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CHAPTER 1

INTRODUCTION

Deana Shenaq, MD

This book was an exciting undertaking and represents the hard work and collaboration of the Young Plastic Surgeons Steering Committee, along with plastic surgery residents and fellows throughout the country. In this updated version of Essentials, there are new chapters focusing on current popular topics in plastic surgery, including lymphedema, gender affirmation surgery and composite tissue allotransplantation. Previous chapters were modernized and now include color photographs. The book was written primarily for medical students and other students of plastic surgery to obtain a brief introduction into the specialty and provide references for further reading. It is not designed to be a comprehensive text, but rather an outline that can be read in the limited time available in a burgeoning curriculum. It is designed to be read from beginning to end.

Plastic surgery had its beginning nearly 3000 years ago, when Sushruta, an Indian surgeon, reconstructed the nose by transferring a flap of cheek and then forehead skin. It is a modern field, stimulated by the challenging reconstructive problems of the unfortunate victims of the World Wars. The advent of the operating microscope has thrust the plastic surgeon of today into the forefront of advances in small vessel and nerve repair, culminating in the successful replantation of amputated parts as small as distal fingers and bypassing of lymphatic vessels, only a few millimeters in diameter. Furthermore, these techniques have been utilized to perform the first composite tissue transplantations of both hands and partial faces. The field is broad and varied and this book covers the many areas of involvement and training of today’s plastic surgeons. The American Society of Plastic Surgeons is proud to provide complimentary online copies of the Plastic Surgery Essentials for Students handbook to all medical students in the United States and Canada.

Continually updated information about various procedures in plastic surgery and other medical information of use to medical students and other physicians can be found at the ASPS/PSF website at www.plasticsurgery.org and the Plastic Surgery Education Network at www.psenetwork.org. The information in this book has also been converted to an app in the Android Market and the Apple App store under the name Plastic Surgery Essentials for Students.
CHAPTER 2

CAREERS IN PLASTIC SURGERY

Chad M. Teven, MD and Ash Patel, MBChB, FACS

Plastic and reconstructive surgery is one of the most diverse specialties within the field of medicine. Unlike many other fields, it is not confined to a single organ system, region of the body, or patient population. The Greek term “plastikos” means to mold or to shape, and this is where our specialty derives its name and underlying principles. By reshaping, remolding, and manipulating both soft and hard tissues, plastic and reconstructive surgeons help patients with a myriad of issues. The goal of our specialty is to restore form and function, and to do so in an aesthetic fashion.

The first known plastic surgery procedures on living patients date back to 600 BCE, when the Indian surgeon Sushruta offered reconstruction to people who had had their noses surgically removed as a form of punishment. As early as 100 BCE, the Romans began performing procedures to improve self-image and cosmetic appearance. Since, plastic surgery has come a long way and now is integral to the treatment of patients with disfigurement due to trauma, burns, scarring, cancer, infection, and more. As opposed to treating specific conditions with a limited set of procedures, plastic surgeons use skills and principles based on anatomy, physiology, and tissue transfer when managing patients.

The challenge of plastic surgery is the combination of the surgeon’s judgment and problem-solving abilities with surgical technique at any given moment. Because of this approach, the plastic surgeon often acts as a “last resort” surgical consultant to surgeons and physicians in the treatment of many wound problems and is often called “the surgeon’s surgeon.”

While plastic surgeons offer life-saving procedures in many situations, an additional important theme in our specialty is the improvement in quality of life for patients. Plastic surgery not only restores body function, but helps to renew or improve a patient’s body image and sense of self-esteem. Along with psychiatrists, plastic surgeons are especially equipped to handle the patient’s problem of body image and to help the patient deal with either real or perceived problems.

Consistent with these far-reaching goals, the scope of the operations performed by plastic surgeons is broad. As outlined by The American Board of Plastic Surgery, “the specialty of plastic surgery deals with the repair, replacement, and reconstruction of physical defects of form or function involving the skin, musculoskeletal system, craniomaxillofacial structures, hand, extremities, breast and trunk, and external genitalia. It uses aesthetic surgical principles not only to improve undesirable qualities of normal structures, but in all reconstructive procedures as well. Among the problems managed by plastic surgeons are congenital anomalies of the head and neck. Clefts of the lip and palate are the most
common, but many other head and neck congenital deformities exist. In addition, the plastic surgeon treats injuries to the face, including fractures of craniofacial skeleton.

Craniofacial surgery is a discipline developed to reposition and reshape the bones of the face and skull through inconspicuous incisions. Severe deformities of the cranium and face, which previously were uncorrectable or corrected with great difficulty, can now be better reconstructed employing these new techniques. Such deformities may result from a tumor resection, congenital defect, previous surgery, or previous injury. Treatment of tumors of the head and neck and reconstruction of these regions after the removal of these tumors is also within the scope of plastic surgery.

Another area of expertise for the plastic surgeon is hand surgery, including the management of acute hand injuries, the correction of hand deformities and reconstruction of the hand. Microvascular surgery, a technique that allows the surgeon to connect blood vessels of one millimeter or less in diameter, is a necessary skill in hand surgery for re-implanting amputated parts or in moving large pieces of tissue from one part of the body to another.

Defects of the body surface resulting from burns or from injuries, previous surgical treatment, or congenital deformities may also be treated by the plastic surgeon. One of the most common of such procedures is reconstruction of the breast following mastectomy. Breasts may also be reduced in size, increased in size, or changed in shape to improve the final aesthetic appearance. Operations of this type are sometimes cosmetic in purpose, but in cases where the patient has a significant asymmetry or surgical defect, the procedure serves important therapeutic purposes.

The most highly visible area of plastic surgery is aesthetic or cosmetic surgery. Cosmetic surgery includes facelifts, breast enlargements and enhancement, nasal surgery, body sculpting, and other similar operations to enhance one’s appearance.

The results of the plastic surgeon’s expertise and ability are highly visible, leading to a high degree of professional and personal satisfaction. Plastic surgery is an innovative specialty. Advances such as transplantation, microvascular surgery, fat grafting, and various medical devices have been spearheaded and advanced by plastic surgeons. The discipline requires meticulous attention to detail, sound judgment and technical expertise in performing the intricate and complex procedures associated with plastic surgery. In addition, plastic surgeons must possess a flexible approach that will enable them to work daily with a tremendous variety of surgical problems. Most importantly, the plastic surgeon must have creativity, curiosity, insight, and an understanding of human psychology.

Students interested in a career in plastic and reconstructive surgery would benefit from rotating on their institution’s plastic surgery service to gain experience in and exposure to the field. In addition, finding a mentor within the field to help guide in one’s development, decision-making, and to answer questions is advised.
The pathway to a career in plastic surgery can follow in one of two ways. The first is through an integrated or categorical plastic surgery residency program. Programs are six years in duration; however, several programs also dedicate one or more years to research. The other route, known as the independent route, is to complete residency in general surgery, otolaryngology, urology, orthopedic surgery, or neurosurgery and then complete a three-year independent plastic surgery residency. After the completion of either the integrated or independent pathway, one is eligible to sit for the plastic surgery board examination. In addition, one can attain further training in a plastic surgery fellowship program. Fellowships are typically one year in duration and are offered for specialty training in hand surgery, craniofacial surgery, microsurgery, facial aesthetics, cosmetic surgery, body contouring surgery, and burn surgery.

Traditionally, plastic surgeons have established their practices in large urban settings. However, there is an increasing need for more plastic surgeons in the smaller communities and rural areas of this country - many metropolitan areas with populations of 65,000 to 268,000 have no plastic surgeons, leaving many areas needing plastic surgery expertise. There are approximately 7,000 board certified plastic surgeons in the United States; many of those currently certified by The American Board of Plastic Surgery received certification in the past ten years. Despite this recent rapid growth, there are opportunities for plastic surgeons in community and academic practice.

Plastic surgery is an old specialty with references that date back thousands of years. It has survived and flourished because it is a changing specialty built by imaginative, creative and innovative surgeons with a broad background and education. The future of this specialty is bright and will continue to progress because of students like you who choose to enter this special field.

Students interested in plastic surgery can find more information from the following:


2) [http://acaplasticsurgeons.org/residency-resources/](http://acaplasticsurgeons.org/residency-resources/)
ADDITIONAL RESOURCES ON THE SPECIALTY OF PLASTIC SURGERY

I. American Society of Plastic Surgeons
   www.plasticsurgery.org
   www.facebook.com/PlasticSurgeryASPS

II. Plastic Surgery Research Council
    www.ps-rc.org

III. American Council of Academic Plastic Surgeons
     www.acapsplasticsurgeons.org

IV. Plastic Surgery Education Network
    www.psenetwork.org
CHAPTER 3

HISTORY OF PLASTIC SURGERY

Rajendra Sawh-Martinez MD, MHS and Ash Patel, MBChB, FACS

“We restore, rebuild, and make whole those parts which nature hath given, but which fortune has taken away. Not so much that it may delight the eye, but that it might buoy up the spirit, and help the mind of the afflicted.”

Gaspare Tagliacozzi (1545–1597)

The origins of Plastic Surgery as a distinct field follows the history of scientific advancements, influenced by the scourge of need from social influences and wartime atrocities. Key historical figures and milestones are described in the long and illustrious history of the development of the modern innovative field of Plastic Surgery.

I. PLASTIC SURGERY ORIGINS IN ANTIQUITY

A. The term “plastic” in plastic surgery comes from the Greek “plastikos” which translates to “moldable”.
B. Ancient plastic surgery has its origins in the management of wounds, with historical reference to sewing wound edges with fibers or wound edges approximated with insect mandibles.
C. References in the Edwin Smyth Papyrus (~3000 to 2500 BC) to plastic surgery cases include descriptions of treating fractures, wounds, dislocations, sores and tumors.
   1. Early wound healing remedies included use of grease, honey, linen and swabs.
D. The first semblance to modern reconstruction is found in India with nasal reconstruction.
   1. Ancient cultures often punished adulterers, thieves and prisoners of war by mutilating their noses as a way of public shaming.
E. Famed Indian surgeon Sushruta wrote in the Ayurvedam (Indian sacred book of medical knowledge) descriptions of transposed flaps for nasal reconstruction (~600 BC).
   1. He is often credited with descriptions of the first forehead flap for nasal reconstruction, but this is controversial and unknown as the first published report of the forehead flap appears to be in 1794 (figure 1).
F. Greek medical influence is grounded in the writings of Hippocrates in the Corpus Hippocraticum (~460 BC) where descriptions of cauterization of raw surfaces, reduction of displaced fractures and trephination for hematoma evacuation are described.
G. Roman influence in plastic surgery can be found in the famous volumes De Medicina (~30AD) by Aulus Cornelius Celsus with descriptions of vessel ligature, lip flap reconstruction and wound closure by an advancement flap.
H. Writings by Claudius Galen (~129-201 AD) are famed for their intricate anatomical descriptions of muscles and cranial nerves.

Figure 1. First reports of the forehead flap for nasal reconstruction  

II. PLASTIC SURGERY IN THE MIDDLE AGES

A. Series of medical texts written by Oribasius (325-403AD) titled *Synagogae Medicae* describe reconstructive procedures aimed at rebuilding the cheek, nose, ears and eyebrows.

B. The middle ages brought about the first independent surgical treatise in the form of *Al Tasrif – On Surgery*, written by Abu-l-Qasim or Albucasis (~936-1013AD) in which surgical instruments, cautery, and the first description of a syringe with a piston was given.

C. The middle ages also brought about the advent of western universities which ushered anatomical classes and cadaver dissections, and anatomists as surgeons.

1. The advent of the printing press (1440AD) also allowed for the widespread dissemination of medical texts and exchange of ideas.
III. PLASTIC SURGERY AND THE RENAISSANCE

A. Andreas Vesalius publishes his anatomical treatise *De Humani Corporis Fabrica* (1543).
B. French surgeon Ambrose Pare (1510-1590) compiled his works in *Les Oeuvres*, in which are described repair of cleft lip and cleft palate, in addition to disputing the practice of “wound cleansing” by hot cauterity and pouring boiling oil into wounds.
C. Gaspare Tagliacozzi (1544-1599) is widely considered the founder of plastic surgery as a distinct discipline.
   1. He is credited with systematizing surgical approaches to nasal reconstruction.
   2. His book *De Curtorum Chirurgia per Insitionem* (On the Surgery of Injuries by Grafting) in 1597 provided step by step guidance and illustration to perform nasal repairs. (Figure 2)
D. After these landmark achievements, there were limited new advances until the 19th Century.

![Figure 2. Nasal reconstruction with the arm flap](From Tagliacozzi G. De Curtorum Chirurgia per Insitionem. Venice: Bindoni, 1597.)

IV. GOLDEN AGE OF PLASTIC SURGERY

A. English surgeon, Joseph Carpue composed in 1794 a letter to the editor which first described the forehead flap. His successful use of this procedure in 1814 marked the dawn of the rebirth of plastic surgery.
   1. Multiple surgeons throughout Europe compared and advanced techniques in rhinoplasty, facial reconstruction, cleft lip and cleft palate repairs including key surgeons such as Carl von Grafe (1787-1840), Johann Dieffenbach (1794-1847) in Germany, and Jacque Delpech (1777-1832) in France.
B. The advent of anesthesia in 1846 introduced new capabilities for all surgical fields and allowed for the blossoming of the golden age of plastic surgery.

C. Key Achievements in the golden age of Plastic Surgery:
   1. Giuseppe Baronio (1758-1811) from Italy first describes the use of autologous skin graft in 1804.
   2. First attempts at closing cleft palate defects by Roux and Von Grafe in France in 1819 and 1820, respectively.
   3. Pietro Sabattini described lip reconstruction with the “lip switch” technique in 1838. (Figure 3)
   4. Bernhard Von Langenbeck (1810-1887) outlines two mucoperichondrial flaps for cleft palate closure, described in 1862.

![Figure 3. Lip Switch technique described by Sabattini (1838)](image)

V. KEY FIGURES AND ACHIEVEMENTS IN MODERN PLASTIC SURGERY

A. World War I created a tremendous amount of disfigured casualties with devastating reconstructive challenges which catalyzed the formation of our modern conceptualization of plastic surgeons as specialists focused on restoring bodies ravaged by war.
   1. Hippolyte Morestin (1868-1919) and Charles Valadier (1873-1931) worked together, pioneering facial reconstructive surgery.
   2. Sir Harold Gilles (1882-1960) – Father of the modern era of plastic surgery, developed a referral center in Europe for causalities of facial disfigurement.
   3. Developed and systematically applied flap reconstructions of facial injuries. 
      a. Employed the tubed flap, skin flaps and bone, cartilage and skin grafts.
   4. Published landmark text *Plastic Surgery of the Face* (1920).
   5. Aesthetic Plastic surgery as a distinct field is initiated with the description of the correction of prominent ears in 1881 by Edward Ely.
a. John Roe (1848-1915), Robert Weir (1838-1927), Jacques Joseph (1865-1934) were early pioneers in Rhinoplasty.

1. Between 1916 and 1918, Johannes Esser (1877-1946) reported on local flaps commonly used today; cheek rotation, bilobed, island and "arterialized" flaps.
2. Training programs began developing after World War I, and spread throughout Europe and North America.
3. The American Society of Plastic Surgeons was founded in 1931 by Jacques Maliniac.
4. The American Board of Plastic Surgery was founded in 1937.
5. The Plastic and Reconstructive Surgery Journal was founded in 1946.
6. The Plastic Surgery Foundation was established in 1948.

B. Since the 1960s, new discoveries have brought about a new wave of reconstructive options.
2. Dawn of microsurgical techniques and advancements in knowledge of anatomy leading to free tissue transfer.
3. Angiosome concept by Taylor and Palmer in 1987 led to the development of perforator flaps (Figure 4).

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<td>Repair of lateral defects of the lower lip using lateral upper lip and corner of the mouth on a labial artery pedicle</td>
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<td>Tubed pedicle cheek flap for nasal reconstruction</td>
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<td>Donner</td>
<td>Tubed pedicle flaps from the cheek, shoulder, and back for nasal reconstruction</td>
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<td>Davis</td>
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<td>1977</td>
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Figure 4. History of flap development
REFERENCES

1. Tagliacozzi G. De curtorum Chirurgia per insitionem. Venice: Bindoni; 1597:43.
2. BL. Letter to the editor. Gentleman’s Magazine 1794;64 891–892
CHAPTER 4

TECHNIQUES AND PRINCIPLES

Nicole A. Phillips, MD and Ash Patel, MBChB, FACS

Plastic surgery is a specialty defined by principles and techniques, rather than by organ system or disease process. The subdivision of plastic surgery into two types of surgical procedures—reconstructive and cosmetic—is another unique aspect of the specialty. While the dividing line between the two is sometimes very clear, there is often a significant amount of overlap. The goal of reconstructive surgery highlights this overlap: “the restoration of form and function.” Both reconstructive and aesthetic surgery rely on a detailed knowledge of anatomy and the foundational principles and techniques outlined below.

I. EVALUATION AND PLANNING

A. Define the defect
   1. What is missing or abnormal?
      a. Tissue layers
   B. Disruption of vascular or neural network
   C. What is left behind?
   D. Is the surrounding tissue healthy, or has it been compromised (i.e. radiation therapy, burns, traumatic injury)?
   E. What local tissues are available for use?
   F. What is the best way to restore form and function?
   G. Replace like with like: the best reconstruction will utilize tissues similar to the missing tissues (i.e., glabrous skin for reconstruction of the weight-bearing sole or fingertips).
   H. “Don’t throw anything away”
      1. Spare parts surgery
      2. Composite grafts
      3. Biological dressings
   I. Designing incisions. Incision design is critical, as the location of scars impacts both their visibility as well as their ability to heal.
      1. Ideal incision placement
         a. Langer’s lines: Langer, a 19th-century anatomy professor in Vienna, first studied and described the relationship between resting skin tension and wounds. However, his studies were carried out on cadavers and were never intended to serve as a guide for surgical technique.
         b. Borges described relaxed skin tension lines in 1962: these lines follow the furrows formed when skin is relaxed and are revealed by pinching the skin. (Figure 1)
         c. Best incision designs usually involve a combination of factors
            i. Allowing for appropriate access
ii. Taking advantage of pre-existent scars or wrinkles
iii. Placement with respect to aesthetic subunits

Figure 1. Relaxed skin tension lines (RSTL) versus other skin lines

2. Aesthetic units and subunits (Figure 2)
   a. Have been described for multiple anatomic regions, including breast, face, and lower extremity
   b. Defined by naturally-occurring concave and convex surfaces
   c. Scars that cross aesthetic subunits are more noticeable than those that are hidden in the boundaries between subunits
Figure 2. Conforming to its underlying skeleton, the surface of the nose is crossed by gentle valleys and low ridges that divide it into topographic subunits. They are the dorsum, tip, sidewalls, alar lobules, and soft triangles.


E. Methods of excision

1. Elliptical
   a. Most common method
   b. Usually designed with length: width ratio of 3:1
2. Wedge
   a. Used for lesions located at or near a free tissue margin
3. Circular
   a. May be utilized when shorter scar is desired
4. Serial
   a. For large lesions which cannot be excised in one stage (i.e. congenital nevi)
   b. Frequently used in conjunction with tissue expansion

II. THE RECONSTRUCTIVE LADDER

A. Conceptual framework for understanding reconstructive options (Figure 3)

1. Starts with most simple option: i.e., healing by secondary intention
2. Progresses to more complex options in a step-wise fashion
B. The “reconstructive elevator” (Figure 4)
   1. Proposed by Gottlieb and Krieger in 1994
   2. Best reconstructive option is not always the least complex
Figure 4. The reconstructive elevator, as proposed by Gottlieb and Krieger. This formulation emphasizes the importance of selecting the most appropriate level of reconstruction as opposed to defaulting to the least complex. From Gottlieb L, et al. From the reconstructive ladder to the reconstructive elevator. Plast Reconstr Surg. 1994;93:1503-1504.

III. PRINCIPLES OF SUTURING

A. Layered closure. Any wound deeper than skin should be closed in layers.
   1. Eliminate dead space
   2. Prevent dehiscence while wound healing is occurring
   3. Precisely approximate skin edges without tension
B. Wound edge eversion
   1. Takes advantage of scar contraction
   2. Allows for optimal wound healing
C. Choosing suture
   1. Permanent versus absorbable
   2. Monofilament versus multifilament
   3. Suture size
   4. Needle type
      a. Cutting
      b. Taper
   5. Surgical glue
   6. Staples
D. Timing of suture removal
   1. Sutures should be removed from face within 5-7 days
   2. Sutures in other anatomic areas should be removed within 7-14 days
   3. Exceptions include wounds that cross joints, wounds that are under significant tension, wounds in irradiated or otherwise damaged surgical fields

IV. SUTURING TECHNIQUE

A. Simple interrupted sutures: most commonly used suture technique (Figure 5)
   1. Needle enters epidermis at 90-degree angle
   2. Needle turned to exit immediately below deep dermis
   3. Care must be taken to enter and exit at same levels on opposite side

Figure 5. Simple Interrupted Suture Technique
A. Running simple sutures
   1. Rely on well-approximated wound edges
   2. Not as precise as interrupted sutures, but faster
B. Subcuticular sutures
   1. Needle passed horizontally through the superficial dermis, parallel to skin
      surface
   2. Can be running or interrupted
   3. Allows close approximation of skin edges without need for external skin sutures
C. Horizontal mattress sutures (Figure 6)
   1. Evertting sutures that spread tension across a wound edge
   2. Needle passed across the wound and then back the other way
   3. Useful in fragile tissue
   4. Also useful in suturing glabrous skin of hands/feet
   5. Can be performed as a running suture

![Horizontal Mattress Suture Technique](image)

Figure 6. Horizontal Mattress Suture Technique

A. Vertical mattress sutures (Figure 7)
   1. Used for increased wound eversion
   2. Far-far near-near suture placement

![Vertical Mattress Suture Technique](image)

Figure 7. Vertical Mattress Suture Technique
REFERENCES

CHAPTER 5

WOUNDS

Tulsi Roy, MD and Michele Manahan, MD, FACS

A wound can be defined as a disruption of the normal anatomical relationships of tissues as a result of injury. The injury may be intentional such as a surgical incision, or it may be accidental following trauma.

I. STAGES OF WOUND HEALING

Wound healing is a complex, highly-regulated, multiphase process involving inflammation, fibroblast proliferation, and remodeling in the setting of tissue injury. Wounds may progress or even regress through these stages based on local and systemic factors.

A. Inflammatory phase (typically 1-4 days, if primarily closed)
   1. Physiology of inflammation
      a. Cytokine release is initiated by affected tissue cells and blood clot, which promotes recruitment of platelets and macrophages to the site of injury
      b. Leukocyte margination and emigration through neighboring vessel walls
      c. Venule dilation and lymphatic blockade
      d. Neutrophil chemotaxis and phagocytosis
   2. Presence of foreign material, bacterial load, and extent of devitalized tissue and bleeding modulate the body’s inflammatory response to tissue damage
   3. As long as the wound remains open, wounds remain in this phase. Reestablishment of epithelium or wound closure is important in order to progress through the remaining phases of wound healing.

B. Proliferative phase (days 4-42)
   1. Platelet degranulation initiates the proliferative phase of wound healing by establishing a fibrin clot that can be used as a scaffold to support angiogenesis and extracellular matrix formation
   2. Synthesis of collagen tissue from fibroblasts
   3. Increased rate of collagen synthesis for 42-60 days
   4. Rapid gain of tensile strength in the wound

C. Remodeling phase (4 weeks-1 year)
   1. Provisional tissue regenerated in the proliferative phase is revised through cellular apoptosis and expression of matrix metalloproteases.
   2. The extracellular matrix and subsequent scar is reorganized largely in response to mechanical tension, inflammation, and genetic phenotype
   3. Maturation by intermolecular cross-linking of collagen leads to flattening of scar
   4. Requires approximately 9-12 months in an adult, longer in children. (Scar revisions may be delayed a year or longer after injury to ensure remodeling is complete.)
5. Tensile strength of a healed scar will peak at approximately 60 days post-injury and achieve up to 80% strength of unwounded skin

II. FACTORS INFLUENCING WOUND HEALING

A. Local factors
   1. Fluid collection: hematoma or seroma
   2. Early wound closure
   3. Blood supply
   4. Temperature
   5. Infection
   6. Technique (gentle handling of tissues, orienting incisions or closures along optimal vectors to disperse mechanical tension) and ideal suture materials

B. Systemic factors – optimize nutrition, palliate or optimize chronic illness, deleterious effects of medications that interfere with wound healing (chemotherapy, steroids, etc.)

III. WOUND CLOSURE

Several general surgical principles are important to keep in mind to expedite wound healing and reduce the incidence of hypertrophic or pathologic scarring. Approaches to wounds should always include adequate debridement, removal of any nonviable tissue and foreign bodies, bacteriologic control, and optimization of systemic factors governing wound healing.

A. Healing by primary intention - wound closure by direct approximation, pedicle flap or skin graft
   1. Debride necrotic or nonviable tissue and irrigate copiously to expedite inflammatory phase
   2. Dermis should be accurately approximated. Skin glue may be used if the wound is limited to partial thickness depth
   3. Scar may be red, raised, pruritic at peak of collagen synthesis
   4. Scar remodeling occurs over approximately 9-12 months in adults, as collagen maturation takes place (may take longer in children)
   5. Final result of scar is dependent on length of time until definitive wound closure, location, and mechanical tension, and factors influencing amount of inflammation.

B. Healing by secondary intention – wound is left open to heal
   1. Myofibroblasts promote contraction of wound edges
   2. Epithelialization proceeds from wound margins towards center at 1 mm/day under ideal circumstances
   3. Secondary healing beneficial in heavily contaminated or “dirty” wounds (e.g. perineum), or wounds in areas that have excellent vascular supply (e.g. scalp)

C. Healing by tertiary intention - delayed wound closure after several days
1. Intentional interruption of healing started as secondary intention
2. May proceed any time after granulation tissue has formed in wound
3. Delayed closure should be performed when wound is not infected. Quantitative culture should demonstrate <100 CFU bacteria/gram of tissue
4. Skin grafting may be considered in larger wounds for definitive closure after adequate debridement and presence of well-vascularized wound bed

IV. MANAGEMENT OF THE CLEAN WOUND

A. Aim to obtain a closed wound as soon as possible to prevent infection, fibrosis and secondary deformity
B. General principles
1. Updated tetanus vaccine. Administer if not within 10 years of booster for clean minor wounds or within 5 years for contaminated wounds
2. Local anesthesia - use lidocaine with epinephrine unless contraindicated
3. Tourniquets may help provide bloodless fields in extremities
4. Surgical prep
   a. Aqueous-based:
      i. Povidone-iodine: one of the few products that is widely effective and safe on nearly all skin surfaces. Must be diluted 1:1 with normal saline when used around the eyes
      ii. Chlorhexidine gluconate (CHG): more sustained antimicrobial activity and more resistant to neutralization by blood products than iodine. Contraindicated on genitalia and open wounds
   d. Alcohol-based:
      i. Ethyl and isopropyl alcohol: quick, sustained, durable, with broader spectrum antimicrobial activity
      ii. When used alone, alcohol is fast and short acting, has broad-spectrum antimicrobial activity, and is inexpensive. Can also be combined with iodine or CHG.
      iii. Highly flammable
      iv. Contraindicated on open wounds and genitalia
5. Debridement and irrigation - Remove clot, debris, and necrotic tissue. Copious irrigation to remove debris and decrease bacterial inoculum
6. Closure - approximate dermis as atraumatically as possible, consider undermining of wound edges to relieve tension
7. Dressing - must provide absorption, protection, immobilization, even compression, ideally without interfering with function
C. Types of wounds and their treatment
1. Superficial abrasions - cleanse to remove foreign material and apply moist dressings or ointments to provide moist environment for re-establishment of epidermis
   a. Remove gross debris buried in dermis within 24 hours of injury to prevent infection, prolonged inflammation, and traumatic tattoos
2. Contusion - evacuate soft tissue hematoma if a fluctuant, organized collection is present or if pressure of hematoma is compromising surrounding tissue (ears, nailbed, etc).
   a. Early - minimize by cooling with ice (24-48 hours)
   b. Later - warm compresses helpful to speed reabsorption of blood
3. Laceration - revise and trim wound edges as necessary, debride, and suture
4. Avulsion
   a. Partial - revise and suture the flap if viable
   b. Total - avulsed tissue may be irrigated, cleansed, defatted, and applied as a full thickness skin graft
5. Puncture wound - evaluate underlying damage, possibly explore wound for foreign body, and obtain x-ray if appropriate. If small, consider leaving open or partially open to promote egress of drainage
6. Bullet wounds – considered clean wounds that require local wound care and healing by secondary intention.
7. Animal bites – debride, irrigate, and close primarily or leave open, depending upon anatomic location, time since bite, etc.
   a. Augmentin is typically a mainstay of antibiotic prophylaxis.
   b. Consider rabies vaccine, if necessary.
8. Wounds of face
   a. Largely develop as result of trauma
   b. Important to first obtain a thorough trauma workup and rule out any intracranial injury, airway issues, or other intra-thoracic or abdominal injury.
   c. The face has reliable and excellent vascular supply, allowing for greater window of opportunity for delayed wound closure
   d. Consider serial examination and delayed closure to evaluate extent of necrotic tissue burden before definitive debridement, particularly if “questionably viable” tissue is in the wound bed.
   e. Reestablishment of symmetry: Carefully align anatomic landmarks such as vermilion border, ala, eyebrow, helical rim
9. Special Wounds
   a. Amputation of parts
      i. Place amputated part in saline soaked gauze in a plastic bag and place the bag on ice. Avoid direct contact with ice to prevent thermal injury.
      ii. Ischemia times for replantation are 12 hours of warm and 24 hours of cold ischemia for digits, and 6 hours of warm and 12 hours of cold ischemia for major replants of upper and lower extremity
   b. Cheek injury - may require exploration for parotid duct or facial nerve injury
   c. Intraoral injuries - tongue, cheek, palate, and lip wounds require approximation with absorbable suture
   d. Eyelids - align grey line and close in layers - consider temporary tarsorrhaphy
   e. Ear injuries
      i. Hematoma requires incision and drainage and well-molded dressing to prevent cauliflower ear deformity
ii. Through-and-through laceration requires 3-layer closure including cartilage
iii. Tapered, noncutting needles are indicated for cartilage repair
iv. Avoid oversuturing
v. May require bolster dressing afterwards to stabilize tissues and avoid hematoma
vi. Large composite defects must be approached in staged fashion

V. MANAGEMENT OF THE “CONTAMINATED” WOUND

A. Guidelines for management of contaminated acute wounds
   1. Majority of civilian traumatic wounds may be closed primarily after adequate debridement
   2. Sharp debridement followed by copious irrigation
   3. Consider healing by secondary intention if:
      a. Heavy bacterial inoculum
      b. Long time lapse since initial injury
      c. Crushed or ischemic tissue - severe contused avulsion injury
      d. Prolonged steroid use
   4. Antibiotics - systemic antibiotics are only of use if a therapeutic tissue level can be reached within four hours of injury or debridement
   5. Wound closure
      a. Buried deep dermal sutures should be used to keep wound edge tension to a minimum. Limit the amount of foreign material in wound as much as possible.
      b. Monofilament sutures less prone to infection
      c. With regard to deep open abdominal wounds, loss of domain can occur when muscle, fascia, or skin necrose or retract, allowing extrusion of viscera
         i. Wound closure attempts to recreate lost domain and reestablish function of abdominal wall
         ii. If in doubt, it is almost always safer to delay closure and consider scar revision at a future date

B. Guidelines for management of contaminated chronic wounds
   1. Debridement and irrigation, may require serial debridement to establish wound stability
   2. Systemic antibiotics of little use, unless patient demonstrates systemic signs of infection
   3. Topical antimicrobial creams - silver sulfadiazine and mafenide acetate
   4. Biological dressings (allograft, xenograft, etc.)
   5. Integra™ (Integra LifeSciences Corporation):
      a. Bilayer wound matrix: superficial layer is a semi-permeable silicone membrane that mimics epidermis, deep layer is a collagen-GAG matrix that mimics dermal layer.
      b. The silicone layer is peeled off after incorporation of deep layer.
c. Useful for wound coverage over devascularized wound bed with limited immediate reconstructive options

6. Final closure
   a. With a delayed flap, skin graft, or flap
   b. Convert the chronic contaminated wound to an acute clean wound by decreasing the bacterial count (debridement)
   c. Quantitative cultures may help guide management with regard to determining appropriateness for definitive closure

VI. WOUND DRESSINGS

Dressings serve to protect the wound from trauma and to provide an ideal environment for healing

A. Antibacterial ointments
   1. Bacitracin, Bacitracin/Neomycin/Polymyxin B, Mupirocin
      a. Provide moist environment conducive to epithelialization. Beware of secondary inflammatory reaction from antibiotic ointment that may mimic infection
      b. Mupirocin effective against MRSA
   2. Silver sulfadiazine and mafenide acetate
      a. Useful for burns
      b. Antibacterial activity penetrates eschar
      c. Mafenide has better penetration of cartilage
      d. Prolonged use of mafenide may promote fungal overgrowth

B. Splinting and casting
   1. Immobilization decreases shear forces on wounds and may help to promote wound healing
   2. Avoid splint for extended period of time to avoid joint stiffness

C. Pressure Dressings
   1. May be useful to obliterate “dead space” or to prevent seroma/hematoma
   2. Do not compress flaps too tightly, may risk compromising vascular supply

VII. NEGATIVE PRESSURE WOUND THERAPY (NPWT)

Beneficial tool for large wounds or contaminated wounds not amenable to primary closure

A. Technique includes application of foam sponge or gauze covered with adhesive dressing applied to vacuum device that provides constant sub-atmospheric pressure (-50 to -175mmHg)
   1. Should not be placed in direct contact with blood vessels or other fragile structures, infected or necrotic wounds, or on denuded bone.
2. NPWT may be placed over bilayer matrices (e.g. Integra™, Integra LifeSciences Corporation) to promote ingrowth of granulation tissue and development of a vascularized wound bed.

3. Dressing should be changed every 48-72 hours to assess wound progress and viability of tissue

B. Mechanism of action
1. Maintains moist wound environment and reduces edema
2. Promotes local wound blood flow and angiogenesis, reduces presence of inflammatory mediators, and speeds overall collagen synthesis and rate of wound closure by offloading mechanical tension on wound

C. Disadvantages: Cost of device, pain or discomfort with dressing changes and peri-wound irritation secondary to adhesive tape

D. Advantages: Can be used as adjunct dressing to serve as bolster for skin grafts and provide exudative management for draining wounds. Wound care regimen is less frequent for patients, making NPWT ideal for more cumbersome or extensive wounds that would otherwise require multiple daily dressing changes.

REFERENCES

CHAPTER 6

GRAFTS AND FLAPS

Chad A. Purnell, MD and Ash Patel, MBChB, FACS

When a deformity cannot be closed primarily, and secondary healing would be unlikely, too slow, or suboptimal, grafts and flaps are employed to restore normal function and/or anatomy.

I. GRAFTS

Grafts are harvested from a donor site and transferred to the recipient site without carrying its own blood supply. It relies on new blood vessels from the recipient site bed to be generated (angiogenesis). A graft MUST have a wound bed of healthy tissue (granulation tissue, muscle, fascia, bone with intact periosteum, tendon with intact paratenon) because the graft is totally dependent on blood supply from the wound.

II. SKIN GRAFTS

A. Thickness (Figure 1)
   1. Full thickness - Full thickness skin grafts (FTSGs) consist of the entire epidermis and dermis.
   2. Split thickness - Split thickness skin grafts (STSGs) consist of the epidermis and varying degrees of dermis. They can be described as thin, intermediate, or thick. A typical intermediate depth STSG is 12/1000 inch in depth.

![Figure 1. Components of a split thickness versus a full thickness skin graft](image)

B. Donor site
   1. Full thickness - The full thickness skin graft leaves behind no epidermal elements in the donor site from which resurfacing can take place. Thus, the donor site of a FTSG is usually closed primarily. It must be taken from an area
that has skin redundancy. It is usually harvested with a scalpel between the dermis and the subcutaneous fat.

2. Split thickness - The split thickness skin graft leaves behind adnexal remnants such as hair follicles and sweat glands, foci from which epidermal cells can repopulate and resurface the donor site. This is typically harvested with a pneumatic or electric dermatome. The donor site is usually covered with an occlusive dressing and left to heal secondarily.

C. Recipient site
1. Full thickness - Full thickness skin grafts are usually used to resurface smaller defects because they are more limited in size. They are commonly used to resurface defects of the face or hand. They provide better color consistency, texture, and undergo less secondary contraction.

2. Split thickness - Split thickness grafts are usually used to resurface larger defects. STSGs undergo more secondary contraction as they heal compared to an FTSG. They typically darken in color compared to the area they were harvested from.

D. Survival
1. Full thickness and split thickness skin grafts survive by the same mechanism.
   a. Plasmatic imbibition (First 24-48 hours) - Initially, the skin graft passively absorbs nutrients from the wound bed by diffusion.
   b. Inosculation - By day 3, the cut ends of the vessels on the underside of the dermis line up with and begin to form connections to those of the wound bed.
   c. Angiogenesis - By day 5, new blood vessels grow into the graft and the graft becomes vascularized.

2. Skin grafts fail by four main mechanisms:
   a. Poor wound bed - Because skin grafts rely on the underlying vascularity of the bed, wounds that are poorly vascularized with bare tendon or bone, or because of radiation or ischemia, will not support a skin graft. Synthetic materials cannot be covered by grafts for this reason.
   b. Shear - Shear forces separate the graft from the bed and prevent the contact necessary for revascularization and subsequent “take”, which refers to the process of attachment and revascularization of a skin graft in the donor site.
   c. Hematoma/seroma - Hematomas and seromas prevent contact of the graft to the bed and inhibit revascularization. They must be drained by day 3 to ensure “take.”
   d. Infection - Bacteria have proteolytic enzymes that lyse protein bonds needed for revascularization. Bacterial levels greater than $10^5$ cells/gram of tissue in the wound will cause graft failure.

E. Skin substitutes - These are used for temporary wound coverage, or as a bridge to another form of reconstruction, typically a skin graft.
1. Allograft/Acellular dermal matrices - Cadaveric skin or dermis
2. Xenograft - Skin from a different species, e.g. pig skin – provides only temporary coverage
3. Synthetic - i.e. Integra™ (Integra LifeSciences Corporation) - collagen/silicone bilayer designed to provide an occlusive environment as granulation forms through the tissue matrix. This granulation will then support a skin graft after the silicone is removed.

III. OTHER GRAFTS

A. Nerve - Most common donor site is the sural nerve
B. Fat - Can be harvested as a structured graft or as lipoaspirate
C. Tendon - Common donor sites are the palmaris longus, plantaris, extensor digitorum longus
D. Cartilage - Common donor sites are the nasal septum, ear conchal bowl, rib
E. Bone - Common donor sites are the calvarium, iliac crest, tibial tuberosity, rib
F. Muscle - Typically small, can be harvested from any muscle
G. Composite - A graft that has more than one component, i.e. cartilage and skin graft, or a dermal-fat graft.

IV. FLAPS

Flaps are tissues transferred with an intact vascular supply to the recipient site. They survive through this blood supply and are not dependent on the wound bed. Flaps can be used when the wound bed is unable to support a skin graft (such as over exposed hardware), or when a more complex, larger, or more aesthetic reconstruction is needed. They are the most versatile type of reconstruction used by plastic surgeons. Flap harvest always leaves a donor site that will need to be closed either primarily, with a graft, or with another flap.

V. CLASSIFICATION

A. By composition - Flaps can be classified by the type of tissue transferred.
   1. Single component
      a. Skin flap - e.g. Parascapular flap
      b. Muscle flap - e.g. Rectus abdominis muscle flap or latissimus dorsi muscle flap
      c. Bone flap - e.g. Fibula flap
      d. Fascia flap - e.g. Temporoparietal fascia
   2. Multiple components - Named by types of tissue
      a. Fasciocutaneous - e.g. Radial forearm flap or anterolateral thigh flap
      b. Myocutaneous - e.g. Transverse rectus abdominis myocutaneous flap or latissimus myocutaneous flap
      c. Osteosecutaneous - e.g. Fibula flap with a skin paddle or medial femoral condyle flap with skin paddle
      d. Composite flap – flap components are directly attached to each other and harvested on single pedicle (e.g. latissimus myocutaneous flap)
e. Chimeric flap – flap components are harvested on their own vessel and freely mobile, with a single feeding vessel. (Figure 2)


B. By location - Flaps can be described by the proximity to the primary defect that needs to be reconstructed.
1. Local flaps
   a. Raised from the tissue adjacent to the primary defect.
   b. Movement into the defect can be described as advancement, rotation, or transposition.
2. Regional
   a. Raised from tissue in the vicinity but not directly adjacent to the primary defect.
   b. Movement is described as transposition or interpolation.
   c. A good example would be a latissimus flap for breast reconstruction.
3. Distant
   a. Raised from tissue at a distance from the primary defect.
   b. Usually requires re-anastomosis of the blood vessels to recipient blood vessels in the primary defect (see “free flaps” below).
   c. There are a few examples of distant pedicled flaps, such as using a groin flap for hand reconstruction.

C. By vascular pattern
1. Random vs. Axial
   a. Random pattern flaps
      i. Do not have a specific or named blood vessel as their blood supply.
      ii. Instead, these flaps are designed based on the size of the flap base.
      iii. Because of the random nature of the vascular pattern, these flaps are limited in dimensions, specifically in a length:base width ratio of 3:1.
      iv. If designed longer than this ratio, a random blood supply often cannot support the flap (Figures 3, 4).
b. Axial pattern flaps
   i. Designed with a specific named vascular system that enters the base and runs along its axis. (Figure 5) This allows the flap to be designed as long and as wide as the territory the axial artery supplies (angiosome).
   ii. Blood supply requires artery and its accompanying vein
   iii. Greater length possible than with random flap

b. Axial pattern flaps
   i. Designed with a specific named vascular system that enters the base and runs along its axis. (Figure 5) This allows the flap to be designed as long and as wide as the territory the axial artery supplies (angiosome).
   ii. Blood supply requires artery and its accompanying vein
   iii. Greater length possible than with random flap

b. Axial pattern flaps
   i. Designed with a specific named vascular system that enters the base and runs along its axis. (Figure 5) This allows the flap to be designed as long and as wide as the territory the axial artery supplies (angiosome).
   ii. Blood supply requires artery and its accompanying vein
   iii. Greater length possible than with random flap

c. All free flaps are axial (see free flap, below)

d. Peninsular – skin and vessel intact in pedicle

e. Island – vessels intact, but no skin over pedicle


Figure 4. (Top) bilobe flap. (Below) Rhomboid flap. Both may be designed as random flaps. From Buchanan P, et al. Evidence-Based Medicine: Wound Closure. Plast Reconstr Surg 2016;138(3S):257S-270S.
Figure 5. Axial/arterial pattern skin flaps

2. Pedicled vs. Free
   a. Pedicled flaps
      i. Flap blood supply remains attached to its source.
      ii. Transferred to the defect with its vascular pedicle acting as a leash.
   b. Free flaps
      i. Detached at the vascular pedicle and transferred from the donor site to a recipient site.
      ii. At the recipient site, the flap artery and vein are anastomosed to recipient vessels.
      iii. This allows more flexibility as tissues can be transferred nearly anywhere but requires microsurgical skill and increased operative time.

3. Perforator Flaps
   a. Supplied by smaller vessels that pass through or in between deep tissues.
   b. Typically harvested without the deep tissues in order to minimize donor site morbidity and to yield only the necessary amount of tissue for transfer.
   c. Can be transferred either as a pedicled or free flap.
   d. Perforators are described by their path from the main vessel to the skin (Figure 6).
   e. Common perforator flaps:
      i. Deep inferior epigastric perforator flap (DIEP)
         a) Skin and fat of the lower abdomen supplied by the deep inferior epigastric artery and vein perforators without the rectus abdominis muscle.
      ii. Anterolateral thigh perforator flap (ALT)
         a) Skin and fat of the anterolateral thigh supplied by the descending branch of the lateral circumflex femoral vessel perforator(s).
      iii. Thoracodorsal artery perforator flap (TAP/TDAP flap)
         a) Skin and fat of the lateral back supplied by the thoracodorsal artery and vein perforator without the latissimus dorsi muscle.
II. CHOOSING THE RIGHT FLAP

A. The primary defect - Recipient site considerations “replace like with like”
   1. Size
   2. Location
   3. Tissue type to be replaced: Muscle can eliminate more dead space, skin is better for resurfacing
   4. Functional and aesthetic considerations

B. The secondary defect - Donor site considerations
   1. Location
   2. Adhere to an angiosome, the territory that is supplied by a given vessel
   3. What type of tissues are needed
   4. Functional and aesthetic morbidity

III. FLAP SURVIVAL

A. Success depends not only on its survival but also its ability to achieve the goals of reconstruction.

B. Flap failure results ultimately from vascular compromise or the inability to achieve the goals of reconstruction.
   1. Tension
   2. Kinking of pedicle
   3. Compression
   4. Vascular thrombosis (typically more often a consideration in free flaps)
   5. Infection
REFERENCES

CHAPTER 7

SKIN AND SUBCUTANEOUS LESIONS

Sabrina Pavri, MD and Cindy McCord, MD

I. BENIGN SKIN LESIONS

A. Acrochordon (skin tag)
   1. Presents as single or multiple small, skin-colored, pedunculated papules
   2. Treatment for symptomatic lesions includes cryotherapy, snip excision, or shave excision

B. Epidermal inclusion cyst (sebaceous cyst, epidermoid cyst)
   1. Result from a proliferation of epidermal cells within the dermis, and arise from the infundibular portion of the hair follicle
   2. Present as skin-colored to yellow, firm, movable nodules, often with a visible central punctum. Usually asymptomatic, but may become infected/inflamed
   3. Well circumscribed by a cyst wall made of stratified squamous epithelium, and communicate with the surface through a small opening, which may contain a keratinous plug or blackhead
   4. Treatment of an acutely infected/inflamed EIC is incision and drainage
   5. Definitive treatment is surgical excision of the entire cyst (including cyst wall)

C. Pilar cyst (trichilemmal cyst)
   1. Originate from the outer root sheath of the hair shaft, and are lined by stratified squamous epithelium, which undergoes keratinization
   2. Present as firm, slow-growing subcutaneous nodules (clinically similar to epidermoid cysts, but they lack the central punctum)
   3. Most common cutaneous cyst of the scalp
   4. Definitive treatment is surgical excision of the entire cyst (including cyst wall)

D. Pilomatricoma
   1. Benign growth composed of hair follicle matrix cells
   2. Classically present as slow-growing, “rock-hard” subcutaneous masses, with a blue hue or ulcerative appearance
   3. Bimodal distribution (first and sixth decades), although more common in children
   4. Definitive treatment is surgical excision of the entire mass

E. Dermoid cyst
   1. Congenital cysts located along lines of fusion in the head and neck region, most commonly along the superior lateral orbital ridge, but also occurring at the scalp and the midline of the nose
   2. Skin-colored, nontender, noncompressible, slow growing, and can arise in the dermis or subcutaneous tissue, or be fixed to underlying periosteum
   3. 40% of midline nasal cysts have intracranial extension through an abnormal foramen cecum, and should undergo MRI preoperatively
   4. Treatment is surgical excision of the entire cyst (including cyst wall)
A. Seborrheic keratosis (verruca senilis, pigmented papilloma)
   1. Present clinically as hyperpigmented, waxy, verrucous papules with a characteristic “stuck-on” appearance
   2. Appear in the fifth to seventh decades of life, usually on the head, neck, or trunk
   3. Arise from the basal layer of the epidermis, are composed of well-differentiated basal cells
   4. Removal is for cosmetic purposes only – cryotherapy, shave biopsy, dermabrasion

B. Cutaneous horn
   1. Hard, cone-shaped cutaneous projections typically caused by excessive epidermal growth and retention of keratin
   2. 20% are associated with premalignant lesions, and 15% are associated with SCC
   3. Treatment is shave biopsy to exclude malignancy, or excision

C. Trichoepitheliomas
   1. Neoplasms of follicular origin that presents as multiple, yellowish-pink, translucent papules distributed symmetrically on the cheeks, eyelids, and nasolabial area
   2. More common in women
   3. Can be confused with basal cell carcinoma
   4. Treatment is excision to differentiate from carcinoma

D. Eccrine poroma
   1. Presents as a solitary lesion (firm papule less than 2 cm in size) usually on the sole of the foot or the palm of the hand in persons older than 40 years. It may also occur on the chest, the neck, or other locations.
   2. Can degenerate into malignant eccrine poroma or porocarcinoma
   3. Treatment is surgical excision

E. Verrucous nevus
   1. Closely set skin colored, brown, or gray-brown verrucous papules that may coalesce to form well-demarcated plaques, usually in a linear configuration along skin tension lines
   2. Hyperkeratosis, acanthosis, and papillomatosis on histology
   3. Treatment is excision to the deep dermis. For more extensive lesions not amenable to excision, treatments may include laser cryotherapy and electrodessication dermabrasion

F. Desmoid tumor
   1. Benign tumor arising from the musculoaponeurotic layer of the abdominal wall
   2. Treatment is excision with 1cm margins

G. Keratoacanthoma (Figure 1)
   1. Rapid growth followed by spontaneous regression over several months
   2. Treatment is recommended because it cannot be reliably distinguished from SCCs

H. Cylindroma
   1. Adult-onset nodules, usually on the face or scalp, with smooth, flesh-colored, possibly telangectatic surfaces
   2. For solitary lesions, treat with excision or electrosurgery
3. For grouped lesions, may need staged excision

I. Pyogenic granuloma (Figure 2)
   1. Appears in early childhood, usually following minor trauma, as a rapidly growing, small (<1cm) red lesion
   2. Friable and prone to bleeding
   3. Treatment is excision (including the dermis)

Figure 1. Keratoacanthoma demonstrating rapid growth over a 3-month period.

Figure 2. Pyogenic granuloma. These lesions occur most commonly on the face (above). (Below) This lesion on the left supraclavicular fossa shows evidence of recent bleeding (arrowhead).

J. Xanthelasma Palpebrarum
   1. Present as asymptomatic yellow-orange papules and plaques, commonly on the medial eyelids
2. Highly associated with hyperlipidemia
3. Treatment is surgical excision for cosmetic purposes
4. Correction of underlying hyperlipidemia is largely ineffective in treating xanthelasma

K. Syringoma
1. Presents as asymptomatic, skin-colored to yellow papules and plaques commonly found on the eyelids and upper cheeks
2. Treatment is for cosmetic concerns, and includes laser therapy, cryotherapy, electrodessication, and excision

L. Nevus of Ota (nevus fuscoceruleus ophthalmomaxillaris or oculodermal melanocytosis)
1. Dermal melanocytic hamartoma that demonstrates bluish hyperpigmentation along the ophthalmic and maxillary divisions of the trigeminal nerve
2. Caused by the failure of complete embryonic migration of melanocytes from the neural crest to the epidermis, resulting in dermal nesting with the resultant dermal melanin causing the Tyndall effect
3. Primarily affects darker-pigmented individuals, more prevalent in females
4. Has a bimodal age incidence, with a peak at 1 year of age and a second around puberty
5. Becomes more prominent with age, puberty, and postmenopausal state
6. 10% association with ipsilateral glaucoma – ophthalmologic examination recommended
7. Malignant degeneration to melanoma occurs in approximately 4% of reported cases
8. Treat with laser therapy – either Q-switched laser with ruby (694 nm), alexandrite (755 nm), or neodymium: yttrium-aluminum-garnet (1064 nm)

M. Nevus of Ito
1. Large blue-grey lesion that characteristically arises over the shoulder region and areas innervated by the posterior supraclavicular and lateral cutaneous brachial nerves
2. Treat with pulsed Q-switched laser therapy

N. Spitz Nevus
1. Benign melanocytic nevus that may resemble melanoma
2. Most frequent in children, usually seen in the head and neck region
3. Clinically presents as a well-circumscribed pink papule that rapidly increases in size
4. Pigmented variant (spindled cell nevus of Reed) is dark brown to black in color with pseudopods at the periphery, giving it a “starburst” appearance
5. Treatment is surgical excision

O. Dermatofibroma (benign fibrous histiocytoma)
1. Present as a solitary, firm, hyperpigmented macule or thin papule on the lower extremity
2. Etiology unknown, 4 times more common in women than in men
3. Surgical excision only for symptomatic lesions (can be painful or pruritic)

P. Lipoma
1. Present as soft, rubbery, nontender, slow-growing subcutaneous nodules that are freely movable on palpation
2. Consist of mature adipocytes surrounded by a thin fibrous capsule
3. Treatment – enucleation or surgical excision for symptomatic lesions

Q. Nevus sebaceous (Jadassohn nevus)
   1. Presents as a hairless, solitary, linear, well-demarcated patch or thin plaque that is pink, yellow, orange, or tan in color, usually on the scalp
   2. During adolescence, hormonal changes cause the lesion to thicken and become more verrucous and nodular in appearance
   3. Risk of degeneration to basal cell carcinoma is approximately 15 to 20%
   4. Keratoacanthoma and squamous cell carcinoma may also develop (less frequently)
   5. Because of the risk of malignant transformation, complete excision is recommended prior to puberty

II. PREMALIGNANT AND MALIGNANT SKIN LESIONS

A. Actinic Keratoses
   1. Erythematous, rough, scaly lesions, typically in sun-exposed areas
   2. 25% chance of progression to squamous cell carcinoma
   3. Treatments include topical imiquimod (Aldara), photodynamic therapy with 5-aminolevulinic acid (Levulan), cryotherapy, 5-fluorouracil, retinoids, and diclofenac gel

B. Squamous Cell Carcinoma (SCC)
   1. Presents as an erythematous, scaly or verrucous papule or plaque
   2. Associated with chronic sun exposure and are more commonly seen with lighter skin, increasing age, and tanning bed use
   3. High-risk – poorly defined borders, recurrent lesion, immunosuppressed patient, site of previous radiation/chronic inflammation (Marjolin’s ulcer), rapid growth, neurologic symptoms, invasion to fat, size >2cm, or size >6mm in the central face, ears, scalp, genitalia, hands/feet
   4. Margins
      a. Low-risk: 4-6mm
      b. High-risk: 10mm or Mohs micrographic surgery
   5. May metastasize (most often with lesions on the ear or lip, lesions > 2 cm in size, and in the immunosuppressed population)
   6. Bowen’s Disease – SCC in-situ (full-thickness epidermal atypia that presents as a thin eczematous, erythematous plaque)
   7. Erythroplasia of Queyrat – SCC in-situ of the glans penis

C. Basal Cell Carcinoma (Figure 3)
   1. Presents as a pink, pearly papule with overlying telangiectasia and rolled borders. Ulceration may be present, giving a characteristic “rodent bite” ulcer
   2. Arises on sun-damaged skin of the head, neck, and upper extremities, with an increasing incidence with age, fair skin, chronic sun exposure, and a history of tanning bed use
3. High risk – poorly defined borders, recurrent lesion, immunosuppressed patient, site of previous radiation, peri-neural involvement, aggressive histology (morpheaform, sclerosing, mixed infiltrative, basosquamous, or micronodular), >2cm in the trunk/extremities, or >1cm in the head and neck

4. Treatment options include surgical excision, Moh’s, ED&C, cryosurgery, Imiquimod (for <2cm BCCs of the trunk, extremities, or neck), photodynamic therapy, 5-FU, and radiation therapy

5. Margins
   a. Low-risk: 4mm
   b. High-risk: 6-10mm (or Mohs micrographic surgery)

6. Vismodegib and Sonidegib (a selective inhibitor of hedgehog pathway activation) are approved for the treatment of metastatic basal cell carcinoma and locally advanced basal cell carcinoma that has recurred after surgery, or in patients who are not surgical or radiation therapy candidates

Figure 3. (Above, left) Superficial basal cell carcinoma. (Above, right) Nodular basal cell carcinoma. (Below, left) Infiltrative basal cell carcinoma. (Below, right) Pigmented basal cell carcinoma. From Lee E, et al. Benign and premalignant skin lesions. Plast Reconstr Surg 2010;125(5):188e-98e.

**D. Melanoma (Figure 4)**

1. 3% of all skin cancers, but 65% of all skin cancer deaths
2. Risk factors include fair hair/skin, history of sunburns/sun exposure, and family/personal history of melanoma
3. ABCDE signs of melanoma include
   a. Asymmetry
b. Border irregularity

c. Color variegation

d. Diameter (>6 mm)

e. Enlarging or evolving

4. Superficial spreading melanoma
   a. Most common subtype of melanoma
   b. Occur particularly on sun-exposed skin and often arise in preexisting nevi
   c. Have a prolonged radial growth phase before developing a vertical growth phase
   d. Initially flat, but can become irregular or raised as the lesions grow

5. Nodular melanomas
   a. Second most common form of melanoma
   b. Commonly seen on the trunk, head, and neck, males > females
   c. Domed-shaped, dark, and may resemble a blood blister

6. Lentigo maligna melanoma
   a. Rare subtype seen in only 4 percent of melanomas
   b. Often arise from pre-existing lentigo maligna lesions, which can be present for many years, growing in a slow, radial fashion, before the vertical growth phase develops
   c. Women>men, often located on the face, head, and neck of older individuals
   d. Commonly present as large, tan lesions with convoluted patterns and multiple amelanotic patches

7. Acral Lentiginous Melanoma
   a. Rarest form of melanoma in Caucasians but 30-60 % of melanoma in dark-skinned individuals
   b. Commonly occur in the palms, soles of the feel, and under the nails

8. Amelanotic
   a. Lack pigment and are often mistaken for other lesions

9. Desmoplastic Melanoma
   a. Has aggressive local growth and less frequent nodal metastases
   b. Often confused with common nevi, blue nevi, Spitz nevi, pyogenic granulomas, or hemangiomas

10. Diagnosis
   a. Gold standard is excisional biopsy
   b. Shave biopsy is often performed by dermatologists but can under-estimate depth, although this hasn’t been shown to affect prognosis/survival

11. Treatment is surgical excision, margins are dictated by Breslow depth
   a. Melanoma in-situ (MIS) – 5mm
   b. < 1mm – 1cm
   c. 1.01-2mm – 1-2cm (generally 2cm where allowable such as the trunk and extremities, and 1cm in more aesthetically sensitive areas like the head and neck)
   d. >2.01mm – 2cm

12. Sentinel Lymph Node Biopsy (SLNB)
   a. Indicated for intermediate thickness (1-4mm) melanomas
b. May also be indicated for high-risk thin melanomas 0.75-1mm thick and thick melanomas

13. Completion Lymph Node Dissection (CLND)
   a. Currently indicated in cases of a positive SLN, or a clinically palpable node

14. Medical treatment for melanoma is currently used for advanced melanoma only (usually Stage 3 or 4) and consists of immunomodulation and targeted molecular therapy toward mutations found in melanocytic lesions
   a. Vemurafenib and dabrafenib (BRAF inhibitors, improved survival but develops rapid resistance with associated relapse)
   b. Interleukin-2 (immunomodulator that activates the host immune system to attack malignant cells, severe side effect profile)
   c. Ipilimumab (monoclonal antibody that suppresses CTLA-4, small but durable response, significant immunologic side effects)
   d. Nivolumumab (anti-PD-1 monoclonal antibody)

Figure 4. ABCDE signs of melanoma. A 75-year-old man with a left cheek lesion presenting with asymmetry, borders irregularity, color variation, and diameter of 2.5 cm evolving over an 8-year period. From Dzwierzynski W. Managing malignant melanoma. Plast Reconstr Surg 2013;132(3):446e-60e.

E. Merkel Cell
   1. Presents as a firm, painless nodule (up to 2 cm in diameter) or a mass (>2 cm in diameter), usually in the head and neck region, classically red in color, but may be flesh-colored or blue, and often enlarges rapidly
   2. Risk factors include exposure to sun and ultraviolet light, immunosuppression, and the Merkel cell polyomavirus
   3. Treatment is surgical excision (1-2cm margins down to investing fascia) and sentinel node biopsy, combined with adjuvant radiation therapy to decrease local recurrence rates

F. Verrucous carcinoma
1. A variant of squamous cell carcinoma – requires wide local excision with negative margins for treatment

G. Paget’s Disease of the Breast
1. Presents with eczematous skin changes of the nipple areolar complex
2. Often associated with ipsilateral breast cancer

H. Extramammary Paget Disease
1. Intraepithelial carcinoma involving the vulvar, perianal, perineal, scrotal, and penile regions
2. Presents as well-defined, moist, erythematous plaques associated with pruritis
3. 7 to 40% rate of associated malignancy – treated with wide local excision

I. Dermatofibrosarcoma protuberans (DFSP)
1. Malignant mesenchymal tumor that arises in the dermis and is characterized by latency in its initial detection, slow infiltrative growth, and local recurrence if not adequately treated
2. 90% of DFSP tumors have the chromosomal translocation t(17;22) that fuses the collagen gene COL1A1 with the platelet-derived growth factor gene
3. Most common on the trunk followed by the proximal extremities
4. Treatment is wide surgical excision (2-3 cm margins). Mohs can be used
5. Molecular targeted therapy with imatinib mesylate (Gleevec) is indicated for unresectable, recurrent, or metastatic DFSP

J. Angiosarcoma
1. Appears as a purple plaque, commonly found in the face and scalp in older Caucasian men
2. 50% in the head and neck, and also commonly found in the breast and extremities, particularly in patients with a history of lymphedema or radiation therapy
3. Treatment is wide local excision, but it is frequently multifocal, and local recurrences are common

K. Stewart-Treves Syndrome
1. Lymphangiosarcoma in post-mastectomy patients
2. Diagnosis is via incisional biopsy
3. Treatment includes WLE if possible with margins of at least 1cm, or isolated limb perfusion with tumor necrosis factor and melphalan

III. VASCULAR ANOMALIES

Vascular anomalies may be divided into hemangiomas and vascular malformations. Hemangiomas are vascular tumors characterized by increased cellular proliferation. Classically, they exhibit rapid growth and slow regression. Approximately 80% of hemangiomas are noted in the first month of life, and 60% occur in the head and neck region. Vascular malformations are present at birth and grow slowly.

A. Infantile hemangiomas
1. May be present at birth (30-50%) but usually appear in the first two weeks of life, with 80% appearing in the first month of life
a. Proliferating phase – 0-9 months (with most of the growth achieved by 3 months)
b. Involuting phase – 9 months to 12 years, but is completed usually by age 4

2. Involution leaves some scar or discoloration in 50% of patients
3. Biopsy is rarely indicated, but IHs are GLUT-1 positive on immunostaining
4. Treatment
   a. Small (less than 2-3cm) well localized IHs can use intralesional corticosteroid
   b. Larger problematic lesions can be treated with medical therapy (oral prednisolone or oral propanolol)
   c. Surgery is indicated in the case of:
      i. Failure or contraindication to pharmacotherapy
      ii. A well-localized tumor in an anatomically favorable area
      iii. If resection will be necessary in the future and the scar would be the same
      iv. Lesions (of any stage) that are compromising function or destroying vital structures

B. Congenital hemangiomas
   1. Arise in the fetus, are fully grown at birth, and do not have post-natal growth
   2. Red-violaceous with coarse telangiectasias, central pallor, and a peripheral pale halo
   3. More common in the extremities, have an equal sex distribution, and are solitary with an average diameter of 5 cm
   4. Two forms:
      a. Rapidly involuting congenital hemangioma (RICH)
         i. Involutes rapidly after birth
         ii. 50% of lesions have completed regression by 7 months of age; the remaining tumors are fully involuted by 14 months
         iii. Affects the head or neck (42%), limbs (52%) or trunk (6%)
         iv. Rarely requires treatment but may leave behind atrophic skin
      b. NICH (Non-involuting congenital hemangioma)
         i. Does not regress (remains unchanged with persistent fast-flow)
         ii. Involves the head or neck (43%), limbs (38%), or trunk (19%)
         iii. Resection can be considered if the scar will be less noticeable than the lesion

C. Kaposiform Hemangioendothelioma
   1. Presents as a large (>5cm), superficial, and diffuse lesion, with the overlying skin deep red-purple, tense, painful, and shiny
   2. Typically involves the trunk and extremities, 50% present at birth (but can appear in childhood)
   3. Kasabach-Merritt phenomenon (thrombocytopenia <25,000, bruising, and bleeding) is common
   4. Regression seen after age 2, although long-term chronic pain and stiffness can persist
   5. Diagnosis is by MRI
   6. First-line treatment is vincristine
D. Capillary Malformations
1. Slow-flowing vascular malformation characterized by ectatic vessels located at various levels within the dermis
2. Treatment is pulsed-dye laser therapy

E. Venous Malformations
1. Most common type of vascular malformation
2. Present at birth, slowly increase in size as the child grows, change size with position, and are prone to thrombosis (phlebolith formation)
3. First line therapy is sclerotherapy (STS, ethanol)

F. Lymphatic Malformations (historically called “cystic hygromas”)
1. Characterized as microcystic, macrocystic, or combined
2. Most commonly occur in the cervicofacial region, axilla/chest, mediastinum, retroperitoneum, buttock, and perineum
3. Diagnosis is by MRI
4. Treatment is reserved for symptomatic lesions that cause pain, significant deformity, or threaten vital structures
   a. First line – sclerotherapy (doxycycline, sodium tetradecyl sulfate (STS), ethanol)
   b. Can also use erbium laser, or surgical excision

G. Arteriovenous Malformations
1. High-flow vascular malformations characterized by warmth, pain, bony destruction, discoloration and sometimes ulceration of the overlying skin
2. Treatment is embolization followed by excision 24-72hrs later

REFERENCES

CHAPTER 8

HEAD AND NECK

Lucas Dvoracek, MD and Sanjay Naran, MD

Problems of the head and neck in the practice of plastic surgery include congenital, traumatic, infectious, neoplastic, and other conditions. A working knowledge of the anatomy of the head and neck is crucial in the diagnosis and surgical treatment of these diseases.

I. TRAUMATIC

A. Facial soft tissue injuries
   1. Stabilize patient and manage concomitant traumatic injuries (ABCDE, primary survey)
      a. Establish airway (may be obstructed by blood clots or damaged parts)
         i. Finger sweep / jaw thrust
         ii. Suction
         iii. Endotracheal intubation
         iv. Cricothyroidotomy or tracheotomy
      b. Control active bleeding by pressure, may need direct ligation in operating room or embolization in interventional radiology suite
   2. Palpate facial skeleton for underlying bone pain and instability; rule out injury to facial nerve, parotid duct, etc.
   3. Radiologic evaluation (Maxillofacial CT scan, C-spine CT, panorex)
   4. Tetanus and antibiotic prophylaxis
      a. Open fractures may require an antibiotic course
   5. Repair as soon as patient’s general condition allows with…
      a. Wounds closed preferably less than 8 hours post-injury, but primary closure may be delayed up to 24 hours
      b. Careful wound irrigation with physiologic solution
      c. Conservative debridement of nonviable tissue and foreign bodies
      d. Meticulous re-approximation of anatomy
      e. Definitive fracture fixation within 2 weeks (while fragments remain mobile)

B. Facial bone fractures
   1. Diagnoses
      a. Consider patient history/mechanism of injury
      b. Physical examination for asymmetry, bone mobility, diplopia, extraocular muscle entrapment, sensory loss, malocclusion, local pain
      c. Old (pre-injury) photographs often useful to assess baseline
      d. Imaging
i. Maxillofacial CT scan with thin slices (now imaging modality of choice)
ii. X-rays: Skull (Water’s view, rarely performed today) and cervical spine
iii. Panorex x-ray may be useful for mandible fractures (but maxillofacial CT usually adequate)

e. When dealing with panfacial fracture, stabilize articulating element (mandible), first by mandibulomaxillary fixation (MMF)
f. Once occlusion is aligned, work systematically, either “outside-in” (Gruss) or “inside-out” (Manson), establishing facial height, width, and projection by aligning key facial buttresses (open reduction) and plating of fractures (internal fixation)

2. Specific Fractures
   a. Frontal sinus
      i. Anterior and/or posterior table
      ii. May require Neurosurgery input for posterior table, monitor for CSF leak
      iii. Management depends on pattern, but may be nonoperative, ORIF, cranialization (removal of posterior table and allowing dura and brain to fill sinus) or obliteration (remove mucosa, fill with bone graft)
   
b. Zygomatic complex (ZMC) and Orbit (Figure 1)
      i. ZMC and orbital floor (blow-out) fractures commonly associated
         (a) Eye exam: Extraocular movements to ensure no entrapment, visual acuity, globe injury
         (b) Periorbital ecchymosis often present
         (c) Ophthalmology consultation if suspicious of globe injury
      ii. Superior orbital fissure syndrome
         (a) Due to injury to contents of superior orbital fissure (CN III, IV, VI)
         (b) Ophthalmoplegia (CN III, IV, VI)
         (c) Proptosis
         (d) Ptosis (CN III)
         (e) Dilated pupil (CN III)
         (f) If also blindness (CN II), called orbital apex syndrome (surgery urgent)
      iii. Indications for surgery
         (a) Entrapment or cranial nerve impingement (emergency)
         (b) Enophthalmos
            (i) globe less projected because of increased intraorbital volume
            (ii) best seen from inferior/worm’s eye view
            (iii) usually require surgery if ≥ 2mm relative to unaffected eye
         (c) Hypoglobus: globe lower than opposite side because of decreased orbital floor support
         (d) Severe displacement creating facial asymmetry (sunken or overprojected cheekbone)
c. Nasal bone and Naso-orbital-ethmoidal (NOE)
   i. Nasal bone fracture most common facial fracture
      (a) Septal hematoma can cause septal necrosis; must be drained immediately
      (b) May be corrected by closed reduction/manipulation and placement of external splint and Doyle splints (internal)
   ii. NOE fractures can lead to telecanthus where the medial edge of the eyelid (canthus) moves away from midline (Figure 2)
      (a) May require ORIF to correct

   \[\text{Figure 1. Fracture of the zygomatic complex}\]
   \[\text{Figure 2. Types of NOE Fractures}\]

   d. Maxilla
      i. Le Fort fractures (Figure 3)
(a) Disrupts vertical maxillary buttresses: major areas of structural stability
   (i) Zygomaticomaxillary
   (ii) Nasomaxillary
   (iii) Pterygomaxillary
(b) Treatment involves open reduction and internal fixation with miniplates to reestablish facial proportions and occlusion

Figure 3. LeFort Fractures

(e) Mandible: often bilateral (ring concept), subcondylar most common (Figure 4)
   i. Clinical signs:
      (a) Malocclusion (“Does your bite feel normal?”)
      (b) Sensation of chin decreased due to mental nerve injury
   ii. Imaging
      (a) Maxillofacial CT scan
      (b) Panorex x-ray
      (c) C-spine imaging: 10-13% of mandible fractures coincide with c-spine fracture; maintain C-spine stabilization until absence of injury can be confirmed
   iii. Initial treatment
      (a) Liquid diet
      (b) Antibiotics and chlorhexidine mouthwash for open fracture (including fracture line involving dentition)
      (c) Re-establishment of normal occlusion is of primary importance (See Disorders of the Jaw below)
      (d) Use of interdental wiring (mandibulomaxillary fixation/MMF) in patients with teeth
(e) Use of patient’s dentures or fabricated temporary dentures in edentulous patient
(f) Depending on fracture location, (parasympysis, body, angle, ramus, subcondyle, condyle), open reduction and internal fixation (ORIF) with plates/screws may be indicated.

Figure 4. Subregions of the mandible, and fracture of subregion fracture

f. Pediatric craniofacial fractures
   i. Usually more conservative with operative repair in this patient population, due to growing facial skeleton and developing dentition.

II. INFECTIONS

A. The head and neck are relatively resistant to infection due to their robust vascularity
B. Routes of spread
   1. Upper aerodigestive infections may track into the mediastinum
   2. Scalp and orbital infections may spread intracranially via the dural sinuses and ophthalmic veins
C. Types
   1. Facial cellulitis: mostly due to staphylococcus or streptococcus - may use a cephalosporin
   2. Oral cavity infections: mostly due to anaerobic streptococcus and bacteroides. Use extended spectrum penicillin or other anaerobic coverage (Augmentin/Unasyn)
   3. Acute sialadenitis: fever, pain, swelling over the involved parotid gland. Seen with dehydration, debilitation, diabetics, poor oral hygiene. Treat with antibiotics, fluids
4. Atypical mycobacteria: seen in enlarged lymph nodes; drainage rarely required. Special cultures may be necessary

5. NEOPLASTIC (exclusive of skin - see Chapter 7)

D. Primarily managed by Otolaryngologists but provide major reconstructive challenges for Plastic Surgeons (see next section)

E. Salivary gland tumors
   1. Classification of tumors by location
      a. Parotid: most common (80%), most are benign (80%)
      b. Submandibular: 55% incidence of malignancy
      c. Minor salivary glands: least common, with highest incidence of malignancy (about 75%)
   2. Diagnosis
      a. Primarily by physical examination
         i. Any mass in the pre-auricular region or at the angle of the jaw is a parotid tumor until proven otherwise
      b. Bimanual palpation: simultaneous intraoral and external palpation
      c. Signs more commonly seen with malignancy
         i. Fixed or hard mass
         ii. Pain
         iii. Loss or disturbance of facial nerve function
         iv. Cervical lymphadenopathy (metastases)
   3. Treatment
      a. For benign tumors
         i. Surgical removal of gland with sparing of adjacent nerves, e.g. facial nerve with parotid; lingual and hypoglossal nerves with submandibular
      b. For malignant tumors
         i. Surgical removal of entire gland with sparing of nerve branches that are clearly not involved
         ii. Radiation therapy if tumor not completely removed
         iii. Cervical lymph node dissection with tumors prone to metastasize to nodes
   4. Pathology
      a. Benign
         i. Pleomorphic adenoma: (benign mixed) high recurrence rate with local excision
         ii. Papillary cystadenoma lymphomatosum (Warthin’s tumor): may be bilateral, (10%) male, age 40-70
      b. Malignant
i. Mucoepidermoid
ii. Malignant mixed
iii. Adenocarcinoma

F. Tumors of oral cavity
1. Classification
   a. Anatomical: malignancies behave differently according to anatomic site 
      and prognosis worsens from anterior to posterior
   b. Histopathologic
      i. Benign: fibroma, osteoma, lipoma, cyst, etc.
      ii. Malignant
         (a) Most are squamous cell carcinoma or variants (may be related to 
             HPV)
         (b) Palate carcinomas are often of minor salivary gland origin
         (c) Sarcomas in mandible, tongue, other sites are rare
         (d) TNM staging is helpful for treatment planning and prognosis (i.e. 
             tumor size, lymph node metastases, systemic metastases)

2. Diagnosis
   a. Examination - including indirect laryngoscopy and nasopharyngeal 
      endoscopy when indicated
   b. Biopsy of any lesion unhealed in 2-4 weeks
   c. X-rays and scans as indicated

3. Treatment
   a. Surgical
      i. Benign: simple excision
      ii. Malignant
         (a) Wide local excision with tumor-free margins with/without lymph node 
             dissection
         (b) Palliative resection may be indicated for comfort and hygiene
         (c) Immediate reconstruction with vascularized flaps when indicated 
             by size and location of defect (see next section)
   b. Radiation therapy
      i. Preoperative
         (a) To increase chance for cure, especially with large lesions
         (b) May make an inoperable lesion operable by shrinking it and reducing 
             involvement with unresectable structures
      ii. Postoperative
         (a) If tumor-free margin is questionable
         (b) For recurrence
         (c) Prophylactic — controversial
   c. Chemotherapy: usually for advanced disease
III. RECONSTRUCTION

A. General principles
   1. Goals
      a. Tension free closure of skin and soft tissue
      b. Maintenance of motor and sensory function
      c. Recreation of aesthetic contours
   2. Reconstructive ladder applied including…
      a. Primary closure (often best option if possible)
      b. Skin graft (full thickness for best color and quality match)
      c. Local flaps (often require two stages) and distant flaps
      d. Tissue expansion (longer term process)
      e. Free tissue transfer (lengthy procedures)

B. Scalp
   1. Main goal: protection of cranium/dura/brain
   2. Primary closure often possible, can be assisted with galeal relaxation incisions (scoring)
   3. Many rotation and advancement flap options
   4. Tissue expansion is an excellent option (can allow up to 50% reconstruction without obvious alopecia)

C. Eyelid
   1. Main goal: tension-free coverage of the globe to prevent exposure keratopathy and ectropion (chronic eyelid irritation)
   2. Defect size determines reconstruction options
      a. ≤30% can be closed primarily, performing lateral canthotomy and cantholysis can allow closure of defects up to 50%
      b. >50% requires local flap rearrangements, which often requiring two or more stages
         i. Many options including:
         ii. Cutler-Beard flap: pass tissue from below the lower lid under it and tack it into the upper lid defect
         iii. Mustarde flap: swing tissue from the malar area to the lower lid

D. Nose
   1. Main goal: Create aesthetic piriform aperture coverage and maintain airway patency and nasal lining
   2. Divided into 9 subunits: single dorsum, tip, columella, and paired sidewalls, soft triangles, and alar lobules (Figure 5)
   3. Often complicated by need for cartilaginous support
   4. Numerous local flap options including…
a. Bilobed flap: for defects up to 1.5cm, Y-shaped tissue pivoted to fill defect and one donor site, with the other donor closed primarily
b. Nasolabial flap: tissue from along the cheek-nose junction swung into defect on the nasal ala or sidewall
c. Forehead flap: workhorse two-staged technique where tissue from the central forehead is swung down to reconstruct part or all of the nose

Figure 5. Nasal Subunits

E. Ear
1. Main goals: Primarily aesthetic reconstruction (symmetry important)
2. Many distinct components with specific reconstruction options for each, mostly involving local rotational flaps when primary closure is not an option
3. Often require cartilage from the other ear or rib
4. Total reconstruction: tissue expansion of nearby skin, and then advancement overlying a cartilage graft construct based on the contralateral ear
5. One of the few facial structures where a prosthesis is a good option

F. Lip
1. Main goal: Recreate oral competence and speech with a sensate aesthetic construct
2. Distinct coloration and contour makes local flaps preferred options
3. Perfect alignment of incisions key, or otherwise can be very noticeable
4. Rule of thirds for defects
   a. ≤1/3rd: Primary closure
   b. ≤2/3rds: local flaps
      i. Abbé Lip Switch flap: two-stage, section of lip swung to fill defect in opposite lip
ii. Estlander flap: lateral end of lip swung up to end of opposite lip
c. ≥2/3rds: Karapandzic flap -- circumoral incision to mobilize lip and cheek, may narrow oral opening (microstomia) (Figure 6)

Figure 6. Karapandzic flap illustrating circumoral incision to mobilize lip and cheek

G. Mandible
1. Main goal: restore speech and mastication
2. Size of defect key
   a. <6cm: avascular bone grafts can work, if adequate soft tissue present
   b. >6cm: vascularized bone graft (free flap)
      i. Fibula osteocutaneous flap: workhorse, can be shaped into an entire mandible and can also provide skin and soft tissue for floor of mouth or tongue reconstruction
      ii. Also can use: scapular flap, iliac crest flap
   c. Metal reconstruction plates are an option for patients who cannot tolerate a free flap operation but extrusion through the tissue is a common problem

H. Tongue, floor of mouth, and pharynx
1. Main goals: prevent salivary contact with neck structures, restore swallowing and speech
2. Can use skin grafts for small posterior pharyngeal defects
3. Partial tongue resection can be closed primarily
4. Pectoralis pedicled flap is very reliable for floor of mouth and pharynx
5. Free flap: free radial forearm, anterolateral thigh (ALT) can be tubularized

IV. MISCELLANEOUS

A. Disorders of the jaw
1. Deformities of the mandible
   a. Classification
      i. Retrognathia: mandibular retrusion with respect to maxilla
ii. Prognathia: mandibular protrusion with respect to maxilla
iii. Micrognathia: underdeveloped, retruded mandible
iv. Open bite: teeth cannot be brought into opposition
v. Crossbite: lower teeth lateral to upper teeth
vi. Micro- and microgenia: under or overdevelopment of chin
vii. Normal occlusion: “mesiobuccal cusp of the maxillary first molar aligns with the buccal groove of the mandibular first molar”

b. Diagnosis
i. Physical examination
ii. X-rays, including a cephalogram (lateral x-ray at a fixed distance) to measure relationships of skull, maxilla and mandible
iii. Dental casts are made (usually by an orthodontist) and “model” or mock surgery is performed on the casts to determine degree of advancement/setback of bone.

c. Treatment
i. Establishment of normal or near normal occlusion of primary importance
ii. Use of osteotomies with repositioning of bone segments, bone grafts as needed, with or without orthodontic corrective measures as needed
iii. Mandibular distraction for severe discrepancies

2. Deformities of the maxilla
a. Most commonly, retrusions or underdevelopment, “dish-face”
b. Must also examine the vertical height of the midface (vertical maxillary excess, VME versus vertical maxillary deficiency, VMD)
c. Diagnosis - as for lower jaw
d. Treatment - as for lower jaw

3. Temporomandibular joint disorder
a. Etiology: previous trauma, arthritis, bone overgrowth, bruxism, tumors
b. Symptoms: pain, crepitus, joint noises, limited opening, occlusion change
c. Diagnosis
i. Consider patient history
ii. Examination: auscultation, opening, occlusion
iii. Imaging
   (a) Tomograms, arthrogram/arthroscopy, MRI
d. Treatment
i. Conservative: joint rest, analgesics, bite plate, etc.
ii. Surgery: seldom indicated

B. Facial paralysis
1. Effects:
a. Very significant asymmetry, deformity of the face, drooling, exposure of the cornea
b. Deformity is accentuated by muscle activity of normal side (if unilateral)

2. Etiology
   a. Congenital
   b. Traumatic
   c. Infectious
   d. Tumor
   e. Vascular (intracranial)
   f. Idiopathic (Bell’s palsy)

3. Diagnosis
   a. Demonstrated by asking patient to raise eyebrow, smile, etc.

4. Treatment includes:
   a. Correct known etiology or supportive (for most Bell’s palsies)
   b. Protect cornea by taping lids, lid adhesions: ophthalmology consultation is critical
   c. Re-establishment of nerve function by primary nerve repair or nerve graft (sural nerve common donor nerve)
      i. Can be done cross-facially by rerouting motor axons from unaffected side but takes months to see improvement and denervated muscles may become unsalvageable
   d. Other measures, such as muscle transfers, static suspension, skin resections, free tissue transfers of muscle, etc.
      i. Free gracilis muscle flap to recreate smile with cross-facial nerve graft or adjacent nerve to masseter innervation (CN V)

REFERENCES


CHAPTER 9

PEDIATRIC PLASTIC SURGERY

Liliana Camison, MD and Sanjay Naran, MD

Children are not just small adults. Conditions, treatment principles, and concerns for certain complications are different and must be considered. The most common pediatric plastic surgery problems comprise congenital craniofacial anomalies, acquired craniofacial problems (such as facial fractures), brachial plexus injuries, hand anomalies, congenital nevi, and vascular malformations. Cleft lip and palate are discussed in a separate chapter.

I. CONGENITAL CRANIOFACIAL ANOMALIES

A. Craniofacial Embryology and Development:
   1. Facial embryology
      a. Facial development occurs between 3-8 weeks of development
      b. Face originates from 5 prominences that appear during 4th week (Figure 1)
         i. Central frontonasal prominence, arising from mesenchyme ventral to forebrain
         ii. Paired maxillary prominences
         iii. Paired mandibular prominences
      c. Paired maxillary and mandibular prominences both arise from neural crest cells migrating from 1st branchial arch
      d. These prominences surround the primitive mouth (stomodeum)
      e. Prominences give rise to
         i. Frontonasal prominence, which divides into:
            (a) Medial nasal process: nasal tip, columella, philtrum and premaxilla
            (b) Lateral nasal process: nasal alae
         ii. Maxillary prominences: upper jaw, upper lip (lateral to philtrum), orbital floor, inferior portion of lateral nasal wall
         iii. Mandibular prominence: lower jaw
      f. Between 5-6 weeks nasal processes enlarge, migrate and coalesce in midline to unite with maxillary process and form upper lip.
Craniofacial embryology. The five facial prominences.


2. Cranial development
   a. Cranium divided into
      i. Neurocranium: structures that surround the brain
      ii. Viscerocranium: structures that surround oral cavity, pharynx, upper respiratory system and face
   b. Ossification
      i. Cranial base (occipital, sphenoid and temporal bones): endochondral ossification
      ii. Cranial vault: intramembranous ossification
   c. Cranial growth responds to increased brain volume
      i. Brain size triples by 1 year
      ii. Brain size quadruples by 2 years
      iii. Cranial vault is 90% of adult size by age 5
      iv. Brain has reached 95% of adult size by age 8-10
   d. Normal cranial growth occurs through
      i. Suture growth: perpendicular to suture
      ii. Appositional growth: bone resorption of the inner surface and bone deposition on the outer surface

B. Craniosynostosis (CS)
   1. Definition: Premature fusion of one or more cranial vault sutures.
   2. Incidence: 1:2,500 live births
   3. Normal suture and fontanelle closure
      a. Suture fusion:
         iii. Metopic: 6-8 months
         i. Sagittal: 22 years
         ii. Coronal: 24 years
         iii. Lambdoid: 26 years
      b. Fontanelle closure:
         i. Posterior: 3-6 months
         ii. Anterior: 9-12 months
4. Characteristic head shape according to suture affected (Figure 2)
   a. Sagittal: scaphocephaly (Gr., *scapho*, meaning boat-shaped)
   b. Metopic: trigonocephaly (Gr., *trigono*, meaning triangular or keel-shaped)
   c. Coronal, bilateral: brachycephaly (Gr., *brachy*, meaning short in AP direction)
   d. Coronal, unilateral: plagiocephaly (Gr., *plagios*, meaning oblique or slanted.
   e. Important: distinguish from *positional* plagiocephaly, where suture is normal. Head deformity is similar, but there is a parallelogram configuration of the head (if seen from above) with anterior displacement of the ipsilateral ear and occipital flattening.

![Figure 2. Craniosynostosis and cranial deformities depending on suture fused](image)

5. Categorized into non-syndromic and syndromic types

6. Non-syndromic CS (most common, 67-80%)
   a. Order of frequency according to suture type
      i. Sagittal (40-50%)
      ii. Metopic (25%)
      iii. Coronal (5-10% bilateral; 15-20% unilateral)
      iv. Lambdoid (<3%)
   b. Longstanding debate as to whether non-syndromic patients have increased incidence of developmental delay. New evidence shows some degree of executive dysfunction in up to 50% of these children.
   c. Treatment indications:
      i. Prevent potential increased intracranial pressure (ICP)
      ii. Correct the cranial deformity and normalize appearance
   e. Treatment:
      i. Vary depending on suture affected and severity
ii. Usually performed within first year of life to take advantage of molding capacity of skull
iii. Minimally invasive procedures: extended strip suturectomy (<6 months) +/- springs, +/- postoperative helmet therapy.
iv. Anterior vault reshaping (fronto-orbital advancement (FOA)/reshaping)
v. Total vault reshaping
vi. Posterior vault reshaping

7. Syndromic CS
   a. Higher incidence of ICP than non-syndromic
   b. Major associated syndromes:
      i. Apert: CS, exorbitism, severe midfacial retrusion, complex syndactyly of the 2-4 digits of the hands/feet.
      ii. Crouzon: CS, exorbitism, midfacial retrusion, no limb anomalies.
      iii. Pfeiffer: CS, exorbitism, midfacial retrusion, broad thumbs and toes)
   c. Characteristic head shape involves turibrachycephaly (Gr., turri, tower)
   d. 50% of Apert syndrome patients have substantial mental delay; Crouzon and Pfeiffer syndrome patients usually have normal mental development.
   e. Genetic defect identified in fibroblast growth factor receptor (FGFR) genes (Apert, Crouzon---FGFR2, Pfeiffer---FGFR1)
   f. Goals of surgery
      i. Expand intracranial volume (83% ICP in Apert, less in others)
      ii. Normalize head shape and appearance
      iii. Correct profound exorbitism to prevent corneal exposure/blindness
      iv. Correct malocclusion
   g. General timeline of surgical interventions: may vary
      i. Suturectomy for decompression of elevated ICP, if present (<3 months)
      ii. Posterior cranial vault distraction/remodeling (6-12 months)
      iii. Anterior/total vault reshaping/fronto-orbital advancement (FOA) (4-12 months)
      iv. Midface procedures: Le Fort III or monobloc advancement (4-12 years).
      v. Orthognathic surgery/canthopexies/other revisions. (12 years - adulthood)
      vi. In general, craniofacial distraction leads to greater advancement, less relapse than conventional procedures.

C. Craniofacial Clefts
   1. Lack of fusion of facial processes that results in abnormal separation of skeletal and soft tissue structures of the face and cranium (alternative theory of lack of mesodermal penetration)
   2. Rare. Estimated 1.4 – 5.1 per 100,000 births
   3. Defined by Paul Tessier, who classified them
   4. Tessier Classification system relates soft tissue to skeletal landmarks (Figure 3)
   5. Any combination of clefts is possible
   6. Facial dysostoses are associated with certain clefts:
      a. Craniofacial/hemifacial microsomia: #7 Cleft (most common facial cleft)
      b. Treacher Collins: Clefts # 6, 7, 8
Figure 3. Tessier Classification of Orofacial Clefts. Soft tissue clefts (above) and bony clefts (below).

D. Facial Dysostoses
1. Treacher Collins Syndrome (Mandibulofacial Dysostosis)
   a. Rare, autosomal dominant, variable penetrance disorder
   b. Affected gene on chromosome 5q
   c. 1st branchial arch, groove and pouch affected
   d. Defined by the bilateral presence of three Tessier Clefts #6, 7, 8, which result in all the phenotypic manifestations
   e. Clinical manifestations:
      i. Hypoplasia/aplasia of the zygomatic arch
         (a) Lateral orbit deficiency
         (b) Midface retrusion
         (c) Lateral canthus hypoplasia/downward slanting palpebral fissures
      ii. Hypoplasia of temporalis muscle
      iii. Coloboma and retraction of lower lid
      iv. Variable ear malformations (microtia/anotia) and deafness
      v. Mandibular hypoplasia with microretrognathia
         (a) Airway compromise due to narrow pharyngeal diameter
         (b) Require tracheostomy and distraction of mandible
vi. Parrot-beak nose +/- choanal atresia  

vii. Normal intelligence  

f. Treatment  
i. Skeletal and soft tissue augmentation of deficient areas with autologous bone grafts (calvaria, rib, iliac crest) and autologous fat/tissue transfer, respectively.  

ii. Mandibular distraction may be necessary for achieving a stable airway  

2. Craniofacial Microsomia (a.k.a. Hemifacial Microsomia)  
a. Spectrum of morphogenetic abnormalities affecting the cranial skeleton, soft tissues and neuromuscular structures derived from the 1st and 2nd branchial arches  
b. Theory is that problems derive from hematoma or thrombosis of stapedial artery  
c. Third-most common congenital malformation (following club foot and cleft lip and palate)  
d. 3:2 male>female  
e. 1:3500-5600 live births affected  
f. Usually unilateral (therefore the more common name hemifacial microsomia)  
g. Manifestations  
i. Hypoplasia of mandibular ramus (uni or bilateral)  
   (a) +/- hypoplasia of the maxilla, zygoma and temporal bone  
   (b) Deviated chin  
   (c) Tilted occlusal plane – malocclusion  

ii. Microtia +/- hearing loss  

iii. Paresis of CN V or CN VII common  

h. Associated with Tessier #7 facial cleft  
i. Pruzansky classification:  
i. I: Small ramus with identifiable anatomy  
ii. II: A functioning TMJ but with abnormal shape and glenoid fossa (IIA glenoid fossa is in acceptable functional position; IIB TMJ is abnormally placed)  
iii. III: Absent ramus and non-existent glenoid fossa  

j. Treatment  
i. Augment deficient areas  
   (a) Skeletal: autologous bone (calvarium, rib, iliac crest)  
   (b) Soft tissue: free flap and/or fat grafting  

ii. Mandibular correction depends upon severity of hypoplasia. Distraction may be necessary to achieve correction of malocclusion versus conventional orthognathic procedures to correct jaw discrepancies in adolescence.  

iii. Bone-anchored hearing aids  

3. Goldenhar Syndrome  
a. Variant of craniofacial/hemifacial microsomia with additional manifestations  
b. Only 5% of hemifacial microsomia cases have these features  
c. Sporadic occurrence
d. Also known as oculo-auriculo-vertebral (OAV) spectrum

e. Manifestations
   i. Prominent frontal bossing, low hairline
   ii. Low set ears
   iii. Anterior accessory auricular appendages
   iv. Colobomas of upper eyelid
   v. Hemifacial microsomia
   vi. Vertebral spine abnormalities
   vii. Abnormalities of heart, kidneys, lungs

E. Pierre Robin Sequence
   1. 1:8,500-20,000 live births
   2. Triad of
      a. Micrognathia
      b. Glossoptosis (tongue retruded back into the pharynx)
      c. Upper airway obstruction
   3. Not a syndrome per se, but a sequence:
      a. Intrinsic/extrinsic disturbance of mandible development (leads to micrognathia)
      b. Arrest of rotation/descent of tongue from between palatal shelves
      c. Lack of palatal fusion or high, U-shaped palate
      d. Tongue remains pressed against posterior pharynx = airway obstruction and feeding issues
   4. May be isolated or associated with certain craniofacial syndromes
      a. Stickler
      b. Nager
      c. Treacher-Collins
   5. Immediate treatment goals in the neonate are establishing a patent airway and improving feeding. Four treatment modalities available
      b. Tongue-lip adhesion: old technique; temporary suturing of tongue to lower lip to address glossoptosis.
      c. Tracheostomy: immediate airway control in emergent scenario, but significant morbidity. Currently used as last resort.
      d. Mandibular distraction: newest procedure preferred by most centers where available. Addresses mandibular hypoplasia; tongue moves anteriorly and relieves airway obstruction.
   6. Requirements
      a. Continuous pulse oximetry and airway monitoring
      b. Multidisciplinary evaluation by genetics, ophthalmology, PRS, feeding specialist
      c. Laryngoscopy/bronchoscopy
      d. Polysomnogram (sleep apnea)

F. Other Embryologic Defects
   4. Branchial cyst, sinus, or fistula
a. Epithelial-lined tract frequently in the lateral neck presenting along the anterior border of the sternocleidomastoid muscle.
b. May present as a cyst or as a sinus connected with either the skin or oropharynx, or as a fistula between both skin and oropharynx openings
c. Treatment: excision
5. Thyroglossal duct cyst or sinus
   a. Cyst in the mid-anterior neck over or just below the hyoid bone, with or without a sinus tract to the base of the tongue (foramen cecum).
   b. Treatment: excision
6. Ear Deformities
   a. Congenital
      i. Anotia: Complete absence; very rare
      ii. Microtia: Vestigial remnants or absence of part of ear
      iii. May present with other mandibular deformities, such as hemifacial microsomia or Treacher Collins
      iv. Abnormalities of position (prominent ears)
      b. Treatment
         i. Anotia or microtia
            (a) Brent technique (several modifications exist): Construction of sculpted ear framework from autologous cartilage graft that is buried under mastoid skin. Usually requires 2-4 operations starting at age 6-8, when rib cartilage is big enough.
            (b) Synthetic implant covered by vascularized fascial flap (Medpor). Risk of extrusion from minor trauma.
         ii. Prominent ears: creation of an antihelical fold and/or repositioning/reduction of concha
         iii. Traumatic loss of part or all of ear: treated similarly to microtia/anotia
         iv. Use of a prosthetic ear may be indicated in some patients

II. PEDIATRIC FACIAL FRACTURES

A. Epidemiology
   1. Uncommon. Comprise less than 15% of all facial fractures.
   2. Frequency increases with age
B. Unique characteristics in children:
   1. At birth, ratio of cranial:facial volume is 8:1; by completion of growth it becomes 2.5:1. This changing ratio produces variation in fracture site frequency with age.
   2. Protective anatomical factors:
      a. Larger fat pads
      b. Decreased pneumatization of sinuses
      c. Skeletal flexibility
      d. Compliant sutures
   3. Fractures that entrap orbital contents (trap-door fractures) are more common secondary to greater bony elasticity
4. Up to 75% can have associated serious trauma (especially cervical spine, neurological, ophthalmologic and abdominal)

C. Causes:
   1. Most common overall (per National Trauma Databank): Motor vehicle crashes (MVC), violence and falls
   2. Some variation by age:
      a. 0-5 years: falls
      b. 6-11: MVC and play or sports
      c. 12-18 years: sports and violence

D. Fracture sites:
   1. Vary with age
   2. Most common overall are orbital or mandible
   3. Cranial vault fractures more common in younger children
   4. Facial fractures more common as they grow older, following midface growth (maxilla, zygomaticomaxillary complex, nasal, mandible)

E. General treatment principles (see Chapter 8):
   1. Growing skeleton possesses inherent plasticity that may render operative intervention unnecessary for a given injury
   2. Usually more conservative with operative repair in this patient population due to fear of altering growing facial skeleton and developing dentition

III. BRACHIAL PLEXUS BIRTH PALSY

A. Anatomy
   1. Spinal roots: C5-T1
   2. Trunks: Upper, middle and lower
   3. Divisions: Anterior and posterior from each trunk
   4. Cords: Lateral, posterior and medial
   5. Terminal branches: Major peripheral nerves of upper extremity: myocutaneous, axillary, radial, ulnar, median

B. Incidence: 1.5 per 100 full-term births

C. Risk factors
   1. Shoulder dystocia
   2. Forceps delivery
   3. Gestational diabetes (macrosomia)
   4. Breech delivery

D. Presentation
   1. Upper root cervical injury in 73%, or Erb-Duchenne palsy
      a. Caused by injury in Erb’s point (convergence of C5-C6 roots as they form upper trunk)
      b. “Waiter’s tip” appearance: lack of deltoid, supraspinatus and biceps functions, causing the arm to hand straight down at side with forearm pronated

E. Evaluation
   1. Passive and active ROM
a. Evaluate with reflexes, such as Moro
2. Imaging usually not helpful
3. Physical therapy to preserve ROM
F. Indications for surgical exploration
   1. Absent biceps or deltoid function by 3-6 months
   2. Absent elbow flexion, wrist, thumb or finger extension by 9 months
   3. Flail limb with Horner’s syndrome
G. Follow patients longitudinally. Partial recoveries might need tendon transfers or nerve grafting (controversial)
H. 90% will have spontaneous resolution within 2 months. If biceps function recovered by 6 months, near-normal function can be expected.

IV. CONGENITAL HAND ANOMALIES

A. Total body examination essential to look for associated deformities and syndromes
B. Upper limbs develop from weeks 5-8
C. Several pathologies exist; many are uncommon. Most common are syndactyly and polydactyly.
D. Syndactyly: congenital fusion of digits
   1. Incidence
      a. 1:200 live births
      b. 10:1 whites > blacks
      c. 2:1 males > females
      d. Bilateral = unilateral
      e. Long + ring finger most common (57%); thumb + index finger most rare (3%)
   2. May be familial (15-40% cases), sporadic simple, or associated with a syndrome (Apert, Poland)
   3. Types
      a. Simple: no bony fusion
      b. Complex: bony fusion
      c. Complicated: associated with syndrome
      d. Incomplete: web recessed
      e. Complete: syndactyly to fingertip
   4. In simple form: ligaments usually normal. Duplicated tendons, nerves, sheaths
   5. In complex or complicated form: various fusion levels, fingernail synechia, abnormal tendons
   6. Evaluation: careful physical examination and X-rays (critical)
   7. Treatment: surgical. Dozens of techniques exist
      a. Usually reassurance and waiting until hand is larger (6-12 months)
      b. Earlier if growth will worsen deformity
E. Polydactyly: more than 5 digits in one hand
   1. Very common
   2. 10:1 African descent > whites
   3. Usually sporadic
4. Ulnar polydactyly (postaxial)
   a. African descent > whites
   b. Classification
      i. Type A: supernumerary digit well developed. Syndromic association in 29%.
      ii. Type B: digit rudimentary and pedunculated. Rarely associated conditions.
   c. Treatment:
      i. Narrow stalk: ligation and autoamputation
      ii. Type A requires operative separation with transfer of structures to adjacent finger
5. Radial polydactyly (preaxial)
   a. May be associated with systemic conditions
   b. Whites > African descent
   c. Radiographic evaluation necessary to determine point of thumb duplication
   d. Surgical reconstruction is more complex as it rarely a “floating finger”
   e. Wassel Classification:
      i. Type I: bifid distal phalanx (3%)
      ii. Type II: supplecated distal phalanx (15%)
      iii. Type III: Bifid proximal phalanx
      iv. Type IV: Duplicated proximal phalanx (43%)
      v. Type V: Bifid metacarpal
      vi. Type VI: Duplicated metacarpal
      vii. Type VII: Triphalangism
   f. Treatment
      i. Floating finger and narrow stalk: ligation
      ii. All others: surgical but wait until 6-18 months. Ulnar side is preserved to keep ulnar collateral ligament

V. VASCULAR ANOMALIES

A. Divided in tumors vs. malformations. Differentiate through physiology:
   1. Tumors: endothelial cell proliferation, malignant or benign (hemangiomas)
   2. Malformations: congenital malformation of vessels (venous, capillary, lymphatic, arteriovenous)

B. Infantile hemangioma
   1. Most common benign tumor of infancy
   2. Incidence 4-10% by 1 year
   3. Female: male 3:1
   4. 60% in head and neck, 25% in trunk
   5. Appears in first weeks of life as a telangiectasia or clustered pinhead red lesions
   6. Phases:
      a. Proliferative: rapid evolution until 0-12 months. 80% of tumor size by 5 months.
b. Involuting phase: lasts 1 to 10 years. Tumor shrinks, color fades, lesion flattens
c. Involuted phase: involution complete at age >10 years. Half of cases will have residual atrophy and contour deformity.

7. Treatment
   a. Conservative: total involution occurs in 50% by 5 years, 70% by 7 and 90% by 9.
   b. Propranolol: induction of apoptosis and fat development
   c. Intralesional steroids: in growth phase they can arrest growth but will not regress. Oral when lesion too large.
   d. Laser: pulsed light and Nd:YAG lasers, mainly for ulcerated hemangiomas and for residual color
   e. Surgery:
      i. Urgent if hemangioma threatens important structures or function (i.e., visual, nasolaryngeal or auditory obstruction)
      ii. Typically delayed until school age otherwise to give time for regression

8. Complications
   a. Bleeding
   b. Ulceration
   c. Infection
d. Kasabach-Merrit: Profound thrombocytopenia with kaposiform hemangioendothelioma
e. High output heart failure if large visceral hemangiomas
f. Emotional distress

9. Associated disorder: Congenital hemangioma
   a. Fully grown at birth
   b. Two forms:
      i. Rapidly involuting congenital hemangioma (RICH): disappears by first year
      ii. Non-involuting congenital hemangioma (NICH): does not respond to pharmacotherapy

C. Vascular malformations
1. Structural and morphologic anomalies resulting from faulty embryologic development
2. Present at birth, grow proportionately with the child, and do NOT regress, unlike hemangiomas
3. Capillary malformations or Port-Wine stains:
   a. 0.3% newborns; females > males 3:1
   b. Face in 80%
   c. Usually affect distribution of V1-V2
d. Treatment: photocoagulation with laser +/- pharmacologic with imiquimod (antiangiogenesis)
e. Associated with
   i. Sturge-Weber syndrome: may be associated with ocular and CNS anomalies
ii. Klippel-Trenauney: in extremity, overlying deeper venous malformation and skeletal hypertrophy
iii. Parkes Weber syndrome: similar to previous, but with associated AV fistula

4. Venous malformation
a. Incidence 1-4%
b. Appear as bluish/purple lesions with spongy texture that swell with dependency and deflate with elevation
c. Treatment: sclerotherapy, compression garments for symptoms, Nd:YAG or argon lasers, surgical resection

5. Lymphatic malformations
a. Previously called hygroma.
b. Can be Macrocystic or microcystic.
c. Soft and compressible
d. Can cause bony overgrowth
e. Combined venous-lymphatic are common
f. Get frequently infected. Aggressive antibiotics crucial

6. Arteriovenous malformations
a. Pulsatile high-flow lesion
b. Anatomy and hemodynamics defined by angiography
c. Varying clinical states:
   i. Quiescent, appearing as only a pink stain
ii. Expansive, with thrill and dilated venous network
iii. Destructive, with cutaneous ulcers, necrosis, bleeding
iv. Decompensated, causing cardiac compromise
d. Treatment: embolization prior to surgical resection; wide local excision (high recurrence), ischemic suture techniques.
e. Complications
   i. Consumptive coagulopathy
ii. Heart failure
iii. Local destruction of anatomy
iv. Bleeding
REFERENCES


CHAPTER 10

CLEFT LIP AND PALATE

Chen Yan, MD and Sanjay Naran, MD

I. ANATOMY AND DEFINITIONS

A. Cleft Lip (CL) alone, Cleft Lip with Cleft Palate (CLP), and Cleft Palate (CP) alone
   1. CL alone and CLP are along the same spectrum of morphology
   2. CP alone is a different entity

   ![Figure 1. Basic anatomy of the palate](image)

   Figure 1. Basic anatomy of the palate

   3. CL occurs anterior to incisive foramen and can involve alveolus, due to failure of fusion of medial nasal processes and maxillary prominence at 4-6 weeks gestation
   4. CP divided into primary and secondary
      a. Primary CP is anterior to incisive foramen, due to failure of fusion of medial and lateral palatine processes
         i. Always involved in CLP
      b. Secondary CP is posterior to incisive foramen, due to failure of fusion of lateral palatine processes at 7-12 weeks gestation
         i. CP alone occurs in this region and soft palate, CLP can extend into secondary palate as well

B. Functional Deficits
   1. Cleft lip
      a. Cannot form fluid/air seal in eating/speech
b. Malocclusion of teeth  
c. Cosmetic deformity with lack of continuity of muscle, skin, mucosa, nasal distortion

2. Cleft palate  
a. Cannot separate nose from mouth so air escapes during speech (velopharyngeal insufficiency)  
b. Cannot suck on breast/bottle well due to poor seal for intraoral negative pressure  
c. Middle ear disease/infections, often chronic

II. CLASSIFICATION

A. CL divided into unilateral/bilateral and incomplete/complete (Figure 2)

![Figure 2. Classification of lip clefts](image)

B. CP alone may be divided into incomplete/complete (Figure 3)  
C. Complete CLP divided into unilateral/bilateral (Figure 3)

![Figure 3. Classification of isolated cleft palates, and combination cleft lip and palates](image)
III. DEMOGRAPHICS

A. Incidence/Demographics of CL alone/CLP
   1. 1:1000 in Caucasians
   2. 1:500 in Asians
   3. 1:2000 in those with African ancestry
   4. 2:1 males:females

B. Incidence/Demographics of CP alone
   1. 0.5:1000 without ethnic variation
   2. 1:2 males:females

C. Genetics
   1. Offspring occurrence risk (Table 1)
      a. Most cases of CL alone or CLP are sporadic, multifactorial, no isolated genetic cause, with only <15% syndromic
         i. Van der Woude’s syndrome, most common syndrome associated with CL
         ii. Multifactorial risk factors include fetal exposure to drugs (phenytoin, EtOH, phenobarbital, diazepam), maternal smoking, advanced paternal age
      b. CP alone is often syndromic
         i. DiGeorge syndrome (most common) and Stickler syndrome

<table>
<thead>
<tr>
<th>Affected Relatives</th>
<th>Predicted Outcomes</th>
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<tbody>
<tr>
<td><strong>CL +/- P</strong></td>
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<tr>
<td>One sibling</td>
<td>= 4%</td>
</tr>
<tr>
<td>One Parent</td>
<td>= 4%</td>
</tr>
<tr>
<td>Sibling and a Parent</td>
<td>= 16%</td>
</tr>
<tr>
<td><strong>CP</strong></td>
<td></td>
</tr>
<tr>
<td>One sibling</td>
<td>= 2-4%</td>
</tr>
<tr>
<td>One Parent</td>
<td>= 2-4%</td>
</tr>
<tr>
<td>Sibling and a Parent</td>
<td>= 15%</td>
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Table 1. Risk of CL +/- P, or CP. Note if congenital lip pits are present, inheritance is autosomal dominant with variable penetrance (Van der Woude’s Syndrome)

IV. TREATMENT
A. Multidisciplinary Care
1. Plastic surgeon, dentist, orthodontist, audiologist, geneticist, social worker, speech/swallow therapist, nutritionist, otolaryngologist (middle ear issues), psychologist, pediatrician

B. Pre-operative techniques to bring cleft closer together
1. Taping across lip segments
2. Lip adhesion by suturing cleft margins together, with definitive repair later
3. Nasoalveolar molding with pre-fabricated oral appliance and weekly adjustment to bring cleft segments closer together, can lengthen columella

C. Goals and Timing of Surgery
1. CL repair – 2-3 months
   a. Repair skin, muscle and mucosa to restore continuity of lip, symmetrical length and function
   b. Simultaneous repair of both sides of a bilateral CL
   c. Can correct nasal deformity at time of CL repair
   d. Millard rotation-advancement repair is most commonly used unilateral CL repair in USA
2. CP repair – 9-15 months
   a. One stage repair of both hard and soft palate
   b. Goal to separate oral and nasal cavities, lengthen palate, and reposition muscles in proper orientation (anomalous insertion of levator veli palatini (LVP) onto the posterior edge of the hard palate rather than decussating in midline)
   c. Furlow palatoplasty (double-opposing Z-plasty) allows for both oral and nasal closure, with lengthening of soft palate and realignment of LVP
3. CL and nasal revisions – 2-4 years
4. Alveolar cleft bone grafting – 6-8 years
   a. Performed at time of eruption of permanent maxillary canines, using most commonly iliac bone graft to fill alveolar defect
   b. Provides bony support to nasal base and incoming permanent teeth
5. Orthognathic surgery to correct malocclusion of teeth – 15-18 years
   a. Lefort I osteotomy to advance maxilla to correct hypoplasia and reach normal occlusion, aided by orthodontics
6. CL revisions and formal rhinoplasty – 14-18 years

REFERENCES
CHAPTER 11

FACIAL PARALYSIS

Shailesh Agarwal, MD and Arash Momeni, MD

The facial nerve innervates a total of 23 paired muscles and the orbicularis oris muscle. The majority of muscles innervated by the facial nerve act in facial expression, and, thus, are critical structures for communication. As a result, facial paralysis can pose both social and physical consequences for the patient. While numerous procedures for facial reanimation exist, it is important to acknowledge that no reconstructive technique is able to reproduce the delicate balance between all mimetic muscles.

I. FACIAL NERVE ANATOMY (CRANIAL NERVE VII)

A. Exits skull base from the stylomastoid foramen
B. Derived from the 2nd branchial arch
C. Landmarks for identification
   1. Tympanomastoid suture – medial to the suture line
   2. Tragus – 1 cm inferior and deep to the tragal pointer
   3. Posterior belly of the digastric muscle – Found midway between muscle and styloid process
D. Travels within the substance of the parotid gland
E. Parasympathetic innervation – Lacrimal, submandibular, and sublingual glands
F. Sensory innervation – Tympanic membrane, external auditory canal, and auricular concha
G. Taste – Anterior 2/3 of the tongue
H. 5 motor branches which control facial mimetic muscles (Figure 1)
   1. Temporal branch
      a. Within the temporoparietal fascia (Figure 2, 3)
      b. Falls on a line from 0.5 cm below tragus to 1.5 cm above the lateral brow (Pitanguy’s line)
      c. Brow depression/elevation
   2. Zygomatic branch
      a. Eyelid closure
      b. Elevation of the oral commissure
   3. Buccal branch
      a. Elevation of the oral commissure
      b. Upper lip elevation
      c. Nostril flaring and compression
      d. Cheek compression
   4. Marginal mandibular nerve
      a. Superficial to the facial artery/vein
      b. Above the inferior border of the mandible when anterior to the facial vessels
      c. Oral commissure depression
d. Chin elevation/dimpling

5. Cervical branch
   a. Oral commissure depression


Figure 3. Anatomic course of the frontal (temporal) branch of the facial nerve. From Agarwal C, et al. The course of the frontal branch of the facial nerve in relation to fascial planes: An anatomic study. Plast Reconstr Surg 2010;125:532-7.

II. MIMETIC MUSCLES

A. Constrictors or expanders of sphincters
   1. Elevators or depressors expand
   2. Constrictors
      a. Orbicularis oculi
      b. Orbicularis oris
      c. Buccinator
      d. Nasalis

B. Innervation from deep surface for most mimetic muscles. Exceptions are buccinators, levator anguli oris, and mentalis which are innervated on superficial surface.

III. CAUSES OF FACIAL PARALYSIS

A. Idiopathic
   1. Bell’s palsy
      a. Most common cause of unilateral facial palsy in adults
      b. Not addressed surgically; managed with steroids and antivirals
      c. May take up to 6 months to resolve
B. Trauma
   1. Neurotransmitter depletion within 72 hours after injury
   2. Repair should be performed within 72 hours so that nerve stimulator can be used
   3. Facial nerve arborizes anterior to the lateral canthus; repair in trauma anterior to lateral canthus is not generally performed. Redundancy exists among muscle innervation by multiple arborizing branches; Exception: temporal and marginal mandibular branches.
   4. Frey syndrome – “gustatory sweating” – is a result of injury to the auriculotemporal nerve, a branch of the trigeminal nerve
C. Infectious
   1. Herpes zoster (Ramsey-Hunt-Syndrome)
   2. Lyme disease – may be bilateral
D. Tumors - Cholesteatoma, neurofibromas, meningioma
E. Congenital - Moebius syndrome
   1. CN VI and VII
   2. CN VI controls eye abduction
F. Iatrogenic – Tumor extirpation or traumatic delivery (facial nerve is more superficial in children)

IV. HISTORY AND PHYSICAL EXAM

A. History
   1. What are the signs/symptoms concerning for the patient?
   2. When did the signs/symptoms begin?
   3. Any inciting factors related to causes (see above)
B. Physical Exam
   1. Forehead/frontalis muscle function
      a. Eyebrow elevation
      b. Symmetric/asymmetric forehead rhytids
      c. Unilateral versus bilateral
   2. Eyes and Eyelids/orbicularis oculi muscle function
      a. Facial nerve controls eyelid closure through the orbicularis oculi muscle
      b. Intact Bell’s phenomenon
      c. Dry eye
      d. Conjunctival injection
      e. Lagophthalmos
      f. Lower lid ectropion
      g. CN III controls levator palpebrae superioris muscle (eyelid elevation)
      h. Sympathetic nerves control Mueller muscle (eyelid elevation)
   3. Ears – change in hearing (stapedius muscle)
   4. Midface/nose
      a. Difficulty breathing through each nostril
      b. Asymmetry of nasolabial folds
   5. Lower face and mouth
a. Asymmetric smile
b. Oral incompetence/drooling
c. Difficult with keeping cheeks puffed out with air
d. Inability to dimple the chin

6. Synkinesis (mass movement) occurs when two different groups of muscles are inappropriately innervated by the same branches of the facial nerve.
a. The more proximal the injury, the greater the expected degree of mass movement.
b. Example: Twitching of the upper lip with eyelid closure

V. MANAGEMENT

A. Testing
1. Electromyography (EMG) – needle insertion into muscle groups.
   a. Fibrillations indicate denervation.
   b. Does not become positive until 2 – 3 weeks after onset of paralysis
      i. Should be considered after 3 weeks
2. Electroneurography
   a. Evaluates the nerve/muscle unit
   b. Compound muscle action potentials (CMAPs) – summation of the action potentials from muscle fibers innervated by queried nerve. Compare normal side with the injured side.

B. Non-surgical management
1. Determine symptoms which pose risk to patient or which bother the patient most
2. Corneal dryness
   a. Risk of ulceration and blindness
   b. Night time lubricant and artificial tears
3. Botulinum toxin
   a. Prevents acetylcholine release across neuromuscular junction
   b. Lasts 2-6 months
   c. Treat facial asymmetry by weakening the normal side
   d. Treat synkinesis by weakening the inappropriate motor function
4. Physical therapy – biofeedback using mirror to rehabilitate during recovery

C. Surgical options
1. Direct nerve repair or nerve grafting can be performed for immediately identified injuries
2. Chronic paralysis
   a. Forehead/upper face
      i. Brow lift
         a) Improve brow asymmetry
         b) Reduce vision obstruction caused by brow ptosis
         c) Beware of causing corneal exposure
      ii. Botox to contralateral side
         a) Improve asymmetry with the paralyzed contralateral side
b) May cause ipsilateral brow ptosis

b. Eyes and eyelids
i. Gold weight in upper lid to improve eyelid closure
ii. Lower lid tightening to treat ectropion
   a) Lower lid canthoplasty
   b) Mid-face lift
iii. Temporalis muscle transfer

c. Midface/Nose
i. Spreader grafts to treat internal nasal valve collapse
ii. Alar grafts to treat external nasal valve collapse
iii. Filler or fat grafting to address nasolabial fold asymmetry
iv. Unilateral facelift to address nasolabial fold asymmetry

d. Lower face and mouth
i. Unilateral facelift to improve symmetry
ii. Static sling using fascia lata, acellular dermal matrix, or other graft material
   a) Attach to the modiolus to raise the corner of the mouth
      i. Muscle transfers
   b) Temporalis muscle transfer procedure (McLaughlin procedure) (innervated by CN V)
   c) Temporalis turnover procedure (Rubin procedure) (innervated by CN V)
   d) Masseter transfer (innervated by CN V)
iii. Nerve transfer
   a) Masseteric or hypoglossal nerve to facial nerve
   b) Requires training to coordinate use of the nerve
   c) Hypoglossal nerve transfer may result in partial tongue atrophy
iv. Free functional muscle transfer
   a) Two-stage vs. single-stage
   b) Cross-facial nerve graft (CFNG)
      i) Two-stage repair
      ii) Sural nerve from contralateral facial nerve tunneled across the upper lip to the ipsilateral side
      iii) Allow 9-12 months for nerve to grow across
      iv) Observe Tinel sign to confirm nerve growth
      v) Free functional muscle transfer (most commonly gracilis muscle)
   c) Single-stage free functional muscle transfer uses the ipsilateral motor nerve to the masseter as a donor
   d) Gracilis muscle
      i) Perform 9-12 months after CFNG (two-stage reconstruction) vs. direct nerve coaptation to ipsilateral motor nerve to the masseter (single-stage reconstruction)
      ii) Innervated by the anterior branch of the obturator nerve
      iii) Blood supply from medial circumflex femoral artery
      iv) Set tension based on resting tension present at the donor site
VI. REFERENCES


CHAPTER 12

BREAST RECONSTRUCTION

Mihaela Rapolti, MD and Michelle Roughton, MD

I. BREAST ANATOMY

A. Mastering breast anatomy is essential for understanding how the breast changes with aging and principles of reconstruction, reduction and mastopexy (Figure 1).

Figure 1. Anatomy of the breast
A. Breast
1. Glandular and adipose tissue are enclosed by superficial fascia and deep fascia overlying chest wall muscles
2. Cooper’s ligaments: suspensory attachment of the breast to the overlying fascia anteriorly. Its attenuation results in breast ptosis.
3. Boundaries for mastectomy:
   a. Superior border is the clavicle
   b. Inferior border is the rectus abdominis fascia/inframammary fold (IMF)
   c. Medial border is the sternum
   d. Lateral border is the anterior border of latissimus dorsi muscle
4. Important elements in planning breast surgery
   a. Breast footprint: superior margin of the breast is lower than the clavicle
   b. Conus: three-dimensional shape, projection, and volume of the breast tissue
   c. Skin envelope: adequate quantity and quality to drape the conus
5. Nipple areola complex (NAC)
   a. Diameter 30-45mm
   b. Aesthetic pleasing breast shape has NAC position - proportion 45:55 (Figure 2)

Figure 2. UPL upper pole line, NM nipple meridian, LPL lower pole line, UPS upper pole slope, LPC lower pole convexity.


B. Vasculature
1. Arterial Supply (Figure 3):
a. Internal mammary artery perforators (60%)
b. Lateral thoracic artery (30%)
c. Thoracoacromial artery: pectoral branches supply pectoralis major muscle and overlying breast tissue
d. Intercostal arteries 3, 4, 5
e. NAC receives blood supply through subdermal plexus and parenchymal vessels, dominant supply from 2nd and 3rd branches of IMA. After nipple sparing mastectomy blood supply is the subdermal plexus. After breast reduction or mastopexy the blood supply is the breast pedicle or parenchyma as you must divide the skin and subdermal plexus to reposition the NAC.

Figure 3. Arterial blood supply to the breast

2. Venous drainage (Figure 4)
   a. Mainly to axillary vein but some to internal mammary and intercostal veins.
   b. Veins are more superficial on the medial breast and deeper on the lateral aspect of the breast
   c. NAC - superomedial/ medial and inferior pedicles contain the most extensive and more reliable venous drainage patterns for NAC.
Figure 4. Resultant venogram of the right breast using plain radiography showing the venous drainage of the breast, including the inferolateral veins (green), superolateral veins (orange), medial veins (pink and purple), and inferior veins (blue). Note the anastomoses between the dominant veins around the nipple-areola complex. *From Le Roux C, et al. Preventing venous congestion of the nipple-areola complex: an anatomical guide to preserving essential venous drainage networks. Plast Reconstr Surg 2011;127(3):1073-1079.*

C. Lymphatics
1. Dominant drainage to axilla (97%)
2. To internal mammary nodes (3%)
3. Level I: nodes lateral to lateral border of pectoralis minor
4. Level II: nodes lying beneath pectoralis minor
5. Level III: nodes medial to medial border of pectoralis minor and extending to apex of the axilla

D. Nerve supply (Figure 1)
1. Cervical plexus: sensory branches of C3, 4 from supraclavicular nerve
2. Lateral branches of intercostal nerves:
   a. Provide sensation to lateral side of breast
   b. Lateral cutaneous branch of the 4th intercostal nerve provides major sensory innervation to nipple (T4 dermatome)
   c. Medial branches of intercostal nerves 2-7 provide sensation to medial breast

II. BREAST RECONSTRUCTION

A. Breast cancer now affects one in eight women over the course of their lifetime and is the leading cause of cancer-related death in women.
B. All patients that have undergone or will undergo mastectomy are entitled to breast reconstructive surgery covered by insurance (Women’s Health Act, 1998). Symmetry procedure for the contralateral breast is also covered.

C. Patients undergoing mastectomy should be offered a preoperative referral to a plastic surgeon.

D. In relation to the timing of the mastectomy, breast reconstruction can be:
   1. Immediate (during the same anesthetic)
   2. Delayed-immediate (uncommon) different anesthesia, different surgery day, but before the skin has fully-healed/retracted
   3. Delayed

E. Possible preferences of patients:
   1. No reconstruction: women may choose to wear an external prosthetic.
   2. Reconstruction of breast mound to attain close to natural breast shape, feel, contour.
   3. Breast mound reconstruction may or may not be followed with NAC reconstruction depending on patient preference.
      a. Mastectomy defects frequently include loss of the NAC (such as in skin-sparing mastectomy). Mastectomy skin necrosis is the unplanned loss of skin due to inadequate blood supply following surgery.
      b. Previous irradiation, such as in BCT, may cause difficulties with wound healing, skin contraction and discoloration, capsular contracture, and fat necrosis
   4. If desired, following unilateral breast reconstruction, the opposite breast can be contoured to obtain symmetry, using mastopexy, reduction or augmentation mammoplasty.

F. Mastectomy options
   1. Breast conservative therapy--BCT (50-70% of breast cancer patients), also called partial mastectomy: removal of the tumor only. Requires post-op radiation. The patients may opt for:
      a. Oncoplastic surgery: i.e. small implants, thoracic anterior perforator TAP flap, muscle-sparing latissimus dorsi flap, intercostal artery perforator flap, lipofilling with or without scar subcision. Timing of this is variable but operating on radiated tissue conveys increased risk.
      b. Contralateral breast reduction for symmetry
   2. Nipple-sparing mastectomy (NSM)/Total skin sparing mastectomy (TSSM): removal of all breast tissue with preservation of all skin, including NAC (Figure 5). The reconstruction can be with autologous tissue or implant-based.
3. Simple (total) mastectomy: removal of all breast tissue, including NAC. Reconstruction in delayed fashion will require tissue expansion or autologous tissue.

4. Modified radical mastectomy: removal of all breast tissue, NAC, pectoralis fascia, as well as Level I and II lymph nodes. Same principles of reconstruction as for simple mastectomy.

5. Halsted radical mastectomy: removal of all breast tissue, nipple/areolar complex, pectoralis major and minor muscles, muscular fascia, Level I, II, and III lymph nodes (this procedure does not improve disease control compared to modified radical mastectomy and is largely of historical interest now)

6. Goldilocks mastectomy: performed through a Wise skin pattern. The inferior de-epithelialized mastectomy flap is folded under the Wise pattern in order to obtain a breast mound. Can be fat grafted for further volume.

G. Techniques of breast reconstruction

1. Choose techniques based on:
   a. Patient preference
   b. Need for adjuvant radiotherapy before or after the breast reconstruction (Figure 6, 7)
   c. Uni- or bilateral mastectomy
   d. Defect of the breast envelope: NSM, total simple mastectomy, mastectomy skin necrosis
   e. Immediate or delayed reconstruction (increased skin requirements)
   f. Habitus of the patient: BMI, size of the breast or desired size of the reconstructed breasts, possible donor sites for autologous reconstruction
2. Implant-based reconstruction
   a. Can be:
      i. Direct-to-implant reconstruction: place implant at time of mastectomy
      ii. Use of tissue expanders (TE) with staged breast implant insertion once adequate skin expansion has occurred.
   b. Acellular dermal matrix (ADM) may be used for partial or total coverage of the device.
   c. Breast implants may be saline or silicone filled devices
3. Autologous reconstruction
   a. Pedicled flaps
      i. Latissimus dorsi myocutaneous flaps (Figure 8). For adequate volume of the reconstructed breast usually combined with:
(a) TE with or without ADM; after completion of the tissue expansion, the TE is eventually exchanged for a breast implant
(b) +/- Fat grafting

ii. Pedicled TRAM flap (Figure 9, 10) using superior epigastric vessels for blood supply (rectus abdominus muscle is used as a “carrier” for the blood vessel)

Figure 8. Three different skin paddles for the latissimus dorsi flap. (Above, left) Oblique skin island design in two different orientations. (Above, right) Vertical skin island design. (Below, left) Horizontal skin island design at the bra line.

Figure 9. Pedicled TRAM flap


Figure 10. Hartrampf’s classification of TRAM flap zonal blood supply. Hartrampf names zone I directly over the muscle pedicle and zone II lying across the midline. (Right) Ninkovic’s classification of TRAM flap and deep inferior epigastric perforator flap zonal blood supply. Holm et al. performed an in vivo study of deep inferior epigastric perforator flaps with indocyanine green and concluded that although zone I remains the most reliably perfused zone, any flow across the midline is less than ipsilateral flow and proposed that Hartrampf’s zone II should be renamed zone III.

b. Free Flaps
   i. Technically more demanding, requiring microvascular techniques. Flap is entirely disconnected from abdomen and sewn to vessels in the chest.
   ii. Recipient vessels may be to internal mammary vessels (or their perforators) or the thoracodorsal vessels (Figure 11).
   iii. As compared to pedicled TRAM, lower risk of partial flap loss and fat necrosis, but potential total flap loss.
   iv. Transverse Rectus Abdominis Myocutaneous (TRAM) flap - rectus abdominus muscle, fat and skin on a transverse paddle, based on inferior deep epigastric artery (DIEA); requires abdominal wall reconstruction/anterior rectus sheath with mesh.
   v. Muscle sparing TRAM flap (MS-TRAM)
      (a) Only a portion of the rectus muscle is harvested (Figure 12)
      (b) In unilateral reconstruction can harvest zones 1-3 (TRAM or MS-TRAM)
   vi. Deep Inferior Epigastric Perforator (DIEP) flap - only skin and fat is harvested on perforating vessels from DIEA, technically more demanding, clinical relevance of not taking any muscle compared with muscle sparing techniques is still under debate. CT angiography may help with perforator harvest planning (Figure 13).
      (a) Unipedicled (Figure 14)
      (b) Double pedicled
   vii. Superficial Inferior Epigastric Artery (SIEA) flap: SIEA has to be of adequate caliber (artery with a palpable pulse, vein >1mm) to be used for anastomosis. Not all patients have an adequate SIEA, may be affected by the position of the lower abdominal incision. SIEV may be the dominant venous drainage and is commonly preserved in case it is needed for venous outflow.
   viii. Gluteal artery perforator (GAP) flap on superior (S-GAP) or inferior (I-GAP) gluteal artery perforators (Figure 15): Typically reserved for patients without sufficient abdominal wall tissue or for those who are not candidates for abdominal based reconstruction.
   ix. Transverse upper (TUG) gracilis flap (Figure 16) and vertical upper gracilis (VUG) flap
x. Turbocharging:
   (a) Vascular augmentation using the vascular sources within the flap territory
   (b) Example: performing a DIEP flap to the recipient internal mammary vessels then anastomosing an additional vessel from this system
xi. Supercharging:
   (a) Vascular augmentation using a distant source of vessels such as axillary or thoracodorsal vessels
   (b) Example: performing a pedicled TRAM flap, then augmenting the flow by anastomosing the deep inferior epigastric vessels to the thoracodorsal vessels.
Figure 11. Exposure of the thoracodorsal recipient vessels (left) and internal mammary recipient vessels (right). *From Macadam S, et al. Evidence-Based Medicine: Autologous Breast Reconstruction. Plast Reconstr Surg 2017;139(1):204e-229e.*

Figure 12. Classification of muscle-sparing free TRAM procedures. MS0 refers to sacrifice of the full width of the rectus muscle, MS1 preserves the lateral segment, MS2 preserves the lateral or medial segments, and MS3 preserves the entire muscle (equivalent to a DIEP flap). MS1 can be further subdivided into MS1-M and MS1-L, depending on whether it is the medial or lateral segment that is spared. *From Macadam S, et al. Evidence-Based Medicine: Autologous Breast Reconstruction. Plast Reconstr Surg 2017;139(1):204e-229e.*
Figure 13. Computed tomographic angiography

Figure 14. DIEP flap
Figure 15. SGAP flap and IGAP flap
H. Fat grafting
   1. Does not increase the risk of breast cancer recurrence
   2. Volume augmentation of a reconstructed breast
   3. Improve shape of the breast, corrects contour deformities, commonly in the upper pole for fullness
I. Ipsilateral corrections include:
   1. Nipple reconstruction
   2. Scar revisions
   3. Volume adjustments either by reduction (i.e. direct resection or liposuction) or by augmentation (i.e. implants, additional flaps or lipofilling)
   4. Shape corrections: can be accomplished by repositioning and/or rotating the flap or implant, skin resections, and adjustments of the inframammary fold or other borders of the flap.
J. Symmetry is the goal of the reconstruction. The contralateral side may be adjusted by the common techniques of:
1. Augmentation
2. Reduction
3. Mastopexy

K. Nipple-areola complex (NAC) reconstruction. NAC is one of the aesthetic units of the breast. For the completeness of the aesthetic of the breast some women chose to have the NAC reconstructed.

1. Nipple sharing (graft from the contralateral nipple) if available and dimensions adequate (Figure 17)
2. Local skin flaps (C-V, C-Y, star flap, skate flap) with or without use of cartilage, ADM graft, revision with fat grafting to improve projection (Figure 18, Figure 19)
3. Intra-dermal color tattoo to match opposite NAC:
   a. May follow nipple reconstruction procedure
   b. 3-D tattoo to simulate also the nipple projection from frontal view (no surgical nipple reconstruction)
4. In-situ or remote-donor skin graft (groin or labia majora/minora) may also be used for areola

Figure 17. Drawings demonstrating the two commonly accepted techniques for nipple sharing. Both harvest 50 percent of the nipple. (Above) Drawings show sagittal harvest for nontubular nipples. The lower half of the nipple is taken and the donor site closed with simple interrupted sutures, leaving an imperceptible scar under the nipple. (Below) Drawings show coronal harvest for tubular, large nipples. Note that the donor site is closed with a purse-string suture, leaving a natural appearing nipple.

Figure 18. C-Y flap diagram

Figure 19. Different techniques for nipple reconstruction. (Above) CV flap. (Center) Star flap. (Below) Skate flap.
REFERENCES

CHAPTER 13

TRUNK RECONSTRUCTION

Kavitha Ranganathan, MD and Michele Manahan, MD

I. ABDOMINAL WALL ANATOMY

A. Layers of the Abdominal Wall (Figure 1)
   1. Skin
   2. Subcutaneous tissue
   3. Scarpas fascia
      a. Superficial fascial system
      b. Approximated during layered closure of abdominal wall to prevent contour
         irregularities within the scar
   4. Lateral Musculature
      a. External Oblique
         i. Muscle fibers run inferomedially
            (a) Inferomedially= same direction as your hands in pockets
      b. Internal Oblique
         i. Muscle fibers run superomedially
         ii. Neurovascular bundles lie between the internal oblique and
             transversus abdominis
            (a) Abdominal wall supplied by ventral rami of T7-L4
      c. Transversus Abdominis

![Figure 1. Abdominal wall anatomy](image)


5. Medial Musculature
a. Rectus abdominis
   i. Above the arcuate line
      (a) Anterior rectus sheath composed of external oblique fascia and internal oblique fascia above the arcuate line
      (b) Posterior rectus sheath composed of internal oblique fascia (splits to contribute to anterior and posterior rectus sheaths) and transversus abdominis fascia
   ii. Below the arcuate line
      (a) Anterior rectus sheath composed of external oblique fascia, internal oblique fascia, and transversus abdominis fascia
      (b) No posterior rectus sheath (layers behind the rectus abdominis muscle at this level are the transversalis fascia and parietal peritoneum)

6. Transversalis Fascia
7. Parietal peritoneum
B. Zones of the Abdominal Wall
   1. Huger zones: used to delineate anatomic regions of perfusion
      a. Zone 1: region between xiphoid, pubic symphysis, and linea semilunaris
         i. Perfused by the deep inferior epigastric arteries
      b. Zone 2: region between anterior superior iliac spines bilaterally, pubic symphysis, and groin creases
         i. Perfused by superficial circumflex femoral arteries, external pudendal arteries, and superficial inferior epigastric artery
      c. Zone 3: region lateral to rectus muscles
         i. Perfused by intercostal perforators

II. GOALS OF RECONSTRUCTION

   A. Provide protection for intra-abdominal viscera
   B. Repair and prevent herniation with strong fascial support
   C. Prevent recurrence and restore abdominal wall function by maintaining innervation allowing for proper contractility

III. COMMON ABDOMINAL WALL PATHOLOGY REQUIRING RECONSTRUCTION

   A. Tumor resection
   B. Infection (ex. necrotizing fasciitis)
   C. Trauma
   D. Recurrent ventral wall hernias
   E. Congenital abdominal wall defects (gastroschisis, omphalocele)

IV. RECONSTRUCTIVE ALGORITHM – ABDOMINAL WALL RECONSTRUCTION
A. Primary closure without prosthetic material
   1. Use for defects that are <3cm
B. Primary closure + prosthetic
   1. Use for defects under tension upon closure
      a. Retro-rectus mesh placement leads to lowest risk of infection compared to other mesh placement locations
         i. If mesh becomes infected or if patient presents with wound concerning for underlying infection, mesh must be removed to eradicate the infection; antibiotics alone are not enough when prosthetic materials are in place
   2. Prosthetic Materials (used in non-contaminated tissue beds):
      a. Many different types are used based on surgeon preference (examples below)
         i. Polypropylene
            (a) Advantages: allows fibrous ingrowth, incorporates into adjacent tissues
            (b) Disadvantages: can erode into bowel and cause fistulas
         ii. PTFE
            (a) Advantages: does not incorporate completely into tissues, so easy to remove if needed
            (b) Disadvantages: does not allow for fluid egress and can lead to wounds
   3. Biologic Materials (used in contaminated tissue beds):
      a. Materials revascularize and incorporate nicely into surrounding tissues, leading to lower risk of contamination
      b. Examples include Alloderm® (Lifecell), Strattice™ (Lifecell), Permacol™ (Medtronic)
C. Components separation + prosthetic
   1. Use for wide defects in which fascia cannot be closed primarily without further release
      a. Release of external oblique at linea semilunaris enables medial transposition of rectus muscle along with internal oblique and transversus abdominis muscles
         i. Advancement attainable: 10 cm in epigastrium, 20 cm at umbilicus and 6 cm in suprapubic region
D. Tissue Expansion
E. Pedicled muscle and myocutaneous flaps (when synthetic mesh and fascial separation are contraindicated)
   1. Antero-lateral thigh (ALT)
   2. Tensor fascia lata
   3. Gracilis
   4. Rectus femoris
   5. Propeller flaps (flaps pedicled on a particular perforator)
F. Free flaps
G. Split thickness skin and/or synthetic mesh directly over bowel (in emergency situations as temporizing measure to facilitate closure; often requires further reconstructive surgery)

H. VAC use can be integrated into the treatment of patients with compromised wound healing
   1. Cases of enteric fistula formation have been associated with the VAC; paradoxically, however, VAC has also been used successfully for the management of fistulae

V. CHEST WALL ANATOMY

A. Skin and subcutaneous tissue

B. Bony structures
   1. Sternum (composed of manubrium, body, xiphoid)
   2. Ribs (12 total)
      a. True ribs (1-7): articulate directly with sternum
      b. False ribs (8-12): articulate with costal cartilages instead of sternum directly
   3. Clavicles
   4. Thoracic vertebrae

C. Musculature
   1. Pectoralis major
      a. Pedicle: thoracoacromial artery, intercostal arteries
   2. Pectoralis minor
   3. Serratus anterior
      a. Pedicle: serratus branch of thoracodorsal artery, lateral thoracic artery
      b. Accessory muscle of respiration
   4. Intercostal muscles
      a. External intercostals: fibers run inferomedially
      b. Internal intercostals: fibers run superomedially
         i. Neurovascular bundles run between the internal and innermost intercostals
      c. Innermost intercostals: fibers run transversely
   5. Diaphragm
      a. Innervated by C3,4,5 nerve roots

D. Pleura
   1. Parietal pleura
   2. Visceral pleura

VI. GOALS OF RECONSTRUCTION

A. Rigid airtight cavity
B. Protection of the thoracic and abdominal contents
C. Optimization of respiration
D. Obliteration of dead space for intrathoracic defects
E. Stable soft tissue coverage
F. Aesthetic reconstruction (whenever possible)
G. Control of infection
H. Removal of foreign bodies

III. COMMON CHEST WALL PATHOLOGY REQUIRING RECONSTRUCTION

A. Trauma
B. Tumor resection
C. Infection (osteomyelitis after cardiac surgery)
D. Congenital anomalies

IV. RECONSTRUCTIVE ALGORITHM – ACQUIRED CHEST WALL DEFORMITIES

(Table 1)

| Size of the defect | • Some authors advocate skeletal reconstruction when the defect is four ribs or more\textsuperscript{38,39}  
| Location of the defect | • Others use the actual size of the defect and believe that defects more than 5 cm in diameter are more likely to benefit from prosthetic reconstruction\textsuperscript{92,100}  
| | • Soft tissue-only reconstruction is often considered adequate for smaller defects  
| Condition of the chest wall | • Defects in the anterior (and anterolateral) chest wall require stability reconstruction more often than posterior chest wall because they more mobile and have stronger impact on the respiratory function\textsuperscript{91}  
| | • Prosthetic stability reconstruction is thought to be required less often in the posterior chest wall than in the anterolateral chest wall because the scapula and its surrounding muscle attachment provide more stability in the former  
| | • Small (<5-cm) posterior defects under the scapula or above the fourth rib can often be closed with soft tissue, without reconstructing the skeletal component\textsuperscript{94}  
| | • Radiation leads to chest wall stiffness and fibrosis; therefore, muscle flaps alone often provide enough stabilization for large irradiated defects without causing flail segments\textsuperscript{92}  

Table 1. Considerations during chest wall reconstruction

A. Remove all foreign bodies including prosthetic materials and sternal wires that commonly harbor biofilm in these settings
B. Primary closure (if possible)
C. Wound vac  
1. Improves respiratory mechanics while patient is undergoing serial debridement prior to definitive coverage
D. Reconstruction of bony defects  
1. Important to reconstruct defects that are >5cm or include removal of 4 contiguous ribs to protect vital structures and improve mechanics of breathing  
   a. Alloplastic Materials  
      i. Ex. polypropylene, methyl methacrylate, titanium plate fixation  
      ii. Pros: no donor site morbidity; rigidity of construct allows for restoration of respiratory dynamics
iii. Cons: foreign bodies increase likelihood of infection requiring explantation; presence of radiation injury also increases complications related to use of prosthetic devices

b. Autologous Materials
i. rib grafts, free tissue transfer
ii. Pros: use of autologous tissue allows for incorporation into surrounding wound bed and minimizes likelihood of infection related to foreign bodies
iii. Cons: donor site morbidity

E. Autologous reconstruction (Figure 2)
1. Loco-regional pedicled flaps
   a. Pectoralis major flap (advancement and turnover flaps possible)
   b. Latissimus dorsi
   c. Rectus abdominis (cannot perform if ipsilateral internal mammary vessels have been used in cardiac surgery cases)
   d. Serratus anterior
   e. Omentum

V. RECONSTRUCTIVE ALGORITHM FOR CONGENITAL CHEST WALL DEFORMITIES

A. Poland’s Syndrome
1. Etiology
   a. Due to kinking of the subclavian artery at week 6 of gestation leading to hypoplasia of the vessel
2. Components
   i. Absence of sternal head of pectoralis major
   ii. Hypoplasia of breast or nipple
   iii. Deficiency of subcutaneous fat and axillary hair
   iv. Bony abnormalities of anterior chest wall
   v. Syndactyly or hypoplasia of ipsilateral extremity
   vi. Shortening of forearm
b. Treatment
   i. For adolescents, can place subcutaneous tissue expander and perform serial expansions to minimize deformity throughout adolescence
   ii. Once fully developed, consider latissimus dorsi pedicled flap with implant reconstruction or other method of breast reconstruction
      (a) Can use innervated ipsilateral latissimus to recreate anterior axillary fold

B. Pectus Excavatum
1. Depression of the sternum that can lead to cardiovascular/respiratory abnormalities and/or cosmetic deformities
2. Treatment
   a. Consultation and collaboration with pediatric surgery team is critical
   b. Repositioning of sternum to normalize contour
      i. Sternal osteotomies for repositioning
(a) May require a posterior strut for support
(b) Nuss procedure utilizes steel bars for support instead
(c) Implantable prostheses can also be used for cases without cardiovascular compromise to restore normal contour to the chest wall

C. Pectus carinatum
1. Excessive protrusion of the sternum
2. Treatment
   a. Reconstruction planned in conjunction with pediatric surgery team
   b. Repositioning of the sternum and abnormal costal cartilage to restore normal contour

![Image of pedicled flaps for chest wall reconstruction]

Figure 2. Common Pedicled Flaps for Chest Wall Reconstruction. (Left) Pedicled flaps commonly used to fill intrathoracic dead space: pectoralis major muscle (1), serratus anterior muscle (2), latissimus dorsi muscle (3), omental (4), and rectus abdominis muscle (5) flaps. (Right) The pectoralis major muscle flap is useful for filling upper chest defects. The latissimus dorsi muscle flap is more useful for filling lateral defects. The omental flap can be passed over the ribs or through the diaphragm.


REFERENCES


CHAPTER 14

PRESSURE INJURIES

Lee Squieteri, MD and Jeffrey Kozlow, MD

I. TERMS

A. “Pressure injury” is now preferred over “pressure/decubitus ulcer” or “bed sore”
B. Decubitus was a term to describe lying position; however, any position that causes sustained pressure to an area (e.g., sitting/side position) can cause a pressure injury. They can also occur due to casting or splinting after surgical procedures.

II. STAGING SYSTEM

A. Stage I: non-blanchable erythema of intact skin
B. Stage II: partial thickness skin loss with exposed dermis
C. Stage III: full thickness skin loss
D. Stage IV: full thickness skin and tissue loss with exposed fascia, muscle, tendon, ligament, cartilage, or bone
E. Unstageable: full thickness skin and tissue loss in which the extent of tissue damage cannot be determined because it is obscured by slough or eschar
F. Deep tissue injury: persistent non-blanchable deep red, maroon, or purple discoloration

III. “ICEBERG PHENOMENON”

A. Since skin can withstand ischemia much better than fat or muscle, a small skin wound on surface can reflect a large amount of deeper tissue necrosis underneath.

IV. EPIDEMIOLOGY

A. Quadraplegics: 60%
B. Bed-bound hospital patients: 10-15%
C. ICU patients: 33%
D. Hip fracture patients: up to 66%

V. COST

A. 5-11.6 billion dollars in annual healthcare costs
B. Additional $7,000-43,000 dollars per hospital stay
C. $21,000-152,000 to treat/heal per pressure injury
D. Medicare is primary payer for ~75% of hospitalizations with pressure injury

VI. AFFECTED BODY AREAS

A. Most common (order varies in literature): ischium, sacrum/coccyx, trochanter, heel
B. Other sites: occipital region, malleoli, spine, shoulder/scapula
C. Spinal cord injury patients: sacrum (acute), ischium (chronic)

VII. RISK FACTORS

A. Extrinsic: nonphysiologic, environmental
   1. Pressure (perpendicular) leads to deep necrosis: can develop after 2 hours of unrelieved pressure
   2. Shear (parallel) leads to superficial necrosis
   3. Friction
   4. Moisture
B. Intrinsic: physiologic
   1. Altered activity/mobility
   2. Cognitive deficit or altered consciousness
   3. Decreased autonomic control (e.g., incontinence)
   4. Infection → sepsis/ischemia
   5. Increased age
   6. Sensory loss
   7. Chronic illness: vascular disease/anemia
   8. Malnutrition
   9. Medications/immunocompromised (e.g., steroids)
C. Braden scale: measures pressure injury risk for adults/children using 6 domains (sensory perception, moisture, activity, mobility, nutrition, and friction/shear)

VIII. NON-SURGICAL TREATMENT

A. Prevention is the best treatment
   1. Keep skin clean and dry
   2. Appropriate nursing care, including turning the patient every 2 hours (avoid dragging/shearing skin of the patient while repositioning)
   3. Optimizing nutrition
   4. Relieving pressure using air mattresses, cushions, heel protectors
   5. Air fluidized beds (Clinitron®) gold standard for pressure injury prevention
B. Systemic infection/sepsis unlikely with pressure injury (unless immunocompromised): look for other source, e.g., urinary tract infection or respiratory tract when patients with pressure injuries present with fevers
C. If localized infection is present (look for signs of local cellulitis) topical antimicrobial agents (Silvadene, Sulfamylon) can be used
D. Bone biopsy best method to assess osteomyelitis versus osteitis
E. Can direct antibiotic therapy to treat osteomyelitis, but virtually impossible to eradicate infection with antibiotics alone
   1. MRI may be helpful as imaging study, while bone scans are often nonspecific due to presence of periostitis associated with open wounds
   2. Long term antibiotics are not indicated
   3. Pressure injury closure may be accelerated using topical protein growth factors
   4. Stage III-IV patients require sharp debridement, highly absorptive dressings (alginites, hydrocolloid beads, foams, hydrogels)
   5. VAC therapy may be beneficial to assist closure

IX. SURGICAL TREATMENT

A. Due to high recurrence rates, surgery tends to be reserved for patients with reversible pathologies
B. Patient motivation is an important determinant of recurrence risk in the alert patient
C. Excisional debridement of pressure injury and bursa and any heterotopic calcification
D. Partial or complete ostectomy to reduce bony prominence – may lead to new pressure injuries elsewhere (be careful when off-loading)
E. Closure of the wound with healthy, durable tissue that can provide adequate padding over the bony prominence (myocutaneous vs. fasciocutaneous flap)
F. Aftercare including appropriate surfaces and wound management are paramount
G. Lifestyle and activity modification often required in order to reduce recurrence risk

REFERENCES

The hand is a gateway to the vast majority of our physical experiences and interactions. However, the hand is a delicately balanced organ, and when one structure is injured, the system as a whole falters. Given the variety of tissues in the hand, including soft tissue, blood vessels, nerves and bone, plastic surgeons are intimately involved in the surgical care of the upper extremity. Plastic surgeons have been pioneers in the field of hand surgery since its earliest days, from traumatic reconstruction to hand transplantation. A basic understanding of hand anatomy and care is essential to not only plastic surgery, but also orthopedics, emergency and family medicine.

I. HAND ANATOMY

A. Nerves
   1. Sensory - median, ulnar, radial (Figure 1)
   2. Motor - intrinsic muscles of hand
      a. Median nerve - thenar muscles, radial lumbricals
      b. Ulnar nerve - interossei, ulnar lumbricals, hypothenar muscles

B. Muscles and tendons
   1. Flexor system (Figure 2)
      a. Extrinsic flexors - Flexor digitorum profundus attaches to distal phalanx and bends the DIP (distal interphalangeal) and PIP (proximal interphalangeal) joints. Flexor digitorum superficialis attaches to middle phalanx and bends PIP joint.
b. Intrinsic flexors - Lumbricals bend the MCP (metacarpal-phalangeal) joints

Figure 2. Flexor tendon system

2. Extensor system (Figure 3)
   a. Extrinsic extensors join the extensor hood at the proximal phalanx, and extend the proximal phalanx at the MCP joint.
   b. Intrinsics (interossei and lumbricals) pass volar to the axis of the MCP joint (where they act as flexors) and move dorsal to the axis of the PIP joint to
insert on the dorsal distal phalanx. They act as extensors to the PIP and DIP joints.

![Extensor tendon system diagram](image1)

Figure 3. Extensor tendon system

C. Skeleton (Figure 4)

![Bony skeleton diagram](image2)

Figure 4. Bony skeleton
D. Wrist – a large number of tendons, nerves and vessels pass through a very small space, and are vulnerable to injury (Figure 5).

II. INITIAL EVALUATION OF THE INJURED HAND

A. Evaluation of ABC’s
   1. Hand injuries are frequently associated with multisystem trauma, the patient must be stable and cleared from a trauma standpoint before further evaluation

B. History
   1. Time and place of accident
   2. Mechanism of injury (position of hand while injured)
   3. First aid given
   4. Right or left-hand dominance
   5. Occupation
   6. Age

C. Examination
   1. Observation
      a. Position of fingers - normally slightly flexed. An abnormally straight finger might indicate a flexor tendon injury (the unopposed extensors hold the finger straight)
      b. Sweating patterns (lack of perspiration may indicate denervation)
      c. Anatomic structures beneath the injury

Figure 5. Extensor compartments of the hand: (1st) APL, EPB; (2nd) ECRL, ECRB; (3rd) EPL; (4th) EDC; (5th) EDQ; (6th) ECU

2. Sensory - must test prior to administering anesthesia
   a. Pin to measure sharp/dull sensitivity, paper clip to measure two point
discrimination in millimeters
   b. Test all sensory territories (median, ulnar, radial)
   c. Test both sides of each finger to test radial and ulnar sided digital nerves
3. Motor
   a. Profundus - stabilize PIP joint in extension, ask patient to flex fingertip
      (Figure 6)
   b. Superficialis - stabilize other DIP joints in extension. This neutralizes
      profundus action.
      i. Ask patient to flex unstabilized finger (Figure 7)
   c. Motor branch of median nerve; test palmar abduction of thumb against
      resistance
   d. Motor branch of ulnar nerve; ask patient to fully extend fingers, then spread
      fingers or cross fingers
   e. Extensor tendons
      i. Ask patient to extend fingers at MCP joints (tests long extensors)
      ii. Ask patient to extend PIP, DIP joints with MPs flexed (tests intrinsic
           extensors)
Figure 7. Testing superficialis function

4. Vascular
   a. Color – nailbed should be pink, blanch with pressure, and show capillary
      refill around two seconds
   b. Temperature – finger or hand should be similar in temperature to uninjured
      parts
   c. Turgor – pulp space should be full without wrinkles
   d. Pulse oximetry/Doppler are useful adjuncts to physical exam findings

D. Early care
   1. Use pneumatic tourniquet or BP cuff inflated to 250mmHg to control bleeding
      for examination and treatment. An awake patient will tolerate a tourniquet only
      for a short period (<30 min)
   2. If there is significant blood loss, apply pressure, wrap with a pressure dressing
      to the affected area and slightly proximally
      a. Do not clamp vessels
      b. Lidocaine with epinephrine can aid in exploration but only after neuro
         examination is completed
      c. Tourniquet may be used as last resort, but must be released intermittently
   3. Splint in safe position if possible (Fig. 6-8)
      a. Position where collateral ligaments are at maximum stretch, so motion can
         be regained with least effort Fig. (6-6)
      b. Positioning - wrist extended (30 degrees), MCP joints flexed (60-70
         degrees), IP joints straight, thumb abducted and rotated in opposing
         position
      c. Proper splinting prevents further injury, prevents vessel obstruction,
         prevents further tendon retraction
   4. All flexor tendon, nerve and vascular injuries, open fractures, and complex
      injuries are managed in the operating room
      a. Many injuries can be treated in the emergency department or clinic using
         field blocks, digital blocks or wrist blocks
   5. Tetanus prophylaxis and antibiotic coverage as indicated

Figure 8. Position of safety

E. Definitive treatment
1. Thorough cleaning of entire hand and forearm, with wound protected
2. Apply sterile drapes
3. Inspect wound - use tourniquet or BP cuff for hemostasis
4. Wound irrigation with normal saline
5. May need to extend wound to inspect all vital structures
6. Assure hemostasis with fine clamps and cautery
7. Nerve injuries should be repaired with magnification
8. Tendons are repaired primarily.
   a. Flexor tendon injuries in Zone II, "no man's land" (Figure 9), should be repaired by a trained hand surgeon
   b. If a hand surgeon is not available, clean and suture the skin wound, splint the hand, and refer as soon as possible for delayed primary repair. Repair should be done within 10 days
9. Reduce fractures and dislocations, apply internal or external fixation if needed
10. Postoperative dressings
    a. Splinting should be in safe position when possible, but alternative positioning may be required to protect tendon or nerve. Splint extensor tendon repairs in extension, and dorsal blocking splint for flexor tendon repairs
    b. Dressings should not be tight

Figure 9. Flexor zones of the hand
III. SPECIAL INJURIES

A. Fingertip - most common injury
   1. Tip amputations
      a. Basic principles - maintain length, bulk and sensibility
      b. Treatment options include secondary healing, skin graft, flap
   2. Nailbed injury
      a. Nailbed typically repaired with fine chromic gut suture
      b. Nail can be cleaned and replaced as a splint, or silastic sheet used as splint
to prevent adhesion of the eponychial fold to the nailbed

B. Amputation
   1. Indications for replantation – thumb, multiple fingers, and children. Single
      finger replantations often not indicated. Must discuss with replant team.
   2. Care of amputated part
      a. Remove gross contamination and irrigate with saline
      b. Wrap part in gauze moistened in saline, place in clean plastic bag or
         specimen cup, seal
      c. Lay container on ice, or float on ice cubes in water. Don’t immerse part
         directly in ice water or pack directly in ice – it may freeze
   3. Care of patient
      a. Do not clamp vessels
      b. Supportive care
      c. X-ray stump and amputated part

C. Burned hand
   1. Initial treatment
      a. Cleanse wound, debride broken blisters
      b. Evaluate blood supply - circumferential full thickness burns may require
         escharotomy
      c. Apply occlusive dressings to reduce pain
      d. Immobilize in safe position
      e. Refer to plastic surgeon if burn is extensive or may require grafting
   2. Hand therapy may be needed to maintain motion

IV. INFECTIONS

A. General principles
   1. Infection can be localized by finding:
      a. The point of maximum tenderness
      b. Signs of local heat
      c. Overlying skin edema
      d. Pain on movement
   2. A fever usually denotes lymphatic involvement
   3. Pressure from edema and purulent fluid in a closed space can produce
      necrosis of tendons, nerves, blood vessels, and joints in a few hours. Extreme
      cases can lead to amputation and even death.
4. Beware and alert for signs of necrotizing soft tissue infection!

B. Treatment principles
   1. Surgical drainage, cultures
   2. Immobilization in safe position, elevation
   3. Antibiotics

C. Special infections
   1. Paronychia - infection of the lateral nail fold Treatment: if early, elevation of skin over nail to drain. If late, with pus under nail, must remove portion of nail
   2. Felon
      a. Infection pulp space of fingertip - closed space without ability to expand - very painful
      b. Treatment is drainage over point of maximal tenderness laterally
         i. Septate of pulp must be divided to ensure drainage
   3. Subcutaneous abscess – incise and drain with care not to injure digital nerve. Be alert to possibility of foreign body
   4. Tenosynovitis – infection of tendon sheath. Should be treated as surgical emergency.
      a. Diagnostic signs (Kanavel’s signs)
         i. Fusiform swelling of finger
         ii. Finger held in slight flexion
         iii. Pain with passive extension
         iv. Tenderness over flexor tendon sheath (look for proximal swelling and pain along palm and forearm)
      b. Treatment is to open and irrigate tendon sheath. Untreated infection can destroy the tendon within hours
      c. Broad Spectrum Antibiotics
   5. Human bite
      a. Have high index of suspicion – patients are often unwilling to admit being in a fight. Most common site over a knuckle
      b. Debride, cleanse thoroughly, culture
      c. Must rule out penetration of joint space – may need to explore in OR
      d. IV antibiotics for severe cases (Ampicillin-sulbactam most commonly used)
      e. Do not close wound

V. FRACTURES

A. General principles
   1. Inspect, palpate, x-ray in multiple planes – AP, true lateral, oblique
   2. Reduce accurately
   3. Immobilize for healing – for appropriate time period
   4. Hand therapy to maintain motion – immobilization leads to stiffness

B. Specific fractures
   1. Metacarpal fractures
      a. Generally unstable fractures, can accept 10-40 degrees of angulation, if no malrotation present.
i. Malrotation/scissoring – have patient make a fist slowly, if involved finger overlaps another, there is rotation at the fracture site which must be reduced.

b. If angulation/malrotation are acceptable – treatment with splinting with buddy taping may be sufficient
c. If fracture is unstable and in poor alignment closed fixation with pins or open reduction with plates and screws may be performed
d. Special fracture - Boxer’s fractures - 5th metacarpal neck.
   i. Misnomer as a boxer does lead their punch with their 5th MCP!
   ii. Frequently a striking injury (look for signs of bite injury)

2. Phalangeal fractures
   a. Unstable fractures require internal or percutaneous fixation
   b. Joint surfaces should be anatomically reduced

3. Tuft fractures (distal phalanx)
   a. If crushed, mold to shape
   b. Repair associated nailbed injury if needed
   c. Splint for comfort (DIP only) for 1-2 wks

VI. JOINT INJURIES

A. Dislocation
   1. If already reduced, test for stability through arc of motion. Test collateral ligament stability with radial and ulnar stressing at affected joint. Test MPJ in flexion and IP joints in extension. Compare laxity to contralateral side to establish baseline.
   2. Most can be treated with closed reduction; open reduction can be necessary if supporting structures prevent the reduction (e.g. metacarpal head through extensor mechanism)

B. Ligamentous injury - usually lateral force
   1. Gamekeeper’s thumb - rupture of ulnar collateral ligament of MCP joint
   2. Wrist injury - multiple ligaments can be involved. Diagnosis may require MRI arthrogram or arthroscopy. Clinical diagnosis by pattern of pain, x-rays, palpation for abnormal movement.

C. Treatment
   1. Immobilize 2-3 weeks for digital joint injury (MP or IP dislocation), then protected motion
   2. Thumb MPJ and wrist sprains require 6 weeks immobilization. (some, e.g. thumb ulnar collateral ligament, might need operative repair)

VII. CONGENITAL DEFECTS

A. Common defects
   1. Polydactyly - most common. Duplication of fingers, usually border digits. Duplication of 5th finger is common autosomal dominant trait in African-Americans. Thumb duplication often requires reconstructive surgery
2. Syndactyly - 2nd most common - May be simple, involving skin only, or complex, involving bone

B. Treatment - goal to decrease deformity and improve function
   1. Some problems are treated in infancy – e.g. splinting for club hand, thumb reconstruction
   2. Some treated by 12 months, before handedness develops – e.g. separation of syndactyly
   3. Some require multi-stage procedures – e.g. club hand

VIII. HAND TUMORS

A. Benign
   1. Ganglion cysts – most common tumor of upper extremity
      a. Synovial cyst of joint or tendon sheath
      b. Most common location scapholunate ligament
      c. Treatment is observation, aspiration or excision (aspiration with high recurrence –less so in children)
   2. Giant cell tumor – 2nd most common tumor, arises from proliferation from tendon sheath
   4. Bone tumors – enchondroma, Osteoid osteoma

B. Malignant
   1. Skin cancers (e.g. basal cell, squamous cell, melanoma)
   2. Malignant bone tumors are uncommon in hand

IX. MISCELLANEOUS

A. Rheumatoid arthritis
   1. Synovial hypertrophy can lead to nerve compressions (carpal tunnel syndrome), joint destruction.
   2. Hand surgeons can perform synovectomy, joint replacement, carpal tunnel release
      a. Disease modifying drugs have lessened the need for surgical intervention in these patients

B. Dupuytren’s contracture
   1. Fibrous contraction of palmar fascia causes flexion contractures of fingers
   2. Treatment is surgical excision, percutaneous release with a needle or Xiaflex (collagenase) for MCP contractures

C. Nerve compressions – compression of nerve by overlying muscle, ligament or fascia
   1. Common examples:
      a. carpal tunnel – compression of median nerve by transverse carpal ligament
      b. cubital tunnel – compression of ulnar nerve at elbow by Osborne’s ligament
2. Diagnosis by symptoms, exam, and EMG
   a. Tinel's sign - percussion of area leads to radiating pain through nerve distribution
   b. Muscle wasting of hands (intrinsics for ulnar nerve compression, thenar for median nerve)
   3. Treatment options include splinting, NSAIDs, steroid injections, surgical release

REFERENCES

CHAPTER 16

LOWER EXTREMITY

Amanda K Silva, MD and Warren Ellsworth, MD, FACS

The plastic and reconstructive surgeon is often called upon to treat many wound problems of the lower extremity. These include leg ulcers of various etiologies, trauma with extensive soft tissue loss or exposed bone, vascular or neural structures, and lymphedema, which is covered in another chapter.

I. CHRONIC WOUNDS

Each wound requires accurate diagnosis. Not all lower extremity wounds will require surgical intervention when appropriate management is pursued. The key to healing is wound hygiene, correction of the underlying problem, treatment of any concurrent infection, optimizing nutrition, and specific surgical intervention when appropriate. The plastic surgeon is an integral member of the treatment team from the onset of the problem. Remember that two different predisposing conditions may occur in the same patient. If so, the treatment must address both conditions.

A. Venous stasis ulcer
   1. Etiology - venous hypertension related to venous valvular incompetence
   2. Appearance
      a. Usually found over medial malleolus
      b. Increased edema
      c. Increased hemosiderin deposition (dark discoloration)
   3. Treatment
      a. Most heal if venous hypertension controlled
      b. Decrease edema with constant bedrest and foot elevation
      c. “Unna boots” may heal ulcers in patients who are noncompliant with bedrest or must continue to work
      d. Surgical treatment requires excision of entire area of ulcer, scar tissue, and surrounding area of increased pigmentation (hemosiderin deposition)
      e. Pressure gradient stocking and a commitment to avoiding standing for long periods of time are necessary for long term success

B. Ischemic ulcer
   1. Etiology - proximal arterial occlusion
   2. Appearance
      a. Usually more distal on the foot than venous stasis ulcers
      b. Most often on lateral aspects of great and fifth toes, and dorsum of foot
      c. No edema
      d. No change in surrounding pigmentation
      e. Painful
   3. Diagnosis
a. Doppler ankle/brachial indices 0.1-0.3 indicates advanced atherosclerotic disease

4. Treatment
   a. Most require revascularization based upon angiographic findings
   b. Usually a skin graft will close the wound; flap closure may be required if critical structures or bone are exposed. A more proximal amputation may be required if revascularization is not possible

C. Diabetic ulcer
   1. Etiology - decreased sensation (neurotrophic) or occasionally decreased blood flow
   2. Appearance
      a. Usually on plantar surface of foot over metatarsal heads or heel
      b. Edema ±
      c. No change in surrounding pigmentation
   3. Treatment
      a. Debride necrotic tissue and use topical and systemic antibiotics to control the infection
      b. Be conservative in care; early amputation is detrimental since patients often have limb-threatening infections in the other leg within a few years
      c. After control of bacterial contamination, small ulcers may be excised and closed primarily; larger ulcers may require flap coverage
      d. Treatment should also include resection of underlying bony prominence
      e. Postoperative diabetic foot care at home is paramount to proper management. Patient education in caring for and examining their feet is extremely important

D. Pyoderma Gangrenosum
   1. Very difficult to treat
   2. May include anti-inflammatory drugs or immunosuppressives, as well as local wound care agents
   3. Success in treatment has been reported with hyperbaric oxygen in conjunction with local wound care
   4. Surgical treatment if often contraindicated as excision causes perpetuation of wounds

II. ACUTE TRAUMA

Lower extremity trauma is frequently very complex, and often requires a team approach involving the orthopedic, vascular and plastic surgeons. Limb salvage with bipedal ambulation, normal weight bearing, and sensation is the goal of all surgical intervention.

A. Initial Management
   1. All patients with lower extremity trauma should be evaluated for associated injuries, and treated according to advanced trauma life support (ATLS) criteria
2. Surgical debridement and irrigation of the wound in the operating room is the proper initial management. Specific management depends upon the level of injury, presence or absence of vascular, nerve, and bony injury

3. Limb threatening injuries, vascular interruption, or open fracture are best assessed and treated in the OR

4. Fasciotomy is often required to maintain tissue perfusion in severe high energy or crush injuries when compartment syndrome exists

B. Fractures of the lower leg are usually classified by the Gustilo system (Table 7-1)

1. Type I and II fractures usually have a good outcome with varied treatment

2. Gustilo Type III injuries have a worse prognosis and often require plastic surgical intervention

<table>
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<tr>
<th>Gustilo Classification of Open Fractures of the Lower Leg</th>
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<tbody>
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<td>Type I</td>
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<td>Type II</td>
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<td>Type IIIA</td>
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<td>Type IIIB</td>
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<td>Type IIIC</td>
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Table 1. Gustilo Classification of Open Fractures of the Lower Leg

III. NON-SURGICAL CARE

A. Wound vac
   1. Applies negative pressure to aid in wound healing
   2. Has decreased the need for flaps in many cases

IV. SURGICAL CARE

A. Indications for limb salvage versus amputation
   1. Assessment tools - none are good enough to use as a guide in management
   2. Consider co-morbidities, concomitant injuries, and whether multi-level injury
   3. Posterior tibial nerve function as the determinate - has been challenged
4. Plastic surgery may still be involved in amputations to preserve length for prosthesis
   a. Fillet flaps - use tissue from amputated part to close surgical site and preserve limb length

B. Essentials
   1. Adequate debridement
   2. Timing
      a. Classic Godina study from 1986 advocated early coverage - within 72 hours
      b. Has been challenged - especially for more extensive injuries, wound vac has probably helped
      c. Cover all vital structures (nerves, vessels, tendons, joints, hardware) within 1 week
   3. Zone of injury
      a. Need to debride all damaged, non-vital tissue
      b. If performing flap coverage, be aware of potential microscopic vessel injury in surrounding tissue. This zone of injury may affect flap or recipient vessel choice.

C. Need for graft vs. flap coverage
   1. Dermal substitutes may help convert a wound requiring flap coverage to a skin graft.

D. Location - Traditional upper, middle, and lower third leg options
   1. Upper third
      a. Gastrocnemius
         i. Blood supply - medial and lateral sural vessels
         ii. May use medial or lateral head, although medial head is more often used due to larger size and more favorable arch of rotation
         iii. Peroneal nerve at risk for injury with lateral head
   2. Middle third
      a. Soleus
         i. Blood supply - popliteal vessels (proximal), posterior tibial vessels (medial belly), and peroneal vessels (lateral belly)
         ii. Divide distally to rotate into defect
         iii. May split in half if necessary
   3. Lower third
      a. Free flap
         i. Considerations with flap selection - pedicle length, OR positioning (concomitant injuries), durability on heel, bony need, sensation
         ii. Recipient vessel selection
            a) Consider preoperative imaging if questionable vascular exam - CTA or angiogram
            b) Consider end-to-side versus end-to-end anastomosis to preserve flow to distal extremity
            c) Vein grafts may be needed
      b. Reverse sural flap
         i. Blood supply - peroneal vessel perforators
         ii. Controversy over whether reliable and truly more simple than free flap
a) If chosen, consider surgical delay to improve the perfusion and delineate viable tissue
c. Medial plantar flap
   i. Blood supply - medial plantar vessels
   ii. Sensate flap
E. Free-style perforator flaps
   1. Locoregional flaps based off perforators near the defect
   2. Not necessarily a simpler option, need to consider zone of injury
F. Bony gaps - may be filled with grafts, flaps, or distraction techniques depending on length and quality or remaining bone
G. Postop care
   1. Elevate extremity
   2. Dangle protocols
      a. Want to slowly get flap used to being in a dependent position, will swell and get congested at first
      b. No hard evidence for best timeline

REFERENCES

I. BROW LIFT (Figures 1 and 2)

A. Open Coronal Brow Lift Technique
   1. Coronal incision is made in the hair-bearing skin
   2. Scalp flap is elevated caudally in a variety of planes towards the orbits, allowing broad exposure of the brow attachments, which can be released and repositioned

B. Endoscopic Brow Lift
   1. Endoscopic ports are made in the hair-bearing skin and endoscopic instruments are used to release the brow attachments under direct camera visualization
   2. The repositioned brow is secured to the bone and/or periosteum through a variety of methods.

C. Hairline Brow Lift
   1. Incision is made along the hairline and a flap is elevated caudally towards the orbits, the brow attachments are released, and the brow is repositioned.
   2. Excess skin is trimmed along the incision line and the incision is carefully closed.

D. Lateral Brow Lift
   1. Incision is made either in the temple hair-bearing skin or along the temple hairline, dissection proceeds to the lateral eyebrow in a plane deep to the superficial temporal fascia.
   2. The lateral brow is re-positioned, excess skin is excised at the incision line and the incision is carefully closed and secured to the deep temporal fascia.

E. Direct Brow Lift
   1. Incision is made in the superior border of the eyebrow and a pre-determined amount of forehead skin is excised between the brow incision and new desired location for the eyebrow.
   2. Once the intervening skin tissue is excised, the incision edges are carefully approximated.

II. NON-SURGICAL (CHEMICAL) BROW LIFT

A. A temporary brow lift can be performed with neuromodulators (Ex. Botox or Dysport) by selectively treating the medial and lateral brow depressor muscles
Figure 1. Incision placement for temporal brow lift
From Codner et al. Blepharoplasty & Brow Lift, Plast Reconst Surg CME. July 2010; 126(1).

Figure 2. Anatomical position of the sentinel vein, temporal branch of facial nerve and ligamentous attachments
III. FACELIFT

A. Facial Aging surface anatomy (Figure 3)
   1. The human face will age in predictable patterns
   2. Common patterns of aging
      a. Descent and deflation of the midface
      b. Development of a prominent tear trough
      c. Deepening of the nasolabial fold
      d. Flattening and lengthening of the upper lip
      e. Development of jowls
      f. Laxity in the neck skin and platysma muscle

![Figure 3. Surface anatomy of the male and female face](image)

(Left) The average of facial surfaces of 116 female subjects aged 20-30 years. (Right) The average of facial surfaces of 100 female subjects, aged 68-91 years (average 76 years). Photographs courtesy of Val Lambros, MD.


IV. FACELIFT ANATOMY

A. Facial Nerve: 5 motor branches which start deep in the lateral face and ascend superficially as they travel medially in the face.
   1. Frontal
   2. Zygomatic
   3. Buccal
4. Marginal Mandibular
5. Cervical
B. Facial Retaining Ligaments

Figure 4. Fresh tissue dissection demonstrates the location of the facial nerve branches and facial artery. Note the location of the branching point of the cervical motor nerve below the mandible and its relation to the angle of the mandible.


C. SMAS (Superficial Musculo-Aponeurotic System)
   1. Superficial muscle-fascial layer in the head and neck
   2. Originates as the platysma in the neck and extends superiorly as a fascial layer just below the subcutaneous fat in the face. Terminates superior to the zygoma as the superficial temporal fascia (aka Temporoparietal Fascia).

V. FACELIFT OPERATIVE TECHNIQUES

A. Subcutaneous only
   1. The facial skin is widely undermined and re-draped. The SMAS is not addressed in this technique.
B. SMAS plication
   1. The facial skin is undermined and the SMAS tissue is sutured together in order to tighten and reposition the SMAS architecture.
C. SMAS-ectomy
   1. The facial skin is undermined and an oblique piece of SMAS is excised, the cut edges of the remaining SMAS are then sutured together.
D. Extended SMAS
1. The facial skin is undermined and a separate flap of SMAS is elevated and sutured superior-laterally to reposition the SMAS.

E. Composite / Deep Plane

1. A limited amount of facial skin undermining is performed and then the SMAS and skin are left adherent to each other as a “composite flap” while a sub-SMAS dissection is performed. Once the composite flap is adequately mobilized, it is repositioned and excess skin/tissue is trimmed.

Figure 5. Ninety degree SMAS plication (left); Extended-SMAS (right)
VI. FACELIFT COMPLICATIONS

A. Hematoma
B. Facial Nerve Injury
   1. The most common branch injured is the buccal branch however cross-innervation from adjacent nerve branches decreases the long-term morbidity of buccal branch injury.
   2. Injury to the frontal branch or marginal mandibular branch result in significant morbidity because they do not have significant cross-innervation from adjacent nerves.
C. Skin sloughing (secondary to excess tension on the skin flap)

VII. BLEPHAROPLASTY

A. Anterior Lamella Anatomy
   1. Skin
   2. Orbicularis Oculi muscle
      a. Pre-tarsal
      b. Pre-Septal
c. Orbital

B. Posterior Lamella Anatomy
1. Tarsal Plate & conjunctiva
2. Levator Palpebrae Superiorus Muscle (upper lid)
3. Muller muscle (upper lid)
4. Orbital Fat
   a. Upper Lid
      i. Medial & Middle fat pads
      ii. Lacrimal Gland is lateral
   b. Lower Lid
      i. Medial, Middle and Lateral fat pads
      ii. Inferior Oblique muscle is between the medial and middle fat pads
5. The orbital septum and orbital fat constitute the middle lamella.

C. Pre-operative evaluation (Upper Eyelid)
1. Eyelid ptosis
2. Brow ptosis
3. Levator and Frontalis muscle function

D. Pre-operative evaluation (Lower Eyelid)
1. Eyelid laxity (distraction test)
2. Eyelid tone (snap back test)
3. Vector analysis (positive/neutral/negative)
4. Eyelid retraction & scleral show

VIII. BLEPHAROPLASTY OPERATIVE TECHNIQUES

A. Upper Eyelid
1. Excess skin is excised +/- a strip of orbicularis muscle
2. In certain cases orbital septum is opened and varying amounts of intraorbital fat is excised.
3. Levator surgery can be performed for patients with ptosis

B. Lower Eyelid
1. Approaches
   a. Transconjunctival
      i. Incision is made on the inside of the lower lid through the conjunctiva
   b. Subciliary:
      i. Incision is made just below the eyelashes on the external eyelid skin
Figure 7. (Left) Approach for transconjunctival blepharoplasty; (Right) Lateral view of transconjunctival blepharoplasty; (Below) Removal of fat from the transconjunctival incision.

From Codner et al. “Blepharoplasty and Brow Lift” PRS CME. July 2010; 126(1)

2. Volume Reduction
   a. In certain cases the intraorbital fat is removed and/or repositioned to blend the lid-cheek junction.

3. Lateral Canthal repositioning for support
   a. Canthopexy
   b. Canthoplasty

IX. BLEPHAROPLASTY COMPLICATIONS

A. Retrobulbar Hematoma
B. Ectropion
C. Lagophthalmos
X. RHYTIDECTOMY AND NECK REJUVENATION

A. Often performed in conjunction with facelift

XI. NECK ANATOMY

A. Pre-Platysma / Subcutaneous Fat
B. Platysma
C. Sub-platysma structures
   1. Submandibular gland
   2. Anterior Digastric muscle
   3. Inter-digastric / Sub-platysemal fat

XII. OPERATIVE TECHNIQUES IN NECK REJUVENATION

A. Anterior Approach
   1. Submental incision
   2. Release of platysmal bands (+/- muscle transection)
   3. Re-approximation of platysma at midline (corset platysmaplasty)
   4. Sub-platysemal structures are addressed if indicated
B. Lateral approach (through facelift incision)
   1. Facelift incision (+/- undermining of neck skin)
   2. Reposition the platysma muscle from a lateral position through a variety of techniques

XIII. ADJUVANT PROCEDURES – FINISHING TOUCHES

A. Fat Grafting
B. Laser Resurfacing
C. Dermabrasion
D. Chemical Peels
E. Implants (chin, jawline, cheek)

XIV. RHINOPLASTY

A. Surface Anatomy & Preliminary Analysis
   1. Skin quality and texture
B. Upper Third
   1. Dorsal Aesthetic Lines
   2. Nasal Bone
   3. Radix
C. Middle Third
   1. Upper Lateral cartilage
   2. Keystone
   3. Internal Nasal Valve
D. Lower Third
   1. Lower Lateral cartilage
   2. External Nasal Valve
   3. Soft Triangle
E. Nasal Septum (also assess for turbinate hypertrophy)

Figure 8. Surface anatomy of the nose
The external and internal nasal valves. The external valve is formed by: (1) caudal edge of the lateral crus of the lower lateral cartilage, (2) soft-tissue alae, (3) membranous septum and (4) nostril sill. The internal nasal valve accounts for ½ of total airway resistance. Borders: (medial) – septum, (inferior) – nasal floor, (lateral) – inferior turbinate.


XV. OPERATIVE TECHNIQUES IN RHINOPLASTY

A. Closed Approach

B. Open Approach

1. Incisions
   a. Skin
      i. Transcolumellar
   b. Alar
      i. Rim Incision (internal incision at border of alar rim)
      ii. Marginal Incision (internal incision at caudal border of lower lateral cartilage)
      iii. Intercartilagenous (internal between upper and lower lateral cartilages)
   c. Septal
      i. Hemi-transfixion
      ii. Full-transfixion
      iii. Killian incision

2. Maneuvers
   a. Septoplasty +/- Turbinate reduction
   b. Dorsal hump reduction or augmentation
   c. Osteotomies
   d. Cephalic Trim
   e. Nasal tip re-structuring
   f. Cartilage grafting for shape, contour and/or support
Figure 9. (Left) Width of dorsal and caudal L strut should be at least 15 mm to ensure long-term support. (Right) Tip suturing techniques: (top) medial crural columellar strut suture, (center) transdomal suture, (below) interdomal suture. From Rohrich et al. “A Practical Approach to Rhinoplasty” Plast Reconst Surg CME. 2016; 137(4).

REFERENCES

Aesthetic surgery of the breast aims to either correct ptosis with a mastopexy, micromastia with augmentation, or macromastia with a reduction. The goals are to create a youthful, well-appearing breast. Important considerations for all patients include an assessment of their breast history including history of breast cancer, mammographic history and measurements of the breast and chest including sternal-notch to nipple, nipple to IMF, internipple distances and breast width and areolar diameter.

I. MASTOPEXY

A. Goals
   1. Correct breast ptosis with skin redraping and parenchymal remodeling of the breast to reduce mismatch between skin and tissue.
   2. Adjust nipple position concurrently.

B. Regnault Classification of breast ptosis based on two important anatomic landmarks: (1) inframammary fold (IMF) and (2) nipple position
   1. Grade I – nipple at level of the IMF
   2. Grade II – nipple below the level of IMF but not at the lowest point of the breast
   3. Grade III – nipple at the lowest point of the breast
   4. Pseudoptosis – nipple at the level of IMF but glandular tissue below the level of the IMF

Figure 1. Ideal nipple position is based on its relationship to the inframammary fold. From Nahabedian MY. Breast deformities & mastopexy. Plast Reconstr Surg 2011 Apr;127(4):91e-102e.
C. Patient concerns
   1. Usually bothered by the “shape” of the breast or size and position of nipple-areola complex.
   2. Often report deflation and sagging of breasts.

D. Periareolar mastopexy
   1. Incision is made around the areola as an eccentric oval
   2. Can be used to raise the nipple up to 2 cm
   3. Good for patients with grade I or II ptosis
   4. Disadvantages include widening of the periareolar scar, flattening and de-projecting of the breast

![Figure 2. Periareolar mastopexy with mesh support](From Hidalgo D, Spector J. Mastopexy. Plast Reconst Surg 2013 Oct;132(4)642e-656e.

E. Circumvertical or Vertical mastopexy
   1. Incision made around the areola and down the meridian of the breast
   2. Can be used for grade II and III ptosis
   3. Usually involves removal of breast skin and underlying tissue from the lower pole of the breast with sutures to bring the medial and lateral pillars of the breast together
4. This helps support the elevated nipple-areola complex at its new, higher position

Figure 3. Vertical mastopexy

F. Wise-pattern mastopexy
   1. Incision made around the areola and across to the medial and lateral edges of the breast and along the IMF
   2. Can be used for grade II and III ptosis and patients with severe excess of skin
   3. Has largest scar burden of any mastopexy technique and can lead to “bottoming out” or recurrent ptosis of the gland when used with an inferior pedicle

Figure 4. Wise pattern or inverted T mastopexy incision types

II. AUGMENTATION

A. Goals: increase the size of the breast parenchyma either using the patient’s own tissue (autologous) or a prosthetic device (saline or silicone implant)
B. Patient concerns
1. Usually bothered by the size of the breasts.
2. Important parameters in pre-operative evaluation
   a. “Base width:” width of the breast on the chest wall
   b. Pinch thickness of the upper pole breast tissue.
   c. Ability of skin to stretch
   d. Chest wall asymmetry
C. Augmentation with an Implant
   1. Planes of placement: implant has to be covered by stable soft tissue, which includes a combination of skin, parenchyma and muscle.
      a. Subglandular – beneath the breast tissue but above the pectoralis muscle fascia
      b. Subfascial – beneath the pectoralis muscle fascia but above pectoralis muscle fibers
      c. Subpectoral – beneath the pectoralis major muscle
      d. Dual plane – pectoralis muscle is released to a variable extent with release of inferior attachments and preservation of medial attachments. Muscle covers the superior part of the implant and breast tissue covers the inferior part of the implant, depending on extent of release of muscle from overlying parenchyma.

Figure 4. Subpectoral implant placement. Release of pectoralis muscle allows the muscle to rise above the implant

2. Incision choices: inframammary, transaxillary, periareolar are the most common.
3. Implants can be round or shaped (anatomic), saline or silicone, smooth or textured
4. BIA-ALCL (Breast Implant Associated Anaplastic Large Cell Lymphoma)
   a. Potential risk with textured devices
   b. An area of evolving research
   c. Presents with a delayed seroma
   d. Cytology: CD30+ and ALK+.
   e. Treatment
      i. Involves at least removal of the implant and complete capsulectomy.
      ii. Requires multidisciplinary team approach including medical oncologist involvement.

D. Augmentation with autologous tissue
   1. Fat –
      a. Can be harvested from another part of the body (i.e. abdomen or thighs) and injected in the various planes of the breast to increase the size of the breast.
      b. Can involve the use of an external tissue expanding device that increase the space within the breast to accept fat as well as neovascularization of the tissue bed (ie. BRAVA system).
   2. Auto-augmentation
      a. Various flaps can be designed on lateral axillary tissue or adjacent tissue and rotated, advanced or folded onto the existing breast mound to reshape and increase the size of the breast.
      b. Commonly performed in massive weight loss patients.
   3. Free flaps and abdominally based flaps

E. Mastopexy-Augmentation
   1. Combined procedure to both increase the size and alter the shape of the breast
   2. Can be staged in two operations or perform simultaneously
   3. Revision rates ~10-30% range

III. REDUCTION

A. Goals
   1. Decrease the size of the breast
   2. Restore nipple location to a more youthful position

B. Patient concerns
   1. Symptomatic patients usually have a history of cervical, shoulder or back pain that have not improved with medications or physical therapy
   2. May also have a history of rashes or hygiene issues
   3. Bra strap grooving as a result of large breasts
   4. Reductions can be done in asymptomatic patients as well for cosmetic reasons.

C. Skin incision patterns and pedicles (which provide blood supply to the nipple areola complex) are two different considerations in breast reduction and can be combined
(for example, Wise-pattern superomedial reduction versus Wise-pattern inferior pedicle reduction)

D. Skin incisions: circumvertical or Wise-pattern

E. Basis of pedicles for blood supply to the NAC:
   1. Inferior – 4th intercostal perforator
   2. Superior – 1st and 2nd intercostal perforators
   3. Superomedial – 2nd intercostal perforator
   4. Medial – 2nd or 3rd intercostal perforator
   5. Lateral – lateral intercostal perforators
   6. Central – musculocutaneous perforators from thoracoacromial artery

Figure 5. Anatomy and blood supply to the breast
F. Other techniques for very large and ptotic breasts include: breast amputation with free nipple grafting

G. Complications
1. Loss in ability to breast feed
2. Changes in sensation.
   a. Can get better, worse or stay the same as the nerve are often on stretch from the weight of breast tissue
3. Loss of the NAC due to inadequate blood supply
   a. If a change in color is noted, must re-open stitches, assess pedicle, etc.
   b. If remains ischemic or congested despite intraoperative maneuvers, an option includes converting the nipple to a free nipple graft with similar principles to skin grafting for survival.

REFERENCES

Body contouring is an umbrella term for aesthetic surgery of the body that includes the breasts, abdomen/trunk and upper and lower extremities. It is also used to describe a set of procedures for patients who have experienced massive weight loss either from bariatric surgery and/or diet and exercise. These patients often have a disproportionate amount of deflated skin secondary to volume loss from atrophy underlying subcutaneous fat/adipocytes. Body contouring involves liposuction, excisional surgery and more recently non-invasive methods like cryolipolysis to achieve the patient’s aesthetic goals.

I. LIPOSUCTION

A. Suction assisted lipectomy (SAL)
   1. Goal – to improve the contour of specific anatomic regions by surgically removing targeted/focal areas of fat.
   2. Surgically removes fat, but will not address excess skin.
   4. In recent years, energy sources have been added to the procedure in hope to break apart fibrous tissue and facilitate the destruction of adipocytes via thermal mechanism (LASER and ultrasound).

B. The total amount of fat in the body is fixed. However, individual adipocytes have the ability to get bigger or smaller. Liposuction can remove fat but the left over fat still can change with fluctuations in weight. Therefore, even if fat is removed, the remaining fat can change in volume and lead to loss of results.

C. Pre-operative evaluation
   1. Ideal candidates are patients who have localized deposits of diet or exercise resistant fat with good overlying skin quality.
   2. Caution in the male abdomen.
      a. Women tend to have extra-abdominal fat meaning the fat deposits in the subcutaneous tissue OUTSIDE the abdominal cavity.
      b. Men tend to have increases in intra-abdominal fat meaning the fat INSIDE the abdominal cavity can increase. Intra-abdominal fat is NOT the target of liposuction. Can be used in male pseudogynecomastia.
   3. Markings are essential as well as discussion of any pre-existing asymmetries or contour irregularities that exist preoperatively.
4. An estimation of volume to be removed can help estimate how much wetting solution, “tumescent” will be needed and how much fat can eventually be removed.

D. Setup: Liposuction involves two major components: tumescent of the areas to be treated and the device being used

E. Tumescent: Doses of lidocaine with epinephrine can be as high as 70 mg/kg because of the large volume of distribution and slow absorption of anesthetic from fat. However, the peak plasma concentration is 10-12 hours after injection so patients after the procedure can experience lidocaine toxicity at home and they and family members should be warned about signs and symptoms to look out for post procedure. With this in mind, the accepted safe dose is typically 35 mg/kg-50 mg/kg.

<table>
<thead>
<tr>
<th>Tumescent Technique</th>
<th>Infiltrate</th>
<th>Estimate of Blood Loss (as a % of volume aspirated)</th>
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<tbody>
<tr>
<td>Dry</td>
<td>No infiltrate</td>
<td>20-45</td>
</tr>
<tr>
<td>Wet</td>
<td>200-300 cc's/area</td>
<td>4-30</td>
</tr>
<tr>
<td>Superwet</td>
<td>1 cc / 1 cc aspirate</td>
<td>&lt;1</td>
</tr>
<tr>
<td></td>
<td>2-3 cc infiltrate per 1 cc aspirate</td>
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</tr>
<tr>
<td>Tumescent Infiltrate</td>
<td>aspirate</td>
<td>&lt;1</td>
</tr>
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</table>

Table 1. Types of tumescent solutions

F. Post-operative considerations
1. Lidocaine toxicity
2. Compression garments are key to providing comfort and helping obtain the final contour after surgery.
3. Large volume liposuction: considered as liposuction > 5L of lipoaspirate in most states
   a. Often warrants admission to the hospital for post-procedure monitoring.
4. Patients can experience large volume shifts after surgery, so special attention should be made in patients with cardiac or renal disease.

II. CRYOLIPOLYSIS

A. New technology that cools pockets of fat using a non-surgical device and induces adipocytes to undergo apoptosis
B. Non-invasive technology
C. Relies on the body’s own system of apoptosis and clearance for removal of fat
III. EXCISIONAL BODY CONTOURING

Focuses on removal of excess skin in addition to fat and can involve the breast, abdomen, arms, thighs, upper and lower back and buttock area. Many patients have hygiene issues with chronic rashes and infections. In the breasts, patients may have associated back pain and cervical neck pain.

A. Breast
   1. Goal: restore normal shape of breast +/- increase or decrease volume based on pre-existing volume of the breast
   2. Often the massive weight loss patient's breasts are associated with distortion of normal anatomy
   3. Breasts can have medially deviated nipples, loss of lateral breast border with excess axillary tissue, hollowing of the upper pole of the breast and a lax inframammary fold
   4. May require either removal of breast tissue (reduction) or rearrangement of existing tissue in a mastopexy (breast lift)
   5. Can also involve placement of an implant to restore volume of shape which can be placed behind the muscle, breast tissue or both (see chapter on breast augmentation).
   6. Mastopexy in massive weight loss patients can use existing lateral breast tissue and rearrangement to restore the shape and preserve volume of the breast

B. Arms
   1. Goal: remove excess skin and fat from the axilla down to the forearm when necessary to improve the contour of the arm
   2. Brachioplasty is an excisional body contouring procedure that removes skin and fat and can extend into the axilla and past the forearm when necessary
   3. Liposuction can be used as an adjunct to help debulk the arm before removal of skin or to help smooth the contour proximally and laterally as well as circumferentially around the arm
   4. A number of scars can be designed and are commonly posterior (straight vertical along the posterior arm) or low along the bicipital groove like the seam of a shirt so the scar is hidden with the arms at the side
   5. Pinch test and “tailor-tacking” are key when determining amount of skin and fat that can be removed
   6. Most common post-operative issue is a large, widened scar
   7. There are several types of important anatomic structures deep to the fascia including branches of the medial antebrachial cutaneous nerve (MABC), which when damaged can lead to paresthesias along the proximal forearm or a painful neuroma
   8. Some surgeons close the skin as they cut each segment to prohibit edema and inability to close the arm. This is a dreaded complication that may require skin grafting.
   9. Post-operative care usually involves a compression garment
C. Abdomen - Panniculectomy vs. Abdominoplasty

1. Goals: restore the normal contour of the abdomen +/- correction of rectus diastasis or widening of the medial edges of the rectus abdominis muscles.

2. Panniculectomy - generally an infraumbilical wedge excision of excess skin and fat. Does not include rectus abdominis muscle plication. May or may not include umbilicoplasty (umbilicus is transposed into the newly mobilized abdominal tissue).
   a. Usually performed to improve hygiene issues as tissue under panniculus can be frequently macerated, ulcerated or infected.
   b. Can be performed in a fleur-de-lis manner to excise not only vertical but horizontal excess.

3. Abdominoplasty – usually infraumbilical and often supraumbilical excision of excess skin and fat. Often includes rectus plication to correct widely separated rectus muscles. Almost always involves umbilicoplasty with transposition of the umbilicus.
   a. Important to rule out existing hernias (umbilical or ventral) that may need repair as well as previous incisions including C-section scars and open cholecystectomy or appendectomy scars.
   b. Commonly combined with lateral flank liposuction.
   c. Has high risk of DVT/PE and patients are often assessed for risk of clot formation with the Caprini score. Many patients are on post-operative chemical DVT prophylaxis.
   d. One of the highest risk cosmetic surgeries.
   e. More commonly being performed without a drain as a “drainless abdominoplasty” in which progressive tension sutures are placed to close down dead space and reduce the risk of seroma formation.
   f. Seromas are a common complication.

4. Circumferential Abdominoplasty (Belt Lipectomy or Lower Body Lift):
   a. Goals: excise excess skin and fat circumferentially including the posterior waistline and flank areas.
   b. Often begins with posterior excision then anterior excision.
   c. Can be staged as two different procedures or performed in one stage.

D. Medial Thigh Lift


2. Can be performed with a number of incisions placed in the medial thigh (inguinal crease) sometimes with a vertical extension down the medial thigh.

3. Results improved with suspension of the superficial fascial system to Colles fascia along the pubic ramus.

4. Liposuction can be used as an adjunct procedure to improve contour.

5. Requires careful dissection over femoral triangle to preserve lymphatics and careful dissection medially to not injure the saphenous vein.

6. Often requires drains but high risk of seroma formation.

7. Involves compression garment post-procedure.

E. Back

1. Goals: excise extra skin and fat that can get be accentuated in clothes like bra’s or dresses and improve the contour of the upper back.
2. Direct excision of back rolls can be performed
3. Excisions can be combined with breast procedures as some patients have excess that courses laterally that is not breast tissue and is considered an “upper body lift”

F. Buttock
1. Goals: improve the contour and volume of the buttock
2. Commonly involves a posterior waistline scar with undermining into the gluteal area
3. The plane of dissection can be altered so that the tissue raised can be placed on top of the existing buttock tissue and used as a form of “auto-augmentation”
4. Gluteal implants, fat grafting and other purse-string suture can be used to augment the buttock which is often ptotic in massive weight loss patients

REFERENCES

CHAPTER 20

INJECTABLES, LASERS, PEELS & NON-INVASIVE PROCEDURES

Sammy Sinno, MD and Jacob Unger, MD

I. INJECTABLES

A. Soft Tissue Fillers
   1. Autologous
      a. Fat
      b. Dermafat grafts
      c. Fascial grafts (ie - fascia lata)
   2. Homologous
      a. Alloderm®
         i. acellular dermal graft is derived from skin obtained from tissue banks
         ii. can be micronized
      3. Synthetic
         a. Radiesse™
            i. microspheres of calcium hydroxylapatite-based implant
            ii. stimulate natural collagen growth, actually causing new tissue development
            iii. is also useful in the treatment of facial lipoatrophy (a stigmatizing effect of HIV), vocal cord deficiencies, oral and maxillofacial defects, as well as scars and chin dimples
         b. Hyaluronic acid-most commonly used fillers today
            i. G prime (or G’)-related to a fillers “hardness”; the smoother the product is to inject the lower the G’
            ii. Restylane™-many different formulations
            iii. Juvederm™-many different formulations
            iv. Perlane™-larger particle size
            v. Belotero™-low-viscosity
            vi. Can be broken down with hyaluronidase
            vii. Common treatment areas include nasolabial folds, lips, cheek/malar area, temples, lower eyelids
            viii. Typically last 6-15 months
         c. Sculptra™
            i. Poly-L lactic avid
            ii. FDA approved for HID-related lipodystrophy

B. Neurotoxin
   1. Botox™
   2. Dysport™
   3. Xeomin™
   4. All work by preventing the binding and release of acetylcholine at the neuromuscular junction
5. Duration of action 4-6 months
6. FDA approved for glabellar wrinkles and crow’s feet

II. LASERS

A. Produce heat in target tissue (chromophore) in the skin
B. Used to improve facial wrinkles and irregular skin surfaces
   1. In many cases, facial wrinkles form in localized areas, such as near the eyes or around the mouth. The depth of laser of treatment can be tightly controlled so that specific areas are targeted as desired
   2. When healing is complete, the skin has a more youthful appearance
C. Can be ablative or nonablative
D. Ablative lasers
   1. CO2
      a. Target chromophore is water
      b. Cause deeper thermal injury
      c. 8-10 day recovery period
      d. Can cause hypopigmentation
   2. Fractionated CO2
      a. Delivers pulse in spaced pixilated pattern
   3. Erbium:YAG
      a. Shorter wavelength than CO2
      b. Target chromophore is water
      c. Less thermal diffusion
      d. Shorter recover time (5-6 days)
E. Non-ablative lasers
   1. Fractional resurfacing
      a. Blue dye on skin is target chromophore
      b. Required multiple treatments
      c. 1-2 day downtime
   2. ND-YAG
      a. Non-specific target
      b. No epidermal ablation; targets the dermis

III. PEELS

A. Chemical peel is especially useful for the fine wrinkles on the cheeks, forehead and around the eyes, and the vertical wrinkles around the mouth
B. The chemical solution can be applied to the entire face or to a specific area – for example around the mouth – sometimes in conjunction with a facelift
C. At the end of the peel, various dressings or ointments may be applied to the treated area
D. A protective crust may be allowed to form over the new skin. When it’s removed, the skin underneath will be bright pink
E. After healing, the skin is lighter in color, tighter, smoother, younger looking
F. Superficial peels include: salicylic acid, Jessner’s solution, and glycolic acid
G. Moderate peels include: TCA peels
H. Deep peels include: Hetter’s formula, Baker-Gordon peel

IV. OTHER NON-INVASIVE MODALITIES

A. Dermabrasion
   1. The surgeon removes the top layers of the skin using an electrically operated instrument with a rough wire brush or diamond impregnated burr
   2. Typical downtime 7-10 days
B. Microdermabrasion
   1. Suction-based device that removes debris and dead skin
   2. Minimal downtime
C. Thermage
   1. Used to tighten skin laxity
   2. Uses periodic radiofrequency and cooling
   3. Results are more modest
D. Ultherapy
   1. Focused ultrasound treatment
   2. Energy given in pulses
   3. Multiple treatments required
   4. Results are more modest
   5. Cryotherapy (i.e. Coolsculpting™) Heat-based Therapy (i.e. Vanquish™) and Ultrasound based Therapy (Ultrashape Power Plus™)
      a. Use focused areas of cooling or heating to target subcutaneous fat
      b. Typically requires several treatments
      c. Minimal downtime
      d. Much less dramatic results compared to surgery (29% Fat Reduction

REFERENCES

CHAPTER 21

INTERNATIONAL PLASTIC SURGERY

Yemi Ogunleye, MD, SM and Richard L. Agag, MD

I. INTRODUCTION

A. Plastic surgery is a surgical specialty with global roots. Records dating back to 800BC in ancient India are the earliest known records of surgical rhinoplasty based on the works of Sushruta, an ayurvedic physician.

B. However, clinical and technological advancements in the past century have not benefited patients in the developing and developed world equally.

II. GLOBAL PRACTICE IN PLASTIC SURGERY

A. Plastic surgery practice in the low and middle income countries (LMIC) focuses largely of chronic wound care, burn care and congenital face and hand deformity reconstruction.

B. There is a lack of access to reconstructive surgery in many parts of the world, especially in children, due to factors ranging from poverty to lack of surgical manpower.

III. INTERNATIONAL PLASTIC SURGERY OUTREACH

A. Plastic surgeons in the United States have sought to bridge this divide by organizing surgical missions to LMICs and providing free surgical care.

B. Plastic surgical charities/NGOs such as Operation Smile, Smile Train, Resurge International (formerly Interplast), Rotaplast and Facing The World spearhead these efforts.

C. Smaller groups of surgeons or individual have also organized short trips to resource-poor countries

D. Surgical missions provide numerous benefits to patients who are otherwise unable to access surgical care.

1. They can provide resources and improve manpower and training for local partners.

2. They improve cost-conscious practice, cultural and clinical skills in volunteers.

3. Concerns exist about the standards of practice, cultural shock and ethics of surgical care delivery.

E. Three common principles described by Garfein et. al. when delivering care in developing countries include

1. Build and support local infrastructure

2. Create long-term, self-sufficient care

3. Adhere to the highest standards.
4. Patel et al have described a diagonal approach to surgical care delivery that takes all these factors into consideration (Figure 1).


**IV. EDUCATION**

A. Plastic surgical trainees also participate in surgical missions and several residency programs have obtained approval from the Residency Review Committee of the ACGME to include global health or international rotations to LMICs as part of surgical training.

B. Such rotations benefit residents by exposing them to different pathologies and innovative operative and treatment approaches.

C. Global surgery fellowships focused on plastic surgery also provide post-residency clinical and research training for advanced trainees with a career interest in global surgery (Table 1).
### Table 1. Global Surgery Fellowship List

<table>
<thead>
<tr>
<th>Fellowship (Web Site)</th>
<th>Organization</th>
<th>Key Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regan Fellowship</td>
<td>Operation Smile</td>
<td>The Regan Fellowship offers residents physician the opportunity to participate in the life-changing work of Operation Smile. Made possible by invaluable donor support, residents in plastic surgery, pediatrics, and anesthesia are invited to attend an international medical mission and work under the supervision and mentorship of veteran Operation Smile physician. While on the medical mission, residents also participate in our research initiatives that will allow for better treatment and prevention of cleft-lip and cleft palate. During this program, residents may network with one another and share their experiences as the annual conference of Regan Fellows.</td>
</tr>
<tr>
<td>Stryker International Fellows Program</td>
<td>Operation Smile</td>
<td>The Stryker International Fellows Program seeks to build a global team of international rising plastic surgeons who have had a unique exposure to the humanitarian programs of Operation Smile and to the surgical management of cleft lip and cleft palate.</td>
</tr>
<tr>
<td>Tsao Global Surgery Fellow</td>
<td>Children’s Hospital Los Angeles, Operation Smile, and the USC Institute of Global Health</td>
<td>Organized by Children’s Hospital Los Angeles, Operation Smile, and the USC Institute for Global Health. The program’s fellows develop academic, clinical, and administrative skills in global surgery, clinical and public health research, and humanitarian aid in resource-poor settings. In addition to gaining master’s degrees in clinical and biomedical investigations, they conduct research and travel around the world to where Operation Smile carries out surgical care missions.</td>
</tr>
<tr>
<td>Sterling-Bunnell Fellowship</td>
<td>American Society for Surgery of the Hand</td>
<td>American Society for Surgery of the Hand for a young hand surgeon to foster national and international relationships.</td>
</tr>
<tr>
<td>Jerome P Webster Fellowship</td>
<td>ReSurge International</td>
<td>The Webster Fellowship offers a newly trained, board-eligible plastic surgeon the opportunity to spend a year with Interplast’s medical colleagues in developing countries such as Bangladesh, Bolivia, Brazil, China, Ecuador, Ghana, Myanmar, Nepal, Nicaragua, Peru, Sri Lanka, Thailand, and Zambia—performing the highest form of medical citizenship through care of those in need.</td>
</tr>
<tr>
<td>The John D. Constable International Traveling Fellowship</td>
<td>American Association of Plastic Surgeons</td>
<td>The John D. Constable International Traveling Fellowship has been an integral part of the American Association of Plastic Surgeons since 2006 and has provided an opportunity for international plastic surgeons to work with leaders in American plastic surgery.</td>
</tr>
</tbody>
</table>

**REFERENCES**

I. INTRODUCTION

A. Microsurgery: type of surgery which uses precision instruments and a microscope to perform intricate surgery on small structures (arteries, veins, nerves, lymph vessels)

B. Examples
   1. Free tissue transfer (aka "free flap" surgery), wherein living tissue is transplanted from one part of the body to another (Figure 1)
   2. Appendage replantation or reconstruction (Figure 2)
   3. Vascularized composite allotransplantation (VCA), wherein living tissue is transplanted from one individual to another (face and hand transplants)

C. Potential tissues transferred: skin, muscle, tendons, bone, nerve, lymph nodes

D. Transferred tissue is selected so it can survive based on a vascular pedicle.
   1. Tissue is transferred to another region of the body
   2. Artery and vein are re-connected ("anastomosed") to blood vessels in the recipient site
   3. Microvascular anastomosis restores blood supply to the transferred tissue

Figure 1. Free tissue transfer ("free flap") for breast reconstruction following mastectomy. Abdominal tissue is transferred to the former breast site, including the Deep and/or Superficial Inferior Epigastric Artery/Vein (DIEA/V, SIEA/V). From Macadam S, et al. Evidence-Based Medicine: Autologous Breast Reconstruction. Plast Reconstr Surg 2017;139(1):204e-29e.
II. FREE FLAP TYPES

A. Isolated tissue (pedicle)
   1. Skin
      a. Deep Inferior Epigastric Perforator (DIEP; deep inferior epigastric artery)
      b. Groin / Superficial Inferior Epigastric Artery (SIEA)
   2. Muscle
      a. Latissimus dorsi (thoracodorsal artery)
      b. Rectus abdominis (deep inferior epigastric artery)
   3. Fascia
      a. Radial forearm free flap (RFFF; radial artery)
      b. Anterolateral Thigh (ALT; descending branch of lateral femoral circumflex artery)
   4. Bone
      a. Fibula (peroneal artery)
      b. Iliac crest (deep circumflex iliac artery)
   5. Nerve
      a. Combined with other tissues as “innervated” flap.
      b. Innervated gracilis (+obturator nerve)
      c. Free toe transfer (+digital nerves) (Figure 2)
   6. Lymph nodes
      a. Groin, omental nodes
      b. Transferred with surrounding fat and supplying blood vessels

B. Composite: combination of tissues taken as a single flap
   1. Myocutaneous
   2. Fasciocutaneous
   3. Osteocutaneous
   4. Innervated myocutaneous

C. Appendage: finger, toe, penis (replant or reconstruction)

D. Allogeneic composite tissue: face or hand transplant

E. For details, see Grafts & Flaps (Chapter 6), Gender Affirmation Surgery (Chapter 23), Composite Tissue Allotransplantation (Chapter 25), and Lymphedema (Chapter 26)
III. SURGICAL INDICATIONS

A. Adequate / acceptable result cannot be achieved by simpler method
B. Severe tissue loss with exposed vital structures such as:
   1. Joint surfaces
   2. Bone without periosteum
   3. Vessels / Nerves
C. Large "dead space" requiring obliteration (filling)
D. Restore form and/or function:
   1. Breast reconstruction after mastectomy (Figure 1)
   2. Loss of digits after blast injury to hand (Figure 2)
E. Congenital deformity: facial paralysis, severe thumb hypoplasia
F. Gender affirmation surgery: female-to-male bottom surgery
G. Lymphedema: cancer patients with lymph nodes removed for staging
H. Other indications for microvascular techniques (magnification, precision tools)
   1. Lymphaticovenous bypass:
      a. Lymph vessel diameter < 0.8 mm
      b. Vessels anastomosed to local vein to redirect drainage around obstruction

2. Peripheral nerve reconstruction:
   a. Nerve fascicle diameter ranges 0.1-1 mm
   b. *In situ* nerve repair / coaptation / dissection needed

IV. CONTRAINDICATIONS

A. No absolute contraindications
B. Age (young or old) less important than overall health
C. Systemic disease: microsurgery contraindicated if patient cannot tolerate prolonged anesthesia, e.g., surgery duration of 8-12 hours
   1. Cardiovascular and respiratory health
   2. Renal insufficiency (high complication rate)
   3. Hepatic function
   4. Bleeding / clotting disorders (hypercoagulable state increases flap failure rate)
D. Smoking:
   1. Digital replantation contraindicated, ~80-90% failure
   2. No increased failure risk for free flaps
   3. Smokers have more wound healing complications
   4. Encourage quitting
E. Previous radiation: Not a contraindication

V. BASIC OPERATIVE TECHNIQUE

A. Details may be finalized intraoperatively, based on:
   1. Defect size / missing tissue after debridement or tumor resection
   2. Caliber and appearance of available vessels
B. Begin with dissection of tissue for transfer (+vessels) and exposure of recipient vessels
C. Team communication is critical (surgeons, anesthesia, nursing)
D. Key portions of the surgery are beyond the limits of human eyesight
   1. Tiny blood or lymphatic vessels: ~0.5-4 mm (Figure 4)
E. Magnification is used during dissection, anastomosis (of artery, vein, and/or lymph vessels), and nerve coaptation
F. Magnification tools:
   1. Magnifying loupes (custom glasses with attached magnifying lenses, 2.5-6.5x)
   2. Surgical microscope (4-40x)
G. Anastomosis:
   1. Blood flow temporarily obstructed with specialized atraumatic “Acland” clamps
   2. Artery or lymph vessel:
      a. Often sewn together by hand (Figure 3).
      b. Interrupted suture technique common
      c. Small caliber suture: 8-0 nylon (0.4 mm) to 11-0 nylon (0.01 mm)
   3. Veins:
      a. Vessel coupler often used (Figure 4)
      b. Vessel ends pulled through rings and edges everted onto sharp pins
c. Opposing pins inter-digitate, sealing the vessels together
4. Clamps released to evaluate flow across anastomosis (Figure 5).
H. Final steps are inset of transferred tissue and closure of donor site.

Figure 3. Hand-sewn arterial anastomosis under the microscope. Interrupted 8-0 nylon sutures (0.04 mm) were used to sew two ~2.5 mm arteries together. A pair of silver “Acland” clamps obstructs blood flow to enable visualization. From Pu, LLQ. A Comprehensive Approach to Lower Extremity Free-tissue Transfer. PRS Go 2017;5(2):e1228.

Figure 4. Vein anastomosis using a vessel coupler. The end of each vein has been pulled through the rings and everted onto the sharp pins. As the coupler is closed, opposing pins will interdigitate to seal the veins together. From Jandali S, et al. 1000 Consecutive Venous Anastomoses Using the Microvascular Anastomotic Coupler in Breast Reconstruction. Plast Reconstr Surg 2010;125(3):792-8.
VI. POST-OPERATIVE MONITORING & COMPLICATIONS

A. Gold standard: Physical exam
   1. Color
   2. Temperature
   3. Turgor
   4. Capillary refill
   5. Handheld Doppler: Signal checked at marked site on flap skin / surface
B. Arterial compromise: pale, cool, flaccid, slow cap refill
C. Venous congestion: purple/blue, tense, brisk cap refill (Figure 6)
D. Most vascular complications occur in the first 48 hours.

Figure 6. Breast reconstruction complicated by venous congestion of the left breast DIEP flap (upper right), while the right flap continued to have a good color (lower right). Congestion was addressed by anastomosis of an additional vein. Preoperative image (upper left) and postoperative image at 28 months (lower left).


**VII. ADJUNCT MONITORING DEVICES**

A. Implantable Doppler
   1. Probe placed around artery or vein during surgery, connects to external flow monitor (Figure 7)
B. Laser Doppler
C. Flow coupler
   1. Similar to implantable Doppler
D. Transcutaneous tissue oximetry
   1. Placed on flap skin paddle
   2. Measures tissue oxygen saturation (StO2)
   3. Early detection of vascular compromise
E. Prompt return to operating room is critical
   1. Evacuate any hematoma
   2. Inspect vessels
   3. Remove thrombus, revise anastomosis, and/or alleviate kinking, vein grafts
   4. Consider leech therapy if vein patent but flap remains congested.
Figure 7. Implantable Doppler used to monitor a left cheek free flap reconstruction. A small silastic cuff probe (left lower) is placed around the vessel (right) and connected to an external flow monitor (upper left).


VIII. MICROSURGERY TRAINING

A. Specialized technical skills necessary
   1. Use of magnifying loupes and/or microscope (Figure 8)
   2. Tissue and vessel handling
   3. Anastomosis techniques
   4. Suturing at microscopic scales (Figure 9)
B. Often taught in a hands-on lab setting
C. Animal-derived or synthetic vessel models
D. May be incorporated into residency training or offered as a stand-alone course
Figure 8. Albany Medical Center residents practicing microsurgical techniques with surgical loupes (foreground) and operating microscope (background).

Figure 9. Hand-sewn microvascular anastomosis model using 1.5 mm chicken dorsal metatarsal artery. Red dye is injected to assess anastomotic patency. (Inset) Anastomosis magnified 10x with operating microscope. Image credit: Paschalia M. Mountziaris. Copyright 2017.

REFERENCES

CHAPTER 23

GENDER AFFIRMATION SURGERY

Jessica Rose DO, and Josef Hadeed MD, FACS

I. GENDER IDENTITY DISORDER

A. Gender dysphoria describes a group of individuals who are dissatisfied with their anatomic gender and want to acquire the secondary sexual characteristics of the opposite gender
   1. Prevalence
      a. 1 in 11,900 males
      b. 1 in 30,400 females
      c. Worldwide population estimated at 15 million
B. Distinct entity from those individuals with congenital disorders of sexual development/ambiguous genitalia
C. Surgery plays a role in relieving their psychological discomfort
D. Gender reassignment surgery is the best option for normalizing their lives
   1. Genetic males are 3X more likely to seek surgical correction
E. Criteria for diagnosis
   1. Desire to dress and live as opposite gender (and make his/her body a closer resemblance to the opposite gender)
   2. Gender identity present for > 6 months
   3. Not a symptom of another genetic or psychiatric disorder
F. Treatment of these individuals requires a multidisciplinary team
   1. Mental health professionals for psychotherapy
   2. Patients need to have life-experiences in their desired gender role
   3. Hormone therapy (monitored by medical physicians)
      a. Feminization via suppression of androgens and induction of female characteristics
         i. GnRH antagonists
         ii. Estrogens
      b. Masculinization
         i. Testosterone: induces clitoral hypertrophy
      c. Special consideration needs to be taken for adolescents, and puberty suppression may be indicated
   4. Surgeon
G. Etiology unknown
   1. Current debate about whether or not this is a biological or psychosocial disorder
H. Likely a spectrum with male and female on either side and various gender identities in between (the gingerbread person)

II. HISTORY
A. Transsexualism is recorded throughout history, particularly in Greek and Roman literature
B. 1923 Transsexualism term coined by Magnus Hirschfeld, a German physician
C. 1931 Dr. Felix Abraham, a German surgeon, first to report staged vaginoplasty
D. Modern use of the word transsexual evolved in the 1940s
E. 1973 “gender dysphoria syndrome” coined describing individuals who had a conflict between their natal and desired gender
F. Current term is “gender identity disorder”
   1. Need to be contrasted from transvestites, who like to dress as the opposite gender but have no desire to change their gender
   2. Does not relate to sexuality or homosexualism
G. 1978 the Harry Benjamin International Gender Dysphoria Association was founded
   1. Plays a major role in research and treatment of these patients
   2. Publishes “Standards of Care for the Health of Transsexual, Transgender, and Gender Nonconforming People” which is now in its 7th edition
   3. Now known as the World Professional Association for Transgender Health (WPATH)

III. MALE TO FEMALE

A. Facial Surgery
   1. Frontonasal-orbital complex is greatest defining feature of male vs female face
   2. Male face:
      a. Increased supraorbital bossing, prominent forehead and glabella, forehead more angled
      b. Hairline with frontotemporal and overall recession (M-shaped)
      c. Squarer chin and jaw, mandible is larger and thicker with greater body height
      d. Malar area flatter but larger
      e. Larger in all dimensions than female face
      f. More muscle bulk
      g. Prominent thyroid cartilage
   3. Female face:
      a. Curved forehead
      b. Pointed chin
      c. Smaller nose
      d. More prominent malar region
      e. Less acute glabellar angle
      f. Rounded hairline
      g. Orbits are higher, more rounded, and larger
      h. Smaller overall than male, about 4/5ths the size
Figure 1. A more acute glabellar angle, a more acute nasal tip angle, a less open nasolabial angle, and a more pronounced chin are preferred in males.


4. Typical procedures
   a. Brow lift
   b. Forehead/supraorbital bar reduction
   c. Frontal hairline advancement/hair transplant
   d. Feminizing rhinoplasty
   e. Genioplasty
   f. Masseter resection/mandibular contouring
   g. Malar implants
   h. Thyroid cartilage reduction
   i. Vocal cord shortening

B. Top Surgery (chest surgery)
   1. Often performed before “bottom surgery”
   2. Breast augmentation
      a. Some breast growth from hormones
      b. Different that natal female: chest is wider, nipple to IMF distance is less, areolae are smaller, pectoralis major more developed
      c. Implant can be placed pre- or sub-pectoral
      d. Any incision is ok: transaxillary, peri-areolar, IMF (most common)

C. Bottom Surgery (Genital reconstruction)
   1. Goals
      a. Need to create normal appearing vagina and mons pubis
      b. Need to create a sensate neoclitoris
      c. Need to create a large and deep enough canal for penetration
   2. Operations
      a. Skin graft creation of a vaginal canal, similar to McIndoe operation or with use of grafts from penile skin
      i. Pros: single stage, can hide scars, skin can be hairless
ii. Cons: Requires use of obturation and dilation

b. Penile inversion technique
   i. First line operation at most centers
   ii. Typically, the last stage of the gender confirming process
   iii. Pedicled penile and scrotal flaps for creation of neovagina and labia
        (from deep external pudendal, superficial perineal, and funicular arteries)
   iv. Anterior penile flap and posterior scrotal flap to create vaginal canal
        a) Can add skin graft for more length
   v. Labia majora from scrotum
   vi. Clitoris from dorsal glans penis
   vii. Labia minora and urethral flap made together from urethra
   viii. Pros: Lined cavity, minimal shrinking, sensate
   ix. Cons: Need for dilation, need for pre-op hair removal, may need revisions/labiaplasty for further feminization

Figure 2. Penile inversion vaginoplasty performed in a 51-year-old transgender woman.
(Above, left) Preoperative genital area. (Above, right) An incision is made along the preoperatively marked pattern. (Second row, left) Blunt dissection of the neovaginal cavity is performed. Caution is taken not to sever the rectum. This is checked by bimanual palpation. (Second row, right) Bilateral orchiectomy is performed. (Third row, left) Penile skin is separated from the penile haft and closed at the distal end. (Third row, right) The dorsal neurovascular bundle is separated from the roof of the corpora cavernosa, and from a part of the glans penis and preputium the neoclitoris and the labia minora are sculptured. (Below, left) A linear incision is made into the raphe of the penile skin, and the penoscrotal flap is imbedded. (Below, right) Postoperative genital area. From Buncamper M, et al. Surgical Outcome after Penile Inversion Vaginoplasty: A Retrospective Study of 475 Transgender Women. Plast Reconstr Surg 2016;138(5):999-1007.
c. Intestinal transplantation
   i. Often used as backup procedure in severe stenosis requiring revision, or as first line procedure in adolescents who do not have adequate penile length
   ii. Pros: vascularized vagina with moist lining, decreasing the need for dilation and lubrication
   iii. Cons: intraabdominal operation, constant lubrication (sometimes excessive) that can be malodorous, diversion colitis, stricture and cancer of the reconstruction

Figure 3. Steps in the procedure of rectosigmoid vaginoplasty. (Above, left) The dissection plane is shown with a marked line that is anterior to the rectum and posterior to the bladder. (Above, right) The area of rectosigmoid colon to be used is shown within the dotted line and based on the superior hemorrhoidal artery. (Below, left) The rectosigmoid portion is harvested with continuity of the bowel established in an end-to-end fashion. (Below, right) Final anatomy of the male-to-female patient after the procedure. From Morrison S, et al. Long-Term Outcomes of Rectosigmoid Neocolporrhaphy in Male-to-Female Gender Reassignment Surgery. Plast Reconstr Surg 2015;136(2): 386-394.
IV. FEMALE TO MALE

A. Top Surgery
   1. Chest wall contouring/mastectomy
      a. Typically, the first surgical procedure in these patients
      b. Need to decrease breast/skin, obliterate IMF, reduction of nipple/areola
      c. Incision choices depend on amount of ptosis
         i. Subcutaneous keyhole or periareolar mastectomy for small breasts with minimal skin excess
         ii. Double incision mastectomy with free nipple grafts for medium and large/ptotic breasts

![Figure 4. Semicircular technique. (Above, left) Incisions and (above, right) scars. (Below, left) Preoperative and (below, right) postoperative photographs. From Monstrey S, et al. Chest-wall contouring surgery in female-to-male transsexuals: a new algorithm. Plast Reconstr Surg 2008;121(3):849-859.](image-url)
Figure 5. Transareolar technique. (Above) Incisions and scars. (Center) Details of nipple resection. (Below, left) Preoperative and (below, right) postoperative photographs. 

Figure 6. Extended concentric circular technique. (Above) Incisions. (Below, left) Preoperative and (below, right) postoperative photographs. 
B. Bottom Surgery

1. Goals
   a. Neourethra capable of voiding
   b. Phallus for sexual penetration
   c. Aesthetically pleasing

2. Operations for phalloplasty
   a. Metoidioplasty/metaoidioplasty
      i. Stretching the hormonally hypertrophied clitoris, lengthening urethra with local flaps
      ii. Complications are usually urethral fistulas/strictures
      iii. Scrotoplasty from labia majora (can be concomitant or staged)
      iv. Pros: tactile and erogenous sensate clitoral tissue, sustained erectile rigidity without prostheses, minor donor site scarring, shorter hospitalization
      v. Cons: very small phallus so unlikely to be able to provide penetration, impaired standing urination

Figure 8. Result of metaoidioplasty and scrotum construction in a slim, 30-year-old female transsexual who has been on hormonal treatment for 6 years. (Left) Preoperatively, (Right) Postoperatively. 

b. Pedicled flaps 
i. Urethroplasties are via tube within a tube design 
ii. More common in the pre-microsurgery era 
iii. ALT 
   a) Pros: Reliable vascular supply, reduced total flap failure risk, hidden donor site, able to be innervated 
   b) Cons: donor site may be too thick 
iv. Inferiorly based abdominal flaps 
   a) Less aesthetic and higher complication rate 
   b) Easier to hide donor site scars 
   c) Diminished sensation 
   d) Variability in vascular pattern 
   e) Limited ability to void standing and unable to provide sexual penetration 
v. Pedicled groin flaps 
   a) Similar to abdominally based flaps 
   b) Insensate 
   c) Functional problems the same as abdominal flaps 
vi. Gracilis flap 
   a) Bipedicled design with two flaps 
   b) Urethra made from skin graft
Figure 9. Illustration of the pedicled anterolateral thigh flap. A tube-within-a-tube design is used. The inner conduit becomes the neourethra, and the outer tubularized tissue represents the neophallus. The semicircular extension at the distal portion of the flap more accurately approximates the circumcised male phallus. From Morrison S, et al. Phalloplasty: A Review of Techniques and Outcomes. Plast Reconstr Surg 2016;138(3):594-615.

c. Free flaps
   i. Radial forearm
      a) Most commonly used
      b) Tube within a tube neourethra
      c) Can be sensate
      d) Pros: aesthetic reconstruction, standing urination, tactile and erogenous sensation
      e) Cons: donor site morbidity, urinary fistulas/strictures, requires prosthesis
   ii. Osteocutaneous fibula
      a) Can be made sensate
      b) Does not require a prosthesis
   iii. Others
      a) ALT
      b) Latissimus flap
1. Can be made functional with inclusion of muscle/nerve and may be able to have erectile function

Figure 10. Illustration of the radial forearm free flap and the fibula osteocutaneous free flap. In the osteocutaneous free fibula flap, the fibula is harvested with a cuff of muscle, the peroneal artery, and either the lateral or posterior sural nerve to create the sensate phallus. The neourethra is created from a groin skin flap. In the radial forearm free flap, the tube-in-tube design is used to create a neophallus and neourethra in a single flap. The radial artery and the antebrachial nerves are harvested to create the sensate phallus. The Norfolk technique is used for the radial forearm free flap to create a glans.

Figure 11. Illustration of the Norfolk technique. A distal circumferential portion of the neophallus shaft is elevated and rolled to create the corona. A split- or full-thickness skin graft is then placed over the defect on the shaft. *From Morrison S, et al. Phalloplasty: A Review of Techniques and Outcomes. Plast Reconstr Surg. 2016; 138(3):594-615.*

Figure 12. Illustration of the gracilis flap. A bipedicled gracilis muscle flap is harvested and pedicled into the groin. Once joined together around a skin graft used for the neourethra, another skin graft is placed around the muscle. *From Morrison S, et al. Phalloplasty: A Review of Techniques and Outcomes. Plast Reconstr Surg 2016; 138(3):594-615.*
d. Other
  i. Penile epithesis
  ii. Penile transplantation

3. Requires vaginectomy, salpingo-oopherectomy, and hysterectomy

4. Scrotoplasty with testicular prosthesis
   a. Labia major flaps +/- implants or tissue expanders
      i. Good color and texture match
      ii. Embryologically from the same area
      iii. Have erogenous sensation
      iv. Can use pump for prosthesis to make one testicle

Figure 13. Illustration of groin flap. The groin flap with or without the iliac bone can be performed in either one or two stages. The two-stage procedure is based on the superficial circumflex iliac artery and the deep circumflex iliac artery. The lateral and medial skin edges of the flap are sutured together, constructing a tube still attached to the body. After some time, the flap is raised on its pedicle. The neourethra is reconstructed using a full thickness skin graft from the contralateral groin. From Morrison S, et al. Phalloplasty: A Review of Techniques and Outcomes. Plast Reconstr Surg 2016; 138(3):594-615.

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CHAPTER 24

THERMAL, ELECTRICAL AND CHEMICAL INJURIES

Maureen Beederman, MD and Eamon O’Reilly, MD, LCDR, USN

Thermal injuries can result from exposure to heat, chemicals, electricity, or cold, and can result in destruction of the skin and cause local and systemic effects. The management of the patient with a major thermal injury requires an understanding of the pathophysiology, diagnosis, and treatment not only of the local skin injury, but also of the derangements that occur in hemodynamic, metabolic, nutritional, immunologic, and psychologic homeostatic mechanisms.

I. BURNS

A. Pathophysiology: Amount of tissue destruction is based on temperature (> 40° C) and time of exposure (Figure 1).

Figure 1. Tissue destruction based on temperature
B. Diagnosis and prognosis
1. Burn size: % of total body surface area (TBSA) burned
   a. Rough estimate is based on rule of 9’s (Figure 2)
   b. Different charts are required for adults and children because of head-chest size discrepancy and limb differentials for ages birth to seven years (Figures 3 and 4).
   c. Patient palm size can be used to estimate TBSA – palm is approximately 1% TBSA

Figure 2. TBSA burn based on rule of 9’s.
2. Age: burns at the extremes of age carry a greater morbidity and mortality
3. Depth: may be difficult to assess initially as injury can evolve and deepen over 24-48 hours
   a. Type and temperature of etiologic agent, and time of exposure helpful
   b. Classification (Figure 5 and Table 1)
      i. First degree: superficial - erythema but no skin breaks, similar to a sunburn
      ii. Second degree: blisters present, red and painful
         (a) Superficial partial-thickness: involves epidermis and upper dermis
         (b) Deep partial-thickness: involves deeper dermis
      iii. Third degree: full-thickness- insensate, charred or leathery in appearance
      iv. Fourth degree: muscle, bone affected
   c. Zones of injury
      i. Coagulation (central): tissues undergo necrosis and are irreparably damaged
      ii. Stasis (intermediate): vasoconstriction and ischemia (can improve or worsen, depending on treatment)
      iii. Hyperemia (peripheral): heals without scarring
4. Location: face and neck, hands, feet, and perineum may cause special problems and warrant careful attention; often necessitate hospitalization and/or transfer to a burn center (See Table 2)
5. Inhalation injury: beware of burns occurring in enclosed spaces, singed nasal/facial hair, carbon particles in pharynx, hoarseness, conjunctivitis - patients may not initially have any signs of airway compromise, so must have high index of suspicion.
6. Associated injuries, e.g. fractures - depending on circumstances surrounding burn, patients may require full trauma workup
Figure 5. Classification of burns by depth
Table 1. Description and features of burns based on depth

<table>
<thead>
<tr>
<th>Degree</th>
<th>Depth</th>
<th>Layers Involved</th>
<th>Features</th>
<th>Healing Time</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>First</td>
<td>Superficial</td>
<td>Epidermis only</td>
<td>Pink, red, brisk capillary refill, painful</td>
<td>&lt;7 days</td>
<td>Symptomatic</td>
</tr>
<tr>
<td></td>
<td>Superficial partial-thickness</td>
<td>Epidermis, papillary (upper) dermis</td>
<td>Pink, red, moist, edematous, brisk capillary refill, very painful</td>
<td>Variable 10-28 days</td>
<td>Daily wound care, debride sloughed skin</td>
</tr>
<tr>
<td></td>
<td>Deep partial-thickness</td>
<td>Epidermis, reticular (lower) dermis</td>
<td>White, pink, red, dry, no blanching, reduced sensation</td>
<td></td>
<td>Daily wound care, surgical excision and resurfacing</td>
</tr>
<tr>
<td>Third</td>
<td>Full thickness</td>
<td>Epidermis, entire dermis</td>
<td>White, brown, dry, leathery, no blanching, insensate</td>
<td>&gt;21 days</td>
<td>Surgical excision and resurfacing</td>
</tr>
<tr>
<td>Fourth</td>
<td>Full thickness</td>
<td>Epidermis, entire dermis, fat, fascia, muscle, bone</td>
<td>Exposed deep tissue</td>
<td>N/A</td>
<td>Amputation, complex reconstruction</td>
</tr>
</tbody>
</table>

7. Co-morbid factors, e.g. pre-existing cardiovascular, respiratory, renal and metabolic diseases; seizure disorders, alcoholism, drug abuse
8. Prognosis: best determined by burn size (TBSA) and age of patient, presence of inhalation injury (Figure 6)
9. Circumferential burns: can restrict blood flow to extremity, respiratory excursion of chest and may require escharotomies
10. Certain criteria are used to make the determination regarding whether transfer to a burn is necessary (Table 2)
**Burn Center Referral Criteria (ABA Guidelines)**

1. Partial thickness burns greater than 10% total body surface area (TBSA) in patients <10 yr or >50 yr
2. Partial-thickness burns greater than 20% total body surface area in all patients.
3. Burns that involve the face, hands, feet, genitalia, perineum, or major joints.
4. Third degree burns in any age group.
5. Electrical burns, including lightning injury.
6. Chemical burns.
7. Inhalation injury.
8. Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality.
9. Any patient with burns and concomitant trauma (such as fractures) in which the burn injury poses the greatest risk of morbidity or mortality.
10. Burned children in hospitals without qualified personnel or equipment for the care of children.
11. Burn injury in patients who will require special social, emotional, or rehabilitative intervention.

<table>
<thead>
<tr>
<th>Table 2. Burn Center referral guidelines</th>
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</table>

**C. Treatment plan**

1. History and physical exam
   a. In pediatric burns where the history does not correlate with the burn injury, consider the possibility of child abuse – contact social work and/or follow hospital-specific protocols.
2. Relieve respiratory distress - escharotomies and/or intubation – see below.
3. Prevent and/or treat burn shock – fluid resuscitation with large bore IV (x2)
4. Monitor resuscitation - Foley catheter and hourly urine output
5. Treat ileus and nausea - N.G. tube if > 20% TBSA burn or intubated
6. Tetanus prophylaxis
7. Thromboembolic prophylaxis
8. Baseline laboratory studies (i.e. CBC, comprehensive metabolic panel, U/A, chest x-ray, EKG, cross-match, arterial blood gases, and carboxyhemoglobin)
9. Cleanse, debride, and treat the burn wounds

D. Respiratory involvement
1. Three major causes of respiratory distress in the burned patient
   a. Unyielding burn eschar encircling chest
      i. Distress may be apparent immediately
      ii. Requires escharotomies (cutting into the eschar to relieve constriction)
   b. Carbon monoxide poisoning
      i. May be present immediately or have a delayed presentation; high level of suspicion if patient exposed to smoke in an enclosed space
      ii. Diagnosed by carboxyhemoglobin levels measured in arterial blood gas
      iii. Important to remember that patients with a history of smoking may have baseline elevated carboxyhemoglobin
      iv. Initial Rx is displacement of CO by 100% O2 by facemask
      v. Hyperbaric oxygen treatment may be of value, but is often not practical in many locations
   c. Smoke inhalation leading to pulmonary injury
      i. Insidious in onset (18-36 hours)
      ii. Due to incomplete products of combustion, not heat (direct thermal injury to lungs occurs only secondary to steam burns)
      iii. Causes chemical injury to alveolar basement membrane and subsequent pulmonary edema
      iv. Initial Rx is humidified O2 but intubation and respiratory support may be required
      v. Secondary bacterial infection of the initial chemical injury leads to progressive pulmonary insufficiency and infection – mucosal barrier of respiratory tract is damaged
      vi. Severe inhalation injury alone or in combination with thermal injury carries a grave prognosis
      vii. Three stages of presentation have been described:
          (a) Acute pulmonary insufficiency (immediately postburn to 48 hours)
          (b) Pulmonary edema (48-72 hours)
          (c) Bronchopneumonia (25 days)
      viii. Monitor respiratory and mental status – be aware of hoarseness, wheezing, stridor. If any concerns about current or future airway involvement, best to intubate patient prior to excessive airway edema

E. Burn shock
1. Massive amounts of fluid, electrolytes, and protein are lost from circulation almost immediately after burning (Table 3)
Table 3. Burn shock

2. Systemic inflammatory response occurs if > 20% TBSA burn in adults, >30% TBSA in children, and >15% TBSA in elderly - results in a hypermetabolic state and increased capillary permeability - causing

3. Resuscitation requires replacement of sodium ions and water to restore plasma volume and cardiac output – initiate if adult burns >15-20% TBSA and pediatric burns >10% TBSA
   a. Many formulas have been reported to achieve resuscitation – these serve only as a guide for initial IVF – must also monitor UOP to appropriately titrate rate
      i. Parkland formula: 2-4cc Ringer’s lactate/Kg/%TBSA burn over the first 24 hours
      ii. 1/2 of the 24-hour fluid requirement should be given in the first eight hours postburn and the remaining 1/2 over the next 16 hours
      iii. Remember to factor in any fluid boluses patient may have received either in ED or at OSH when performing calculations
   b. Administration of colloid during or after resuscitation can vary from burn center to burn center
   c. Children – should receive maintenance fluids (weight-based 5% dextrose in half-normal saline), as well as resuscitative fluids

F. Monitoring resuscitation
   1. Urine output 0.5-1cc/kg/hr in adults and 1cc/Kg/hr in children < age 12
   2. A clear sensorium, pulse < 140/min, BP >90/60 mmHg, HCO₃ > 18meq/L, cardiac output > 3.5 L/min/M²
3. CVP in acute major burns is unreliable – use if myocardial disease, age > 65, inhalation injury, fluid requirements > 150% of expected

G. Metabolic considerations
1. Increased metabolic demands in patients with burn injury (hypermetabolic state)
2. High carbohydrate/high protein diet – dietician and tube feeds as needed
3. Early feeding (start at 12 hours) – prevents mucosal atrophy, ulceration, bacterial translocation in gut
4. Measure prealbumin to determine nutritional status

H. Treatment of the burn wound
1. Wound closure by the patient’s own skin (autograft) is the ultimate goal of treatment
   a. By spontaneous healing
   b. Autograft
   c. Allograft
   d. Xenograft
   e. Artificial skin
   f. Cultured epithelial cells
2. Specific treatment of the burn wound differs from one burn center to another
   a. The most commonly employed topical antibacterials are silver sulfadiazine (Silvadene®) and mafenide acetate (Sulfamylon®)
   b. Dressing changes at least 1x/day with soap and water – no need to scrub or remove blisters
   c. Status of burn wound bacterial colonization and effectiveness of topical antibacterial treatment can be monitored by biopsies of wound for quantitative and qualitative bacteriology
   d. Systemic antibiotic therapy only used for sepsis, not prophylactically (breeds resistant organisms)
3. Necrotic tissues or any burned tissue not expected to heal within 2-3 weeks may be removed by any of several techniques
   a. Formal excision
   b. Tangential (layered) debridement
   c. Enzymatic debridement
   d. Hydrotherapy - a useful adjunct
4. Autografts should be applied to priority areas first, such as the hands, face and important joints, as well as the neck for possible tracheostomy placement
5. Once healed, pressure with elastic support/compression garments is usually necessary to reduce hypertrophic scarring
6. Physical therapy/occupational therapy - important adjunct in burn care, helps prevent contractures, especially for burns that cross joint surfaces

I. Complications: can occur in every physiologic system or secondary to burn injury (Table 5)
1. Renal failure
   a. From hypovolemia
   b. Beware of nephrotoxic antibiotics in the burn patient
c. Myoglobinuria associated with compartment syndrome

<table>
<thead>
<tr>
<th>Risk Factors in Burn Wound Infection</th>
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<tbody>
<tr>
<td><strong>I. PATIENT FACTORS</strong></td>
</tr>
<tr>
<td>A. Extent of burn &gt; 30% of body surface</td>
</tr>
<tr>
<td>B. Depth of burn: full-thickness vs. partial-thickness</td>
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<tr>
<td>C. Age of patient (very young or very old at higher risk)</td>
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<td>D. Pre-existing disease</td>
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<td>E. Wound dryness</td>
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<tr>
<td>F. Wound temperature</td>
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<tr>
<td>G. Secondary impairment of blood flow to wound</td>
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<tr>
<td>H. Acidosis</td>
</tr>
<tr>
<td><strong>II. MICROBIAL FACTORS</strong></td>
</tr>
<tr>
<td>A. Density &gt;10⁶ organisms per gram of tissue</td>
</tr>
<tr>
<td>B. Motility</td>
</tr>
<tr>
<td>C. Metabolic products</td>
</tr>
<tr>
<td>1. Endotoxin</td>
</tr>
<tr>
<td>2. Exotoxins</td>
</tr>
<tr>
<td>3. Permeability factors</td>
</tr>
<tr>
<td>4. Other factors</td>
</tr>
<tr>
<td>D. Antimicrobial resistance</td>
</tr>
</tbody>
</table>

Table 5. Burn wound infection risk factors

2. Gastrointestinal bleeding
   a. Curling’s ulcer: gastric ulcer that results from large TBSA burn due to decreased plasma volume - causing ischemia of gastric mucosa. More likely in burns over 40% TBSA.
   b. Usually remains subclinical
   c. Antacids and H₂ blockers
   d. Increased risk with burn wound sepsis

3. Burn wound sepsis
   a. Monitored by tissue biopsy - qualitative and quantitative
   b. Must keep bacterial count < 10⁶ bacteria/gm of tissue
   c. Clinically suspect global sepsis with
      i. Sudden onset of hyper or hypothermia
      ii. Unexpected congestive heart failure or pulmonary edema
      iii. Development of the acute respiratory distress syndrome
      iv. Ileus occurring after 48 hours postburn
      v. Mental status change
      vi. Azotemia
vii. Thrombocytopenia
viii. Hypofibrinogenemia
ix. Hyper or hypoglycemia is especially suspect if burn > 40% TBSA
x. Blood cultures may be positive but in many cases are not

4. Progressive pulmonary insufficiency
   a. Can occur after:
      i. Smoke inhalation
      ii. Pneumonia
      iii. Cardiac decompensation
      iv. Sepsis from any cause
   b. Produces:
      i. Hypoxemia
      ii. Hypocarbia
      iii. Pulmonary shunting
      iv. Acidosis

5. Overresuscitation
   a. If IVF rate remains elevated despite adequate UOP, increased risk for
      i. Abdominal compartment syndrome
      ii. Extremity compartment syndrome

6. Wound contracture and hypertrophic scarring
   a. Largely preventable
   b. Active range of motion of involved and adjacent joints is encouraged to
      prevent joint contractures from the outset
      i. Splints and passive range of motion are used if active range of motion
         is unable to be performed, e.g. elbow and knee are kept in extension
         and MCP joints of fingers in flexion
      ii. Limb elevation and range of motion are useful for reducing edema and
          maintaining movement
   c. Timely wound closure with adequate amounts of skin should largely
      eliminate these problems
   d. Continued postoperative splinting and elastic pressure supports are of
      value in the remodeling of collagen with prevention of hypertrophic scars
   e. Contractures may require future revision and reconstructive procedures
      months to years after original burn injury.

II. CHEMICAL BURNS

A. Pathophysiology
   1. Tissue damage secondary to a chemical depends on:
      a. Nature of agent
      b. Concentration of the agent
      c. Quantity of the agent
      d. Length of time the agent is in contact with tissue
      e. Degree of tissue penetration
      f. Mechanism of action

B. Diagnosis
1. Chemical burns are deeper than initially appear and may progress with time
   a. Alkali burns may be more severe due to ability to deeply penetrate tissues
   b. Fluid resuscitation needs often underestimated
   c. Watch for renal/liver/pulmonary damage

C. Treatment
1. Initial treatment is dilution of the chemical with tap water
   a. Copious irrigation for 30 minutes
   b. Exception – cement/concrete/powdered lye/other powders should be brushed off dry
2. Special attention to eyes – after copious irrigation with saline, consult ophthalmologist
3. After 12 hours initial dilution, local care of the wound with debridement, topical antibacterials, and eventual wound closure is same as for thermal burn

D. Of particular note are:
1. Gasoline
   a. Excretion by lung
   b. May cause large skin burn, if immersed
   c. Watch for atelectasis, pulmonary infiltrates; surfactant is inhibited
2. Phenol
   a. Dull, gray color to skin, may turn black
   b. Urine may appear smoky in color
   c. Spray water on burn surface
   d. Wipe with polyethylene glycol
   e. Direct renal toxicity
3. Hydrofluoric acid
   a. Irrigate copiously with water
   b. Subcutaneous injections of 10% calcium gluconate, or intra-arterial infusion in affected extremities
   c. Serial EKG and BMP monitoring of patients - may become hypocalcemic
   d. Pulmonary edema may occur if subjected to fumes
   e. Monitor effectiveness of treatment by frequent pain assessment – if treatment is working, pain should decrease
4. White phosphorous burn
   a. Do not allow to desiccate - may ignite
   b. Each particle must be removed mechanically
   c. Copper sulfate (2%) may counteract to make phosphorous more visible (turns black in color)
   d. Watch for EKG changes (Q - T interval and S - T and T wave changes)
   e. May cause hemoglobinemia and renal failure

III. ELECTRICAL INJURIES

A. Pathophysiology
   1. Effects of passage of electric current through the body depend on:
      a. Type of circuit
b. Voltage of circuit
c. Resistance offered by body
d. Amperage of current flowing through tissue
e. Pathway of current through the body
f. Duration of contact
2. Tissue resistance to electrical current increases from nerve (least resistant) to vessel to muscle to skin to tendon to fat to bone

B. Diagnosis
1. Types of injury
   a. Arc injury: localized injury caused by intense heat, current does not run through patient’s body
   b. Injury due to current
      i. Due to heat generated as current flows through tissue
         (a) Injury more severe in tissue with high resistance (i.e. bones)
         (b) Vessels thrombose as current passes rapidly along them
      ii. Effect of current may not be immediately seen

C. Special effects of electrical injury
1. Cardiopulmonary
   a. Anoxia and ventricular fibrillation may cause immediate death
   b. Early and delayed rhythm abnormalities can occur
   c. EKG changes may occur some time after the burn in a delayed fashion – need serial EKG monitoring
2. Renal
   a. High risk of renal failure due to hemoglobin and myoglobin deposits in renal tubules – important to see if myoglobinuria is present
      i. Requires higher IVF/urine flow (75cc/hr in adults)
      ii. Must alkalinize urine to keep hemoglobin and myoglobin in more soluble state
      iii. Mannitol may be useful to clear heavy protein load
3. Musculoskeletal
   a. Tetanic muscle contractions may be strong enough to fracture bones, especially spine
   b. Although there may be minimal external damage, have a high suspicion for compartment syndrome in extremities
4. Spinal Cord Damage
   a. Can occur secondary to fracture or demyelinating effecting of current
5. Abdominal effects
   a. Intraperitoneal damage can occur to G.I. tract secondary to current
6. Vascular effects
   a. Vessel thrombosis progresses with time
   b. Delayed rupture of major vessels can occur
7. Cataract formation - late complication
8. Seizures

D. Treatment
1. CPR if necessary
2. Fluids - usually large amounts
a. No formula is accurate because injury is more extensive than can be predicted by skin damage
b. Alkalinize with NaHCO₃, if myoglobinuria or hemoglobinuria present

3. Monitoring
   a. CVP or pulmonary wedge pressure helpful since total capillary leak does not occur as it does in a thermal burn
   b. Maintain urine output at 75-100cc/hr until all myoglobin and/or hemoglobin disappears from urine

4. Wound Management
   a. Topical agent with good penetrating ability is needed [i.e. silver sulfadiazine (Silvadene®) or mafenide acetate (Sulfamylon®)]
   b. Debride non-viable tissue early and repeat as necessary (every 48 hrs) to prevent sepsis
   c. Major amputations frequently required
   d. Technicium-99 stannous pyrophosphate scintigraphy may be useful to evaluate muscle damage

5. Treat associated injuries (e.g. fractures)

IV. COLD INJURIES

The two conditions of thermal injury due to cold are local injury (frostbite) and systemic injury (hypothermia)

A. Frostbite
   1. Pathophysiology
      a. Formation of ice crystals in tissue fluid
         i. Usually in areas which lose heat rapidly (e.g. extremities)
      b. Anything which increases heat loss from the body such as wind velocity, or decreases tissue perfusion, such as tight clothing, predisposes the patient to frostbite
      c. Ability of various tissues to withstand cold injury is inversely proportional to their water content
   2. Treatment
      a. The key to successful treatment is rapid rewarming in a 40° C water bath
         i. Admission to hospital usually required
            (a) Tetanus prophylaxis
            (b) Wound management
            (c) Physical therapy
               (i) Maintenance of range of motion important
               (ii) Daily whirlpool and exercise
            (d) Sympathectomy, anti-coagulants, and early amputation of questionable value in controlled studies
         ii. Usually wait until complete demarcation before proceeding with amputations. Non-viable portions of extremities will often autoamputate with good cosmetic and functional results.

B. Hypothermia
1. Diagnosis
   a. Core temperature < 34° C
   b. Symptoms and signs mimic many other diseases
   c. High level of suspicion necessary during cold injury season

2. Treatment
   a. Must be rapid to prevent death
   b. Monitor EKG, CVP, and arterial blood gases and pH during warming and resuscitation, maintain urine output of 50cc/hr
   c. Begin Ringer’s lactate with 1 ampule NaHCO₃
   d. Oral airway or endotracheal tube if necessary
   e. Rapidly rewarmed in 40° C hydrotherapy tank (requires 1-2 hours to maintain body temperature at 37°C)
   f. Treat arrhythmias with IV Lidocaine or Amiodarone drip if necessary
   g. Evaluate and treat any accompanying disease states

V. LIGHTNING INJURIES

A. Cutaneous effects - lightning strikes may cause cutaneous burn wounds
   1. Contact burns from clothing on fire or contact with hot metal (i.e. zippers, etc)
   2. Entry and exit burns are usually small, may be partial or full thickness
   3. Lightning burns are not the same as electrical burns – don’t get deep tissue injury

B. May have temporary ischemic effects on extremity - pallor or neurologic deficits. Spontaneous recovery after a few hours is the rule - probably due to local vasoconstriction

C. Systemic effects can occur such as arrhythmias, cataracts, CNS symptoms (similar to electrical injuries)

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CHAPTER 25

COMPOSITE TISSUE ALLOTRANSPLANTATION

Helene Retrouvey, MDCM and Jon Ver Halen, MD, FACS

I. INTRODUCTION

A. Also referred to as Vascularized Composite Allotransplantation (VCA) or Composite Tissue Allograft (CTA)
B. Allograft (graft from cadaveric donor) composed of different tissue types (e.g. skin, muscle, bone, nerves, vessels)
C. Introduced for major reconstruction of tissue defects from surgical excision of tumors, traumatic injury and congenital malformations
   1. Option for limb replacement and reconstruction of other non-reconstructible tissue defects including facial, abdominal wall and others
D. Couples the principles of microsurgical reconstruction with those of human organ transplantation
E. Goal: improve function, quality of life, integration with society

II. IMMUNOGENICITY

A. CTA are histologically heterogeneous, composed of different tissue types including skin, fat, muscle, nerves, lymph nodes, blood vessels, bone, cartilage, ligaments, and bone marrow
B. Each tissue has different antigenicity
   1. Skin most antigenic, likely owing to dendritic cell population and antigen variety
   2. CTA graft is not the sum of immunogenicity of its different components
   3. Whole limb allograft elicits a less intense immune response than does allografts of each of the individual components
C. CTA elicit a stronger immune response as compared to solid organ transplants
D. Split tolerance phenomenon: simultaneous tolerance to one tissue and rejection of another from the same donor

III. DEVELOPMENT

A. Rapidly progressing field
B. > 150 composite tissue allograft worldwide to date
C. An estimated 7 million people per year in the United States could benefit from composite tissue allotransplantation
D. Online international registry of hand and composite tissue transplantation: https://www.handregistry.com/
IV. HISTORY

A. 348 AD: ’The legend of the black leg’ / Legend of Saints Cosmos and Damien
   1. Tale of twin brothers Cosmas and Damian who replaced the diseased leg of a man with that of a recently deceased person
B. 1954: first successful human organ isograft, a kidney donated between identical twins; USA (Joseph E. Murray, John P. Merrill, and J. Hartwell Harrison)
C. 1964: first case of hand CTA, failed due to rejection after 3 weeks; Ecuador (Robert Gilbert)
D. 1990s: progress in immunosuppression → composite tissue renaissance
E. 1998: first successful hand CTA; France (Jean-Michel Dubernard)
F. 2000: first successful bilateral hand CTA; France (Jean-Michel Dubernard)
G. 2005: first successful partial face transplantation; France (Devauchelle, Dubernard)

V. TYPES OF CTA PERFORMED TO DATE

A. Hand / upper extremity
   1. Most common transplant
      a. First 4 successful cases:
         i. Right hand – transplanted in Lyon, France, on September 23, 1998
         ii. Left hand – transplanted in Louisville, KY on January 23, 1999
         iii. 2 right hands – transplanted to two individuals in Guangzhou, China, on September 21, 1999
   2. > 80 upper limbs reported in 2015
B. Partial or total face
   1. first partial face transplant in 2005 in France
   2. > 25 cases reported in 2015
C. Abdominal wall
   1. Typically, in the setting of abdominal multi-organ transplant
D. Knee
E. Larynx
F. Flexor tendon apparatus
G. Peripheral nerve
H. Tongue
I. Trachea
J. Esophagus
K. Scalp
   1. In the setting of active malignancy, and with simultaneous solid organ transplantation
L. Penis
M. Uterus
   1. with the first successful pregnancy following uterus transplant in 2015 in a Swedish woman
VI. ADVANTAGES

A. Replace “like with like”
B. An option when standard reconstructive options are exhausted or autologous tissues are not available
C. Can achieve structural, functional, esthetic, and psychological result
D. Avoidance of any donor site morbidity
E. Good functional outcomes
   1. for example, for hand transplants:
      a. Sensibility recovered 6-12 months
      b. Motor function allows return to most daily activities (eating, driving, grasping objects, riding a bicycle or a motorbike, shaving, using the telephone, and writing)
F. Majority of patients are satisfied and report improved quality of life

VII. DISADVANTAGES / LIMITING FACTORS

A. Need to determine appropriate indications
B. No consensus on which physical defects justify reconstruction with CTA

VIII. DONOR-RECIPIENT MATCHING CRITERIA

A. ABO
B. Graft size
C. Skin pigmentation
D. Age
E. Gender

IX. DETERMINATION OF ORGAN ALLOCATION

A. Patient level
   1. complex decision making
   2. important discussion with recipient regarding donor and recipient skin pigmentation, age mismatch, and gender
B. Society level
   1. no current policy
   2. societal values variable on the benefits and risks of CTA
C. Need for indefinite immunosuppressive
   1. Always at risk for rejection
   2. Predominant limiting factor: side effects of immunosuppression
D. Opportunistic infections
E. Drug toxicity
1. Metabolic disorders
2. Nephrotoxicity
3. End-organ damage

F. Malignancies
1. Heightened antigenicity of composite tissues → difficult to develop an effective yet nontoxic immunosuppressive protocol

G. Uncertain long term outcomes
1. Limited long term experience
2. Unknown if CTA will undergo chronic rejection leading to diminished functional capacities

X. FUTURE DIRECTIONS

A. Results of the first clinical cases are encouraging, but still controversial.
1. Significant progress in the last 2 decades
2. Novel immunosuppression and medication regimens may improve motor and sensory function, and decrease episodes of rejection
3. Novel donor-specific tolerance regimens (i.e., simultaneous bone marrow transplantation to induce bone marrow chimerism and central tolerance)

B. Increasing application for a wider group of patients as surgery is streamlined, tolerance regimens are perfected, and outcomes are improved.

REFERENCES

CHAPTER 26

LYMPHEDEMA

Imran Ratanshi, MD, MSc, FRCS(C) and Andres Maldonado, MD, PhD

I. DEFINITION

A. Chronic, progressive swelling in tissues due to insufficient drainage of interstitial fluid

II. EPIDEMIOLOGY

A. Over 300 million cases worldwide
B. By etiology: 90% secondary lymphedema (*lymphatic filariasis* is the most common cause worldwide), 10% primary lymphedema
C. Primary lymphedema is relatively rare – incidence 1.2:100,000 for persons age < 20 years (Schook et al, 2011)
D. By location: 90% lower extremity, 10% upper extremity, < 1% genitalia
E. Becoming a major public health issue in developed countries due to increased cancer survivorship

III. ETIOLOGY

A. Loss of lymphatic outflow can be due to *primary lymphedema* (inherent channel dysfunction) or *secondary lymphedema* (acquired insult causing physical interruption or dysfunction)
B. Primary lymphedema (can also be part of syndrome or vascular malformation)
   1. **Lymphedema Congenita**: resents age < 1-2 years, often bilateral limb involvement
      a. **Milroy Disease**: hereditary form presenting at birth with family history of lymphedema congenita
         i. Genetic mutation in vascular endothelial growth factor receptor-3 (VEGFR
      b. **Lymphedema Praecox**: often unilateral limb involvement
      c. Most common form of primary lymphedema
      d. **Meige disease**: adolescent onset, usually with family history
      e. **Lymphedema Tarda**: presents later in adult life, usually 3rd or 4th decade
C. Secondary lymphedema
   1. Infection
      a. #1 cause in developing countries
      b. **Lymphatic Filariasis**: blood borne nematode infection that directly obstructs lymphatic channels
i. Common organisms: *Wucheria bancrofti, Brugia malayi, Brugia timori*

2. Lymph node dissection (sentinel lymph node biopsy, nodal basin dissection)
   a. #1 cause in developed countries
   b. Cancer related lymphedema is one of the most distressing complications of sentinel lymph node biopsy or complete nodal basin dissection

3. Tumor excision
4. Trauma
5. Obesity
6. Vascular anomaly

IV. PATHOPHYSIOLOGY

A. Chronic disease of lymphatic system characterized by fluid accumulation and deposition of fibroadipose tissue

B. Evolution of chronic lymphedema generally involves
   1. Mechanical disruption of channel flow preventing outflow of interstitial fluid
   2. Accumulation of protein rich fluid leading to pitting edema and limb heaviness
   3. Chronic fluid stasis activates inflammatory pathways
   4. Long-standing inflammation promote deposition of fibroadipose tissue leading to non-pitting edema
   5. Skin trophic changes result from chronic dermal inflammation

V. DIFFERENTIAL DIAGNOSIS: LIMB ENLARGEMENT

A. Lipidema
B. Lipodystrophy
C. Hemihypertrophy
D. Edema
   1. Cardiac insufficiency
   2. Renal insufficiency
   3. Hepatic insufficiency
   4. Venous insufficiency
   5. Nutritional insufficiency
E. Deep vein thrombosis
F. Thyroid disease with pre-tibial myxedema

VI. CLINICAL PRESENTATION

A. Subjective
   1. Pain
   2. Swelling
   3. Heaviness of involved limb
   4. Inability to find properly fitting clothing
5. Decreased use of involved limb
6. Aesthetic concerns
B. Objective
   a. Doughy swelling of the extremity
   b. **Stemmer sign:** inability to grasp skin on dorsum of second digit of foot
   c. Edema – pitting vs. non-pitting
   d. Skin trophic changes: hyperkeratosis, acanthosis, skin ulcerations, plaques
   e. Recurrent soft tissue infections – cellulitis, erysipelas, lymphangitis
C. Source of multiple, hospital admissions; potentially prolonged duration
D. **Can be life threatening!!!**

VII. **CLASSIFICATION**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 0 (or Ia)</td>
<td>Latent or subclinical condition where swelling is not evident despite impaired lymph transport. It may exist months or years before overt edema occurs (Stages I–III)</td>
</tr>
<tr>
<td>Stage I</td>
<td>Early accumulation of fluid relatively high in protein content that improves with limb elevation. Pitting may occur.</td>
</tr>
<tr>
<td>Stage II</td>
<td>Limb elevation alone rarely reduces tissue swelling and pitting is manifest.</td>
</tr>
<tr>
<td>Stage II (late)</td>
<td>Loss of pitting edema due to fibrosis and deposition of lymphadiposal tissue.</td>
</tr>
<tr>
<td>Stage III</td>
<td>Lymphostatic elephantiasis. Gross limb enlargement with absent pitting. Trophic skin changes develop, such as acanthosis, fat deposits, and warty overgrowths.</td>
</tr>
</tbody>
</table>

Table 1. International Society of Lymphology (ISL) Classification

A. Newer classifications consider limb circumference or estimated limb volume
B. Limb circumference should be measured at defined points (reduces variability between evaluators)

VIII. INVESTIGATIONS

A. Lymphatic Filariasis (if patient is from or recently travelled to endemic region)
B. Nocturnal blood smear: microscopic identification of microfilariae with Giemsa or hematoxylin & eosin (H&E) stain. Blood collection should be done at night to coincide with peaks levels of microfilariae.
C. Serum antifilarial IgG4 Assay: serologic study that detects elevated levels of antifilarial IgG4 in blood of patients with active filarial infection.
D. Standardized limb circumference measurements: measure circumference in office at set intervals (e.g. 4 cm) from tip of 2nd toe (lower extremity) or long finger (upper extremity). Allows for calculation of estimated limb volume.
E. Direct lymphangiography: visualize lymphatic channels using oil-based iodine contrast agent directly injected into lymphatics (rarely done today).
F. Lymphocintigram: nuclear medicine technique to visualize lymphatic channels using injection of filtered colloid of Technetium-99m (Tc\textsuperscript{99m}) subdermally into limb. Tc\textsuperscript{99m} selectively taken up by lymphatics to allow visualization.
G. Laser angiography with indocyanine green (ICG): similar technique to lymphocintigram but uses ICG injection that can be performed in office or operating room settings. It is commonly used intraoperative to plan the location of the lymphaticovenular anastomosis (Figure 2).
H. MR-Lymphangiogram: high resolution imaging of lymphatic system. Injection of ferumoxytol can help distinguish lymphatics from veins
I. Lymphedema Index (L-Dex): office-based device measures differences in fluid accumulation between limbs.
J. Other possible investigations
   1. Ultrasound: if clinical concerns of DVT
   2. Fungal culture: if concerns of superimposed fungal infection
Figure 2. Imaging used to identify lymphatic channels. Magnetic resonance lymphangiogram showing (A) veins with a smoother appearance and (B) lymphatic channels with beadlike characteristics. Laser angiography with subdermal injection of indocyanine green (ICG) can also be used to observe lymphatic channels (C) that preferentially take up ICG in the interstitium. Arrows demonstrate shadow from an overlying vein that has not taken up ICG in this study.


**IX. MANAGEMENT**

A. **Non-operative Management**
   1. Medical management for lymphatic filariasis, if infection present
      a. Diethylcarbamazine (DEC) - #1 choice in North America
      b. Ivermectin – kills only microfilariae but not adult worm
      c. Albendazole
      d. Doxycycline
   2. Limb elevation
   3. Compression garment
   4. Decongestive therapy
      a. Massage/manual drainage
      b. Intermittent pneumatic compression
   5. Complete decongestive therapy: combination of lymphatic massage and compression
   6. Weight loss
   7. Skin and foot care
      a. Low pH, water-based lotion to prevent fungal infection
      b. Topical +/- systemic antifungal agents
   8. Experimental
a. Low level laser therapy
b. VEGF-C therapy – not indicated in cancer pts as can promote tumor growth
c. Stem cell therapy with bone marrow or adipose-derived mesenchymal stem cells
d. *Note: Recent evidence suggests that even with aggressive nonsurgical therapy and a fully compliant patient, lymphedema disease progression can still occur resulting in morbidity.

B. Operative Management
1. Excisional Procedures: remove chronic lymphadiposal tissue
2. Liposuction – useful for reducing fat accumulation
3. Serial Excision – useful for skin/fat accumulation and removal of festoons
4. Charles Procedure – radical excision of lymphedematous tissue, limb resurfaced with split thickness skin graft
5. Physiological Procedures: restore lymphatic outflow
   a. Axillary vein decompression
   b. Lymphaticovenular anastomosis (LVA) – anastomose lymphatic channel to venule for improved outflow
   c. Vascularized lymph node transfer (VLNT) – selective transfer of lymph nodes on a vascular pedicle from non-lymphedematous region
      i. Potential lymph node donor sites: omentum, groin, axilla, supraclavicular, submental
      ii. Reverse lymphatic mapping an option to prevent unintentional harvest of sentinel lymph nodes, reducing the risk of lymphedema in donor site
   d. Vascularized lymphatic channel transfer (VLCT) – transfer of vascularized lymph channels, replaces segmental gap in lymphatics

![Schematic and intraoperative view](image)

Figure 3. Schematic (left) and intraoperative view through a microscope showing anastomosis of a lymphatic channel to a small vein (venule). From Ishiura R, et al. Comparison of lymphovenous shunt methods in a rat model: supermicrosurgical lymphaticovenular anastomosis versus microsurgical lymphaticovenous implantation. Plast Reconstr Surg 2017;139(6):1407-1413.
X. COMPLICATIONS

A. Limited mobility and functional disability
B. Lymphorrhea
C. Recurrent infections – result in multiple hospital admissions, can be life threatening
D. Slow- or non-healing wounds
E. Chronic cutaneous changes
F. Psychosocial morbidity
G. Venous insufficiency
H. DVT
I. Malignancy
J. **Stewart-Treves Syndrome**: cutaneous angiosarcoma that develops in the setting of chronic lymphedema. Best treated by wide local excision or amputation.

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