reactions associated with free radical activity is different for different vascular beds.[71]

Monovalent compounds formed in the reduction of oxygen are the cause of persistent dilation of arterial vessels. Longer exposition to free oxygen radicals leads to vascular bed damage. The damage consists of highly intensified protein permeability of vascular endothelium and focal endothelium damages. These compounds are able to cross the cellular membrane into the cell interior, where their survival time elongates significantly while at the same time intensifying their vasodilating effect.[25,85] Free oxygen radicals belong to the most crucial microcirculation regulators in the body. They also mediate the most pathological responses of the vascular bed in stress conditions effect.[133] They are released when the cell metabolic reserves become depleted. They also affect acetylcholine-dependent vessel relaxation; therefore, the destructive effect of these compounds on the synthesis of NO is possible.[64,88,89,133]

Nitric Oxide and Its Effect on Microcirculation and Factors Affecting Micropolarizaton of Vascular Smooth Muscles

L-arginine is a physiological precursor of nitric oxide, synthesized and released by the vascular endothelium. Biological presence of nitric oxide was confirmed due to Furchgott and Zawadzki's discovery (1980) of the compound they called endothelium-derived relaxing factor (EDRF).[57,58,125,152]

Upon biosynthesis of nitric oxide, L-arginine is converted with molecular oxygen in the presence of NO synthetase (NOS). Endothelial formation of NO seems to be crucial for regulation of systemic vascular resistance. Administration of nitric oxide inhibitors results in a local decrease in blood flow and leads to an increase in arterial blood pressure.[132,154] Changes in L-arginine metabolism and NO formation may be responsible for the etiology or may be the factor facilitating vascular insufficiency.[96,111,152]

As mentioned above, capillaries in various organs, not excluding the brain, show their ability to spontaneously constrict and dilate in a characteristic pattern, which is manifested by oscillations of blood flow at the local level. The mechanism of this phenomenon has not yet been fully explained.[8,20,55,83] In 1964, Folkow suggested that special trigger cells present in smooth muscle, forming a spreading oscillating membrane, are responsible for this phenomenon.[49,50] Research by Hundley and Bouskel proved, however, that the explanation of this question is much more complex.[20,83]

It is known, however, that nitric oxide is continually released from the vascular endothelium and probably has a modulating role in the act associated with the change of the vessel caliber described above. Another observation regarding the analyzed aspect of the issue is related to nitric oxide synthase and the activity of its inhibitor – L-NG-nitro arginine (L-NOARG).[117,149]

It turns out that the change in vessel diameter is pH-dependent. Upon hypercapnia, vessel dilation is caused by a decrease in the pH value, just as vessel constriction may occur following inhibition of NO synthetase by L-NOARG. Upon the pH drop due to hypercapnia, however, there is no stimulation and increased activity of NO synthetase or an increased production of cGMP, which suggests that low pH is the only factor responsible for vessel dilation in these situations.[79,80,87,118] Currently, a phenomenon known as hyperpolarization of vascular wall smooth muscle starts to play an increasingly important role among the factors responsible for vascular tension, besides the role played by the endothelium.[62]

According to Garland et al., exogenous nitric oxide may cause hyperpolarization of smooth muscle. This phenomenon directly affects the activity of endothelium, which releases NO in response to hyperpolarization.[62,110] On the other hand, research by Chen et al. proved the presence of phenomena in which vascular relaxation is not directly associated with the activity of nitric oxide and enzymatic cyclooxygenase products, as hyperpolarization continues to cause relaxations following administration of their inhibitors.[29]

Therefore, it is suggested that a not fully identified hyperpolarization-causing factor exists, independent of NO and prostacyclin, but dependent on the epithelium – endothelium-derived hyperpolarizing factor (EDHF).[32,131,152]

The Effect of Morphotic Blood Elements on Microcirculation with Special Attention on Their Effect on Capillary Network

Morphotic blood elements are important elements affecting the blood flow dynamics in microcirculation. As white blood cells are larger in volume and undergo less deformation than red blood cells, white cells may play an important role in microcirculation blood flow regulation.[11,19] Observing the survival of the white cells in microcirculation, one can clearly see that they are of spherical shape. They can undergo passive deformation under hemodynamic stress conditions and flow along the capillary network in this time.[21] In normal conditions, these cells do not undergo any active change in their shape during transport through the circulatory system. Spontaneous deformation pertains to nondifferentiated forms and occurs in vivo during the cells' migration through the endothelium along the chemotactic gradient and during phagocytosis.[28,31]

The overall outline of white cells is spherical; however, the corrugated cellular membrane creates an excess surface, allowing for deformation of a cell without increasing its surface area.[31,39,41]

In 1946, Nicoll and Webb recorded a transient blockade in the blood flow at the capillary level caused by the presence of white blood cells.[123] This research was confirmed in 1970 by Chen and Skalak and again in 1980 by Schmid-Schonenbein, who observed the blood flow in small vessels in vivo.[28,139]

White blood cells affect the blood flow in the microvessels by the following mechanisms: adhesion to the vascular wall, closure of the vascular lumen during the slowed flow, including complete closure of the vessel patency, by mutual interaction of microvessels in which the resistance was increased, as well as by their presence in the extracellular space.[124,126,138]

In small capillaries, the white blood cells have to undergo deformation, sometimes even down to half of their original dimension, and their further flow is possible only owing to encapsulating the white cell structure by a plasma "sleeve." White cells also affect the blood flow at sites where new vessels branch off, causing their occlusion at the very start of ramification. If a white cell enters the lumen of a vessel, which has a diameter smaller than the white cell size, it is obvious that a definite time required for blood cell deformation must pass to allow the cell to get through the vessel lumen. During this deformation, the vessel is completely occluded, which makes any blood flow through its lumen impossible. This blood flow occlusion may have a significant effect on the distribution of blood in the capillaries, depending on how long the deformation process is.[137,141,147] The question to be asked is which of the elements – deformation time or occlusion time – is more important in terms of affecting the microcirculation blood flow? Mathematical formulas developed by Needham and Hochmuth in 1989 and Fenton in 1985 provided no definite answer to this question. Partially, they suggest that the higher the pressure force of the inflowing blood stream, the shorter the deformation time.[48,122] Adhesion of white cells to the vascular wall is more frequent when the blood flow in the vessel is slowed down.[4] This promotes margination of white cells, which thus become more susceptible to chemotactic substances.[78]

It is known that the higher volume and viscosity of white blood cells compared with red blood cells (one white cell is equivalent to 700 red cells) leads to much faster mechanical arrest of blood flow in vessels 5 μm in diameter.[107,108]

Other in vivo studies proved that the increase in resistance in the capillary network and the incidence of lumen occlusion by leukocytes are dependent on the capillary network structure. In the actual capillary structure, the slow pressure increase is actually five times higher along the vessels occupied by occluding leukocytes than in the starting conditions. Variation of pressure values in the capillary network, depending on the number of occlusions, also depends on intervascular canals within the studied network. A higher number of canals within the capillary network leads to lower variation of pressure values, in consequence elongating the time of vessel occlusion by leukocytes. Reversely, in borderline conditions where no canals between the capillaries are found within the network, the pressure increase will correspond to the pressure value at the capillary network entrance, and the leukocyte occlusion will be very quickly removed.[108,124,126]

Another important feature of the capillary network geometry is the number of ramifications within the network, as they affect the leukocyte behavior.[162,163] Experimental models of microcirculation in skeletal muscle showed that a significant number of vessels must become occluded before a delayed vascular resistance increase is observed in the network.[61,139,147] For instance, a network model comprising 60 parallel vessels shows no signs of resistance increase until at least five main paths of this network are totally occluded. Above this number, a slight increase in the vascular resistance, not exceeding 9% of the baseline value, was recorded. In the tree-branched vascular network, occlusion of a small number of capillaries causes a high increase in flow resistance, since in this structure a much larger number of capillaries remains devoid of blood flow.[137,146,147]

Differences in capillary network structure are observed in the tissues, which may significantly affect flow disturbances and change the so-called capillary reserve, depending on the organ being analyzed or subjected to surgery.[138,157]

The effect of white blood cells on the increase in flow resistance is also dependent on changes in viscosity. Medications and substances as well as pathological lesions formed in the system and affecting white cell deformation and viscosity must change the capillary network resistance values.[120,137]

In addition, white cell occlusion affects further white cell distribution in the capillary network and is directly responsible for the phenomenon of highest flow pathway selection.[156] When one of the vessels is occluded, white blood cell flow is transferred to another capillary network. The more vessels are occluded, the more white cells are dispersed within the entire network.[28,31,60]

Effects of Anesthetics on Microcirculation

Volatile Anesthetics

Microcirculation behavior upon halothane anesthesia was studied in the bat wing membrane skin. Shallow halothane anesthesia with an inhaled vapor pressure of 0.81% vol. caused a significant pressure drop and dilation of arterial vessels to the level of arteriole A2 ramification, without changing the diameter of postcapillary venule. Deep halothane anesthesia (1.42% vol.) was again responsible for the pressure drop and increased arteriole dilation, as well as for significant dilation of venules.[100]

Nitrous oxide used as a component of volatile or combined anesthesia caused arteriole constriction in rabbit ears when used with oxygen in 80% N_2O and 20% O_2 mixture.[101] Rat cremaster muscle and its microcirculation were observed during exposition to 80% N_2O and 20% O_2 mixture following induction of anesthesia with pentobarbital as intraperitoneal injection. The third-order arteriole (starting diameter, 36 μm) was constricted within the first 15 min of breathing with nitrous oxide. After 20 min of breathing with N_2O and O_2 mixture, the arterial pressure value and the arteriole dimension returned to the baseline values. At the same time, the third ramification of the venule (in counting order) showed no deviations compared to baseline.[99]

In an in vivo study, Leon et al. observed the behavior of end arteriole and the density of functioning capillaries in the region supplied by this arteriole in the diaphragm muscle of rats following exposure to increasing concentrations of halothane and isoflurane in the respiratory mixture (0.5; 0.75; 1.0 MAC). The main difference between the effects of halothane and isoflurane on microcirculation within the diaphragm muscle was marked by a distinct reduction of the arteriole A4 diameter following administration of halothane. Isoflurane administration had no effect on this parameter. The mechanism behind this phenomenon is unclear. The authors suggest a process of passive constriction of end arteriole as an effect of significant drop in MAP in the halothane group. Contrary to the above, in comparison studies of halothane where equivalent doses of isoflurane were used, no similar phenomenon was observed at the arteriole A4 level, although MAP in the isoflurane group was significantly lower than that in the halothane group. Analysis of these phenomena may lead to a suggestion that there is a threshold MAP value at which arteriole constriction takes place; however, this value varies depending on the anesthetic agent used. In addition, reduction of the arteriole A4 diameter was accompanied by a simultaneous drop in the density number of capillaries with effective blood flow. This finding was also reported by other authors.[74,95]

Seyde and Longnecker proved that in rats breathing halothane and isoflurane at a concentration of 1 MAC, there were no changes in spleen and gastrointestinal blood flow, whereas the changes proceeded in a parallel fashion in the skeletal muscle and in the diaphragm and consisted in flow reduction with an accompanying increase in vascular resistance. The authors suggest that at this stage of research, explanation of differences between the effects of halothane and isoflurane on microcirculation must form the basis for an analysis of cellular phenomena in vascular beds of different organs.[155]

Yano et al. used microspheres labeled with radioactive ^{51}Cr, ^{125}I, and ^{46}Sc to study the effects of structural isomers of enflurane and isoflurane on microcirculation in various organs in dogs. The research was performed at two anesthesia levels, namely, 0.75 MAC, and 1.5 MAC. At the same time, behavior of arteriovenous canals under the influence of enflurane and isoflurane anesthesia was observed. Enflurane anesthesia at 0.75 MAC resulted in reduction of the capillary flow by 35% in the thyroid gland, by 59% and 50% in left and right myocardium wall, respectively, by 59% in right adrenal gland, by 63% in the liver, by 56% in the spleen, by 35% in the pancreas, by 20% in the network, and by 60% in the small intestine. Further reductions of the flow at this level during anesthesia were observed at 1.5 MAC by 15% in the thyroid gland, by 31% and 32% in left and right myocardium wall, respectively, by 42% in the adrenal gland, by 47% in the liver, by 31% in the spleen, by 23% in the pancreas, by 20% in the network, by 45% in the stomach, and by 54% in the small intestine. No significant changes in blood flow in the brain, kidney, large intestine, and skeletal muscle were observed. A reduction in the number of open arteriovenous canals in the kidney from the baseline of 12.1% to 3.8% at 0.75 MAC and to 2.5% at 1.5 MAC of enflurane was observed. On the other hand, upon isoflurane anesthesia, the capillary blood flow did not change, excluding the thyroid gland and the right ventricular wall, in which the flow was reduced respectively by 43% and 74% at a concentration of 1.5 MAC. However, significant increases of flow through the arteriovenous canals in the brain were observed, from the baseline value of 12.0% to 29.7% and 33.0% at 0.75 MAC and 1.5 MAC of isoflurane anesthesia, respectively. The same process applied to the skeletal muscles and globally to the entire systemic circulation.[160] Isoflurane can also significantly increase the blood shunt at the skeletal muscle level.[94]

Sigurdsson et al. studied the effect of halothane and isoflurane on microcirculation in the musculocutaneous flap in small pigs (23–28 kg) in normovolemia and controlled hypovolemia settings. The choice of animals was guided by the fact that the vascularization of a pig's skin is similar to that in humans, whereas vascularization of a rodent's skin is completely different. In addition, rodents have a much higher concentration of xanthane oxidase enzyme in their skin, therefore, producing a higher amount of free oxygen radicals upon ischemia and subsequent reperfusion than humans or pigs. Secondly, the systemic and coronary circulation in pigs is significantly similar to that in humans. The anesthetics were used in equivalent concentrations, that is, 1 MAC in pigs is 0.7% vol. of halothane and 1.4% vol. of isoflurane. The authors intended to provide experimental conditions that would be significantly similar to clinical conditions. Combination anesthesia was also performed using halothane or isoflurane with nitrous oxide and opiates, since volatile anesthetics are seldom used as the only agents during anesthesia.[145]

Ischemia and necrosis of musculocutaneous flaps used for reconstruction of damaged tissues remain an important clinical issue. Little knowledge regarding the effect of anesthetics on hypoperfusion of the transplanted cutaneous and muscle lobes remains the cause of necrosis-associated complications. The influence of halothane and isoflurane on microcirculation in the musculocutaneous flap in pigs was compared to their effect on undamaged skin and muscle in pigs. Measurements were made under normovolemia and moderate hypovolemia conditions (loss of 5%, 10%, and 15% of the circulation blood volume). Microcirculation blood flow was measured using a multichannel laser Doppler flowmeter (LDF) and electromagnetic flowmeter (EMF) to measure the global blood flow through the lobe. The microcirculation flow in normovolemia conditions was well controlled during both halothane and isoflurane anesthesia. Following slight and moderate blood loss, the microcirculatory blood flow (MBF) decreased significantly when halothane was used, whereas isoflurane did not significantly affect the flow in these conditions.

The analysis of the reasons for microcirculation flow reduction in hypovolemia seems to suggest that deeper hypotension in the halothane group is the cause of the increased concentrations of circulating catecholamines and, in consequence, intensified vascular constriction. These anesthetics have a specific effect on endothelial cells and the flexibility of erythrocytes and leukocytes during ischemia and reperfusion in the cutaneous flap.[22,144,145]

Vascular endothelium is not only able to release compounds responsible for vascular lumen relaxation but also balances those with the formation of vasoconstricting compounds, such as endothelin I (ET-1).[7] In the case of the mechanism of action of halogen anesthetics, each of these agents influences the release of eNO to some degree, depending on concentration, and may change this reaction by affecting the activity of reactive oxygen species so that vasoconstricting endothelin becomes predominant in the circulation. In this aspect, sevoflurane is particularly characteristic, as its mechanism of action involves responsibility for releasing peroxide anion from the vascular wall. Since peroxide anion is responsible for the drop in eNO production in the presence of sevoflurane, the vasodilating effect of sevoflurane is doubtful, as it mediates the increase in ET-1.[7] This type of vascular endothelium dysfunction may have serious microcirculation blood flow-related clinical implications. It is currently known that halogen anesthetics may suppress NO activity by disabling NO binding by a heme prosthetic group or by an allosteric effect on the enzyme involved in this binding process. It is believed that the factor inhibiting the NO activity in the presence of isoflurane and halothane – the latter in particular – might be the effect of the anesthetic agent on the release of free oxygen radicals, as in the case of sevoflurane.[17,18,103] However, it is worth mentioning that isoflurane and halothane do not alter the function of adenyl cyclase, which is the first messenger stimulating NO release for vessel relaxation.[82,97] Very important, from a clinical point of view, are observations suggesting that in the experimental studies of free musculocutaneous flaps in halothane and isoflurane anesthesias, an increased blood flow through the capillaries was observed only at normal partial oxygen pressure in the blood of animals; the use of a particular inhaled anesthetic agent had no effect on the microcirculation status in the case of hypoxemic groups.

The behavior of postcapillary venule was studied and evaluated together with the morphotic blood elements flowing through this vessel upon halothane and isoflurane anesthesia. The postcapillary venule is the primary blood reservoir, receiving all the morphotic blood elements, which passed through the capillary network and to which the postcapillary venule is the anatomically prescribed drainage site.[162] At the same time, the vessel plays the primary role in the case of venous insufficiency of the studied flap – in the presented case, among others, the cremaster muscle.[5,128] Blocking the flow in part of the postcapillary venules results in congestion in the isolated or transferred muscle lobe with slowed arteriole blood flow and is often associated with irreversible changes due to severe metabolic disturbances in cells of the region affected by pathology.[2,141] Behavior of leukocytes in the lumen of the postcapillary venule is affected by the character of blood flow in the lumen. A significantly lower number of rolling leukocytes in the lumen of postcapillary venule was observed during halothane anesthesia compared with that in the isoflurane group; as the experiment went on, an increase in the number of leukocytes that adhered to the endothelium was documented in the isoflurane group.

Reduction of leukocyte accumulation in the postcapillary venule has a direct effect on the improvement of capillary perfusion and significantly extends the time of endothelial edema development, a final consequence of which is the complete blockade of blood flow. This finding supports the hypothesis that changes in the activity of the Ca^{++} pump system, along with the changes in Ca^{++} ions concentration in leukocyte cytosol decrease their activity during halothane anesthesia, as halothane produces such effects.[31,52,53,103,159] Activity of leukocytes in the postcapillary venule could, thus, depend on the levels of active Ca^{++} ions. Simultaneously, Dirnagl et al. and Warnke et al. believed that the increased activity of leukocytes is associated with hypoperfusion of the studied region.[39,157] Isoflurane was explicitly responsible for hypoperfusion of the cremaster muscle, which was manifested by a reduction in the number of capillaries participating in blood flow and a reduction of the diameter of the observed venules with simultaneous increase in the erythrocyte flow rate. Many

substances are responsible for the increased activity of leukocytes within the oxygen-deficient tissues. Leukotrienes and peroxides deregulate the complex of glycoproteins adhering to the leukocyte surface. Peroxides also cause a change in microcirculation leukocyte behavior via the 140-kD granule membrane protein (GMP-140) molecule released in such circumstances by the endothelium, causing intensification of leukocyte adhesion to the vascular wall. Tumor necrosis factor (TNF) and interleukin-1 are responsible for production of platelet activation factor with the stimulation of monocytes/macrophages, which are bound to their specific receptor in the endothelium. All these phenomena lead to increased adhesion of leukocytes to the vascular lumen endothelium, facilitating its destruction, microvessel occlusion, and release of toxic compounds (free oxygen radicals), platelet activating factor (PAF), which results in microvascular stasis, and migration of leukocytes into the tissue outside the vessel lumen. A modulatory role is played by halogen anaesthetics in this process.[39,107,108,152]

This opinion is shared by Blaise and Akata, who argued that in particular situations the inhaled anesthetics, including halothane and sevoflurane, may be responsible for the vascular spasm and increased permeability by the release of free oxygen radicals.[3,17,18] The effect of these agents on endothelial guanylyl cyclase can be potentially responsible for this phenomenon in the case of both anesthetics.[35,54,56,80] The effect on leukocyte distribution in the capillary network is also demonstrated by sevoflurane, which to a significant degree reduces the absolute number of leukocytes rolling through the lumen of postcapillary venule and increases the leukocyte deposits in the extravascular space.[81,120]

It was explicitly proven that halogen anesthetics directly affect the production of prostacyclin (PGI_2), both in the endothelial and in the platelet formation. It was proven that sevoflurane, but not isoflurane, has a significant inhibiting effect on PGI_2 production by the vascular endothelium. The effect of these last two anesthetics is also expressed by inhibition of neutrophil apoptosis. In this context, sevoflurane is characterized by a strong inhibitory effect. Such effects of halogen anesthetics may have their consequence in postoperative leukocytosis, which was observed in a clinical setting.[103,152]

Other Anesthetic Agents

Barbiturates show a specific effect on microcirculation. Hershey et al. studied the effect of thiopental on network microcirculation in dogs. They found that thiopental causes a dilation of arterioles and vivid blood flow in the venules upon moderate anesthesia. On significant deepening of anesthesia, maximum arteriole dilation and very slow venule blood flow were observed.[76] Longnecker et al. studied the effect of pentobarbital or thiopental in peritoneal injections on the behavior of the second-order arteriole (according to nomenclature: A-2) sized 35–45 μm and the venule sized 70–100 μm in bat wing muscles. Pentobarbital, urethane, and chloralose caused a reduction in mean arterial pressure with simultaneous dilation of arteriole A-2.[99,101] The effect of subanesthetic and anesthetic doses of ketamine on the function of microcirculation components in bats was also studied. Ketamine caused dilation of arteriole A-2 by 25% without noticeable changes in the caliber of the postcapillary venule, with unchanged values of MAP.[102]

Holzmann et al. assumed that propofol induces blood redistribution phenomena at the level of skeletal muscle microcirculation, increasing the blood flow through the collecting venules. The cited authors provide no explanation for this phenomenon.[16]

This phenomenon is explained by an experiment conducted later. Using optical Doppler flowmeter to measure the erythrocyte flow, a slight increase in the erythrocyte blood flow in all studied arterioles and a slight decrease in the erythrocyte blood flow in the collecting venule were observed on propofol infusion.[92] The increase in erythrocyte flow rate was accompanied by a successive decrease in vessel diameters and significant drop in the number of capillaries participating in blood flow. These three phenomena were responsible for the transfer of blood from precapillary to postcapillary areas of the muscle, as observed in the earlier studies by Holzmann et al.[16] An important role in this process is played by a significant reduction of the number of capillaries participating in blood flow during propofol infusion.[14,92] The result of this phenomenon is ischemia of certain areas of the skeletal muscle, which in consequence must lead to local metabolic acidosis. Infusion of propofol is responsible for causing a dramatic reduction in the number of leukocytes and lymphocytes

rolling through the lumen of the postcapillary venule. At the same time, the number of leukocytes and lymphocytes adherent to the endothelium and crossing outside the lumen of the postcapillary venule increased significantly. These observations were accompanied by a significant increase in the postcapillary venule endothelial edema marker.[14,92] The adhesion ability of leukocytes is proof of their increased chemotactic activity and of the flow rate reduction at the microcirculation level.[9,15] This process explicitly explains the drop in the number of leukocytes rolling through the lumen of the postcapillary venule. These leukocytes are a potential deposit of important inflammatory mediators, which may be chaotically activated at any moment, becoming a cause of a generalized uncontrolled inflammation process and formation of reactive oxygen species. A "time bomb" was, therefore, deposited in the microcirculation of the studied muscle, and the consequences of the bomb's detonation may have calamitous effects if such phenomena would pertain to microcirculation of other important factors.[92] A sudden drop in the number of rolling lymphocytes indicates a well-pronounced immunological reaction inhibiting the cellular activity, occurring in the presence of propofol. In a clinical setting, an increase in the amount of blood lymphocytes was observed during propofol infusion; the lymphocytes were identified as T-helper cells. This phenomenon was considered to be desirable.[18] Contrary to the results obtained in a population of healthy volunteers, propofol in intensive care patients was responsible for a reduction in lymphocyte proliferation at drug blood level values close to those used in a clinical setting.[3] Therefore, special consideration must be given in cases of choosing propofol as the anesthetic to be used during organ transplant and tissue or free flap transfer, with simultaneous use of an immunosuppressive drug setting.[23,24,92,93]

A few general conclusions can be made on the basis of the presented findings. In many cases, the arteriole condition and diameter correlate with variable values of mean arterial pressure, cardiac output, or variable heart rate. However, no reaction of the other microcirculation component – the postcapillary venule – is often observed on administration of anesthetics. However, it must be noted that the depth of the anesthesia plays an important role here. Many reports ignore important issues such as capillary behavior during anesthesia, which deals with determining the capillary's ability to maintain blood flow in a defined area of the studied muscle or cutaneous flap. Common reasons for this are technical shortages and poor underestimating of the importance of this problem. Analyses regarding the affects of anesthetics on individual morphotic blood elements, especially the leukocyte system in the postcapillary venule, are frequently omitted in research. Yet microsurgical experience shows that it is often a critical point of microcirculation, at which the fates of operated tissues are decided.[141,143] A frequently asked question regarding the explanation of the situations in which the mean arterial pressure values do not correlate at all with the individual A-1, A-2, A-3, A-4 arteriole diameters remains unanswered. It is quite common that only one technique used for microcirculation analysis – currently the laser Doppler flowmeter – is considered sufficient and conclusive. This certainty is completely unjustified in the light of the doubts presented here. The problem of vascular endothelium condition during prolonged anesthesia is not fully documented in the literature. Can the anesthetics directly and indirectly affect the endothelial activity and can they – to a lesser or greater extent – damage its delicate structure by different mechanisms at the cellular level?

The above considerations show that anesthetics are responsible for the course of the treatment in as large degree as the surgery and the surgical technique. Anesthesia, being a therapeutic process, may significantly influence the final outcome. Therefore, the appropriate choice of an anesthetic can play a crucial role in achieving final therapeutic success.

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51

Experimental Composite Tissue Transplantation Models

Maria Siemionow and Serdar Nasir

Summary

Advanced microsurgical techniques have allowed performing different composite tissue allotransplantations (CTA), although immunology and pharmacotherapy still have many unresolved questions. Different CTA models were developed to explore and answer these questions during the past 25 years. Most experimental transplants to date have been performed in rats, and the immune system of the rats has fundamental differences from that of the humans, such as the lack of expression of class II antigens on the endothelial cells.[7] Over the past 20 years, we have developed 17 CTA models of the rat in our laboratory in an effort to characterize differences in the mechanism of graft acceptance and rejection. These CTA models were tested to (1) determine the mechanism of CTA acceptance and rejection; (2) determine the immunologic pattern of responses of different tissue types in CTA; (3) evaluate the immunologic response of transplant grafts of different dimensions and skin components of the most antigenic of CTA tissues[10]; (4) test the effects of vascularized bone marrow transplantation (VBMT) on immunotolerance; (5) develop new immunosuppressive and immunomodulatory treatment protocols; and (6) correlate histological, morphological, and functional responses of CTA with different immunosuppression protocols. We overwrite CTA models according to the following classification: vascularized bone marrow transplant models, vascularized skin allograft models, composite allograft transplantation models, and other models (Tables 51.1 and 51.2).

Abbreviations

CsA	Cyclosporine-A
CTA	Composite tissue allotransplantation
SVC	Superior vena cava
VBMT	Vascularized bone marrow transplantation

Vascularized Bone Marrow Models

Vascularized bone marrow transplantation may be a component of composite tissue allograft (CTA) transplants; and there is evidence that bone marrow cells are tolerogenic and may induce chimerism. Over the years, different models of VBMTs have been introduced and are outlined below.

Hind-Limb Transplant Model

Limb transplantation was the first VBMT model for this purpose. Black et al.[5] achieved the first

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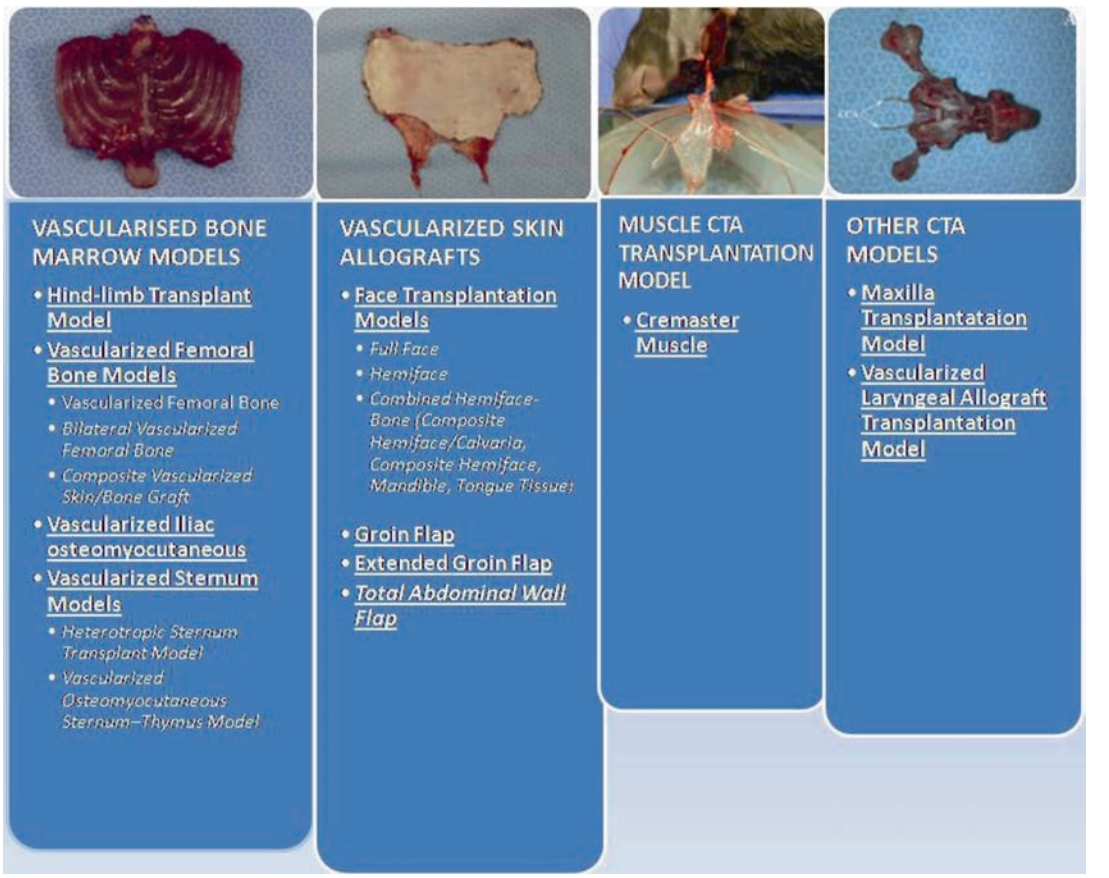
Table 51.1. Classification of experimental CTA models.



successful semiallogenic limb transplantation in rats in 1982 under the protocol of long-term immunosuppression with cyclosporine-A (CsA) immunotherapy. Zhang et al.[25] developed an experimental limb transplantation model in mice in 1999. We were the first to report tolerance induction in limb transplants across MHC.[15]

Surgical technique: A limb tissue allograft consisting of distal femur, knee joint, tibia–fibula, and surrounding muscle and skin is harvested using a similar technique in both small[18] and large animals.[11] In the donor rat, a circumferential skin incision is made in the proximal one third of the femur. The femoral artery and vein are dissected, clamped, and transected proximal to the superficial epigastric artery. The femoral nerve is dissected and transected 1 cm distal to the inguinal ligament. The biceps femoris muscle is transected to expose the sciatic nerve. The nerve is then transected proximal to its bifurcation. The limb is amputated at the mid-femoral level. The recipient is prepared using a similar technique. The limb is amputated at the mid-femoral level. The donor limb is attached to the recipient limb by a 20-gauge intramedullary pin and a simple cerclage wire. All large muscle groups are sutured in juxtaposition. Vessels are anastomosed using end-to-end microsurgical anastomosis while a conventional epineural technique is performed for nerve coaptation (Figure 51.1).

Advantages: The limb grafts are vascularized carriers of donor bone marrow and proved to be an important model for elucidating the mechanisms of transplantation tolerance. Limb transplants are composed of many key tissues such as skin, subcutaneous tissue, neuromuscular tissue, and mesenchymal tissues such as bone, bone marrow, muscle, fascia, and cartilage, and the same tissues are a part of many clinical CTAs, including hand or face, larynx, knee, abdominal wall, and so forth.

Disadvantages: Hind-limb transplantation is technically complex and leaves the recipient with

Table 51.2. Summary of experimental CTA models.

Experimental models	Transplant pedicle	Recipient vessels
Vascularized femoral bone	Femoral artery and vein	Femoral artery and vein or abdominal aorta and vena cava inferior
Bilateral vascularized femoral bone	Abdominal aorta and vena cava inferior	Abdominal aorta and vena cava inferior
Composite vascularized skin/bone	Femoral artery and vein	Femoral artery and vein
Vascularized iliac osteomyocutaneous	Abdominal aorta and iliolumbar vein	Femoral artery and vein
Heterotropic sternum	Bilateral common carotid artery and unilateral superior vena cava	Abdominal aorta and vena cava inferior
Vascularized osteomyocutaneous sternum–thymus	Unilateral common carotid artery and external jugular vein	Femoral artery and vein
Full face	Bilateral common carotid artery and bilateral external jugular vein	Bilateral common carotid artery and bilateral external jugular vein
Hemiface	Unilateral common carotid artery and unilateral external jugular vein	Unilateral common carotid artery and unilateral external jugular vein
Composite hemiface/calvaria	Unilateral common carotid artery and unilateral external jugular vein	Unilateral common carotid artery and unilateral external jugular vein
Composite hemiface, mandible, tongue tissue allograft	Unilateral common carotid artery and unilateral external jugular vein	Femoral artery and vein
Vascularized groin	Femoral artery and vein	Femoral artery and vein
Extended groin	Femoral artery and vein	Femoral artery and vein
Total abdominal wall	Bilateral femoral arteries and veins	Bilateral femoral arteries and veins
Cremaster muscle	Bilateral femoral arteries and veins	Unilateral common carotid artery and unilateral external jugular vein
Maxilla	Unilateral common carotid artery and unilateral external jugular vein	Femoral artery and vein

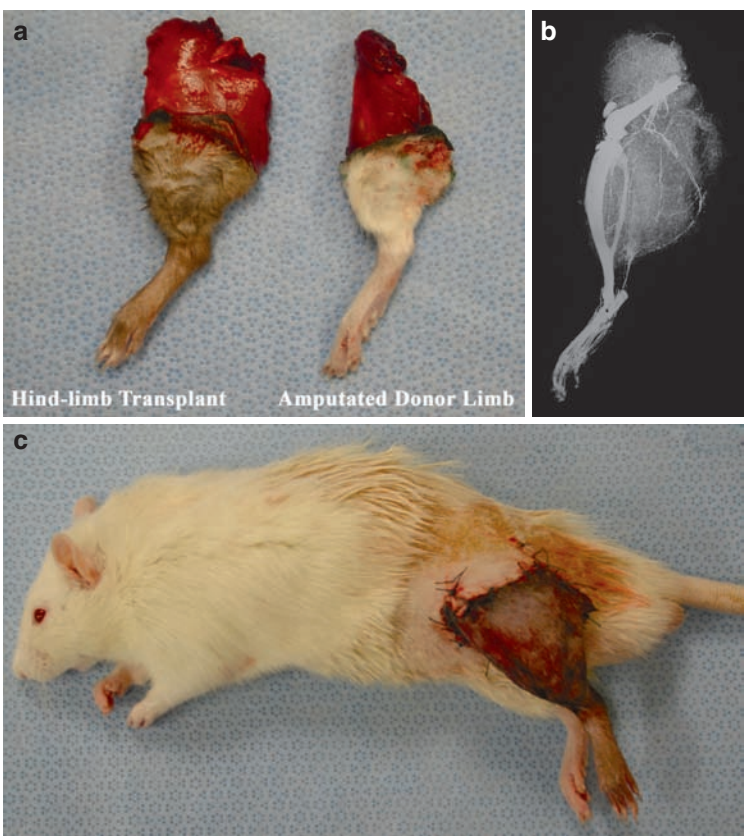


Figure 51.1. (**a**) Hind-limb model and amputated recipient and is the same size as that of the transplant. (**b**) Microangiographic view of hind-limb transplant. (**c**) Recipient rat after transplant transfer.

a functional deformity. In this complex model, half of the femur and the entire tibia from the donor are sources for VBMT. Technically, the tibia is transplanted as an intact part of the graft; in contrast, the distal half of the donor femur is rigidly fixed to the proximal half of the recipient's femur. The fixation using a pin and steel wire often damages bone marrow, and as a result the femoral bone is not intact and is not able to adequately contribute to VBMT.[4] Some investigators have reported major complications such as respiratory failure, bleeding, and thrombus formation. As a result, the published mortality rates range between 20 and 30%.[3,8]

Vascularized Femoral Bone Models

Vascularized Femoral Bone Model

Suzuki et al.[20] described a vascularized femoral bone marrow transplant model, and in this model, the femur was placed in the intraperitoneal space. Tai et al.[21] developed an extraperitoneal VBMT model that was placed in a subcutaneous pocket over the anterior abdominal wall of the recipient to obviate the surgical difficulties of intraperitoneal placement. We modified Tai et al.'s technique to reduce recipient morbidity, and no subcutaneous pockets were created and no sutures were applied for bone fixation.[1]

Surgical technique: The skin covering the thigh region of the donor limb is degloved. The femur is disarticulated from the knee joint. Anterior, posterior, and lateral thigh muscles are removed and the periosteum is preserved. The adductor muscles are gently dissected to protect the muscular branch of the femoral artery and superior genicular artery, which supplies blood to the femoral bone. Finally, the hip joint is disarticulated, medial and lateral circumflex arteries are ligated, and femoral vessels are prepared for anastomosis. In the recipient, the transplant is transferred to the intra-abdominal space[20] or groin region,[21] and abdominal aorta and inferior vena cava or femoral vessels, respectively, are used as recipient vessel (Figure 51.2a).

Bilateral Vascularized Femoral Bone Model

For augmentation of transplanted bone marrow cells, we have made further modifications by creating bilateral femoral bone allografts based on the common pedicle of the abdominal aorta and inferior vena cava.[2] In this technique, VBMT must be transferred into the abdominal cavity due to insufficient space for both femoral bones in the groin region of the recipient (Figure 51.2b).

Composite Vascularized Skin/Bone Graft Model

To augment the antigenic load of a vascularized femoral bone model, we simultaneously harvested the groin skin flap based on the common arterial–venous pedicle. Furthermore, the skin component of the flap facilitated monitoring of the viability of the vascularized bone as well as providing visual indications of rejection. In this model, the vascularized femoral bone is elevated using the standard technique described above while preserving the superficial epigastric artery during groin flap elevation to protect its blood supply (Figure 51.2c).

Advantages of vascularized femoral bone models: Transplantation can be made safely, easily, and without complications. In this transplantation, no nerves, muscles, or bones are transected, and the recipient can use the limb immediately after transplantation without significant morbidity.

Disadvantages of vascularized femoral bone models: Transplant viability cannot be monitored in vascularized femoral bone models without the skin component, because vascularized bone is embedded under the skin and is not able to be visualized for assessments. Bone marrow cell load in this model is lower compared with the limb transplant model (Table 51.3). Vascularized femoral bone transfer does not simulate CTA transplant, because it contains only one tissue component and a lower antigenic load when compared with the more characteristic types of CTA.

Vascularized Iliac Osteomyocutaneous Transplant Model

To monitor the tolerogenic effect of VBMT, an experimental model should attain both a high antigenic load and be associated with low mortality and morbidity. To achieve this aim, we introduced an iliac osteomyocutaneous VBMT model that contains iliac bone as well as a large abdominal wall muscle component and large skin component.

Surgical technique: In the donor rat, a square-shaped skin island, measuring approximately 8 × 8 cm, is marked over the flank and lateral

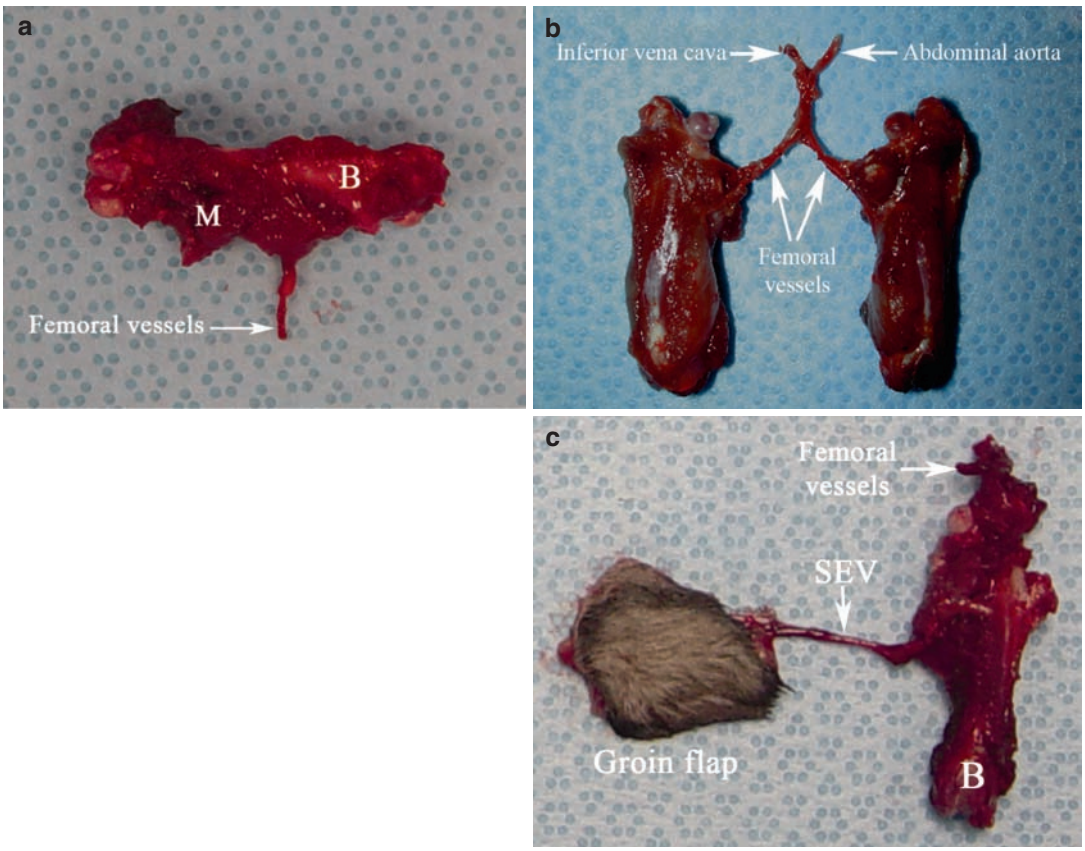


Figure 51.2. Vascularized femoral bone models. (**a**) Vascularized femoral bone. (**b**) Bilateral vascularized femoral bone. (**c**) Composite vascularized skin/bone model. M, muscle component, B, femoral bone. SEV, superficial epigastric vessels.

aspect of the hip of the rat. The skin paddle is raised as an island flap based on the cutaneous perforator of the iliolumbar vessels. Incising the abdominal muscles adjacent to the iliac bone, the vascular pedicle of iliolumbar vessels is exposed and dissected to their origins from the abdominal aorta and the inferior vena cava. Iliac bone osteotomy is performed, and whole iliac bone measuring 3 × 2 cm is elevated with the flap. All components of the iliac osteomusculocutaneous flap, including the whole iliac bone, adjacent gluteus maximus, iliacus, lateral abdominal wall muscles, and the overlying skin island, are elevated on the iliolumbar vascular pedicle as a single unit (Figure 51.3).To avoid the smaller caliber iliolumbar artery, the abdominal aorta is used for continuity of pedicle and is transected at the level below the renal artery. However, the iliolumbar vein is used as the pedicle vein, because its diameter is adequate for microvenous anastomosis. In the recipient rat, the groin region is preferred as a recipient area, and the flap pedicle is anastomosed to femoral vessels using end-to-end technique.

Advantages: Iliac bone contains a rich supply of bone marrow cells, and thus it is a viable model for VBMT studies, with a straightforward surgical approach in the recipient and lower rat mortality when compared with limb transplant and sternum models. An iliac osteomusculocutaneous transplant with the largest skin component may present a model of a high antigenic load compared with previously described VBMT models (Figure 51.4) (Table 51.3). Furthermore, it successfully simulates clinical CTA models.

Disadvantages: In our study, we found a lower chimerism level in peripheral blood despite the high bone marrow load found in transplanted bone. In addition, the difference in vessel size

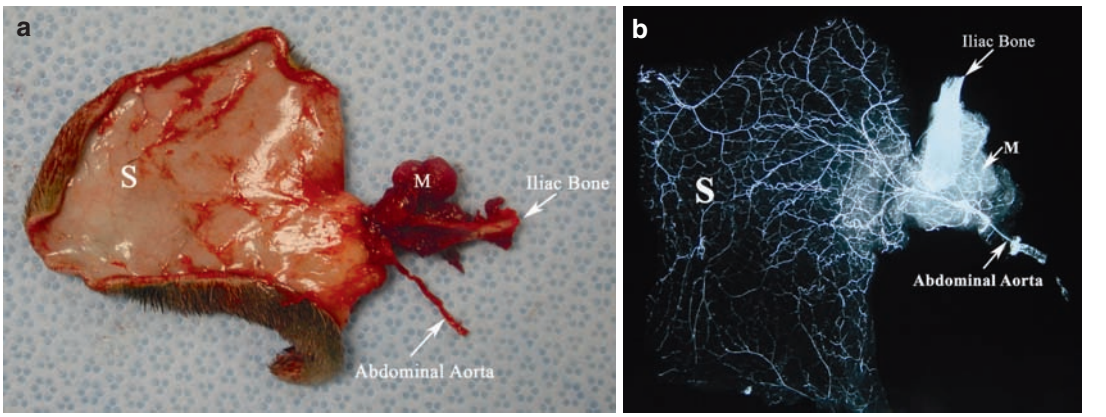


Figure 51.3. (**a**) Vascularized iliac osteomyocutaneous model. (**b**) Microangiographic view of transplant components. M, muscle component, S, skin.

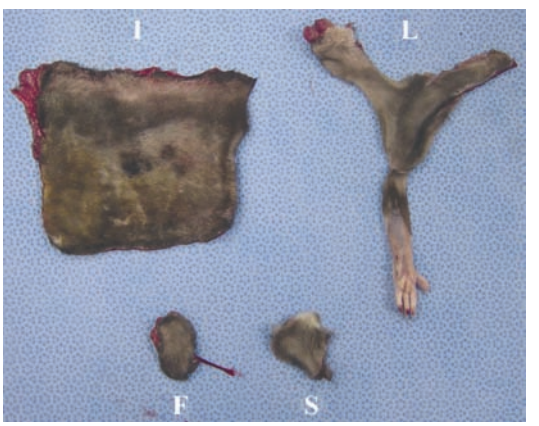


Figure 51.4. Comparison of skin islands of vascularized bone marrow models. Iliac osteomusculocutaneous model has the largest skin island, which is followed by hind limb. I, iliac osteomusculocutaneous, L, limb, F, vascularized femoral bone, S, osteomyocutaneous sternum–thymus model.

Table 51.3. Comparison of VBMT models

VBMT models	Diameter of skin island (cm^2)	Weight (g)	Bone marrow cell populations (10^6)
Iliac	57.96	15.70	25
Limb	24.12	18.84	48.75
Femur	5.4	14.80	50
Sternum	5.04	2.4	7.5

between the transplant pedicle and recipient may produce additional challenges during microsurgical anastomosis.

Vascularized Sternum Transplantation Models

Heterotropic Sternum Transplant Model

Santiago et al.[14] described a vascularized sternum transplantation method in which the transplant was transferred into the abdominal cavity as a heterotopic graft.

Surgical technique: In the donor rat, the skin is incised in the midline from the suprasternal space to just above the pubis, and pectoral muscles are divided from the thoracic wall. After entering the peritoneal cavity, the diaphragm is incised, and the thoracic cavity is entered under the manubrium sterni. From the xiphisternum, the ribs on both sides of the sternum are divided 0.5 cm away from the lateral sternal border up to the clavicle. The entire sternum is then turned cephalad, and the superior vena cava (SVC) is dissected on both sides up to the formation of the brachiocephalic veins. To create a single venous orifice for anastomosis, one SVC is divided distal to the preserved vein and venovenous end-to-side anastomosis is carried out between the left and right SVC. The right and left carotid arteries and subclavian arteries distal to the

branch of the sternum are ligated and divided. The arteries supplying the sternum are taken with the aortic arch, which is closed distally with a ligature, leaving the proximal end open for the arterial anastomosis. Once the artery and vein are ready, the sternum is separated from the clavicles and soft tissue with a sharp dissection. All raw areas on the graft (cut ends of ribs, manubrium, and divided muscles) are cauterized with caution to control bleeding after transplant transfer (Figure 51.5a). In the recipient, for vessel preparation, the abdomen is opened, followed by isolation of the infrarenal aorta and the inferior vena cava. Sternal transplant pedicle is anastomosed to the recipient vessel the using end-to-end technique.

Vascularized Osteomyocutaneous Sternum–Thymus Model

To avoid the disadvantages of the heterotopic sternum transplant model and increase antigenic load, we have recently developed a vascularized osteomyocutaneous sternum–thymus model in our laboratory.

Surgical technique: In the donor rat, the skin island and pectoral muscle on the thoracic cage are isolated from the heterotopic sternum transplant, and they are reached through the thoracic cavity by transecting the diaphragm. Brachiocephalic vessels are ligated in the axilla. The ribs on both sides of the sternum are divided from the lateral sternal border up to the clavicle, and the sternum is turned cephalad. A Vessel connection between the thymus and sternum is protected, and the distal ends of the thoracic aorta and inferior vena cava are ligated below the heart. A different method, unique from the previously described model, does not involve dissection of the superior vena cava, and the common carotid artery and external jugular vein are prepared as a transplant pedicle on one side of the neck region. After pedicle preparation, the osteomyocutaneous sternum–thymus composite transplant is divided from the clavicles and soft tissue by sharp dissection (Figure 51.5b, c). In the recipient rat, the groin region

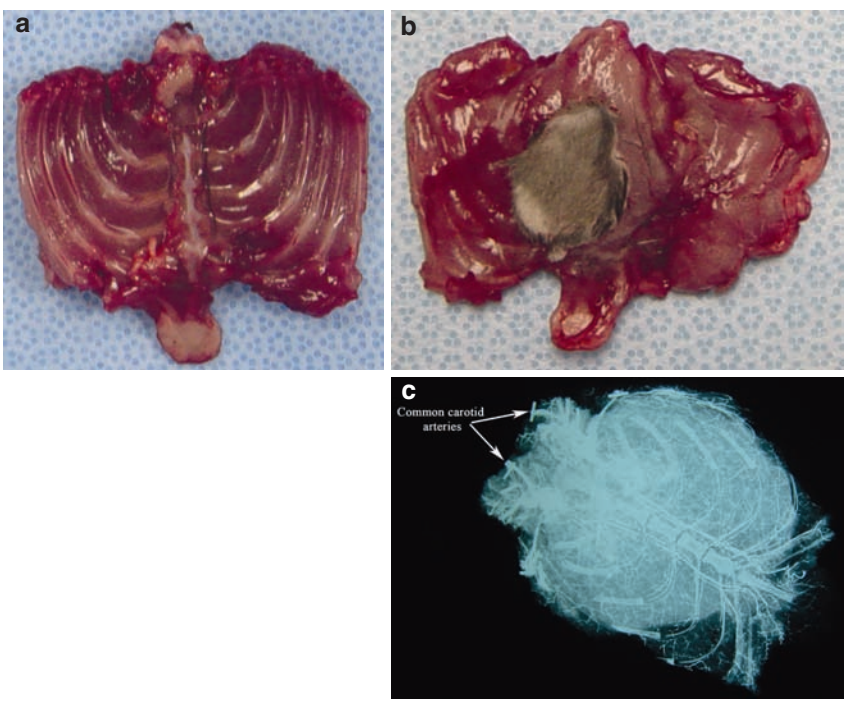


Figure 51.5. Vascularized sternum transplantation models. (**a**) Vascularized sternum. (**b**) Vascularized osteomyocutaneous sternum–thymus. (**c**) Vascular network of vascularized osteomyocutaneous sternum–thymus in the microangiographic view.

and femoral vessels are prepared for transplant transfer, and an end-to-end technique is used for microanastomosis.

Advantages and disadvantages of vascularized sternum transplantation models: The sternum is a marrow-rich bone and a valuable VBMT model. A salient disadvantage is that the vein used in the vascular pedicle preparation before flap transfer requires an extra microvenous anastomosis between the right and left SVC. Furthermore, pedicle anastomosis with recipient vessels in the abdominal region requires advanced microsurgical skills. Another disadvantage of this model is that assessment of the heterotopic sternum transplant during allotransplant follow-up is impossible due to its intra-abdominal placement. The vascularized osteomyocutaneous sternum–thymus model allows avoidance of a laparotomy and permits externalization of the transplant for convenient observation during the follow-up period, because the transplant is transferred to the groin region. This model presents a high antigenic load due to multiple tissue components, including skin, muscle, and thymus, as well as vascularized bone marrow. Although there is an increased antigenic load in this model, it is still lower when compared with that of the limb transplant model, which has large skin, muscle, and bone components. We found that this model had the lowest bone marrow cell load compared with those in other VBMT models (Figure 51.6) (Table 51.3).

Vascularized Skin Allografts

Most of the experimental CTA models contain a skin component that induces strong immunologic response from the host. To resolve this immunologic challenge, different vascularized skin allograft transplants are described.

Face Transplantation Models

Recently, we have described for the first time full-face and hemiface allotransplantation in a rodent model.[6,17] Subsequent studies included incorporation of different cranial bones (calvarial bone[24] and mandible[9] into the facial transplants as a source of VBMT without recipient conditioning.

Full-Face Transplantation Model

Surgical technique: In the donor rat, first, a median skin incision is made in the neck region. Branches of the external jugular vein between the cranial base and above the clavicle are ligated and the vessel dissected from the subcutaneous tissue. The Sternocleidomastoid muscle is divided and common carotid artery dissected from the vagus nerve. The stylohyoid and omohyoid muscles and the greater horn of the hyoid bone are resected. The full-face transplant is supplied by two main vessels, the facial artery and the posterior auricular

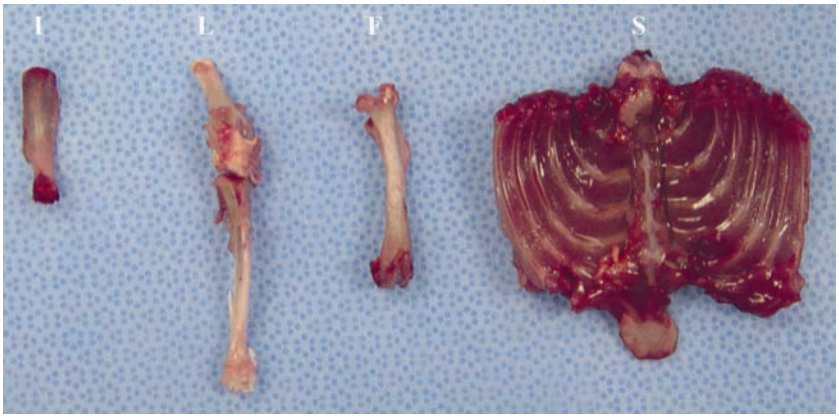


Figure 51.6. Comparison of bone compartment of vascularized bone marrow transplant models. Iliac osteomusculocutaneous and vascularized femoral bone models contain whole bone. Hind-limb transplant models include whole tibia and half of femoral bone, whereas vascularized sternum models covered near-total thoracic cage. I, iliac osteomusculocutaneous, L, limb, F, vascularized femoral bone, S, osteomyocutaneous sternum–thymus model.

artery, which are branches of the external carotid artery. Dissection of these vessels is the most important part of the face harvesting procedure. After ligation of the internal carotid artery and the cervical branches of the external carotid artery in the neck region, the facial artery is dissected up to the point of its emergence from the external carotid artery. This dissection is continued to the nasal region, which is called the angular artery at this level. After circular neck incision, to facilitate dissection of posterior auricular artery, the ear is detached at the level of the external cartilaginous auricular canal, and this vessel is elevated with skin from the ear. At the same level, the branches of the posterior facial vein, which continue as the external jugular vein, are divided from the pterygoid and pharyngeal vein plexuses. Then the facial allograft transplant, which includes ear, neck, and facial skin without eyelid and nose skin, is completely freed from all surrounding tissues based on the bilateral common carotid artery and external jugular vein. The dissections of the branches of the facial or trigeminal nerve are optimal. In the recipient, the same size of facial skin is excised. Bilateral common carotid arteries and external jugular veins of the full-face transplant pedicle are anastomosed with the recipient bilateral common carotid arteries and external jugular vein respectively, using the end-to side technique (Figure 51.7a, b, c).[17]

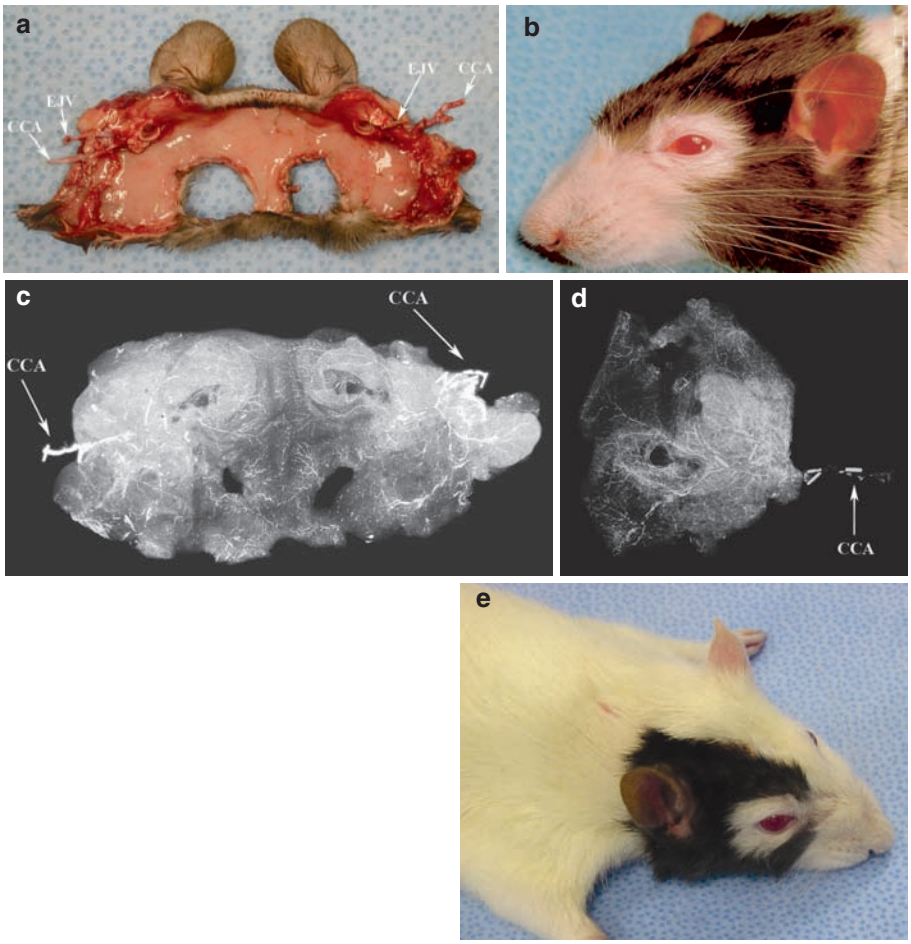


Figure 51.7. Face transplantation models. (**a**) Harvested full face (**b**) Transplanted full-face model. (**c**, **d**) Microangiographic views of full- and hemiface transplants. (**e**) Transplanted hemiface. CCA, common carotid artery; EJV, external jugular vein.

Hemiface Face Transplantation Model[6]

Surgical technique: All surgical procedures are the same as those in the full-face transplantation. However, the transplant contains only the hemiface skin, and, therefore, preparation of a unilateral common carotid artery and external jugular vein is sufficient as a transplant pedicle. Transplant arteries are anastomosed to the recipient common carotid using the end-to-side technique, whereas the vein anastomosis is performed using an end-to-end technique (Figure 51.7c, d).[6]

Advantages and disadvantages of facial transplantation models: Full-face allograft transplantation in the rat model has higher complication and mortality rates as a result of brain ischemia and vascular complications. In this technique, the main vascular structures of the head and neck are used bilaterally on carotid arteries and jugular veins in the recipient. To avoid this hazardous disadvantage and for the purpose of reducing mortality in facial allograft recipients, we have introduced another modification to the face allotransplantation model, in which a total facial allograft flap was based on bilateral common carotid arteries and was vascularized based on the unilateral common carotid artery of the recipient.[22] In the hemiface models, arterial circulation of the brain was not significantly affected, because only one major vessel of the head and neck was used, and the flap was connected to the recipient's pedicle via the end-to-side technique. This model may be more suitable for immunologic studies instead of the full-face model, because it contains the same tissue components as a full-face model without the associated morbidity and mortality found in the full-face model.

Combined Hemiface–Bone Transplantation Models

In order for our experimental face transplantation studies to achieve longer survival time, facial bone components were added to the hemiface models. Furthermore, we achieved face and vascularized bone transplantation at the same time, and thus we intended to examine the benefits of heterotopic bone marrow on immune tolerogenicity.

Composite Hemiface/Calvaria Transplantation Model

Surgical technique: In this model, the parietal bone of a rat is added to the hemiface transplant. The hemiface is elevated using the technique described previously. However, connections between the periosteum of the parietal bone and transplant tissues are protected, and the bone is harvested combined with a hemiface transplant. The composite flap is transferred using the common carotid artery and external jugular vein with the same microsurgical technique used in the hemiface transplant (Figure 51.8a, b).[24]

Composite Hemiface, Mandible, Tongue Tissue Allograft Model

The flap harvesting technique is the same as that in the hemiface model, and the same vessels are used to transplant the pedicle. A unique step that differs from the previous technique is whereby the hemiface is elevated in combination with both the hemimandible and tongue tissue. During transplant elevation, connections between the transplant and hemi mandible and tongue are maintained while preserving the blood supply, and these parts are harvested in combination with the hemiface transplant. The groin region and femoral vessels are prepared as recipient, and the transplant is transferred using the end-to-end technique (Figure 51.8c, d).[9]

Advantages and disadvantages of combined hemiface–bone transplantation models: Both methods are used successfully for bone marrow-based therapy studies, as demonstrated by prolonged survival time and increased chimerism levels. Furthermore, the mandibular segment of the composite hemiface, mandible, tongue tissue allograft model is suitable for prospective orthognathic surgery. However, creation of the same defect in a recipient rat in this model would make survival impossible, and thus there is no chance for orthotopic transplantation.

Vascularized Groin Flap

A groin flap (or inferior epigastric flap) is the first described experimental free flap model.[18] Although it is accepted as a vascularized skin

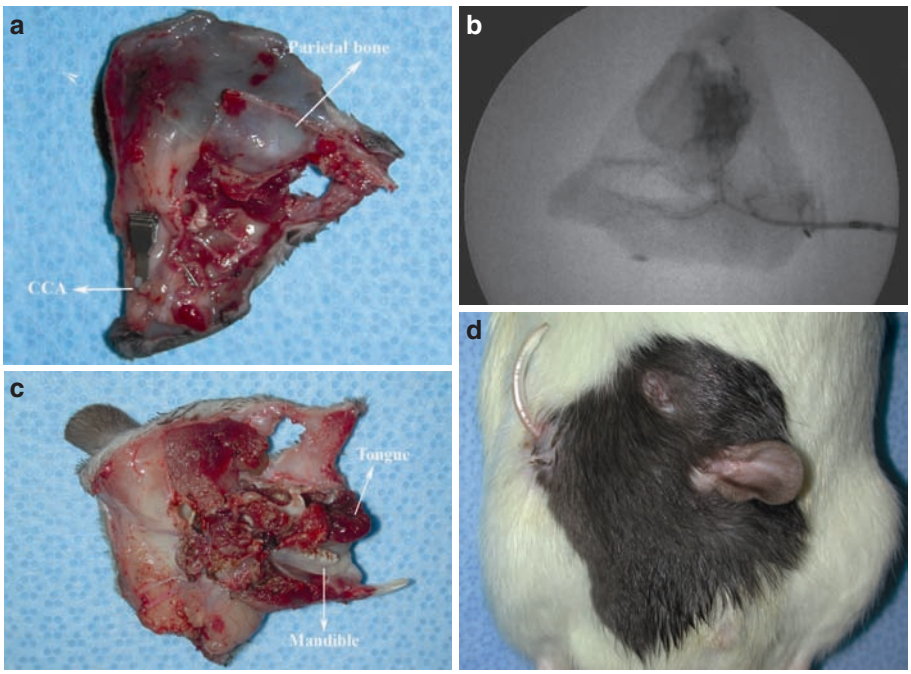


Figure 51.8. (**a**) Composite hemiface/calvaria. (**b**) Microangiographic view of composite hemiface/calvaria transplant. (**c**) Composite hemiface, mandible, tongue tissue. (**d**) Transplanted composite hemiface, mandible, tongue model. CCA. common carotid artery.

allograft, the inguinal fat pad constitutes an important part of flap volume.

Surgical technique: In the donor rat, the groin and lower abdominal skin are used as the vascularized allograft design. After the skin incision, a skin island with inguinal fat pat is elevated on the anterior abdominal wall muscle. The groin transplant pedicle has a superficial inferior vessel, which is dissected at its origin on the femoral vessels. Due to the large vessel diameter and a pedicle length, femoral vessels are ligated from the distal region of the superficial epigastric vessels' origins and divided during transplant elevation (Figure 51.9).

Advantages: This model is very useful for immunotolerance studies because of its simple surgical dissection, low rat morbidity, and mortality.

Disadvantages: When the skin island section is extended on the contralateral side and passes the abdominal midline, partial necrosis is observed, because its pedicle provides blood supply only to the same side lower abdominal and groin skin. Thus, maintaining a large skin island section is impossible using this flap.

Figure 51.9. Transplanted groin flap in recipient rat.

Extended Groin Flap Model

Although definitive data has not yet been published, it is thought that larger amounts of skin tissue in CTA transplants may increase the antigenic load. An experimental model that has a large skin component may be used to test this hypothesis. We have described such a model using an extended groin flap to provide a large skin island for transplantation study.

Surgical technique: The Skin island is planned measuring approximately over the abdominal wall and flank regions in the donor rat. The borders of the skin island are defined superiorly by the costal cage, inferiorly by the inguinal ligament, and 3 cm below the iliac spine in flank regions, respectively, medially by abdominal midline, and laterally by dorsal midline. After skin incision, elevation starts at the abdominal section on the anterior abdominal wall, and dissection is continued with flank section elevation over the lateral abdominal wall muscles. Iliolumbar vessels are found close to the upper border of the iliac bone and are cauterized and separated so that their connection with the superficial epigastric artery maintains blood supply to the flank region. After this step, the flap harvesting procedure is the same as that with the previously described groin flap (Figure 51.10).

Advantages: This model has a short operation time and minimal rat mortality. Although a large amount of skin is used, the design of this skin flap makes it possible to harvest two flaps from the same animal and thus reduces the number of necessary donors.

Disadvantage: Seromas may collect under transplant tissue because of the large dead space that underlies it. To avoid seroma complications, a few sutures may be placed between the flap and basement tissue to decrease the amount of dead space.

Total Abdominal Wall Transplant Model

In all experimental models in the rat, the total abdominal wall transplant model entails the largest skin component. We described this model to evaluate immunologic response against a large skin size.[12]

Surgical technique: In the donor rat, the skin island of this model covers the entire abdominal skin region. The border of the skin island extended from the xiphoid process and arcus costa to the pubic tubercle and inguinal ligaments and between the two anterior axillary lines. Following incision of the skin island, the total abdominal transplant was elevated over the anterior abdominal

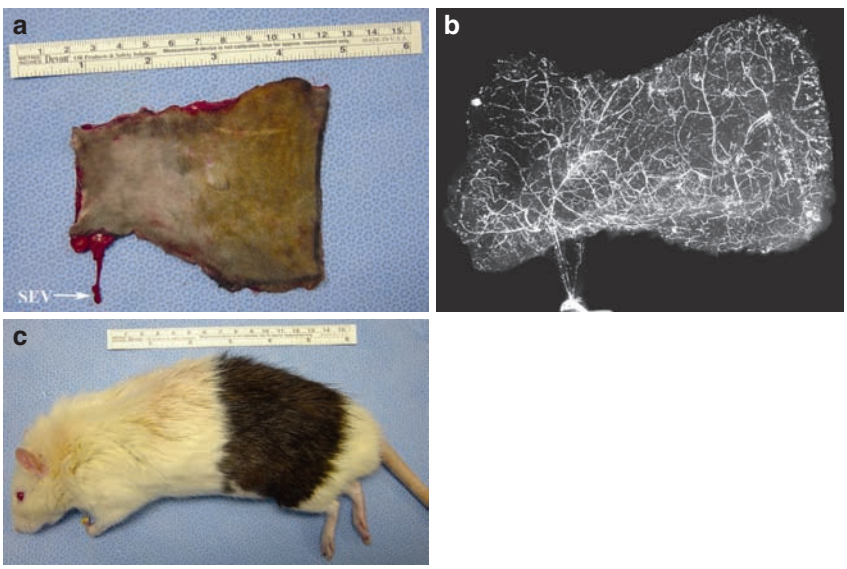


Figure 51.10. (**a**) Extended groin flap includes wide skin island, which covers groin, lower abdomen, and flank regions. (**b**) Microangiographic view of extended groin flap. (**c**) Recipient rat after transplantation after long term follow-up.

wall muscle. Pedicle dissection is the same as that with the groin flap, but bilateral superficial epigastric vessels are prepared as the flap pedicle. In the recipient rat, all of the anterior abdominal skin is harvested, and the bilateral femoral vessels are dissected for flap transfer. Bilateral flap pedicle is anastomosed to the femoral vessels on both sides using the end-to-end technique. A few fixation sutures are placed between the transplant and abdominal muscle in the midline for prevention of seroma collection (Figure 51.11).

Advantages: The surgical procedure is very similar to that in the previously described groin flap procedure, with the exception that there is a need for bilateral microanastomosis. Furthermore, the surgery required in the preparation of the large vascularized skin transplant is relatively simple. This is the first model demonstrating that an abdominal wall transplant could be used to evaluate causes of high rejection rates in clinical transplants.

Disadvantages: This model led to a higher morbidity and mortality rate compared with the groin and extended groin transplants. The large recipient skin excision needed to create the defect for transplant transfer and large tissue transfer may have resulted in severe hemodynamic imbalance in the recipient.

Muscle Transplantation Models

Cremaster Muscle CTA Transplantation Model

This CTA transplantation model was developed to study the microcirculatory changes during acute allograft rejection and ischemia/reperfusion

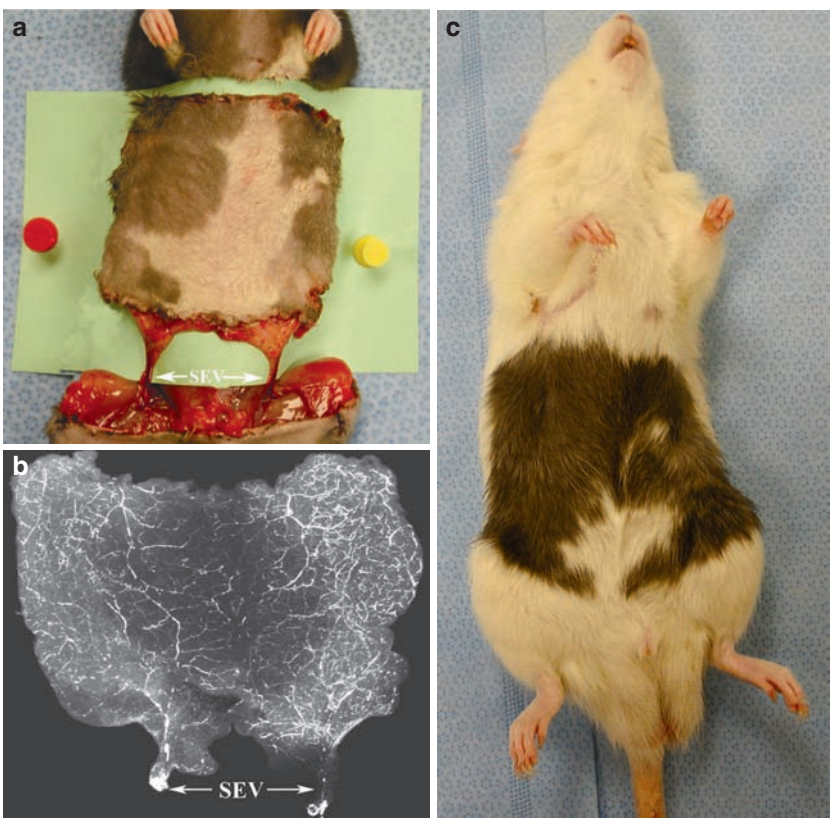


Figure 51.11. (**a**) Total abdominal wall transplant model based on bilateral superficial epigastric pedicles. Entire abdominal skin of recipient is elevated for total abdominal wall transplant. (**b**) Wide microvascular network of vascularized transplant model. (**c**) Recipient rat after transplantation after long-term follow up. SEV, superficial epigastric vessels.

injury in our laboratory.[13] This model is the first described one performed on the mouse cremaster transplant model.

Surgical technique: An anterior longitudinal skin incision is made starting from the testicle to the xiphoid in the donor rat. After identification of the cremaster muscle on its vascular pedicle, the testicular vessels are ligated and the testicular contents are extracted. The cremaster muscle is isolated as a tube flap. The pudic-epigastric branch of the femoral artery, the main feeding branch of the flap, is identified, and the side branches are ligated. The distal branch of the femoral artery and the vein are tied, and the cremaster muscle is elevated on the common iliac artery and vein. The cremaster muscle CTA model is transplanted to the neck region of the recipient, and the pedicle is anastomosed to the recipient's ipsilateral carotid artery and external jugular vein using the end-to-end technique (Figure 51.12).

Advantages: Mouse studies of pathophysiologic states have some advantages over those with other animals. Housing, maintenance, and treatment costs of mice are much less, compared with other laboratory animals. Treatment modalities applied per body weight of the animal are 5–10 times less expensive in mice than those in rats.

Disadvantages: The vessel size of the donor and recipient arteries is very small compared with that in rat, and this surgery needs advanced microsurgical skill. Mice are less tolerant to surgical trauma, blood loss, and anesthesia and have a higher mortality rate than rats. Meticulous attention should be paid to minimize blood loss during surgery.

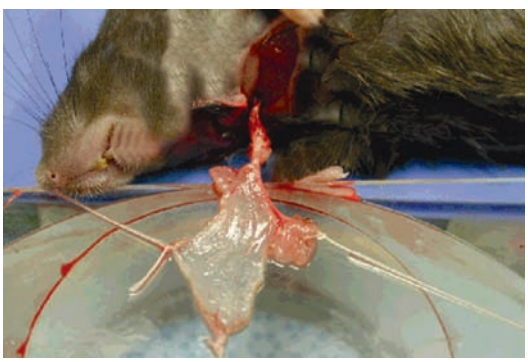


Figure 51.12. Transplanted cremaster muscle flap in the neck region of the muscle.

Other CTA Models

Maxilla Allotransplantation Model

Face bone transplantation may serve as an alternative option in clinical transplantation in the future. With this in mind, we have described a maxilla allotransplantation model to evaluate the immunologic responses and growth properties of transplanted face bones.[23]

Surgical technique: In the donor rat, after skin incision, the external carotid artery and its branches are dissected by the midline neck approach. After isolating the common carotid artery and the bifurcation, the external carotid arteries are ligated and divided bilaterally. External jugular veins are dissected and the side branches ligated from the supraclavicular region to under the cranial base. Finally, the maxilla is dissected along Le-Fort II osteotomy lines based on the common carotid artery and external jugular vein and transplanted to the anterior abdominal wall using end-to-end anastomosis with femoral vessels in the recipient rat (Figure 51.13).

Advantages: Using this model may allow for evaluation of the growth properties of teeth, facial bone, and cartilage in allografts and could provide valuable information with potential clinical applications.

Disadvantage: Orthotopic transplantation for this model is impossible due to the high mortality related to animal suffocation and nutritional problems.

Vascularized Laryngeal Allograft Transplantation Model

In 1992, Strome et al.[19] developed a vascularized laryngeal transplantation model to reexamine the potential for laryngeal transplantation.

Surgical technique: For transplant harvesting from donor rat, the skin incision extends from the hyoid bone to the sternal notch. The salivary glands, sternohyoid, and thyrohyoid muscles are excised, and the omohyoid muscle is divided to expose the internal carotid arteries. The external carotid arteries are ligated and severed above the superior thyroid arteries, with the common and internal carotid arteries left intact. Internal carotid artery and ascending pharyngeal arteries are ligated a few millimeters superior to the carotid

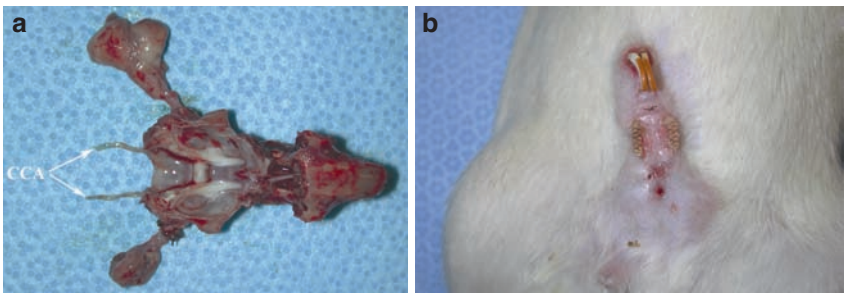


Figure 51.13. (**a**) Maxilla allotransplantation model. (**b**) Transplanted maxilla allotransplantation in the groin region.

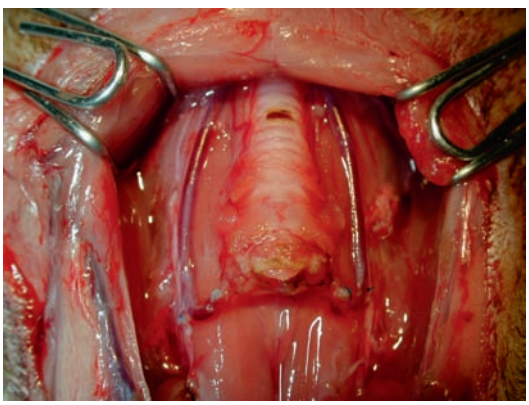


Figure 51.14. Laryngeal allograft transplantation model.

bifurcation. This procedure is performed on the other side, and the lower border of the larynx is freed by transection of the trachea at the second tracheal ring. Then the larynx is dissected from the esophagus from cephalad to the superior thyroid artery until the larynx is connected to the donor rat only by its vascular pedicle. The larynx is removed following bilateral common carotid artery transection. The recipient neck region and vessel techniques are performed as described for the donor rat, with the dissection being limited to the left side. The right common carotid artery is anastomosed to the left common carotid artery using the end-to-side technique. Next, the left common carotid artery of the donor is anastomosed end-to-side to the external jugular vein of the recipient (Figure 51.14).

Advantages: This model is suitable for large trials to determine the fundamental criteria for graft life.

Disadvantage: This procedure needs advanced microsurgical skill and has high postoperative morbidity.

Conclusion

To open a new category for CTA transplantation in the reconstructive ladder in plastic surgery practice, the experimental CTA model is a very important tool for transplantation studies.

Different models have allowed investigators to evaluate the immunogenicity and possible clinical implications of CTA transplantation using different techniques. Continued research in the development of these and other models will further delineate the role of CTA transplants in plastic and reconstructive surgery.

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52

Clinical Experience with Hand Transplantation

Chad R. Gordon and Maria Siemionow

Summary

Since 1998, 40 hand transplants (HTs) in 29 patients have been performed in countries such as France, United States (US), China, Austria, Malaysia, Italy, Belgium, Poland, and Spain. Now, with recent improvements in immunosuppression, the clinical applicability may expand, and the ethical obstacles may soon be overcome, allowing multiple US institutions to perform hand allotransplantation. Therefore, the objective of this chapter is to provide a comprehensive review of hand transplantation and to assist multidisciplinary teams in creating an Institutional Review Board (IRB) protocol for their hospital. Using a variety a published reports, a comprehensive literature review of hand transplantation was performed in May 2008. Due to its inherent complexity, hand transplantation should be attempted only at institutions capable of orchestrating a specialized multidisciplinary team, and the IRB protocol used should be extremely detailed and individualized. Without question, further research is warranted, and surgeons and scientists alike have yet to identify the clinical role of hand transplantation.

Abbreviations

US	United States
IRB	Institutional Review Board
HT	Hand transplant
U/l	Unilateral
B/l	Bilateral
IRHCTT	International Hand Transplant and Composite Tissue Transplant Registry
HTT	Hand transplant team
OPO	Organ Procurement Organization
GVHD	Graft-versus-host-disease
HLA	Human leukocyte antigen
EMG	Electromyography
PR	Public relations
CMV	Cytomegalovirus
EBV	Epstein–Barr virus
HIV	Human immunodeficiency virus
CT	Computed tomography
MRI	Magnetic resonance imaging
U/S	Ultrasound
ICU	Intensive care unit
UW	University of Wisconsin
STSG	Split-thickness skin graft
PTD	Post-transplant day
ROM	Range of motion
EPM	Early protective motion
PCA	Patient-controlled apparatus

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ASA	American Society of Anesthesiologists
ESRD	End-stage renal disease
CTA	Composite tissue allotransplantation
TCR	T-cell receptor
MMF	Mycophenolate mofetil
PTLD	Post-transplant lymphoproliferative disease
ATG	Anti-thymocyte globulin
POD	Postoperative day
QID	Four times daily
PO	per os (oral route)
fMRI	Functional magnetic resonance imaging
MCP	Metacarpophalangeal
HTSS	Hand Transplant System Score
IRS	International Registry Score
MRC	Medical Research Council
ADL	Activities of daily living
ASSH	American Society for Surgery of the Hand
DASH	Disabilities of the arm, shoulder, and hand Questionnaire
IRSS	Hand Transplantation Comprehensive Outcome Questionnaire
HTC	Hand transplant coordinator

Introduction

The first documented attempt at unilateral hand transplantation (HT) was performed in Ecuador (1964). Unfortunately, the hand allograft was removed in 3 weeks due to rejection (limited immunosuppression available). The world's first modern day unilateral (U/l) hand allotransplantation was performed in Lyon, France (September 1998). The world's first bilateral (B/l) hand transplant (HT) also took place in France (January 2000). Since that time, 40 hand transplants have been performed in 29 patients, in countries such as France, United States (US), China, Austria, Malaysia, Italy, Belgium, Poland, and Spain (Table 52.1).[1] In fact, the most recent patient (15 years old) received B/l hand transplantation in Valencia, Spain, in November 2007.[2]

Table 52.1. Recent timeline of unilateral/bilateral hand transplants performed around the world.

Date of operation	Unilateral	Bilateral
September 1998	Lyon, France	
January 1999	Louisville, Kentucky (US)	
September 1999	Guangzhou, China	
January 2000	Nanning, China[a]	Lyon, France
March 2000		Innsbruck, Austria
May 2000	Kuala-Lumpur, Malaysia	
September 2000		Guangzhou, China
October 2000	Milan, Italy	
November 2000	Nanning, China[a]	
January 2001		Harbin, China
February 2001	Louisville, Kentucky (US)	
October 2001	Milan, Italy	
June 2002	Bruxelles, Belgium, Harbin, China	
July 2002	Nanjin, China	
October 2002		Harbin, China
November 2002	Milan, Italy	
February 2003		Innsbruck, Austria
April 2003		Lyon, France
June 2006		Innsbruck, Austria
February 2006	Wroclaw, Poland	
November 2006	Louisville, Kentucky (US)	
December 2006		Valencia, Spain
February 2007		Lyon, France
November 2007		Valencia, Spain

(US) = United States[a] Two patients

In 2002, the International Hand Transplant and Composite Tissue Transplant Registry (IRHCTT) was created to provide a worldwide registry for HT. Their inaugural report was published in 2005,[3] and their second (most recent) review was in July 2007. Although hand allograft survival has shown a combined US/European 5-year graft survival rate greater than 90% (survival better than solid organ transplantation) and offers hand/forearm amputees an attractive option, factors such as expensive lifelong-mandated immunosuppression (and its accompanying adverse effects), a limited number of potential donors and restricted number of recipients, the high cost of this "nonlife saving" surgery and immunosuppression, and its extensively required rehabilitation have all limited its widespread clinical application.[4]

Hand Transplant Anatomy

Throughout the world, different levels of successful HT have been performed, ranging from wrist to distal forearm to proximal forearm. Nonetheless, the level of transplantation and relevant anatomy are predetermined by each patient's unique deficit (Figure 52.1). Pertinent anatomy for implantation includes the forearm bones (radius/ulna), nerves (median, ulnar, and radial), arteries (radial/ulnar), veins (cephalic/basilic), and tendons (flexor/extensor groups) (Figure 52.2). Interestingly, B/l HT can be performed at different levels on either side depending on a patient's preexisting disability.[5]

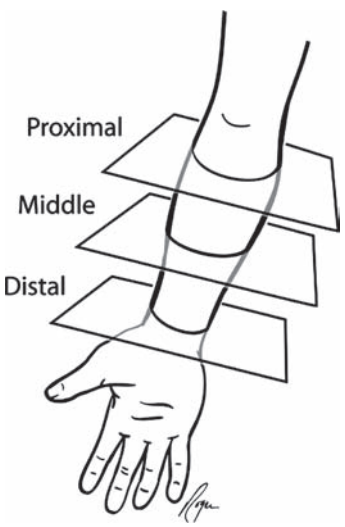

Figure 52.1. Schematic drawing representing various levels of hand allotransplantation.

Technical Description

Establishing a new hand transplant program is both challenging and rewarding. Numerous hospital specifics and team personnel must be arranged before completing a successful hand transplant.

Recipient Recruitment

National recruitment of potential HT patients can be made public by way of print, on-line advertisement, meetings/presentations to various organ/tissue donation groups, and professional communication to prospective recipients. Once identified, the potential candidate must undergo a screening phone call by a transplant coordinator to determine any preexisting contraindications. If the phone interview is successful, the patient is mailed a "screening consent form" and, in return, will be required to mail his/her medical records and photographs to the hospital. Once his/her file is reviewed by a specified member of the hand transplant team (HTT), U/l and B/l amputees must then undergo "medical screening", which includes a complete physical and psychiatric evaluation. All subject data will then at this time be evaluated by the HTT "patient selection committee."

Eligible amputee(s) will then be invited to the hospital to review an informed consent. For those still interested, enrollment into a Hand Transplant education program will be mandatory (in order to fully comprehend the entire procedure including immunosuppression, potential risks, complications, etc.) before completing the consent form. Patients eligible for involvement will then undergo official screening for all required pretransplant tests, will be waitlisted for HT in cooperation with the local organ procurement organization (OPO), and a search will be initiated for an appropriate donor. In addition, a special information program will be needed to acknowledge families in the local area of their ability to donate their loved ones' hand(s). Also, as suggested by the Louisville

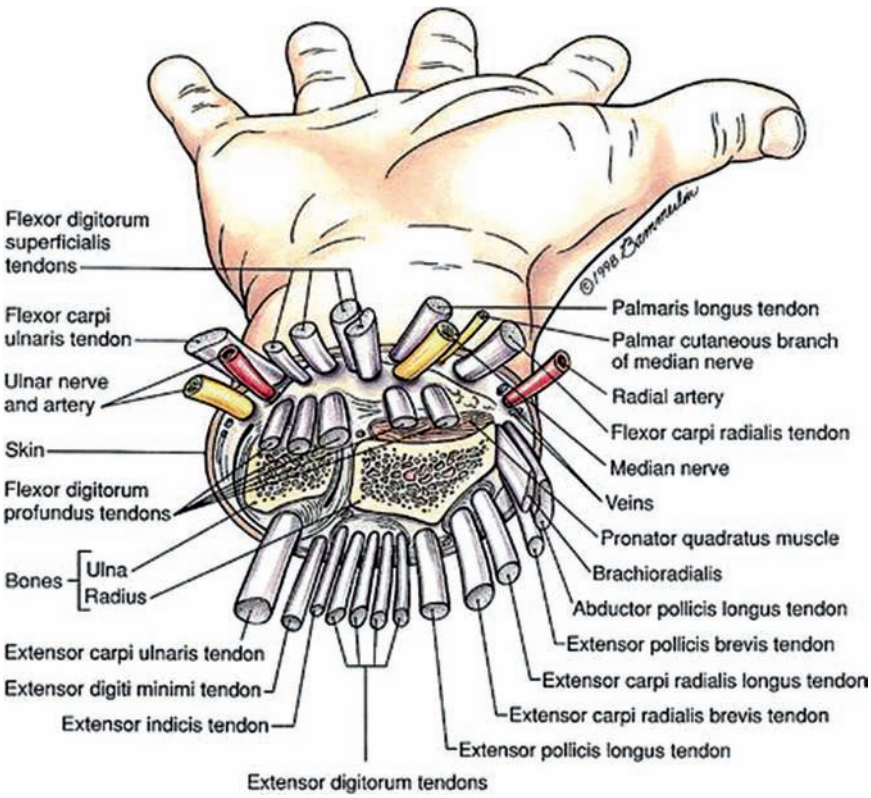

Figure 52.2. Pertinent hand allograft anatomy for transplantation. (Reproduced with permission from the Kleinert, Kutz and Associates Hand Care Center, Jewish Hospital and University of Louisville.).

team, the hospital should offer all donor families the option for prosthesis insertion to allow an open casket funeral.[6]

Meanwhile, your OPO will approach all potential families in connection to any recently pronounced "brain-dead" patients. The OPO will use the inclusion/exclusion criteria initiated by the HTT. Each donor family will be assured of complete confidentiality during the entire process, and the hospital should pay for all donation-related expenses incurred following declaration of "brain death." Once a family is in agreement with hand donation, the OPO will organize either U/l or B/l hand procurement to be performed by the HTT. In the case of a local donor, the HTT will perform the procurement within your main hospital. For all other instances, the HTT will need to arrive to and from via air travel and follow procurement guidelines stated within your protocol. Once the procurement process starts, the designated HTT Coordinator will immediately contact the planned recipient and set up rapid transportation to the hospital.

Consent Process

All potential risks and complications must be fully disclosed to the patient and his/her family, including, but not limited to, additional surgery, graft failure/rejection, immunosuppression-related complications, metabolic abnormalities, cancer, graft-versus-host-disease (GVHD), and/or death.

Consent Details

1. The consent should be written and videotaped.
2. It must address in detail pre-, intra-, and post-operative risks.
   a. Graft failure/rejection
   b. Death

c. Immunosuppression-related complications
d. Immunosuppression protocol and compliance importance
e. Opportunistic infections
f. Cancer
g. Metabolic complications
h. Psychosocial risks
i. Available alternatives (i.e., prosthesis)
j. Salvage strategies in the event of failure/rejection

3. If possible, it must be obtained in the presence of his/her social support system.

Preoperative Recipient Evaluation

A complete preoperative evaluation is needed to optimize proper patient selection. We have learned from previous hand transplants the devastating consequences of poor compliance, and, therefore, it is our sole responsibility as surgeons to select the proper recipient.

Physical Evaluation

a. History and physical examination
b. Routine laboratory work
c. Blood typing/cross matching
d. Human leukocyte antigen (HLA) typing
e. Panel-reactive HLA antibody testing
f. Epstein–Barr virus (EBV) testing
g. CMV testing
h. Human immunodeficiency virus (HIV) testing
i. Viral Hepatitis testing
j. Toxoplasmosis testing
k. U/l or B/l upper extremity radiographic imaging (sizing for osteosynthesis)
l. U/l or B/l upper extremity angiography (to predetermine vascular anomalies)
m. U/l or B/l upper extremity electromyography (EMG)
n. U/l or B/l upper extremity nerve conduction velocity testing

Psychological Evaluation

a. Clinical interview
b. Emotional/cognitive evaluation for transplant potential
c. Decision-making capacity
d. Rorschach/Thematic Apperception tests
e. Family support/social/financial status evaluation
   - Social support system
f. Medical compliance history
   - Degree of motivation
   - Level of realistic expectations
   - Potential for psychological regression
   - Perceived body-image adaptation
   - Anticipated comfort with cadaveric hand

Ethical Evaluation

a. Patient advocate (i.e., clergy, friend) is determined preoperatively by recipient
   1. Reviews the informed consent with patient and significant other (i.e. spouse)
   2. Receives and reviews peer-reviewed publications describing all risks versus benefits of hand transplantation

Social Worker Evaluation

a. Assignment of patient to a psychologist
   1. Performs regularly scheduled and PRN psychoanalysis
b. Assignment of patient to an HT social worker
   2. Coordinates all necessitated appointments, supportive measures, and ancillary staff interactions

Medical Insurance Evaluation

a. Prior to listing the recipient onto the HT candidate list, a certified letter is written to the recipient's medical insurance company inquiring about the posthand transplant medical coverage for the following:
   1. Tissue biopsies
   2. Laboratory blood work
   3. Medication cost
   4. Rehabilitative therapy
b. All costs (including surgery, facility fees, etc.) for the first 3–6 months should be covered by the hospital.

Institutional Media

At the time of recipient identification, an immediate meeting will be scheduled between a representative from both the HTT and the Public Relations (PR) office. Afterwards, a designated PR individual is expected to schedule and attend all press conferences, personnel interviews, and any other public media sessions. In addition, all potential hand transplant candidates should

be admitted to the hospital under an alias, in order to allow full adherence to current privacy regulations and to allow optimal protection from the press.

Donor Selection Criteria

Due to the fact that HT is performed on patients with a "non-life-threatening" disability, careful prevention of transplanting any unwanted disorder, disability, or disease to the recipient is of utmost importance. Therefore, careful selection of the hand transplant donor is necessary and should follow certain selection criteria.

Donor Selection

Ideally, the HT donor and recipient should be matched based on similar blood type, race, gender, bone size, hairiness, and skin color/tone. After locating a suitable donor within reasonable distance (long travel time must be accounted for with regard to total ischemia time), matching tests (lymphocytotoxic, HLA, B- and T-cell) are performed.

Donor Inclusion Criteria

1. Documented brain death
2. Hemodynamic stability
3. Complete medical history and physical examination
4. Routine laboratory studies
5. Blood typing/cross matching
6. Human leukocyte antigen typing
7. Panel-reactive HLA antibody testing
8. Serology testing for Epstein–Barr virus
9. CMV/EBV testing
10. HIV testing
11. Viral Hepatitis testing
12. Toxoplasmosis testing
13. U/l or B/l upper extremity radiographic imaging (sizing for osteosynthesis)
14. U/l or B/l upper extremity ultrasound (U/S) or angiography (to predetermine vascular anomalies)

Donor Exclusion Criteria

1. Congenital upper extremity disorder
2. Connective tissue disorder
3. Peripheral neuropathy
4. Traumatic upper extremity deformity
5. Evidence of end-organ failure
6. History of carcinoma
7. Active smoker (>1 pack year history)
8. Younger than 18 or older than 60 years
9. Documentation of previous trauma or surgery to hand(s) resulting in limited range of motion (ROM)
10. Documentation of hand disability (motor and/or sensory deficit)

Additional Preoperative Workup

All U/l and B/l amputees on the Hand Transplant list should undergo preoperative testing to analyze the exact specifications necessary for allograft retrieval. These include computed tomographic (CT) scan (chest, abdomen/pelvis, U/l or B/l upper extremity), CT angiogram (vessel mapping), magnetic resonance imaging (MRI) (to rule out preexisting bone abnormalities), U/S (to clarify exact bone length for osteosynthesis), and EMG (nerve/muscle status). Using these preoperative findings, an individualized protocol will then be constructed for each specific HT patient and will be detailed accordingly.

In addition to an exhaustive history and physical examination allowing complete medical clearance, each patient must undergo a complete dental (rule out dental abscess) and oropharyngeal examination (rule out carcinoma). Also, all patients older than 50 years are obliged to have undergone preoperative upper/lower gastrointestinal endoscopy (to rule out undiagnosed pathology such as polyps/carcinoma) and females older than 40 years must have undergone annual mammography. For obvious reasons, a hand transplant recipient in the perioperative period found to have an undiagnosed carcinoma (i.e., breast, gastrointestinal, etc.) would become quite difficult to manage.

Hand Allograft Recovery Process

Prior to entering the operating room for hand allograft recovery, a phone call will be placed to the HTT leader. This will begin an immediate phone-call algorithm, led by the hand transplant coordinator (HTC). This involves notifying the rest of the HTT surgical team, anesthesia, tissue

typing laboratory, public relations, and the transplant intensive care unit (ICU) nursing staff.

After all parties have been alerted, the hand procurement team will need to work in concert with all organ transplant teams involved with the procurement. Exact details, with regard to the organ/hand harvest sequence and the clamping of the aorta, need to be discussed preoperatively. In 2007, it was reported that 50% of the transplanted hand allografts had been harvested in the operating room before solid organ retrieval (i.e., liver, kidneys, etc.) and that 50% of the hand allografts were procured after organ harvest had already been completed. The Louisville group recommends dissecting the hand last in an unstable patient (after all other organs have been dissected) and then retrieving the donor graft before the cross-clamping of the aorta.[6] An upper extremity tourniquet is used for hemostasis. University of Wisconsin (UW) solution is used primarily for cold flush and limb preservation. The distal upper extremity is wrapped in moist sterile gauze for transport. The distal limb(s) should be kept on ice (≈4°C) in a secure, sterile container (supplied by the OPO) and then immediately transported via air to the hospital. While en route, the distal limb should be constantly infused with cold UW solution using a brachial artery cannula. Cold ischemia times have ranged from 30 min to 13 h (mean = 5.3 h). At the end of the harvest operation, a unilateral or bilateral premeasured prosthesis should be placed on the donor routinely for the purpose of an open-casket funeral. Obviously, this type of operation mandates a large, multifaceted two-team approach, with one team performing the U/l hand procurement (or two teams in the case of B/l), while an additional team simultaneously prepares the recipient.

Hand Transplant Procedure

While performing the graft recovery, a second team prepares the recipient's limb(s) simultaneously to minimize total ischemia time. An upper extremity tourniquet for hemostasis will also be used for transplantation. The recipient's limb is dissected carefully, identifying (and labeling) viable bone, nerve, tendon, and vessel endpoints, excising excess skin, and creating skin flaps for closure. All pertinent endpoints are cut to lengths per estimates determined by the recipient's preexisting deficit. Prior to implantation, all UW fluid is flushed from the allograft and replaced with cool lactated Ringer's (LR) solution.

Similar to replantation, a hand transplant first begins with osteosynthesis. If for some reason, ischemia time is prolonged during procurement, a vascular shunt should be placed before bone fixation. Hand allograft osteosynthesis involves both the radius and ulna, depending on the patient's level of amputation. The ulna should be fixated dorsally and the radius ventrally, using multiple eight-hole, 3.5-mm dynamic compression plates. Next, an intraoperative microscope with 3.5× magnification is used to anastomose the two main arteries (radial/ulnar), along with one or two large veins (with the option to repair some of the veins later). Tendons are then attached using either a group, an individual, or mixed method (as per the Louisville group, the Pulvertaft weave technique is optimal in comparison to an end-to-end repair).[6] Median and ulnar nerves should be repaired next, followed by the radial nerve (if possible). All remaining veins, if applicable, are then anastomosed. Skin incisions are offset by 90° to assure a tension-free closure of donor/recipient skin edges. If incomplete, an autologous split-thickness skin graft (STSG) is harvested (from a donor site with similar hairiness and tone) and used for closure. The transplanted limb is then immobilized carefully using a long arm splint, to allow physiotherapy to commence on post-transplant day (PTD) 1 or 2 (depending on patient's overall clinical condition).[5]

The main objective at this time is to minimize unwanted joint stiffness and adhesions and to achieve good ROM at the same time preventing injury to healing structures. Rehabilitation should include an "Early Protective Motion (EPM)" program, which has been shown to be favorable in previous HT patients. Exercises are adjusted daily to maintain optimal tendon lengths and tension and to allow cortical re-education (motor/sensory). A sensory exercise protocol (as described by Perfetti) is emphasized from the beginning to optimize protective sensation. Intrinsic muscle exercises begin on or after PTD 21. EMG biofeedback training and active finger movements are introduced after 1–3 months, as well as a variety of other exercises, depending on your hospital's post-transplant rehabilitation protocol. The HT recipient requires a wide array of splints, ranging from daytime and nighttime to passive and active.[5]

Pain Management

General anesthesia is used in conjunction with a preoperatively placed regional nerve block. Regional anesthesia, as pertaining to hand transplantation, may decrease an upper extremity sympathetic response and limit unwanted vasoconstriction. In addition, a locally injected anesthetic (i.e., marcaine) can be used intraoperatively.

Postoperative pain is controlled using intravenous narcotics via a patient-controlled apparatus (PCA). Once applicable, an oral pain medicine regimen is initiated using primarily a long-acting narcotic derivative. The HTT should attempt to de-escalate all prescribed pain medications in a timely fashion, in order to decrease the risk of developing physical dependence. A pharmacologist is assigned to the HTT to help oversee all pharmacological regimens, limit unwanted drug reactions and/or side effects, and to make sure dosing and serum levels are within optimal range.

Bilateral Hand Transplantation

The HT protocol is similar for B/l HT in regard to all details, except for the fact that this type of transplantation requires an even larger multidisciplinary team (ideally three designated teams) due to obvious ischemia-related time constraints. Each double-hand transplant can be performed at different levels on either side, depending on the patient's preexisting disability.[7] For example, the most recently reported B/l hand transplant (Valencia, Spain, November 2007), consisted of a proximal forearm transplant on the left (just distal to the elbow), and a middle-third forearm transplant on the right.[2]

In addition, many hand surgeons are more supportive of B/l hand transplantation, as opposed to U/l transplantation, due to its overwhelming, inherent disability. This was recently spotlighted in a recent survey conducted by Mathes et al. (2008), which reported that 78% of 473 hand surgeons surveyed support B/l amputation as an indication for hand transplantation (vs. 31% support for U/l amputation).[8] Another advantage seen with B/l hand transplantation is that the recipient is unable to compare his/her native hand versus a transplanted hand, as seen in the case of U/l hand transplantation. This seems to make rehabilitation less complicated and/or disappointing, and the patient seems to be more appreciative.

On the contrary, bilateral hand transplantation inherently carries a larger risk for all perioperative complications, such as malunion and anastomotic thrombosis. Fortunately, overall graft rejection/failure risk remains the same, since this complication is of systemic etiology.

Recipient Selection

Since 1998, a wide variety of patients have benefited from HT.[7] As per our literature review, we have assembled a novel set of inclusion/exclusion criteria for U/l and B/l HT recipient selection.

Recipient Inclusion Criteria

Absolute Indications

1. Strong desire to proceed with transplant
2. Accepts dedicating at least 2 years towards postoperative rehabilitation
3. U/l or B/l distal upper extremity amputee (HT has been performed in five nondominant hands)
4. Between the ages of 18 and 60 years (youngest, 19 years; oldest, 52 years)
5. Minimal coexisting medical illness or trauma
6. Elapsed injury-to-transplant time > 6 months (appreciate transplant?) or less than 15 years (is it necessary?)* *(Previous times range from 2 months to 22 years)
7. Displays "severe" disability secondarily to loss of hand(s)
8. Screening by an assembled multidisciplinary team (i.e., plastic/hand surgeon, transplantologist, physical/occupational (hand) therapist, psychiatrist, infectious disease specialist, pathologist, pharmacologist, competent ancillary staff, etc.)
9. Amputation level distal to elbow joint and proximal to wrist
10. Reports a "suboptimal" outcome from at least one documented attempt using a myoelectric prosthesis
11. Cardiac, pulmonary, hepatic, and renal functions within normal limits

Recipient Exclusion Criteria

Relative Contraindications

1. Current smoker* *(HT has been done in three smokers)
2. Active infection
3. CMV mismatch (positive donor and negative recipient)
4. History/current evidence of alcohol abuse
5. Insulin-dependent or Type I diabetes mellitus (hyperglycemia, infections)
6. Connective tissue disorder (vessel patency)
7. American Society of Anesthesiologists (ASA) 4 (perioperative morbidity, anesthesia limitations)
8. Younger than 18 years (maturity)
9. Older than 60 years (risk-to-benefit ratio)
10. Renal insufficiency (nephrotoxic drugs)
11. Evidence of significant cardiac, pulmonary, and/or liver disease (life expectancy)
12. Remote history (>5 years) of carcinoma

Absolute Contraindications

1. Record of poor medical compliance (rehabilitation, immunosuppression)
2. Unable to receive immunosuppression either due to geographic or financial limitations (inevitable graft rejection)
3. Unable to follow strict rehabilitation schedule (inevitable graft failure)
4. Geographical limitations precluding close follow-up (ineffective management)
5. Documented psychological disorder(s) or incomplete psychological clearance (compliance)
6. ASA 5 (perioperative mortality)
7. End-stage renal disease (ESRD) (life expectancy)
8. Evidence of metastatic carcinoma (life expectancy)
9. Acquired Immune Deficiency Syndrome or Immunosuppressed status (opportunistic infections)
10. Blindness (sight needed for early limb protection)
11. Amputation level proximal to elbow joint
12. Neuroma proximal to amputation level
13. U/l or B/l hand amputation was suicide related
14. Active cancer (with potential need for chemotherapy)
15. History of significant psychiatric disorder
16. History of documented suicide attempt(s)

Hand Allograft Rejection

There are a variety of rejection types that may occur in hand transplant patients. These include primarily hyperacute, acute, and chronic graft rejection.

Hyperacute Graft Rejection

Preoperative screening for preformed antibodies, which are usually either anti-ABO or anti-HLA I and/or II, is performed immediately before transplantation in an effort to prevent hyperacute graft rejection. In detail, an antibody screen is performed by using a mixture of donor lymphocytes and recipient serum. It was Patel and Teraski[9] who first described the utility of "donor-specific cross matching".[9] If present, hyperacute rejection usually occurs within seconds to minutes after the allograft/transplant inflow vessels are anastomosed.[10]

Acute (Immediate) Graft Rejection

To date, all HT patients have received systemic immunosuppression to prevent acute allograft rejection. Published reports show that 100% of all hand transplant patients have suffered at least one or more episodes of acute graft rejection. Although antibody-mediated (humoral) rejection is possible, T-cell-induced (cell-mediated) rejection is the common etiology of acute graft rejection in composite tissue transplantation. This typically presents with some degree of dermatitis and/or allograft edema and is screened for and diagnosed by way of punch biopsy. This typically occurs within days to months (usually within the first 6 months posthand transplant). After a graft's first acute rejection, it is rare to have further episodes of acute rejection unless the patient is noncompliant with his or her immunosuppression. Evidence has shown that those patients experiencing at least one episode of acute graft rejection are unfortunately at an increased risk of subsequent chronic allograft rejection.[10]

Chronic Graft Rejection

Due to the relatively recent clinical introduction of HT, chronic graft rejection has yet to be identified, and therefore, scientists have no effective treatment. It happens to be the most common cause of late graft failure, usually appearing months to years after surgery. Histologically, chronically rejected allotransplants display fibrosis, graft parenchyma atrophy (i.e., functional decline of a hand allograft), and a progressive arteriopathy, with little to no infiltrating lymphocytes unlike acute rejection. In addition, scientists have recently demonstrated numerous benefits with Rapamycin (Sirolimus) in regard to solid organ transplantation (i.e., kidney), and current research will define its role in CTA.[10]

Multiple hand transplant teams have used concomitant cellular bone marrow and stem cell transplantation during the post-transplant period in an attempt to achieve hematopoietic chimerism (either peripheral or central).[11] This was encouraged by Starzl and colleagues after they reported microchimerism in recipients with long-term graft survival.[12] Interestingly, none of the following studies was able to induce donor-specific tolerance (i.e., the "Holy Grail"), but there was an associated decreased incidence of acute and chronic rejection.[13]

Fortunately, there have been no reported cases of graft-versus-host disease in any CTA patients. GVHD develops when the donor lymphocytes recognize the recipient's antigens as foreign. Symptoms include fever, dermatitis, and pancytopenia. Although this phenomenon has yet to be seen within the specialty of CTA, many scientists feel that if we were to begin using HLA-matching prudently for recipient screening, then we would be able to significantly reduce the risk of developing GVHD.[10]

Immunosuppression Therapies

The first documented hand transplant was attempted in Ecuador in 1964. Due to limited available immunosuppression at this time, the transplanted hand suffered severe acute rejection and was amputated at 2 weeks post-transplant. Since then, surgeons have employed a wide range of immunosuppression therapies, which include various methods of induction (Table 52.2), maintenance (Table 52.3), and rejection (Table 52.4), as well as various drugs for opportunistic infection prophylaxis (Table 52.5).

Induction Therapy

As similar to solid organ transplantation, induction therapy is used to prevent acute graft rejection for the first 2–3 weeks post-transplant. In this case, hand transplant immunosuppression, for both induction and maintenance, is most similar with regard to kidney transplant protocols. It is quite obvious that there is currently no single drug approved for accomplishing donor-specific suppression, but many believe a drug that could effectively interfere with the T-cell receptor (TCR) would be ideal. However, secondary (or costimulatory) signals on the T cell have been effectively inhibited by various drugs, such as antibodies to CD28, B7, CD154, and CD40. Therefore, transplantologists currently use a combination of multiple immunosuppressive drugs (with various mechanisms) to achieve a synergistic effect, and at the same time, minimize single-drug toxicity.[10]

The typical immunosuppressive protocol used (similar to solid organ transplantation) for induction includes the following four components: (1) Basiliximab, Thymoglobulin, Rapamycin, Campath-1H, or anti-thymocyte globulin (ATG), (2) Corticosteroids (methylprednisone/prednisone), (3) FK-506 (tacrolimus), and (4) mycophenolate mofetil (MMF). This regimen may or may not be used in conjunction with a topical immunosuppressant, for it has been shown that the integration of tacrolimus is of significant importance since it accelerates nerve regeneration.[14–16]

Also, there is an undefined role for autologous bone marrow cell infusion (harvested from the donor) in an effort to obtain "donor-specific tolerance." For instance, this technique was recently utilized by the French transplant team when performing the world's first partial face allotransplant.[17] Further research in this area, as well as a newer subset known as vascularized bone marrow transplantation, is currently underway.[18] Once either of these approaches has been clinically proven, a concomitant bone marrow transplant may be used.

Table 52.2. Variety of induction therapies used for hand transplantation.

Drug	Mechanism	Major side effect
Azathioprine	DNA replication and clonal expansion inhibitor	Bone marrow suppression
Cyclosporine (CsA)	IL2-inhibitor	Difficult enteric absorption, nephrotoxic, diabetogenesis, hyperlipidemia, hypertension, neurotoxicity
FK-506 (Tacrolimus)	Direct calcineurin inhibitor	Difficult enteric absorption, nephrotoxic, diabetogenesis, hyperlipidemia, hypertension, neurotoxicity
Basiliximab	Anti-IL2 receptor alpha monoclonal antibody	Post-transplant lymphoproliferative disease (PTLD)
Daclizumab	Anti-IL2 receptor alpha monoclonal antibody	Post-transplant lymphoproliferative disease (PTLD)
OKT3	Murine anti-CD3 monoclonal antibody	Febrile response, bronchospasm, pulmonary edema, circulatory collapse, rebound rejection, post-transplant lymphoproliferative disease (PTLD)
Rapamycin (Sirolimus)	Indirect calcineurin inhibitor	Minimum nephrotoxicity, delays wound healing
Campath-1H	Anti-CD52 antibody	Lymphopenia, post-transplant lymphoproliferative disease (PTLD)
Anti-lymphocyte globulin (Thymoglobulin, ATG)	Polyclonal antibodies	Serum sickness, nephritis, leukopenia, thrombocytopenia, allergic manifestations, post-transplant lymphoproliferative disease (PTLD)
Steroids (Methylprednisone)	Cytokine gene inhibitor	Hypernatremia, water retention, hypertension, fat dystrophy, psychosis, diabetogenesis, hyperlipidemia, adrenal suppression, cataracts, peptic ulcer disease, osteoporosis, aseptic necrosis of femoral/humeral heads, dermal atrophy
MMF	Inosine monophosphate dehydrogenase (purine synthesis) inhibitor (i.e., antiproliferative)	Gastrointestinal complications, leucopenia

Table 52.3. Maintenance drugs used in various combinations for hand transplantation.

Drug	Dosage	Target levels
FK-506[a] (Tacrolimus)	3–6 mg PO twice daily	15 ng/ml until POD 60, 12 ng/ml until POD 180, 10 ng/ml until 1 ½ years, and ultimate goal is 6–8 ng/ml
Mycophenolate[a] mofetil (MMF)	500 mg four times daily	3–5 ng/ml
Corticosteroids[a] (Prednisone)	5–10 mg	0 mg (Steroid-free goal)
Rapamycin (Sirolimus)	2–3 mg daily	6–8 ng/ml
Everlimus	1.75 mg daily	6–8 ng/ml

[a]Most common regimen used for hand allotransplantation.

Table 52.4. Rejection therapy drugs used for the reversal of acute graft rejection.

Drug	Mechanism	Route
Corticosteroids	Gene cytokine inhibitor	IV or topical
FK-506 (Tacrolimus)	Direct calcineurin inhibitor	IV or topical
Rapamycin (Sirolimus)	Monoclonal antibody	IV
Simulect	Anti-CD25 monoclonal antibody	IV
Campath-H	Anti-CD52 monoclonal antibody	IV

Table 52.5. Prophylactic drugs used for hand transplantation.

Drug	Indication	Dosage	Duration
Broad-spectrum antibiotics (skin flora)	Surgical-site infections, osteomyelitis	Variable	Variable
Antifungals	Fungal infection prophylaxis	Variable	Variable
Bactrim	Pneumocystis carinii pneumonia	5 mg/kg TID IV or PO	18 months
Gangcyclovir	Cytomegalovirus (CMV)	10 mg/kg IV, 1000 mg PO	First 3 months post-transplant
Valgancyclovir	Cytomegalovirus (CMV)	900 mg/day	First 3 months post-transplant

Maintenance Therapy

Maintenance therapy is used to prevent delayed acute and chronic graft rejection. It includes a regimen of either FK506, MMF, and prednisone or FK506, Rapamycin, and prednisone. Tacrolimus and Basiliximab are started simultaneously, and then Tacrolimus is tapered down at 4 weeks postoperatively. Tacrolimus target levels are 15 ng/ml until postoperative day (POD) 60, 12 ng/ml by POD 180, 10 ng/ml by 1.5 years, and an eventual goal of 8 ng/ml. The optimal maintenance regimen includes tacrolimus 3 mg BID, MMF 500 mg four times daily (QID) (or sirolimus 3 mg QD), + /− prednisone 5 mg QD (many prefer steroid-free maintenance therapy). After 2 years (or when graft function has peaked), sirolimus/everolimus is started at 2/1.75 mg/day (target trough levels of 6–8 ng/ml), and tacrolimus is tapered off.[6]

All tacrolimus dosages are adjusted based on the recipient's 12-h trough (if >20% above or below the target range). Trough concentrations are measured using an enzyme-linked immunosorbent assay. If using MMF, which is started preoperatively, its dose is adjusted to reach a target plasma concentration between 3 and 5 ng/ml, with a maximum dose of 3 g/day. MMF dosing should also be adjusted based on the recipient's WBC count (if <4, reduce by 50%; <2.5, discontinue). IV Solumedrol (500 mg) must be given intraoperatively, with an option to taper via the per os (oral route) (PO) route. If steroids are chosen by the HTT, doses postoperatively should start at 2 mg/kg/day on POD 1 and then be tapered slowly and cautiously to 10 mg/day (by POD 90).[6]

Tacrolimus

To date, the most common combination (maintenance) therapy used for hand transplantation includes a regimen consisting of tacrolimus (FK506), MMF, and corticosteroids. Tacrolimus is a macrolide antibiotic that was first introduced in 1992. It is derived from a fungus, Streptomyces tsukubaensis, which prevents both T-cell and B-cell activation by way of inhibiting calcineurin (similar to cyclosporine). In vitro studies have shown it to be 100 times more potent than cyclosporine, and animal experiments have demonstrated a nerve regeneration benefit when compared with other immunosuppressive agents. Recently, it has been used clinically via topical and/or systemic delivery for both the reversal of acute graft rejection and for maintenance therapy.[14]

Mycophenolate Mofetil

Mycophenolate Mofetil (MMF) is an antiproliferative drug that works by way of selective inosine monophosphate dehydrogenase inhibition. Since its first introduction in 1995, its most common use has been in conjunction with tacrolimus and corticosteroids. Essentially, MMF prevents de novo synthesis of guanosine nucleotide, thereby preventing B-cell and T-cell lymphocyte proliferation.[14]

Corticosteroids

Corticosteroids are the most commonly used immunosuppressive drugs used within the field of CTA, and their mechanism is basically cytokine gene inhibition. Common drugs used within this class are prednisolone and methylprednisone. Cellular function and protein synthesis in inflammatory cells are inhibited at a subcellular level, once the steroid receptor complexes bind to their DNA. Newer therapy regimens are trying to eliminate steroids chronically due to their overwhelming amount of adverse effects, which include hypernatremia, water retention, hypertension, fat dystrophy, psychosis, diabetogenesis, hyperlipidemia, adrenal suppression, cataracts, peptic

ulcer disease, osteoporosis, aseptic necrosis of femoral/humeral heads, and dermal atrophy.[14]

Rejection (Rescue) Therapy

Episodes of acute graft rejection have been encountered in all HT patients (100%). Almost all (11/12, 92%) patients examined by Lee in 2003 had a rejection-associated rash and skin biopsies consistent with lymphocytic infiltration. Two hand allografts demonstrated irreversible rejection in noncompliant patients and warranted subsequent amputation.[7] In the case of graft rejection, additional immunosuppression in compliant patients is effective in obtaining reversal and rescuing the hand allograft (100% success rate). This has been accomplished in multiple ways, including systemic steroids with or without topical steroids/tacrolimus, Rapamycin, Simulect, and Campath-H.[15]

Some have suggested that an early diagnosis of rejection allows easier reversal, thereby instilling the importance of close follow-up. In addition, the hand allograft allows the physician "visual transplant monitoring" (vs. liver or kidney), which accounts both for a higher incidence of rejection episodes (easier to diagnose) and a quicker diagnosis (hypothetically increasing the rate of successful graft salvage). By having access to the graft at skin level, it also affords the transplant surgeon a new method of drug delivery, topical immunosuppressants, thereby allowing a reduction in systemic dosing.[4] Tacrolimus ointment has been an impressive weapon, since it acts on the epidermal dendritic cells, thus limiting both the initiation and promotion of rejection.[15]

In conclusion, the two major problems with immunosuppressive therapy are their accompanying side effects (infection, organ toxicity, and malignancy) and significant cost, which in turn lead to a undeniably high incidence of noncompliance. Composite tissue allotransplantation (CTA), and specifically hand transplantation, will undergo a dramatic increase in utilization once a new, more effective method of achieving donor-specific tolerance and selective immunosuppression is identified.[10]

Prophylaxis Therapy

All immunosuppression drugs have accompanying side effects, such as weight gain, diabetes, and opportunistic infections, including bacterial, fungal, and/or viral. Opportunistic infections are prevented by using a variety of prophylactic medicines. For example, intravenous broad-spectrum antibiotics and antifungals are used perioperatively to decrease the risk of surgical site infection and/or osteomyelitis, and then they are switched to an oral route (when applicable) and continued postoperatively. Bactrim is given for the first 18 months for Pneumocystis carinii pneumonia prophylaxis. Intravenous Gangcyclovir (10 mg/kg) is given perioperatively (every 12 h) for CMV prophylaxis to POD 7 and then switched to 1,000 mg PO every 8 h (or Valgancyclovir 900 mg) on discharge (discontinued at POD 90).[5,16]

In addition, scientists know from studying solid organ transplantation that 80% of solid organ transplant recipients develop some type of post-transplant infection and that approximately 40% of post-transplant deaths are infection related. In detail, 55% are of bacterial origin, 30% are viral, and 15% are viral.[19] As of yet, CTA-related opportunistic infections have been seen at a significantly lower incidence (around 12%), which may be due to the fact that CTA (hand) transplant recipients are, on the average, younger with less comorbidities than patients needing a kidney transplant, for example.[20]

Post-transplantation Data Review

The largest collection of post-HT data, in regard to independent (unbiased) physical assessment, was completed during a world-wide traveling fellowship sponsored by the American Society for Surgery of the Hand (ASSH) WP Andrew Lee, M.D., Traveling Fellow 2003.[3] At this time, all HT patients were male. Five of the 14 patients had had bilateral HTs. Of the nine unilateral HTs, all but one replaced their dominant hand (8/9, 89%). Ages ranged from 21 to 46 years and were evenly distributed between each of the 3 decades. All HT patients had displayed "severe disability" preoperatively secondary to the loss of one or both hand(s). A total of 11 HT patients, with 16 transplanted hand allografts, were examined. Amputation-to-HT intervals ranged from 1 to 15 years (median, 4 years). Different levels of amputation/HT were noted: distal forearm (n = 11, 79%), middle forearm (n = 2, 14%), and proximal forearm (n = 1, 7%).

Human leukocyte antigen testing was performed in all cases (n = 14) between the donor and the recipient. In detail, six recipients had a

0/6 HLA match, two recipients had a 1/6 match, one recipient had a 2/6 match, three recipients had a 4/6 match, and one recipient had a 4/6 match. This data simply illustrates the fact that successful hand allotransplantation is not dependent on HLA matching and happens to be a significant difference compared with living-related kidney transplantation. In addition, all hand allografts reviewed at this time had been matched to the recipient's bone size, gender, and skin color.

There were no life-threatening, adverse systemic effects in any of the patients examined within this group. This study was limited in its evaluation of chronic rejection due to the fact that many patients had only a relatively short, transpired postoperative course.[9]

Postoperative Management

To date, there have been 40 transplanted hands in 29 patients. Patient survival remains at 100% in all HT patients. Graft survival remains at 100% in all patients at 1 and 2 years. Graft failure at more than 2 years was found in eight HT patients due to poor compliance.[21] Within the first year after HT, 12 episodes of reversible, acute rejection in compliant patients have been reported, with no malignancies or life-threatening complications.[1,2]

Functional Recovery

The ultimate goal of HT is to attain functional motor recovery of the transplanted hand superior to myoelectrical prosthesis and to achieve sensory function for discrimination and tactile sensation, while at the same time, maintaining minimal adverse effects secondary to unavoidable immunosuppression. In addition, both the hand procurement and transplantation require optimal preservation of neural, muscular, and sensory end-organ components, while post-transplant goals include timely reinnervations of neural targets and reestablishing several degrees of cortical reorganization.[5]

All hand allografts, when viable, present with normal-appearing skin color and texture. Hair and nail growth are also within normal limits. Arterial and venous patencies have also been consistent in all HT patients thus far (except for one HT patient who had multiple arterial–venous fistulae at 6 months and was corrected with surgical ligation).[3] Surgeons have found that the extrinsic muscle function (i.e., finger flexion/extension, grasp, pinch) seems to recover at a faster rate versus intrinsic muscles (i.e., thumb opposition, lumbricals, interossei) which show a slower, delayed pattern of recovery (≈12 months). Although this equates to limited postoperative mobility, many HT patients are able to perform most of their preoperative ADLs (i.e., reaching, grasping, moving, positioning, and turning objects). Some surgeons use electromyography to confirm these findings, whereas functional MRI (fMRI) has been performed both pre- and postoperatively. Data from fMRI in HT patients shows global cortical rearrangement. Interestingly, some HT patients retain similar-side dominance pre- and post-HT versus others who switch to the contralateral side for dominance. Therefore, "dominance" may not be a factor in hand allotransplantation.[5]

In 2007, the Registry published a "functional score system", with the intent to objectively analyze each HT using a standardized system. According to their report, all patients (n = 24) had achieved protective sensation (100%), and 17 of them maintained some form of discriminative sensation (71%). Approximately 90% of the patients reported intrinsic/extrinsic function, thereby allowing them to return to work. Fifteen (63%) HT recipients reported an improvement in their "quality of life".[4]

In terms of bone healing, many feel that the rate in HT patients is equivalent to those hands of replanted patients. More surprisingly, nerve function return (sensory and motor) in transplanted hands has been reported as "above average" when compared with baseline function in replanted hands.[4]

Postoperative Motor Function

Of the eleven HTs independently assessed in 2003, all showed significant postoperative mobility of the wrist, fingers, and thumb. It was also reported that none of the HTs had approached "normal" range of motion (Table 52.6). Some patients, on average, demonstrated a mean active wrist extension of 40° and an active wrist flexion of 33°. Average finger flexion was near 174°, with an average extension lag of 35°. Pulp-to-distal palmar crease displayed a wide range of 1–7 cm, with an average of 3.7 cm.[7]

Table 52.6. Independent assessment of function identified in 11 transplanted hands.[9]

Motor/sensory function	Range of motion (ROM)
Wrist extension	40°
Wrist flexion	33°
Total finger flexion	174°
Finger extension lag	35°
Pulp-to-distal palmer crease	3.7 cm
Digital localization	6/10
Two-point discrimination	>15 mm, 8/10 HTs

Intrinsic muscle function of those HTs independently assessed was "poor." A few HTs had palpable contraction of their thenar muscles, but no palmar abduction. Only two of the 11 had significant thumb abduction or opposition against gravity. At the time of examination (postoperative examination time widely ranged due to global limitations), thumb adduction and finger abduction/adduction were severely limited.[7]

The IRHCTT report confirmed Lee's findings with regard to delayed intrinsic muscle recovery and specifies 12 months post-transplantation as a recovery starting point. This was further confirmed with EMG in some HT recipients. These patients report using their transplanted hands to perform a variety of ADLs, such as eating, driving, grasping objects, motorbike/bicycle riding, shaving, holding a telephone, and writing. Functional MRI has shown that the brain regains the cortical site corresponding to the hand knob area in these patients when compared with normal subjects.[3,4]

Postoperative Sensory Function

In Lee's independent review, examination showed uniform, near-complete return of protective sensation, with 10/11 showing protective digit sensibility. Six out of eleven were able to accurately localize digits upon touch. Two displayed median versus ulnar nerve confusion. A majority of HTs distinguished two points 15 mm apart.[7] As per the registry's most recent report, all hand allografts recover some degree of protective sensation (i.e., ability to detect pain, thermal stimuli, and gross tactile sensation), but each allograft recovers a varying amount of discriminative sensation.[4]

The Innsbruck team reports that for their B/l HT patients (n = 2), nerve regeneration reached the center of the palm at 3 months and their finger tips by 6 months. On average, they report that active ROM in all of the transplanted hands (n = 4) is about 60%.[5]

Psychological Assessment

Nine of eleven HT patients surveyed reported a desire to return to a "normal" life, and 9/11 had returned to modified job tasks. For those more than 1 year post-transplant, 8/9 had returned to work (Lee and colleagues warn of selection bias, since the most motivated patients were chosen for surgery).

When comparing HT patients to hand-replantation patients, by way of the Carroll test, HTs (n = 4) scored in a range between "average to above-average" (mean, 62). Two patients were given the Michigan Hand Questionnaire and reported the following: complete lack of pain, activities of daily living (ADLs) comparable to preoperative carpal tunnel patients, a high aesthetic score, and an overall high score.

Unilateral recipients (n = 9) reported their "greatest benefit" to be "improved body image and enhanced ability to engage in social interactions" (n = 4, 44%), "increased hand function" (n = 3, 33%), and "greater self-reliance" (n = 2, 22%). Bilateral HT patients reported "greater self-reliance" (n = 3, 33%) and "the ability to feel their world again (i.e., touching their wives)" (n = 2, 22%).

HT disappointments were "none" (n = 4), "less function than they had hoped in their HT" (n = 4), "medication side effects" (n = 3), "rehabilitation/recovery length" (n = 2), and one unsatisfied patient stated that he would not go through with this HT again (This is a patient who lost his hand secondarily to rejection and had suffered significant ischemic pain).[7]

Rehabilitation

In Louisville, the hand therapy team recommends the following: a hand therapist meets with the patient preoperatively to assess all functional needs, take pertinent measurements, and begins the education process on what to expect postoperatively. In addition, they feel that inserting electrodes, at the end of the operation or soon thereafter, into the median and ulnar nerves (proximal to the anastomoses) is optional.[6]

Dynamic/static casting and bracing are applied postoperatively by an experienced orthotist

or therapist, since efficient hand allograft therapy is imperative for obtaining optimal graft function. A crane outrigger with static metacarpophalangeal (MCP) flexion and dynamic finger/thumb interphalangeal extension is indicated. Its purpose is to limit metacarpal joint flexion and at the same time allow active interphalangeal flexion/extension, which hypothetically limits adhesion formation and promotes tissue healing. This crane's utility, as described by Scheker et al., was first displayed in hand replantation patients. Hand exercise programs are based on the specific tendon repair technique used by the hand transplant surgeon. Scar management, which may include the use of compression glove, may be initiated at 1 month post-transplant. In addition to the outrigger, a hand-based anticlaw splint can be used intermittently in conjunction with the outrigger PRN at 3 weeks post-transplant. Additionally, electrical muscular stimulation can be used to improve tendon gliding.[6]

According to the French surgeons, they initiate rehabilitation 12 h after surgery, schedule physiotherapy BID, and include electrostimulation and occupational therapy. They believe that the rehabilitation, in combination with patient compliance, establishes functional recovery and that starting complex rehab early is optimal. They have found that muscular power and range of motion can be improved with constant, targeted exercise and strong motivation during physiotherapy, which they note as a significant difference in comparison with solid organ transplantation (Table 52.7).[16]

Functional Assessment

A "Hand Transplantation System Score (HTSS)" or "International Registry Score (IRS)" was recently created for the purpose of standardizing hand allograft function recovery. The score evaluates six different aspects of recovery and then totals for a score out of 100 points (appearance, sensibility, movement, psychological/social acceptance, daily activities/work status, and patient satisfaction/general well-being). Outcome scores are categorized as 81–100 (excellent), 61–80 (good), 31–60 (fair), and 0–30 (poor). A small study was performed on eight European HT recipients (U/l = 4, B/l = 4) using this scoring system. The survey results, for a total of 12 hand allografts, were the following: "Excellent" ($n = 5$), "Good" ($n = 5$), "Fair" ($n = 2$), and "Poor" ($n = 0$).[3,4]

In addition to the IRS score, each Hand Transplant Center should use (as suggested by the Innsbruck team) hot/cold temperature testing, the Weber static, two-point discrimination test, the Dellon moving two-point discrimination test, shape/texture identification tests, volumetric analysis, muscle strength testing by way of the Medical Research Council's (MRC) grading system, range of motion measurements, Kapandji opposition testing, visual analog scale, Perfetti's tactile/kinesthetic test, ADL testing, a Nine Hole Peg Test, arm disability measuring, and most importantly, the Carroll test as a method of assessing the allograft's global function and recipient integration (maximum of 99 points). The allograft's grip and pinch strength should be measured using a Jamar dynamometer and a pinchometer (tip, three-jaw chuck, and key pinch), respectively. ASSH guidelines are used to measure active/passive ROM. Tinel's sign is assessed periodically until it reaches the allograft's fingertips. Semmes Weinstein monofilament testing (pressure threshold), vibration (30 and 250 cps), and static/moving two-point discrimination are also performed regularly. The IRS score (as previously mentioned) should be calculated on a regular basis, and all data needs to be reported to the International Hand Registry. The patient is to be asked to complete, on a regular basis, the disabilities of the arm, shoulder, and hand questionnaire (DASH) and the Hand Transplantation Comprehensive Outcome Questionnaire (IRSS). Nerve conduction studies/EMG are used to assess motor function (a grade will be given) in the abductor pollicis brevis and abductor digiti minimi muscles, and the index and fifth fingers are used to measure sensory function.[4,5,20]

Table 52.7. Average rehabilitation therapy schedule for HT patients.[9]

Post-transplant timeline	Intensity
First 1–2 months	6–8 h/day
Following 6–12 months	4–7 times/week
After 1 year	1–3 times/week

Graft Assessment

Obviously, the most serious postoperative graft complication is rejection/failure. Therefore, periodic skin and muscle (i.e., punch) biopsies are taken either during scheduled follow-up (days 0, 5,

7, 10, 14, 21, 30, and Qmonthly) or when graft rejection is clinically suspected (dermatitis, edema, scaling, blisters, etc.). Other important findings of clinical concern to suggest rejection include erythema, escharification, and necrosis. All pathology specimens are to be reviewed by a transplant pathologist. The pathologist will assign an "Acute graft rejection score" using the Louisville Skin Rejection Scoring System as well as examine the specimen for evidence of chronic graft rejection (intimal hyperplasia or subintimal foamy histiocytes in allograft skin or muscle).[22]

Physical examination is also used to assess for any signs of GVHD. Blood work is assessed regularly for hematologic/renal status (i.e., CBC, basic metabolic profile) as well as for donor-specific chimerism (immunohistochemistry analysis for monoclonal antibodies specific to host or donor antigens). The antibody panel consists of the following: control antibodies, T-cell antibodies (CD3, CD4, CD8), B-cells (CD19), NK cells (CD56), and myeloid markers (CD33, CD45). Radiographic hand allograft imagery includes periodic ultrasound, CT angiogram, functional MRI, and angiography, depending on each patient's clinical course.[6]

Ancillary Staff

A diverse multidisciplinary team is needed for optimal outcomes. A large responsibility is delegated to a wide variety of nonsurgical staff, which includes a hand transplant coordinator, an internist, a social worker, an ethics committee, and a psychiatrist.

Hand Transplant Coordinator

A HTT needs to assign a designated hand transplant coordinator for each hand transplant patient. This person shall be well informed as to the intricacies of hand transplantation and must either be a physician, registered nurse, or physician's assistant. Their main objective is to coordinate each and every detail throughout the entire process, and the magnitude of this role cannot be underestimated. Their duties include aiding in the screening of potential candidates, coordinating all transplant-related activities, overseeing test results, and helping to arrange follow-up care either within your health system or at a patient's local hospital, if warranted. Prior to assignment, this person should successfully demonstrate adequate knowledge of the hand transplant protocol and transplant drugs/services, summarize pertinent procedures, and prove an understanding of potential complications seen within the area of hand transplantation.

For the first three post-transplant months, the HTC will need to be on-call and available 24 h/day and 7 days/week. A "backup" HTC system may need to be implemented if coverage becomes an issue. Their duties include monitoring and presenting daily immunosuppression drug levels and laboratory work to the HTT, since obvious abnormalities are of utmost importance and need immediate attention. All pharmacological dosing changes and/or additions also need to be overlooked by the HTC. Since a majority of the hand transplant recipients will most likely not reside in close proximity to your hospital, the HTC will need to arrange post-transplant housing for the first 90 days (at a distance within 10 miles), which should be subsidized. Their follow-up visits, at a Q3month interval for the first year and annually thereafter, are also the responsibility of the HTC.

The HTC will also prearrange a "satellite" medical team for all patients living at a significant distance away from the hospital. This satellite team, which should obviously be in close proximity to the patient, will include a local surgeon (preferably a hand surgeon), an internist, and a physical/occupational therapist. This would obviously be of great value to the patient if for some reason the HTT wanted to request a tissue biopsy, medical examination, and/or alter any specific hand therapy.[20]

Medical Management

Hand transplant patients should be followed by an internist at your institution. He or she will orchestrate all nonsurgical details, visits, and or medical examinations as needed. In addition, internists must also coordinate and refer all hand transplant patients to the various medical specialists, as they see necessary (i.e., nephrology, cardiology, etc.).

Social Worker

A transplant social worker should also be assigned to HTT, for their involvement is critical for patient adjustment pre- and post-transplant.

Their responsibilities include coordinating all daily appointments, confirming supportive measures as ordered by the HTT, and orchestrating all pertinent ancillary staff interactions with the hand transplant recipient on a day-to-day basis.

Medical Ethics

An ethics committee should be consulted either pre- or post-transplant for any and all ethical decisions in question, as they pertain to hand transplantation.

Public Media

All interactions with the media will be a collaborative effort between the HTT and a designated representative from a Public Relations department. Every effort should be made to conceal each and every hand transplant patient's identity. All photographs and videos relating to hand transplantation are the property of the hospital and must receive written approval from both the HTT and the hospital before public release.

Psychiatric Management

A transplant psychiatrist should also be assigned to the HTT. His/her responsibilities will include the following:

1. Preoperative assessment and selection of patients
2. Prescribe and guide all indicated psychiatric treatments for any candidate listed for hand transplantation
   a. Pretransplant psychopharmacological
   b. Psychotherapeutic
   c. Chemical dependency treatment
   d. Social/family/marital interventions
   e. Transplant education
3. Minimize psychiatric morbidity throughout the entire hand transplant process
4. Help the recipient integrate his or her foreign hand(s) both physically and psychologically. Psychiatric and psychosocial postoperative assessment is mandatory and follows the following schedule: Q3months for the first post-transplant year, Q6months for the second post-transplant year, and then annually thereafter

Financial Cost and Funding

The overall cost attributed to either unilateral or bilateral hand transplantation is dependent on a variety of factors, such as the country of origin for which the surgery is performed and the hospital's geographic location/size.

Hospital Expenses

For example, the Louisville group (Kentucky, US) reports the average expense for unilateral hand transplantation to be approximately 2,25,000.[23] This includes the cost of surgery, an average stay of 5 days within the hospital, hotel room expenses thereafter (approximately 3 months), all related medications, pertinent laboratory expenses, and rehabilitation therapy. This estimation would obviously be more for bilateral transplantation due to its coexisting necessities.

In addition, since this type of transplant surgery is still considered "experimental," the inpatient bill will be, for the most part, not covered by the insurance company, and all incurring costs will be absorbed by the hospital. This financial deficit can, however, be offset by a combination of endowments, research grants, and departmental funds.

Patient/Insurance Expenses

For unilateral or bilateral hand transplantation, your hospital covers all expenses occurring within the first 90–180 days of surgery, which by these estimations, would equal approximately $3,00,000 for each unilateral hand transplant. From this point forward, all charges would then be forwarded to the recipient's medical insurance company. This includes all required post-transplant medications, which can average up to $20,000/year. As for mandatory rehabilitation therapy, an adjusted cost schedule is provided to the recipient after 90–180 days depending on the medical insurance coverage and his/her financial status. Unexpected occurrences such as skin biopsies, laboratory tests, and rejection therapies are usually covered by insurance, since they are obviously classified under "medical necessity."

Graft Survival

Over the last 10 years, hand allograft survival has been excellent (>90% 5-year survival in compliant patients). However, due to the fact that hand transplantation is a "life-enhancing" surgery, rather than "life threatening," its acceptance into the United States has been minimal. It is also important to understand that the amputated hand allografts (since 1998) were simply a consequence of suboptimal compliance and unpreventable graft rejection.

Compared with solid organ allotransplantation, for which nonliving-related kidney transplantation is accepted by many as the most successful, hand transplantation still displays an attractive success rate. For instance, a report by Leichtman et al.[24] stated that the 5-year survival rate for nonliving-related kidney transplants performed between 2000 and 2005 in the United States ranged between 55% and 70%, depending on their selection criteria. Surprisingly, this is significantly lower than the reported survival rate for transplanted hand allografts.[24]

Complications

The IRHCTT's report in 2007 listed postoperative partial skin necrosis (STSG graft), arterial thrombosis (thrombectomy), and multiple arterial–venous fistulae (ligation) as causes for additional surgery. Other reports include hyperglycemia, chronic headaches, serum sickness, ulnar artery thrombosis, and ulnar osteomyelitis, with the latter requiring hardware removal and long-term antibiotics (at 6 months posttransplant).[4] As for serious complications such as malignancy, cardiovascular-related disease, nephrotoxicity, and gastrointestinal adverse effects, none has been reported thus far.[15]

Infection-related side effects include cytomegalovirus (CMV) reactivation, papilloma virus-associated skin lesions, *Clostridium difficile* colitis, Herpes simplex blisters, cutaneous mycosis, ulnar osteomyelitis (*Staphylococcus aureus*), opportunistic fungal infections, and reversible neutropenia. Metabolic complications include transient hyperglycemia, renal insufficiency, Cushing's syndrome, weight gain, dermatitis, and avascular hip necrosis (this particular HT patient required B/l hip replacement).[4]

Acute graft rejection typically presents in postoperative months 2–6, with skin lesions ranging from asymptomatic faint, pink macules to erythematous, infiltrated, scaly papules. Skin biopsies reveal perivascular/interstitial dermal mononuclear cell infiltrates, mixed with a small percentage of FoxP3 + Treg cells. Therefore, in most cases, steroids (oral or intravenous) are often used as the first-line treatment for reversal along with some topical creams/ointments (clobetasol, tacrolimus, and/or steroid). Additionally, some have reported using ATG, Basiliximab, and Campath-1H. All rejection episodes have resolved clinically and histologically within 10–15 days in compliant patients. All hand allograft rejection episodes were associated with some degree of skin involvement, thereby demonstrating the need for close dermatological follow-up. A CTA grading scale has been created for skin rejection and will be used by the IHRCTT (Table 52.8).[22]

Chronic rejection is of significant concern in CTA patients due to their relatively young age, but study has been limited due to obvious reasons. To date, the longest viable hand allograft (Louisville patient) at approximately 9 years, displays no signs of chronic rejection.[15] Although this phenomenon has yet to be truly identified, it does not mean, however, that hand allotransplants are not at risk. This may be due to the fact that punch biopsies of skin, subcutaneous tissue, and muscle are suboptimal for diagnosing chronic rejection in composite tissue transplantation (i.e., hand), but this obviously requires further research.[6]

Table 52.8. Skin rejection grading scale used in hand and composite tissue allotransplantation.

Rejection severity	Grade	Histological findings
Minimal	I	Perivascular lymphocytic/ eosinophilic infiltrates
Mild	II	Interphase epidermal reaction +/– adnexal structures
Moderate	III	Diffuse lymphocytic epidermal/ dermal infiltration
Severe	IVA	Necrosis of a single keratinocyte with focal dermal–epidermal separation
Severe	IVB	Necrosis/loss of epidermis

Additionally, wound healing may be of significant concern in HT patients due to their required immunosuppression. For example, a recent U/l HT performed in Poland (February 2006) was complicated by an open forearm wound. The patient, a 32 year-old man (Injury-to-transplant interval, 14 years) received a right-hand transplant (dominant side) at the mid-forearm level. Total ischemia time was 10.5 h. His postoperative course was complicated by an open wound, with a coexisting fungal infection. As per the surgical team, the healing time was "very slow" (as expected while on immunosuppression). Traditional dressings were applied daily, and delayed closure was accomplished using a split-thickness skin graft. Total healing time was prolonged at 64 days.[25]

The most serious complication associated with immunosuppression is the occurrence of post-transplant lymphoproliferative disease (PTLD) (malignancy). In kidney transplant patients, a risk ranging from 4% to 20% has been reported in the literature, but recent authors such as Baumeister et al. suggest a true incidence of around 3%. Thankfully, these cancers also seem to be less frequent in CTA as opposed to solid organ transplantation.[26]

Conclusion

Hand transplantation, along with its parent specialty known as composite tissue allotransplantation, has progressed tremendously since the first documented attempt in 1964. Since 1998, many patients around the world have benefited from hand transplantation, and a majority of them report their outcomes as "excellent." Their postoperative function seems to be approximated at 60–70% (vs. their native hand), and recovery timelines are more attractive when compared with those of replantation.[4] Since the first successful modern-day hand transplant (1998), scientists around the world, through a collaborative process conducted by the IHRCTT (International Registry for Hand and Composite Tissue Transplantation) have identified a few necessities limiting the efficacy of hand allotransplantation. These key factors include complete patient compliance with regard to immunosuppression, extreme psychological stability/social support, aggressive rehabilitation therapy, and constant patient motivation to succeed.

In conclusion, hand transplantation should be performed only at medical institutions capable of orchestrating a multidisciplinary team of specialists due to its inherent complexity, and the Institutional Review Board (IRB) protocol used for this type of difficult procedure should be extremely detailed and individualized. Further investigation is necessary to determine the clinical utility of hand allotransplantation.

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